Travell & Simons'

Myofascial Pain and Dysfunction

The Trigger Point Manual

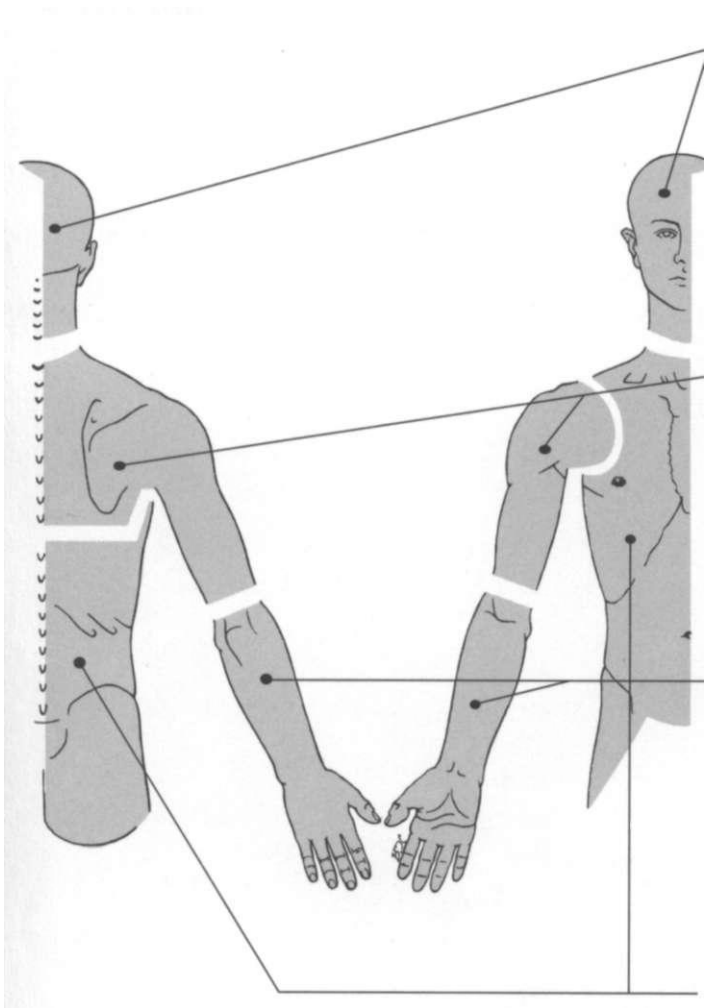
VOLUME 1. Upper Half of Body

Second Edition

DAVID G. SIMONS, M.D.
JANET G. TRAVELL, M.D.
LOIS S. SIMONS, P.T.

Illustrations by Barbara D. Cumming

Pain Patterns



Pictorial index. The muscles that are likely to refer pain to an illustrated region of the body are listed in the Pain-and-muscle Guide to the corresponding Part of the Manual. A Guide is found at the beginning of each Part, which is marked by red thumb tabs.

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BALTIMORE • PHILADELPHIA • LONDON • PARIS • BANGKOK
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[t] Dr. Janet Travell's genius and medical insight identified in the first edition the clinical picture of individual myofascial pain syndromes and many perpetuating factors. In addition, we were most fortunate to have had the benefit of her advice in preparing some of this edition. She emphasized the importance of including a new chapter that covers the respiratory muscles and supplied unique pearls of clinical wisdom that sprinkle this revision.

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This Volume is dedicated to
Janet G. Travell, M.D.
1901 - 1997
To whom we all owe a great debt of gratitude
for her inspirational pioneering of this field
and for heading us in the right direction.

Foreword

In my 1992 Foreword to the now-famous mate, *Volume 2,* of the *Trigger Point Manual Volume 1,*1 boldly opined that Volume 2 was "... even better than the other [Vol.1] because it reflects an enormous new recharging of energy that further experience, interaction, and thought have stimulated.... This new volume," I went on to say, "has the distinction of going beyond those areas [emphasized in the first edition of Volume 1] to discuss rationale, new principles arising from a ground-swell of experience, and the unique place of myofascial pain syndrome in the spectrum of musculoskeletal disorders."

"Myofascial trigger points and their significance in painful conditions are no longer the rather controversial subject they were before Volume 1 appeared, nor are the treatment methods taught by Drs. Travell and Simons. These are firmly established and are increasingly being validated by once skeptical clinical investigators ...[Volume 2] goes beyond and opens up new ground in sensitizing clinicians to the important interfaces between myofascial pain syndromes and articular (somatic) dysfunctions on the one hand and fibromyalgia on the other hand. I applaud the wise manner in which these issues are addressed, assessed, and integrated."

How can this scribbler do any better than that eulogy to summarize his opinion of the macho younger mate of Volume 1 when the latter now produces an offspring that outshines both its parents? My scanning of the manuscript before its birth permits me to predict a magnificent success. This is a gargantuan publishing effort that would have done Rabelais credit. But this *Gargantua* is not fantasy; it is hard-headed facts and a wise explication of many current ideas and new findings.

The new edition clarifies for me the overlap of confusing similar conditions that must be distinguished. It illuminates improved testing methods and clearly puts others into their place, often outside. The authors are forthright and precise in the diagnostic criteria for an active trigger point for general clinical use as a "... *circumscribed spot tenderness in a nodule of a palpable taut band and patient recognition of the pain evoked by pressure on the tender spot as being familiar."*

The thorough discussion of the nature of trigger points and their electrodiagnostic characteristics is very valuable and timely, as is the coverage of the histogenesis of trigger points. Updating and expansion throughout the volume now make the two volumes together one of the most impressive medical publishing efforts of modern times. Truly it becomes a *tour de force.* I am proud to be its "godfather."

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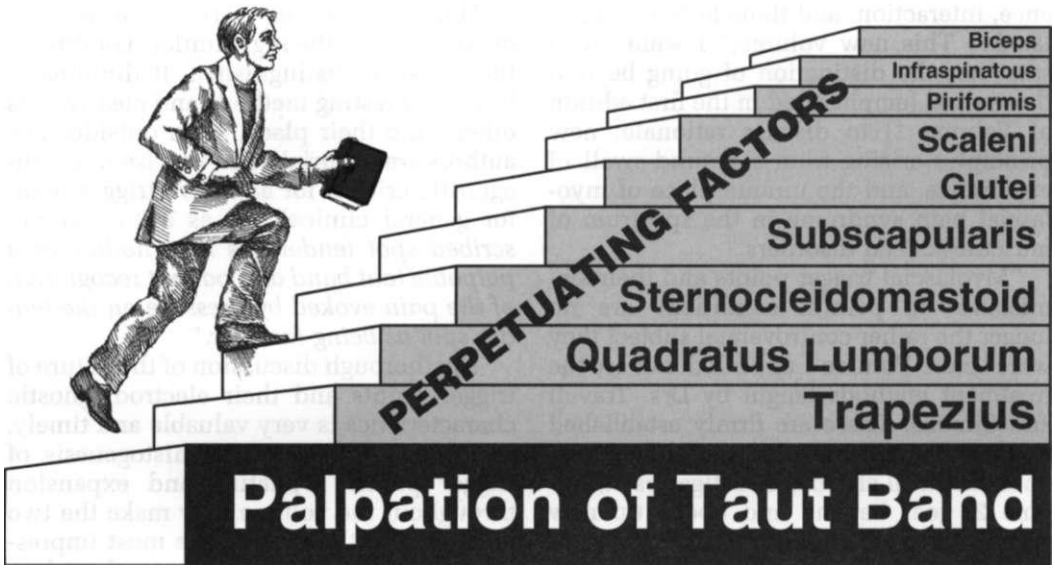

At present, the only way to make a definite diagnosis of a trigger point is by physical examination. This figure emphasizes the fact that the ESSENTIAL first step is to learn how to recognize by palpation the nodule and taut band that are characteristic of a trigger point. Each muscle has individual characteristics which the examiner needs to learn. When active trigger points in any muscle fail to respond to treatment, with few exceptions one or more perpetuating factors need to be identified and resolved.

Preface

The passing of Janet Travell, M.D., on 1 August 1997 at the age of 95 marked the end of the era when she gave life to the concept of myofascial trigger points (TrPs) and nurtured it through childhood. Others must now lead the concept through adolescence to maturity.

As a memorial and tribute to Dr Travell's unique contribution, the following selection is quoted from the preface of the first edition of this volume: "Dr. Travell describes in detail her introduction to myofascial trigger points in her autobiography, *Office Hours: Day and Night.* Although she was brought up on the unitary concept of disease that all of the patient's symptoms should be explained by one diagnosis, she soon learned that life is not like that. The man who has both heart disease and pulmonary tuberculosis may suddenly die of cancer of the lung. Patient complaints that originate in the musculoskeletal system usually have multiple causes responsible for the total picture.

"Early in her medical career, Dr. Travell served simultaneously on pulmonary, cardiology, and general medical services. On all the services, the major complaint she encountered among the patients was pain. The patient might be dying of a serious illness, but when asked, "How are you?" would answer, "Well, ok, except I have this terrible pain in my shoulder. I can't sleep. I can't lie on that side." When asked the cause of the pain, the resident on the pulmonary service would say that it was reflex from the lung. On the cardiology service, in another hospital, patients had the same complaint of shoulder pain, but the resident explained the pain as reflex from the heart, of course. In the general medical clinic, a secretary who spent all day typing and pulling heavy file drawers would describe precisely the same pain complaint; but its origin was said to be "psychosomatic." In none of these patients did the doctors find objective evidence of disease to account for the patient's pain, but the skeletal muscles had not been examined. When Dr. Travell examined these patients, all three groups had isolated tender spots in muscles which, when compressed, reproduced the patient's pain in the shoulder, arm or chest. The common ailment was an unrecognized myofascial trigger point syndrome.

"Fortunately, these observations were made in an environment rich in experimental expertise. She regularly taught pharmacology to medical students whom, to answer their questions, she inspired to perform the appropriate experiments for themselves in the laboratory. The inquisitive students and faculty at the Cornell University Medical College helped Dr. Travell formulate her investigation of the nature of trigger points and how they function.

"She herself was inspired by the interchange of ideas and the criticism of leaders in basic and clinical research at the New York Hospital, Cornell Medical College Center. Foremost among these were Drs. Harry Gold, McKeen Cattell, Vincent du Vigneaud, Ephraim Shorr, Harold G. Wolff, Eugene F. Dubois, and the renowned neurologist, Frank Fremont-Smith, Director of the Josiah Macy Foundation. During the many years of their association, she was especially indebted to her cardiologist collaborator, Seymour H. Rinzler.

"The successful care rendered Senator Kennedy five years prior to his election as President led Dr. Travell to the position of White House Physician under Presidents John F. Kennedy and Lyndon B. Johnson. Except for that one short detour, she never strayed from her primary focus on the diagnosis and management of myofascial pain syndromes due to trigger points."

TRANSITIONS

This second edition of volume 1 of *The Trigger Point Manual* is transitional in a number of ways. Most important, it marks the transition of the trigger point concept from the status of a syndrome of unknown etiology to that of an experimentally established neuromuscular disease entity. Electrophysiological and histopathological evidence presented in Chapter 2 now makes it clear that dysfunctional motor endplates of skeletal muscle fibers are at the heart of the pathophysiology that characterizes myofascial trigger points (TrPs). Many important details remain to be resolved.

This second edition is transitional also because it addresses the close interaction between myofascial TrPs and articular (somatic) dysfunctions. This edition calls attention to that relationship in practically every chapter and presents it some detail in Chapter 16, Posterior Cervical Muscles. At present, these are often treated as unrelated problems that should be integrated in clinical practice for the patient's sake. An osteopathic physician who is well acquainted with myofascial TrPs has achieved this integration in her practice and has contributed to most of the chapters. This edition can only call attention to specific examples of the closeness of the muscle-joint relationship. It barely scratches the surface of what is needed.

This edition marks the beginning of a transition from a volume by two authors to a volume with significant contributions by others. The subject matter is rapidly outgrowing the comprehension of only two individuals.

This edition presents the major progress in our understanding of the pathophysiological basis for many of the clinical phenomena associated with myofascial TrPs. We now know, based on histopathology, that a palpable nodule and an associated taut band are essential features of a myofascial TrP (and also of myogelosis). The importance of the nodularity was not emphasized in the first edition of volume 1. Emphasis is now shifting from pain as the cardinal feature of a myofascial TrP to increased muscle tension and its consequences.

Previously no distinction was drawn between trigger points located in the middle portion of the muscle belly (central TrPs) and those located in a region of muscle attachment (attachment TrPs). The tenderness of each depends on different pathophysiological processes with significant therapeutic implications that have yet to be adequately explored.

In the first edition, an attempt was made to document what we knew about myofascial TrPs, almost all of which was based on clinical observations. There is now the beginning of a peer-reviewed body of literature with blinded, controlled studies that are scientifically credible. Many more are urgently needed. Such studies of the effectiveness of TrP treatment by skilled clinicians should contribute greatly to a more widespread recognition of the importance of myofascial TrPs as a major source of musculoskeletal pain. To facilitate this transition, the present edition now calls attention to specific clinical conditions worthy of investigation (*see* "Research Opportunities" in the Index).

CHANGES IN THIS EDITION

This edition incorporates a number of changes applicable to most chapters. All treatment sections (Section 12) have been extensively rewritten and now include a number of trigger point release techniques in addition to spray and stretch. Section 12 in many of the chapters has been enhanced by the extensive experience and insight of Mary Maloney, P.T., and her daughter Jill Maloney Newman, P.T. Successful inactivation of active TrPs depends on restoring full range of pain-free motion. The most effective technique(s) for achieving that goal depends on an appreciation of the trigger point source of the pain, which muscle is involved, the patient's response, the clinician's training and skill, etc. This change in emphasis is reflected in the new title of section 12, Trigger Point Release. A rewritten Section 11 of every muscle chapter now is called Differential Diagnosis and incorporates the material under its previous title, Associated Trigger Points, as a subheading.

Recent surface electromyographic studies confirm and emphasize the importance of the motor dysfunctions associated with

TrPs. This fundamental and essentially unexplored effect of TrPs may be as important as, or even more important clinically than the referred pain that they cause.

Differences in several individual chapters are noteworthy. Chapter 2 has been completely rewritten and presents a new understanding of the nature of myofascial TrPs. It also considers the role of TrPs in the family of syndromes related to occupational overload of muscles.

The considerable changes in the diagnostic and treatment sections of Chapter 3 reflect the new understanding of the nature of myofascial trigger points. Evidence of muscular dysfunctions as well as the identification of referred pain patterns are emphasized, and effective physical treatments include many more that are now available.

A considerable part of the section on systemic perpetuating factors in Chapter 4 was rewritten and updated by Robert Gerwin, M.D., based on clinical experience and research projects of his own.

Most of the introduction to masticatory muscles in Chapter 5 was written by a dentist, Dr. Bernadette Jaeger, and includes a comprehensive section on the contribution of TrPs to many different kinds of headache. The masticatory muscle chapters themselves, Chapters 8-12, have benefitted greatly from her expertise as well as that of Mary Maloney, P.T., who is well acquainted with the management of myofascial TrPs in masticatory muscles.

Chapters 16 (Posterior Cervical Muscles) and 17 (Suboccipital Muscles) emphasize the close relation between **articular dysfunctions** and TrPs in those muscles.

Chapter 20 (Scalene Muscles) presents (in Section 11) new insights into the controversial and frustrating subject of **thoracic outlet syndrome.**

Chapter 21 (Supraspinatus muscle) reviews (in Section 11) **rotator cuff disease** and considers its relation to the motor and sensory disturbances caused by TrPs.

In Chapter 36 (Supinator Muscle), Section 10 explores the contribution of TrPs to entrapment of the deep radial nerve, and Section 11 examines the close relation between TrPs and **tennis elbow** or lateral epicondylitis.

Chapter 45 is an entirely new and extensive chapter specifically on the diaphragm and the many functions of the intercostal muscles. The chapter includes respiratory mechanics and the contribution of other muscles to respiration.

OVERVIEW OF THIS EDITION

Myofascial trigger points are a frequently overlooked and misunderstood source of the distressingly ubiquitous musculoskeletal aches and pains of mankind. This manual assembles in one place the information necessary for the student and the practitioner to recognize and treat *one* major source of musculoskeletal pain complaints, myofascial TrPs. Many other sources of muscle pain and their neurophysiological basis are presented in another book, *Muscle Pain,* by Mense and Simons, that will be published by Williams & Wilkins.

This first volume of *The Trigger Point Manual* presents introductory general information on all TrPs and also detailed descriptions of single-muscle syndromes for the upper half of the body.

This book is specifically addressed to health care professionals who are concerned for patients with musculoskeletal pain problems and who have been *well* trained in (or are prepared to learn): muscle anatomy, physiology, kinesiology, necessary palpation skills, and how to recognize (and resolve) perpetuating factors. It is NOT intended as a procedural "cook book." It presents a basis for understanding the cause of the patient's pain and ways of eliminating the source of the pain, not just alleviating it.

Usually, acute single-muscle syndromes are easily managed. Often, however, the patient presents with pain that represents a composite pattern referred from several muscles; the practitioner must become a sleuth and recognize the component parts. Much of the detective work lies in tracking down not only what specific stress or stresses initiated the patient's trigger points, but also what *additional* factors may be perpetuating them. Chapter 4 of this manual reviews many of these factors. The identification of perpetuating factors can require a thorough knowledge of body

mechanics, kinesiology, and skillful medical detective work in areas that are often neglected or dismissed as unimportant.

This manual describes individually the component parts of the myofascial jigsaw puzzle. The reader must piece them together to fit the clinical picture of each patient, remembering that no two persons are exactly alike.

The handy listing of muscle pain patterns, alphabetically arranged, is located inside the front cover.

The primary clue as to which muscle has a TrP is the restriction of passive and active range of motion due to pain. In addition, the pattern of pain referred from an active TrP is a valuable guide as to the TrP source of the pain.

Since the pain referred from most myofascial TrPs often appears at some distance from the trigger point, the practitioner can benefit from a guide that identifies which muscles most commonly refer pain to a particular area of the body. The pictorial index on the front fly leaf illustrates what part of the body is included under each of the four main parts of this volume that follow the introduction. Each part is identified by a red thumb tab and begins with a pain-and-muscle guide. This guide illustrates the areas within that region and lists the muscles that are most likely to refer pain to each area. For convenience, the pain-and-muscle guide also lists the chapter number of the chapter that deals with a muscle.

Every muscle chapter is uniformly arranged with fourteen headings and begins with HIGHLIGHTS, a summary of key points in that chapter. The sections of introductory Chapter 3, *Apropos of All Muscles,* have the same fourteen numbered headings. Thus, each section of Chapter 3 serves as a general introduction to the corresponding section for every muscle. It presents information applicable to all muscles. *Information in Chapter 3 that is critical to the management of a patient's myofascial pain syndrome may not be repeated in the individual muscle chapter.*

Acknowledgments

We are especially indebted to four individuals who made considerable contributions to this revision and who are listed on the Contributor page and are identified in chapters to which they made a substantial contribution. They are Robert Gerwin, M.D., Bernadette Jaeger, D.D.S., Mary Maloney, P.T., and Roberta Shapiro, D.O. In addition, Michael Kuchera, D.O., and I. Jon Russell, M.D., PhD. meticulously reviewed and made helpful contributions to Chapter 16.

The research collaboration with Professor Chang-Zern Hong, M.D., in performing both human studies and animal studies resulted in the electrodiagnostic characterization of TrPs that was essential to the identification of their pathophysiology. The support of Phyllis Page, M.D., Chief, Physical Medicine and Rehabilitation Service, Veterans Affairs Medical Center, Long Beach, California and of Raghavaiah Kanekamedala, Director of the Electromyography Laboratory of the same service, was essential to the accomplishment of the electrodiagnostic studies of TrPs in human subjects. The enthusiastic support of Professor Robert Blanks, PhD, Departments of Anatomy and Neurobiology, University of California, Irvine, for the use of his laboratory facilities and the support by Professor Jen Yu, Chairman of the Department of Physical Medicine and Rehabilitation, University of California, Irvine, made possible our investigation of the electrodiagnostic characteristics of trigger spots in rabbits.

In general, the long discussions with Professor Siegfried Mense over many years concerning the nature of myofascial TrPs, and in particular their referred pain neurophysiology, helped greatly in the development of our understanding of the pathophysiology of TrPs. Specifically, the discussions with Professor Mense concerning the nature of the potentials being observed at active loci contributed significantly to steering us in the right direction, and his critical review of Chapter 2 was most helpful.

Dr. Hong's numerous controlled clinical studies have helped greatly to give research substance to the clinical impressions of TrP characteristics.

We are especially grateful to Jason Lee for carrying the major responsibility of typing most of the revisions, for obtaining and organizing references, and for doing some editing. Jason was a veritable genius with the care and feeding of the computers and exhibited a priceless talent for finding essential items that we had misplaced from time to time. Our thanks also go to Barbara Zastrow, who provided the secretarial services at the beginning of the revision, and to Frances Denmark, who competently and cheerfully provided the assistance that enabled us to complete the work.

The observations made and questions raised by Jochen Sachse, M.D., while translating the first edition into German identified many necessary corrections. His efforts and expertise are much appreciated.

The visit to the office of Michel Bouve, M.D., D.C. in Belgium redirected our attention to the critical functional importance of the restricted range of motion caused by TrPs even when the patient presents with little or no pain complaint. We now better appreciate both theoretically and clinically how increased muscle tension can be the fundamental, primary effect of TrPs and that pain can be a less constant, secondary phenomenon.

We express our deep respect and gratitude to Barbara D. Cummings for completing most of the drawings for this revision, and to Diane Abeloff for contributing the rest of the drawings. The computer-generated figures are a tribute to the computer skills of Jason Lee. It was a pleasure

to work with our Project Editor, Jeffrey Myers, who kindly made the additional changes necessary in page proof and provided much additional information needed to ensure a quality product.

Last, but by no means least in importance, words cannot adequately express our deep gratitude to our Managing Editor, Linda Napora, for her close support and encouragement throughout the enormous undertaking of this revision. Her patient persistence and understanding helped to sustain and encourage us, buoyed up by her delightful sense of humor which made us laugh through the tears at times when we desperately needed laughter.

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Contents

PART 1
INTRODUCTION

CHAPTER 1
Glossary

The glossary comes first to ensure that the reader knows what a term means as it is used in this manual and to help the reader become acquainted with unfamiliar terms. The glossary is in the front of the book to encourage frequent reference to it, whenever needed. Comments concerning a definition are added in *italics.*

Abduction: Movement away from the midline. For **fingers,** it is movement away from the midline of the middle digit. For the **thumb,** it is movement perpendicular to, and away from, the plane of the palm. For the **hand,** at the wrist, it is radial deviation of the hand, which is away from the midline of the body in the anatomical position. For the **arm,** at the shoulder joint, abduction moves the elbow in the frontal plane away from the midline of the body. For the **scapula,** it is a gliding movement across the posterior thorax away from the vertebral column.

Active Locus (of a Trigger Point): A minute region in a muscle that exhibits spontaneous electrical activity (often characterized as endplate noise) and that may or may not also exhibit spike activity characteristic of single fiber action potentials.

Active Range of Motion: The extent of movement (usually expressed in degrees) of an anatomical segment at a joint when the movement is produced only by voluntary effort of the subject to move that part of the body being tested.

Active Myofascial Trigger Point: A myofascial trigger point that causes a clinical pain complaint. It is always tender, prevents full lengthening of the muscle, weakens the muscle, refers a patient-recognized pain on direct compression, mediates a local twitch response of muscle fibers when adequately stimulated, and, when compressed within the patient's pain tolerance, produces referred motor phenomena and often autonomic phenomena, generally in its pain reference zone, and causes tenderness in the pain reference zone. *To be distinguished from a latent myofascial trigger point.*

Acute: Of recent onset (hours or days).

Adduction: Movement toward the midline. For **fingers,** it is movement toward the midline of the middle digit. For the **thumb,** it is movement perpendicular to, and toward, the plane of the palm. For the **hand,** it is ulnar deviation at the wrist. For the **arm,** at the shoulder joint, it is movement of the elbow toward the midline of the body, movement in the frontal plane from the abducted position of the arm. For the **scapula,** it is a gliding movement across the posterior thorax toward the vertebral column.

Agonists: Muscles, or portions of muscles, so attached anatomically that when they contract they develop forces that complement or reinforce each other.

Allodynia: Pain due to a stimulus that does not ordinarily provoke pain (decreased

pain threshold; the response is a different kind of sensation than that normally evoked by the stimulus).

Analgesia: Absence of pain in response to stimulation which would normally be painful.

Anatomical Position: The erect position of the body with the face forward, each arm at the side of the body, forearms supinated so that the palms of the hands are facing forward, fingers in extension, feet together with the toes directed forward. *The terms posterior, anterior, lateral, medial, superior, inferior, etc. are applied with the body in this position.*

Antagonists: Muscles, or portions of muscles, so attached anatomically that when they contract they develop forces that oppose each other.

Anterior: Toward the front of the body (ventral); opposite of posterior (dorsal).

Arm: In this volume, the arm includes only the segment of the upper limb between the shoulder and elbow, and does not include the entire upper limb.

Associated Myofascial Trigger Point: A trigger point in one muscle that occurs concurrently with a trigger point in another muscle. *One of these associated trigger points may have induced the other, or both may stem from the same mechanical or neurologic origin.*

Atlantoaxial Joint: Junction of the atlas (CJ and the axis (C_2).

Attachment Trigger Point: A trigger point at the musculotendinous junction and/or at the osseous attachment of the muscle that identifies the enthesopathy caused by unrelieved tension characteristic of the taut band that is produced by a central trigger point.

Bruxism: Clenching of the teeth, resulting in rubbing, gritting, or grinding together of the teeth, usually during sleep.[6]

Caudad: Away from the head, toward the tail; usually synonymous with inferior; opposite of cephalad.

Central Myofascial Trigger Point: A myofascial trigger point that is closely associated with dysfunctional endplates and is located near the center of muscle fibers.

Cephalad: Toward the head; usually synonymous with superior; opposite of caudad.

Chronic: Long-standing (weeks, months or years), but NOT necessarily irreversible. *Symptoms may be mild or severe.*

Composite Pain Pattern: Total referred pain pattern of two or more closely adjacent muscles. *No distinction is made among the referred pain patterns of the individual muscles.*

Contract-Relax: As used in this volume, it is a gentle voluntary muscle contraction followed by relaxation with encouragement of elongation of the muscle. *To be distinguished from Hold-Relax, which is isometric.*

Contraction (of muscle): Activation of the contractile elements of muscle fibers by propagated action potentials. *To be distinguished from Contracture.*

Contracture (of muscle): Sustained intrinsic activation of the contractile elements of muscle fibers. With contracture, muscle shortening occurs in the absence of motor unit action potentials. *This physiological definition, as used in this manual, must be differentiated from the clinical definition, which is shortening due to fibrosis. Contracture also must be distinguished from contraction and spasm.*

Coordinated (normal) Respiration: Expansion of the chest with simultaneous contraction of the diaphragm which increases intraabdominal pressure and protrudes the abdomen during inhalation. *To be distinguished from paradoxical (abnormal) respiration.*

Coronal Plane: A frontal (vertical) plane that divides the body into anterior and pos-

terior portions and lies at right angles to a sagittal plane.[10]

Deep: Farther from the surface; *opposite of superficial.*

Distal: Farther from the trunk or point of origin; *opposite of proximal.*

Dysesthesia: An unpleasant abnormal sensation, whether spontaneous or evoked.

Enthesitis: "Traumatic disease occurring at the insertion of muscles where recurring concentration of muscle stress provokes inflammation with a strong tendency toward fibrosis and calcification."[7] *The enthesopathy referred to in this book may, in time, develop into an enthesitis.*

Enthesopathy: A disease process at musculotendinous junctions and/or where tendons and ligaments attach into bones or joint capsules. *It is characterized by local tenderness and may, in time, develop into enthesitis.*

Erector Spinae Muscles: This group of muscles consists of the spinalis, the longissimus, and the iliocostalis muscles,[1] which are the longest, most longitudinal, and most superficial of the paraspinal musculature.

Essential Pain Zone (Area): The region of referred pain (indicated by solid red areas in pain pattern figures) that is present in nearly every patient when the trigger point is active. *To be distinguished from a spillover referred pain zone.*

Extension: In general, straightening of hinge joints. In the upper limb, it is movement in the posterior direction in a sagittal plane. In the case of the thumb, it is movement in the radial direction in the plane of the palm.

Fibrositis: An outmoded term with multiple meanings. *Many authors in the past used it to identify what were myofascial trigger points. Other authors have used the term very differently (see Chapter 2). We avoid using the term because of its ambiguity.*

Flat Palpation: Examination by finger pressure that proceeds across the muscle fibers at a right angle to their length, while compressing them against a firm underlying structure, such as bone. *Flat palpation is used to detect taut bands and trigger points. To be distinguished from pincer palpation and snapping palpation.*

Flexion: In general, bending of hinge joints. In the upper limb, it is movement in the anterior direction in a sagittal plane. In the case of the thumb, it is movement in the ulnar direction in the plane of the palm.

Function (of a muscle): In this edition of volume 1, the actions (movements) of a muscle are included under its function. *No sharp distinction is made between function and action.*

Functional Unit: A group of agonist and antagonist muscles that function together as a unit because they share common spinal-reflex responses. *The agonist muscles may act in series or in parallel. Previously identified as a Myotatic Unit.*

Hold-Relax: As used in this volume, a gentle voluntary isometric muscle contraction followed by relaxation. *To be distinguished from Contract-Relax, in which movement takes place.*

Horizontal Abduction: Movement of the elevated arm about a longitudinal axis in the transverse plane, away from the midline of the body.

Horizontal Adduction: Movement of the elevated arm in the transverse plane toward the midline of the body.

Hyperesthesia: Increased sensitivity to stimulation, excluding the special senses.

Hyperpathia: A painful syndrome characterized by abnormally painful reaction to a stimulus, especially a repetitive stimulus (both threshold and response are increased).

Hyperalgesia: An increased pain response to a stimulus that is normally painful

(stimulus and response are in the same mode).

Hypoalgesia: Diminished pain in response to a normally painful stimulus.

Incisal Path: The path of a point in the groove between the lower central incisor teeth in relation to the sagittal plane as the jaws are opened and closed.

Inferior: Toward the soles of the feet; synonymous with caudal in the trunk; *opposite of superior.*

Involved Muscle: A muscle that has developed one or more active or latent trigger points.

Ischemic Compression: Now revised and identified as Trigger Point Pressure Release.

Joint Play: Small movements within a synovial joint that are independent of, and cannot be induced by, voluntary muscle contraction. *Essential for normal, pain-free, nonrestricted movement of the articulation.*[2]

Jump Sign: A general pain response of the patient, who winces, may cry out, and may withdraw in response to pressure applied on a trigger point. *This term has been used erroneously to describe the local twitch response of muscle fibers to trigger-point stimulation.*

Key Myofascial Trigger Point: A trigger point responsible for activating one or more satellite trigger points. Clinically, a key trigger point is identified when inactivation of that trigger point also inactivates the satellite trigger point.

Latent Myofascial Trigger Point: A myofascial trigger point that is clinically quiescent with respect to spontaneous pain; it is painful only when palpated. *A latent trigger point may have all the other clinical characteristics of an active trigger point and always has a taut band that increases muscle tension and restricts range of motion.*

Lateral: Farther from the midsagittal plane of the body or from the midline of a structure; *opposite of medial.*

Lateral Rotation (External Rotation, Rotation Outward): Rotation of the anterior surface of the limb away from the midline of the body. For the scapula, it is upward rotation about an anteroposterior axis, with the inferior angle moving laterally and the glenoid cavity moving cranially.

Local Twitch Response: A transient contraction of a group of tense muscle fibers (taut band) that traverse a trigger point. The contraction of the fibers is in response to stimulation (usually by snapping palpation or needling) of the same trigger point, or sometimes of a nearby trigger point. *Sometimes the local twitch response has been erroneously called a jump sign.*

Low Back Pain: Pain in lumbar, sacral, and/or gluteal areas; *a descriptive term that does not identify a diagnosis or cause.*

Lumbago: Pain in the mid and lower back; *a descriptive term that does not identify a diagnosis or cause.*

Medial: Closer to the midsagittal plane of the body or to the midline of a structure; *opposite of lateral.*

Medial Rotation (Internal Rotation, Rotation Inward): Rotation of the anterior surface of the limb toward the midline of the body. For the scapula, it is downward rotation about an anteroposterior axis, with the inferior angle moving medially and the glenoid cavity moving caudally.

Motor Endplate: Soleplate ending where a terminal branch of the axon of a motor neuron makes synaptic contact with a striated muscle fiber (cell).

Muscular Rheumatism [Muskel Rheumatismus): Muscular pain and tenderness attributed to "rheumatic" causes (especially exposure to cold). *Sometimes used to identify myofascial trigger points. To be distinguished from articular rheumatism.*

Myalgia: Pain in a muscle or muscles.[8] The term is used in two ways to signify: (1) diffusely aching muscles due to systemic disease, such as a virus infection, and (2) the spot tenderness of a muscle or muscles as in myofascial trigger points. *The reader must distinguish which use an author has in mind.*

Myofascial Pain Dysfunction Syndrome: A controversial, largely outmoded term that has been considered to mean a syndrome largely of muscular origin, a complex psychophysiological phenomenon, or a syndrome primarily due to disturbed occlusal mechanics.

Myofascial Pain Syndrome (Myofascial Syndrome): 1. (as used in this book) The sensory, motor, and autonomic symptoms caused by myofascial trigger points. *The specific muscle or muscle group that causes the symptoms should be identified.* 2. (as sometimes confusingly used by others) A regional pain syndrome of any soft tissue origin.[13] *To avoid confusion, we recommend that when anyone uses the term myofascial pain syndrome, that person should specify which meaning applies—*file *general or specific definition.*

Myofascial Trigger Point (clinical definition of a central trigger point): A hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is painful on compression and can give rise to characteristic referred pain, referred tenderness, motor dysfunction, and autonomic phenomena. *Types of myofascial trigger points include: active, associated, attachment, central, key, latent, primary, and satellite. (Note especially the distinction between central and attachment myofascial trigger points). Any myofascial trigger point is to be distinguished from a cutaneous, ligamentous, periosteal, or any other nonmuscular trigger point.*

Myofascial Trigger Point (etiological definition of a central trigger point): A cluster of electrically active loci each of which is associated with a contraction knot and a dysfunctional motor endplate in skeletal muscle.

Myofascitis (Myositis Fibrosa): Induration of a muscle through an interstitial growth of fibrous tissue.[9] *Sometimes used erroneously in the past as synonymous with myofascial trigger points.*

Myogelosis: Circumscribed firmness and tenderness to palpation in a muscle or muscles associated with the patient's pain complaint. *The name is derived from the concept that the regions of circumscribed firmness were due to localized gelling of muscle proteins. Focal tenderness and palpable taut muscle fibers and nodules are also characteristic of myofascial trigger points. Most patients diagnosed as having myogelosis also would be diagnosed as having myofascial trigger points.*

Myotatic Unit: See Functional Unit.

Occipitoatlantal joint: Junction of the occiput (C_0) and the atlas (CJ,[3] sometimes referred to as the atlanto-occipital (joint).[5]

Occlusal Disharmony: Occlusal contacts that interfere with centric occlusion of the teeth or with functional mandibular excursions from centric occlusion.[4,12]

Palpable (Taut) Band: *See* Taut Band.

Paradoxical (abnormal) Respiration: Simultaneous expansion of the chest and contraction of abdominal muscles that pulls the abdomen inward during inhalation. *To be distinguished from coordinated (normal) respiration.*

Passive Range of Motion: The extent of movement (usually tested in a given plane) of an anatomical segment at a joint when movement is produced by an outside force without voluntary assistance or resistance by the subject. *The subject must relax the muscles crossing the joint.*

Pincer Palpation: Examination of a part by holding it in a pincer grasp between the thumb and fingers. *Groups of muscle fibers are rolled between the tips of the digits to detect taut bands of fibers, to identify trigger point nodules and tender spots in the*

muscle, and to elicit local twitch responses. To be distinguished from flat palpation and snapping palpation.

Posterior: Toward the back of the body (dorsal); *opposite of anterior.*

Pressure Release: See Trigger Point Pressure Release.

Primary Myofascial Trigger Point: A central myofascial trigger point that was apparently activated directly by acute or chronic overload, or repetitive overuse of the muscle in which it occurs and was not activated as a result of trigger-point activity in another muscle.

Prone: Lying face downward; *opposite of supine.*

Proximal: Closer to the trunk or point of origin; *opposite of distal.*

Reactive Cramp: Synonymous with shortening activation.

Reference Zone: see **Zone of Reference.**

Referred Autonomic Phenomena: Vasoconstriction (blanching), coldness, sweating, pilomotor response, ptosis, and/or hypersecretion that occur in a region separate from the trigger point causing these phenomena. *The phenomena usually appear in the same general area to which that trigger point refers pain.*

Referred (Trigger-Point) Pain: Pain that arises in a trigger point, but is felt at a distance, often entirely remote from its source. The pattern of referred pain is reproducibly related to its site of origin. *The distribution of referred trigger-point pain rarely coincides entirely with the distribution of a peripheral nerve or dermatomal segment.*

Referred (Trigger-Point) Phenomena: Sensory and motor phenomena such as pain, tenderness, increased motor unit activity (spasm), vasoconstriction, vasodilatation, and hypersecretion caused by a trigger point, which usually occur at a distance from the trigger point.

Release (of muscle tightness): Any procedure that reduces the resting muscle tension (or muscle stiffness).

Sagittal Plane: A vertical anteroposterior plane that divides the body into right and left portions. The midsagittal plane divides the body into right and left halves.

Satellite Myofascial Trigger Point: A central myofascial trigger point that was induced neurogenically or mechanically by the activity of a key trigger point. *Distinguishing the mechanism responsible for the key-satellite relationship can rarely be resolved by examination alone. The relationship usually is confirmed by simultaneous inactivation of the satellite when the key trigger point is inactivated. A satellite trigger point may develop in the zone of reference of the key trigger point, in an overloaded synergist that is substituting for the muscle harboring the key trigger point (key muscle), in an antagonist countering the increased tension of the key muscle, or in a muscle linked apparently only neurogenically to the key trigger point. Previously, only a trigger point that developed in the referred pain zone of another trigger point was identified as a satellite trigger point.*

Scoliosis: Lateral curvature of the spine.

Screening Palpation: Digital examination of a muscle to determine the absence, or presence, of palpable bands and tender trigger points using flat and/or pincer palpation.

Secondary Trigger Point: Term previously used, but rarely in this edition. Trigger points previously identified as secondary trigger points are now classified as satellite trigger points. *A secondary trigger point was previously identified as one that developed in a synergist or an antagonist of the muscle harboring the key trigger point.*

Shortening Activation: Activation of latent myofascial trigger points by unaccustomed

shortening of a muscle during stretch therapy of its antagonist. *An activated trigger point increases tension of its muscle and can cause severe referred pain.*

Shoulder Joint: Glenohumeral joint.

Snapping Palpation: A fingertip is placed against the tense band of muscle at right angles to the direction of the band and suddenly presses down while the examiner draws the finger back so as to roll the underlying fibers under the finger. (The motion is similar to that used to pluck a guitar string, except that the finger does not slide over the skin but moves the skin with it.) *To most effectively elicit a local twitch response, the band is palpated and snapped at the trigger point, with the muscle positioned to eliminate slack. To be distinguished from flat palpation and pincer palpation.*

Spasm: Increased tension with or without shortening of a muscle due to nonvoluntary motor nerve activity. Spasm is identified by motor unit potentials that cannot be terminated by voluntary relaxation. *To be distinguished from contracture.*

Spillover Pain Zone (Area): The region where some, but not all, patients experience referred pain beyond the essential pain zone, due to greater hyperirritability of a trigger point. *The spillover zone is indicated by red stippling in the pain-pattern figures. To be distinguished from an essential referred pain zone that is solid red.*

Strain: Tissue and psychological reaction to prolonged stress.

Stretch: Any procedure that elongates the muscle fibers. *With trigger points, the goal of the procedure is to release the increased muscle tension by elongating the shortened sarcomeres of contraction knots.*

Stress: 1. A physical or psychological overload that produces a tissue or psychological reaction. 2. "The resisting force set up in a body as a result of an externally applied force."[11] 3. A force that tends to produce distortion.

Square Brackets [ ]: In this volume square brackets identify comments or interpretations by the authors.

Stripping Massage (Deep-stroking Massage): As described in Chapter 3 Section 12.

Suboccipital Decompression: A tension-release procedure for the upper cervical region. It is performed with the patient in the supine position and the examiner's fingertips placed in the suboccipital recess bilaterally; initially, pressure is applied anteriorly (toward the ceiling) so as to induce regional extension at the OA, C_{1}, and C_{2} junctions. Then traction is applied in a cephalad direction.

Superficial: Closer to the surface; *opposite of deep.*

Superior: Toward the vertex of the head; usually synonymous with cephalad; *opposite of inferior.*

Supination: A movement of the forearm that positions the palm anteriorly when the body is in the anatomical position.

Supine: Lying face upward; *opposite of prone.*

Synergistic Muscles: Muscles that reinforce or complement each other when they contract.

Taut Band: The group of tense muscle fibers extending from a trigger point to the muscle attachments. *The tension of the fibers is caused by contraction knots that are located in the region of the trigger point. Reflex contraction of the fibers in this band produces the local twitch response.*

Thoracic Outlet: The triangular aperture bounded anteriorly by the scalenus anterior muscle, posteriorly by the scalenus medius muscle, and interiorly by the first rib. *Some other authors include all of the superior opening of the thoracic cage.*

Transverse Plane: A horizontal plane that divides the body into upper and lower portions.

Trigger Area: Sometimes used in this volume as synonymous with an attachment trigger point.

Trigger Point (Trigger Zone, Trigger Spot, Trigger Area): See Myofascial Trigger Point.

Trigger Point Pressure Release: Application of slowly increasing, nonpainful pressure over a trigger point until a barrier of tissue resistance is encountered. Contact is then maintained until the tissue barrier releases, and pressure is increased to reach a new barrier to eliminate the trigger point tension and tenderness. *In this edition, Trigger Point Pressure Release replaces the term Ischemic Compression that was used in the first edition. Other versions (some of which are painful) are identified elsewhere as Acupressure, Myotherapy, Shiatzu, and as "Thumb" Therapy.*

Trigger Point Release: Release of muscle tension by inactivating the trigger points that are causing the taut bands which are responsible for the increased tension. *Many methods of release are described in Section 12 of Chapter 3.*

Zone of Reference: The specific region of the body at a distance from a trigger point, where phenomena (sensory, motor, and/or autonomic) caused by the trigger point are observed.

REFERENCES

1. **Clemente CD. *Gray's Anatomy,* 30th ed. Philadelphia: Lea & Febiger, 1985:466-469, 472 (Fig. 6-21).**
2. **Greenman PE. *Principles of Manual Medicine.* Baltimore: Williams & Wilkins, 1996:99.**
3. **Ibid. (p. 175).**
4. **Mahan PE. Personal communication, 1981.**
5. **Maigne R. *Diagnosis and Treatment of Pain of Vertebral Origin: A Manual Medicine Approach.* Baltimore: Williams & Wilkins, 1996:54-55.**
6. **McDonough JT Jr. *Stedman's Concise Medical Dictionary,* 2nd ed. Baltimore: Williams & Wilkins, 1994:141.**
7. **Ibid, (p.339).**
8. **Ibid. (p. 659).**
9. **Ibid. (p. 664).**
10. **Ibid. (p. 793).**
11. **Ibid. (p. 966).**
12. **Shaber EP. Personal communication, 1981.**
13. **Simons DG. Myofascial pain syndrome: one term but two concepts: a new understanding [editorial]. *JMusculoske Pain* 1995;3(1):7-13.**

Acronyms

AA: atlantoaxial (joint).

ACh: acetylcholine

ATP: adenosine triphosphate

ATPase: adenosine triphosphatase

ATrP: attachment trigger point

C: centigrade (degrees)

C_2: second cervical spinal nerve

Ca^{2+}: ionized calcium

CBC: complete blood count

Cbl: cobalamin (vitamin **B_{12}**)

CK: creatine kinase

cm: centimeter

CTrP: central trigger point

DNA: deoxyribonucleic acid

EMG: electromyographic

ESR: erythrocyte sedimentation rate

ETK: erythrocyte transketolase

F: Fahrenheit (degrees)

FIGLU: formiminoglutamate

FMS: fibromyalgia syndrome

FT_3: free triiodothyronine

FT_4: free levothyroxine

g: gram

GABA: gamma aminobutyric acid

GOT: glutamic oxaloacetic transaminase

GPT: glutamic pyruvate transaminase

h: Hour

HTC II: holo-transcobalamin II

Hz: Hertz (frequency)

INH: isonicotinic acid hydrazide (isoniazid)

IP: Interphalangeal (joint)

Kilo: kilogram

kg: Kilogram, a unit of weight equal to 1,000 grams; *approximately 2.2 pounds.*

LLLI: lower limb-length inequality

LTR: local twitch response

m: Meter, a defined measure of distance; *approximately 39 inches.*

MCP: metacarpophalangeal (joint)

MCV: mean corpuscular volume

Me-Cbl: methylcobalamin

Meq: milliequivalent

Me-THF: methyltetrahydrofolate

mg: milligram (1/1,000 of one gram)

Mg: magnesium

(Mg): magnesium concentrations

mg/dl: milligrams per decaliter

MIU/L: milli international units per liter

mm: Millimeter, 1/1,000 of a meter; *approximately 1/25 inch.*

Mm: millimolar (0.001 moles of solute per liter of solution)

MP: metacarpophalangeal (joint)

MPD: myofascial pain dysfunction (syndrome)

MPS: myofascial pain syndrome

msec/div: milliseconds per division (of sweep speed)

ng**/ml:** nanogram per milliliter (10^{-9} or 1/1,000,000,000 of one gram)

OA: occipitoatlantal (joint)

oz: ounce

pg**/ml:** picograms per milliliter (10^{12} or 1/1,000,000,000,000 of one gram)

PSIS: posterior superior iliac spine

RBC: red blood cell

RDA: recommended (required) daily allowance

RDG: Robert D. Gerwin **RDI:** recommended

dietary intake **SEA:** spontaneous electrical

activity **SI:** Sacroiliac (joint)

SR: sarcoplasmic reticulum *(see* Figs. 2.5 and 2.13) **sTSH:** sensitive thyrotropin (test) **T_3:** 3,5,3'-triiodothyronine **T_4:** levothyroxine

$T_{„}$: fourth thoracic vertebra or spinal nei

TBG: thyroxine-binding-globulin

THF: tetrahydrofolate

TM: temporomandibular

TMD: temporomandibular disorders

TMJ: temporomandibular joint

TOS: thoracic outlet syndrome

TPP: thiamine pyrophosphate

TRH: thyrotropin releasing hormone

TrP: trigger point

TrPs: trigger points

TSH: thyroid-stimulating hormone

(jisec: microsecond, or 0.001 sec

(xV: microvolt, a measure of electrical] tential: 10^6 volt, or 0.0000001 volt

CHAPTER 2
General Overview

HIGHLIGHTS: **BACKGROUND** (Section A) summarizes the prevalence and importance of myofascial trigger points (TrPs) and then reviews their place in the medical literature of this century. Many overlapping and some confusingly similar conditions currently identify muscle pain syndromes. Clear distinctions are important. The most distinctive **CLINICAL CHARACTERISTICS OF TRIGGER POINTS** (Section B) are a history of pain related to muscular activity and characteristic physical findings. Examination of the muscle reveals circumscribed spot tenderness in a nodule that is part of a palpably tense band of muscle fibers, patient recognition of the pain evoked by pressure on the tender spot as being familiar, pain referred in the pattern characteristic of TrPs in that muscle, a local twitch response (LTR), painful limitation of stretch range of motion, and some weakness of that muscle. Promising *TESTING* methods that demonstrate the presence of TrPs include a specific needle electromyographic (EMG) technique, ultrasound, surface EMG, algometry, and thermography. Referred motor dysfunctions during activity can be tested using surface EMG techniques. Appropriate *TREATMENT* of patients for TrPs may involve many forms of stretch, several techniques to augment muscle release, injection of TrPs, management of perpetuating factors, and a home self-treatment program. Recommended *DIAGNOSTIC CRITERIA* of an active TrP for general clinical use are circumscribed spot tenderness in a nodule of a palpable taut band and patient recognition of the pain evoked by pressure on the tender spot as being familiar. The *DIFFERENTIAL DIAGNOSIS and CONFUSIONS* section lists many conditions mimicked by TrPs. It emphasizes the importance of understanding and examining for the distinguishing characteristics of fibromyalgia and articular dysfunctions as compared to myofascial TrPs. **MUSCLE STRUCTURE AND FUNCTION** (Section C) examines the motor unit, motor endplate zone, and neuromuscular junction in some detail. It updates an understanding of muscle pain. **NATURE OF TRIGGER POINTS** (Section D) first reviews the newly discovered *Electrodiagnostic Characteristics of Trigger Points* which include the demonstration of spontaneous electrical activity and spikes at active loci that are closely associated with dysfunctional motor endplates. Then it presents the newly identified *Histogenesis of Trigger Points* that recognizes contraction knots as the key feature which apparently are closely related to active loci. This leads to an *Integrated Hypothesis of Trigger Points* that postulates a local energy crisis which results from the dysfunctional endplates at active loci. *Other Hypotheses* are considered unlikely. The extensive research on the *Local Twitch Response* is summarized.

A. BACKGROUND

Prevalence

Myofascial trigger points (TrPs) are extremely common and become a painful part of nearly everyone's life at one time or another. *Latent* TrPs, which often cause motor dysfunction (stiffness and restricted range of motion) without pain, are far more common than the *active* TrPs, which in addition cause pain.

Among 200 unselected, asymptomatic young adults, Sola, *et* a/.[261] found focal tenderness representing latent TrPs in the shoulder-girdle muscles of 54% of the female, and 45% of the male subjects. Referred pain was demonstrated in 25% of these subjects with latent TrPs. A recent study of 269 *unselected* female student nurses with or without pain symptoms[228] showed a similar high prevalence of TrPs in masticatory muscles. A TrP was identified by palpating a taut band for spot tenderness of sufficient sensitivity to cause a pain reaction. No effort was made to distinguish active and latent TrPs, but a considerable number of TrPs were likely active because 28% of subjects were aware of pain in the temple area. In masticatory muscles, TrPs were found in 54% of right lateral pterygoid muscles, in 45% of right deep masseter, in 43% of right anterior temporalis, and in 40% of intraoral examinations of the right medial pterygoid muscle. Among the neck muscles, TrPs were identified in 35% of the right splenius capitis muscles and in 33% of right upper trapezius muscles. The insertion of the right upper trapezius was also tender in 42% of those muscles with TrPs. Enthesopathy of this muscle was common.[228]

Frohlich and Frohlich[84] examined 100 asymptomatic control subjects for latent TrPs in lumbogluteal muscles. They found latent TrPs in the following muscles: quadratus lumborum (45% of patients), gluteus medius (41%), iliopsoas (24%), gluteus minimus (11%), and piriformis (5%).

Reports of the prevalence of myofascial TrPs in specific patient populations are available and, together, indicate a high prevalence of this condition among individuals with a regional pain complaint. The reports that follow are summarized in Table 2.1.

In an internal medicine group practice,[257] 54 of 172 patients presented with a pain complaint. Sixteen [30%] of the pain patients met the criteria for myofascial TrPs. Four of these sixteen patients had pain duration of less than 1 month, three had pain for 1 to 6 months, and nine had pain duration of more than 6 months.

A neurologist examining 96 patients from a community pain medical center[90] found that 93% had at least part of their pain caused by myofascial TrPs and in 74%, myofascial TrPs were considered the primary cause of the pain.

Among 283 consecutive admissions to a comprehensive pain center, a primary organic diagnosis of myofascial syndrome was assigned in 85% of cases.[80] A neurosurgeon and a physiatrist made this diagnosis independently, based upon physical examination "as described by Simons and Travell."[255]

Of 164 patients referred to a dental clinic for chronic head and neck pain of at least 6 months duration, 55% were found to have a primary diagnosis of myofascial pain syndrome caused by active TrPs.[83]

Five lumbogluteal muscles of 97 patients complaining of pain in the locomotor system were examined in an orthopedic clinic.[84] Forty-nine percent of the patients presented with latent TrPs and 21% presented with active TrPs in the piriformis muscle.

The wide range in prevalence of myofascial pain caused by TrPs that is reported in different studies is likely due in part to differences in the patient populations examined and in the degree of chronicity. Probably even more important are differences in the criteria used to make the diagnosis of myofascial TrPs and, most important, differences in the training and skill level of the examiners. Few of these studies gave a detailed description of the diagnostic examinations employed. A summary of prevalence[242] excluded papers that used the general definition[241] of a myofascial pain syndrome. Active myofascial TrPs are clearly very common and are a major source of musculoskeletal pain and dysfunction, but poor agreement on appropriate diagnostic criteria has been a serious handicap. A study has critically tested interrater reliability for 5 manual examinations in 5 different muscles[94] among four

Table **2.1.** ***Prevalence of Trigger Point Pain in Selected Patient Populations***

Region	*Practice*	*Number Studied*	*% with Myofascial Pain*	*Source*
General	Medical	172(54)	30%	Skootsky, *etal.,* 1989[257]
General	Pain Med. Center	96	93%	Gerwin, 1995[90]
General	Comprehensive Pain Center	283	85%	Fishbain, *etal.,* 1986[80]
Craniofacial	Head & Neck Pain Clinic	164	55%	Fricton, *etal.,* 1985[83]
Lumbogluteal	Orthopedic Clinic	97	21%	Frohlich and Frohlich, 1995[84]

experienced and trained examiners. The study demonstrated good to excellent agreement for all muscles and for all examinations except for one examination, which was not highly reliable for all muscles tested.

In a population of hospitalized and ambulatory Physical Medicine and Rehabilitation Service patients with the fibrositis syndrome (mostly TrPs), the greatest number were between 31 and 50 years of age.[155] These data agree with our clinical impression that individuals in their mature years of maximum activity are most likely to suffer from the pain syndromes of active myofascial TrPs. With the reduced activity of more advanced age, the stiffness and restricted range of motion of latent TrPs tend to become more prominent than the pain of active TrPs.

Importance

Voluntary (skeletal) muscle is the largest single organ of the human body and accounts for nearly 50% of body weight.[93,91,73] The number of muscles counted in the body depends on the degree of subdivision that is considered one muscle and on the number of variable muscles that are included. Not counting heads, bellies, and other divisions of muscles, the *Nomina Anatomica* reported by the International Anatomical Nomenclature Committee under the Berne Convention,[136] lists 200 paired muscles, or a total of 400 muscles. Any one of these muscles can develop myofascial TrPs that refer pain and motor dysfunction, often to another location.

The clinical importance of myofascial TrPs to practitioners has been described in the literature for acupuncturists,[111, 187,] anesthesiologists,[208- 23, 260] chronic pain managers,[221] dentists,[83, 102, 140, 271] family practitioners,[184204] gynecologists,[213] neurologists,[87] nurses,[25] orthopedic surgeons,[6,10, 46] pediatricians,[1168] physical therapists,[199, 200] physiatrists,[31,139,219,220,223] rheumatologists,[82,89,215] and veterinarians.[143]

Yet the muscles in general and TrPs in particular receive little attention as a major source of pain and dysfunction in modern medical school teaching and in medical textbooks. This manual describes a neglected, major cause of pain and dysfunction in the largest organ of the body. The contractile muscle tissues are a primary target of the wear and tear of daily activities, but it is the bones, joints, bursae and nerves on which physicians usually concentrate their attention.

Severity. The severity of symptoms caused by myofascial TrPs ranges from the agonizing incapacitating pain caused by very active TrPs to the painless restriction of movement and distortion of posture due to latent TrPs that are so commonly overlooked. The potential severity of pain from acute activation of a TrP is illustrated by one housewife who, while bending over cooking, activated a quadratus lumborum TrP that felled her to the kitchen floor and caused pain so severe that she was unable to reach up and turn the stove off to prevent a pot from burning through its bottom. The patients with myofascial TrP pain in a general medicine practice reported visual analog scale ratings of pain as high as or higher than pain due to other causes[257].

Patients who have had other kinds of severe pain, such as that due to a heart attack, broken bones, or renal colic, say that the myofascial pain from TrPs can be just

as severe. Despite their painfulness, myofascial TrPs are not directly life threatening, but their painfulness can, and often does, devastate the quality of life.

Cost. Unrecognized myofascial headache, shoulder pain and low back pain that have become chronic are major causes of industrial lost time and compensation applications. Bonica[21] pointed out that disabling chronic pain costs the American people billions of dollars annually. Low back pain alone costs the people of California $200 million annually. Analgesics to relieve chronic pain are costly and can be a significant cause of nephropathy.[97] A considerable portion of the chronic pain due to myofascial TrPs could have been prevented by prompt diagnosis with appropriate treatment.

How many more people not included in these studies do carry on, yet bear the misery of nagging TrP pain that would respond if it were diagnosed and treated for what it is? When the myofascial nature of pain is unrecognized, such as the pain caused by TrPs in the pectoral muscles that mimics cardiac pain, the symptoms are likely to be diagnosed as neurotic, psychogenic, or behavioral. This adds frustration and self-doubt to the patient's misery and blocks appropriate diagnosis and treatment. Active myofascial TrPs are largely responsible for that enigmatic scourge of mankind, musculoskeletal pain. The total cost is incalculable, but enormous, and most of it is unnecessary.

Historical Review

The history of growth in our understanding of musculoskeletal pain is the history of the identification of specific sources and causes of pain, including neuropathic sources, articular dysfunction, muscular origins, and modulation of central nervous system processing of pain. The history of muscle pain was reviewed for much of this century[216'235] and recently has been updated.[238]

This review[235] identifies, and Table 2.2 lists, a number of historically noteworthy publications that provide a background to our present understanding of myofascial pain caused by TrPs. Progress has been slow and spotty. Pain and/or tenderness of the muscles may have distinctly different causes that can produce confusingly similar symptoms. The medical community is just beginning to sort out this complex puzzle. One way of clarifying what TrPs *are* clinically is to define more clearly what they *are not* and how other diagnoses are related. Major progress has been made this past decade by clearly distinguishing a central cause of muscle pain and tenderness, fibromyalgia, from the primarily muscular dysfunction, TrPs. The relation between articular dysfunction that responds to manual therapy and TrPs remains to be as clearly delineated. The updated review[238] shows that repeatedly, an author or the adherents to a school of thought will concentrate on part of the total clinical picture of myofascial TrPs, introduce a new name, and overlook the rest of the picture.

Froriep[85] is a pre-twentieth century author who identified *Muskel Schwiele* as extremely tender, palpable hardenings in muscles that, when treated, afforded the patient much pain relief. By the turn of the century Adler,[2] in America, used the English term muscular rheumatism and included the concept of pain radiating from the tender spot. In England, Gowers,[101] Stockman,[264] and Llewellyn and Jones[172] introduced the term fibrositis for the same symptom complex. In Germany, Schmidt[229] used the German counterpart to muscular rheumatism, *Muskelrheumatismus.* Other authors used the term *Weichteilrheumatismus* literally "soft-parts rheumatism" which is commonly translated into English as *nonarticular rheumatism.* The cause of the diagnosis remained controversial in every case.

In 1919, Schade[226] reported that the hardness of previously tender ropiness in muscles persisted during deep anesthesia and after death until rigor mortis obscured the difference. This finding discredited a nerve-activated, muscular contraction mechanism as the cause of the palpable bands, but is consistent with an endogenous contracture of sarcomeres being responsible. Schade[227] later postulated a localized increase in the viscosity of muscle colloid and proposed the term "Myogelosen," literally translated as "muscle gellings" and identified in English as myogelosis. In the same year, two orthopaedic surgeons in Munich, F. Lange and G. Eversbusch[163] described tender points associated with regions of palpable hardness in muscles, that they termed "Muskel-

Table 2.2. ***Historical Muscle Pain Papers***

Term Used	*Muscular Findings*	*Authorship, Year, & Reference*
Muskelschwiele [Muscle callus]	Tender tight cord or band	Froriep, 1843[85]
Muscular rheumatism	Tender, elongated infiltrations, radiating pain	Adler, 1900[2]
Fibrositis	Tender fibrous beaded chains	Gowers, 1904[101]
Chronic rheumatism	Nodules: histologically, inflamed connective tissue	Stockman, 1920[264]
Fibrositis, Myofibrositis	Tender nodules with radiating pain	Llewellyn and Jones, 1915[172]
Muskelrheumatismus, Myalgie [muscular rheumatism, myalgia]	Tender, contracted muscle bundles	Schmidt, 1916[229]
Myogelose [muscle gelling]	Tender muscle indurations (persisted after death)	Schade, 1.919[226]
Muskelharten [muscular indurations]	Tender indurations with or without muscular contraction	F. Lange, 1925[162]
Muskelharten, Myogelosen [muscular indurations or gelling]	The first "trigger point manual;" referred pain not mentioned	M. Lange, 1931[164]
Muskelharten [muscular indurations]	Introduction of ethyl chloride spray	Kraus, 1937[156]
Referred pain	Experimental demonstration of pain referred from muscle	Kellgren, 1938[149]
Muscular rheumatism	Spot tenderness in indurated region, pain reaction, and referred pain	Gutstein, 1938[112]
Idiopathic myalgia	Spot tenderness, referred pain, decreased ROM (her first description of TrPs)	Travell, era/., 1942[276]
Fibrositis	Tender nodule, referred pain	Kelly, 1941[151]
Myofascial TrPs	Tender spot, referred pain, 32 pain patterns	Travell, R, 1952[278]
Myofascial TrPs	Early recognition of importance of TrPs in patients with pain	Bonica, 1953[20]
Trigger Areas	Electromyographic activity of trigger areas first reported	Weeks and Travell, 1957[288]
Fibrositissyndrom [fibrositis syndrome]	Reported non-specific dystrophic pathology in more severe cases	Miehlke, era/., I960[193]
Fibrositis syndrome	Generalized chronic pain with multiple tender points (redefinition)	Smythe and Moldofsky 1977[258]
Fibromyalgia	Renamed the 1977 redefinition of fibrositis	Yunus, era/., 1981[300]
Myofascial TrP	Publication of Volume 1 of the Trigger Point Manual	Travell and Simons, 1983[279]
Pressure pain threshold	Introduction of an algometer for measuring trigger point tenderness	Fischer, 1986[72]
Fibromyalgia	Official diagnostic criteria for fibromyalgia	Wolfe, era/., 1990[294]
Myofascial TrPs	Publication of Volume 2 of the Trigger point Manual	Travell and Simons, 1992[280]
Myofascial TrPs	Electromyographic activity characteristic of TrPs reported	Hubbard and Berkoff, 1993[133]
Localized twitch response	Value of the rabbit as an experimental model for the local twitch responses characteristic of myofascial TrPs	Hong and Torigoe, 1994[128]

Table 2.2. *Historical Muscle Pain Papers (Continued)*

Term Used	*Muscular Findings*	*Authorship, Year, & Reference*
Active Loci	Use of the rabbit as an experimental model to study the elecrical activity of TrPs	Simons, *etal.,* 1995[249]
Myofascial TrPs	New research data for selection of diagnostic criteria; experimental basis for the new dysfunctional endplate hypothesis	Simons, 1996[242]
Myofascial TrPs	Interrater reliability; identified TrP diagnostic criteria	Gerwin, *etal.,* 1997[94]
Myofascial TrPs	Identification of likely pathogenesis	Simons, 1997[244]

harten," literally translated into English as "muscle hardenings" or "indurations." In 1925, F. Lange[162] described the local twitch response. His student, M. Lange,[1"4] later equated these muscle hardenings to Schade's myogeloses. M. Lange used fingers, knuckles, or a blunt wood probe to apply forceful, ecchymosis-producing massage (Gelotripsie). His comprehensive clinical book[164] also presented the history and experimental basis of the concept of myogeloses (prior to the discovery of the actin-myosin contractile mechanism). This work essentially ignored the referred pain aspect of TrPs.

Before coming to the United States from Germany, Hans Kraus, who was an early pioneer in this field, first reported the therapeutic use of ethyl chloride spray, for relief of *Muskelharten* in 1937,[156] and for the relief of fibrositis in 1952,[157] and for the relief of TrPs in 1959.[158] He continued to promote the importance of exercise and TrPs until his recent death.

In 1938, Kellgren,[149] working under the influence of Sir Thomas Lewis, published a major milestone paper. He established unequivocally for most major postural muscles of the body that each muscle and many fascial structures had a characteristic referred pain pattern when injected with a small amount of painful salt solution. Shortly after this, three clinicians on three continents simultaneously and independently published a series of papers in English emphasizing four cardinal features: a palpable nodular or band-like hardness in the muscle, a highly localized spot of extreme tenderness in that band, reproduction of the patient's distant pain complaint by digital pressure on that spot, and relief of the pain by massage or injection of the tender spot. Each author reported pain syndromes of specific muscles throughout the body in large numbers of patients. All three had identified myofascial TrPs. However, each used different diagnostic terms, were apparently unaware of one another, and the commonality of their observations passed unnoticed for decades.

One of the three, **Michael Gutstein,** was born in Poland and first published as Gutstein from Berlin, then Gutstein-Good and finally as Good from Great Britain. In the 12 or more papers that he published in Britain between 1938[112] and 1957,[99] he used many diagnostic terms to describe the same condition: myalgia, idiopathic myalgia, rheumatic myalgia, and nonarticular rheumatism. He illustrated the referred pain patterns of many patients as case reports. He repeatedly held that the process responsible for the "myalgic spots" was a local constriction of blood vessels due to overactivity of the sympathetic fibers supplying the vessels.

Michael Kelly lived and published in Australia. Throughout his series of nearly a dozen papers, all on fibrositis between 1941[151] and 1963,[152] he was impressed by both the palpable hardness of the "nodule" associated with the tender point in the muscle and by the distant referral of pain from the afflicted muscle. Kelly published numerous case reports with referred pain patterns. He gradually evolved the concept

that fibrositis was a functional, neurological disturbance that originated at the myalgic lesion. He envisioned little or no local pathology, but a central nervous system reflex disturbance that caused the referred pain.

Janet Travell lived and published in the United States. Her more than 40 papers on myofascial TrPs have appeared between 1942[276] and 1990,[275] and the first volume of *The Trigger Point Manual* was published in 1983 followed by the second volume in 1992. She and Rinzler in 1952 reported the pain patterns of TrPs in 32 skeletal muscles, as "The myofascial genesis of pain,"[278] which quickly became the classic source of this information. It was her opinion that any fibroblastic proliferation was secondary to a local muscular dysfunction and that any pathologic changes occurred only after the condition continued for a long time. She believed that the self-sustaining characteristic of TrPs depends on a feedback mechanism between the TrP and the central nervous system. Of those three pioneers, only Travell's influence withstood the test of time.

To date, only two biopsy studies are known of sites selected specifically as myofascial TrPs. One study[253] reported biopsies of TrPs in the leg muscles of dogs and the other[214] reported findings in human biopsies of myogelosis located at TrP sites. Biopsy studies of the tender nodule of myogelosis or of fibrositis must have included many myofascial TrPs. The study by Miehlke, *et al.*[W3] of the *Fibrositissyndrom* (fibrositis) was the most extensive and thorough. They reported minimal findings in mild cases and increasingly marked nonspecific dystrophic findings in progressively more symptomatic cases. If the pathophysiology of TrPs is primarily a dysfunction in the immediate region of individual motor endplates, there is no reason to expect routine histological studies to reveal the cause. However, a recent histological study of the palpable nodules associated with myogelosis at TrP sites found substantiating evidence of contracture of some individual muscle fibers.[214]

Throughout most of this century, the term fibrositis described a condition that was compatible with myofascial TrPs, although ambiguously so.[216] In 1977, Smythe and Moldofsky[258] added another and very different meaning to the many meanings that had become associated with fibrositis.[216] The 1977 authors[258] identified a condition of generalized pain marked by multiple tender points when tested by palpation. Four years later, Yunus, *et al.*[300] proposed the term fibromyalgia as a more appropriate name for the 1977 redefinition of fibrositis. Since the diagnoses of either myofascial TrPs or fibromyalgia now accounted for nearly all of the patients previously diagnosed as having fibrositis, this became an outmoded diagnosis. At that time, it was not clear how closely the pathophysiology of fibromyalgia and TrPs related to each other; the etiology of both was highly speculative.

By 1990, rheumatologists under the leadership of F. Wolfe[294] officially established diagnostic criteria for fibromyalgia. The criteria were simple and the examination easily and quickly performed, which helped focus the attention of the medical community on this syndrome. Since then, remarkable progress has been made toward identifying its cause. It is now firmly established that a central nervous system dysfunction is primarily responsible for the increased pain sensitivity of fibromyalgia.[224]

In the mid 1980s, A. Fischer[71,74] produced a pressure algometer that provided a method for measuring the sensitivity of myofascial TrPs and of fibromyalgia tender points.

An important milestone of progress was reached by Hubbard and Berkoff in 1993 when they convincingly reported needle EMG activity characteristic of myofascial TrPs.[133] Weeks and Travell[288] had illustrated the phenomenon 36 years earlier. The following year Hong and Torigoe[128] demonstrated that the rabbit was a suitable experimental model for studying the LTR that is characteristic of human TrPs. In 1995, Simons, *et* ai.[248] confirmed in rabbit experiments the electrical activity reported by Hubbard and Berkoff. These rabbit studies, and a concomitant human study,[249] strongly implicated a dysfunctional endplate region as the prime site of TrP pathophysiology.[242]

Another important step of progress was the report by Gerwin, *et al.*[9]* of an interrater reliability study that demonstrated reliable identification of myofascial TrP criteria in 5 muscles. The integrated hy-

pothesis found in section D of this chapter moves our understanding of TrPs another major step forward.[244]

Related Diagnostic Terms

The cause of muscle pain syndromes and of musculoskeletal pain in general, has perplexed the medical community for more than a century. The subject has been plagued by a multitude of terms that emphasized different aspects of basically the same condition[235] and that were reported in different languages. A brief review of some of the more important diagnostic terms currently encountered will help to put the available literature into perspective.

Anatomically Oriented Terms. Through the years, many authors "discovered" a "new" muscle pain syndrome related to a specific part of the body and gave it a name corresponding to that region. Characteristically, unrecognized myofascial TrPs contributed significantly to the pain syndrome identified. Common examples are tension headache,[140, 272, 287] occipital neuralgia,[103] the scapulocostal syndrome,[192,203,204] and tennis elbow *[see* Chapter 36).

Fibromyalgia. Fibromyalgia is fundamentally a different condition than TrPs, but often presents with symptoms that are confusingly similar to those caused by chronic myofascial TrPs. Fibromyalgia is characterized by a central augmentation of nociception which causes generalized deep tissue tenderness that includes muscles. It has a different etiology than myofascial TrPs but many of the tender points diagnostic of fibromyalgia are also common sites for TrPs and many patients have both conditions. In the German literature, fibromyalgia is usually equated with *Generalizierte Tendomyopathie* (generalized tendomyopathy). Fibromyalgia is considered in more detail later in this chapter.

Fibrositis. The term fibrositis appeared in the English literature in 1904[101] and was soon adopted into German as the *Fibrositis-syndrom.* For most of the century, fibrositis was characterized by a tender palpable "fibrositic" nodule by most of the authors using the term fibrositis. Many of these patients had TrPs. In time, fibrositis became an increasingly controversial diagnosis because of multiple definitions and no satisfactory histopathological basis for the nodule. The diagnosis was completely redefined in 1977,[258] and the condition described by the 1977 definition was officially established in 1990 as fibromyalgia.[294] According to the current definition of fibromyalgia, it is a totally different condition that is unrelated to the original concept of fibrositis. Fibrositis is currently an outmoded diagnosis.

Muskelharten. By 1921 the term *Muskelharten*™[3] was well recognized in German literature and still appears in German occasionally, but rarely in English. It literally means "muscle indurations" and refers to the palpable firmness of the tender nodule responsible for the patient's pain. Another German term, *Myogelosen*™[4] (literally "muscle gellings") refers to the same phenomena and the two terms have frequently been used interchangeably. The term *Muskelharten* is often used to characterize the physical findings and the term *Myogelosen* to identify the diagnosis.

Myofascial Pain Syndrome. This term has acquired both a general and a specific meaning. The two meanings need to be distinguished.[241] The general meaning includes a regional muscle pain syndrome of any soft tissue origin that is associated with muscle tenderness[160,298] and is commonly used in this sense by dentists.[14] The other meaning is specifically a myofascial pain syndrome caused by TrPs. This is a focal hyperirritability in muscle that can strongly modulate central nervous system functions and is the subject of this book.

Myofascitis. The term myofascitis is now rarely (and should not be) used as synonymous with myofascial TrPs. Myofascitis is properly used to identify inflamed muscles.

Myogeloses. The term myogeloses is the English form of a German term, *Myogelosen,* which is still commonly used and is generally considered synonymous with *Muskelharten [see* above). The name myogeloses was based on an outmoded hypothesis to account for muscle contraction that was proposed before the actin-myosin contractile mechanism was discovered. A recent study[214] indicates that myogeloses and TrPs identify the same condition approached from somewhat different diagnostic points of view using different terminology.

Nonarticular Rheumatism. /Monarticular rheumatism is a commonly used, but not very clearly defined, general term for

soft tissue pain syndromes that are not associated with a specific joint dysfunction or disease. The term is generally considered as synonymous with soft tissue rheumatism, which is the English translation for the German term *Weichteilrheumatismus.* This term was commonly used to describe a range of conditions that also include myofascial pain caused by TrPs. Currently, the term nonarticular rheumatism is used[218] to identify muscle pain syndromes that are *not* fibromyalgia and are *not* attributed to myofascial TrPs. The literature reviews of nonarticular rheumatism by Romano[218] include conditions such as adhesive capsulitis, periarticular arthritis, bursitis, epicondylitis, insertion tendinosis, and tennis elbow, which are frequently myofascial TrPs masquerading as another diagnosis.

Osteochondrosis. This term is used by Russian vertebroneurologists as an inclusive term to cover the interaction of neural and muscular conditions, such as fibromyalgia, myofascial TrPs, and spinal nerve compromise.

Soft Tissue Rheumatism. This term is usually used synonymously with nonarticular rheumatism, described above.

Tendomyopathy. Tendomyopathy is the English version of the German term that is divided into general and local categories. General tendomyopathy is considered synonymous with fibromyalgia.[65] The localized form often includes myofascial TrPs but is not as clearly defined.

B. CLINICAL CHARACTERISTICS OF TRIGGER POINTS

The clinical characteristics of TrPs under the headings of symptoms, physical findings, and treatment are introduced and presented only in overview in this section. The rationale for each physical finding is noted in that subsection. Testing techniques that are useful for experimental purposes and some that have potential routine clinical application are dealt with in more detail here rather than in the muscle chapters because they have yet to be established as a part of routine clinical practice.

Diagnosis and treatment of acute single-muscle myofascial pain syndromes can be simple and easy. When an acute myofascial TrP syndrome is neglected and allowed to become chronic, it becomes unnecessarily complicated, more painful, and it becomes increasingly time-consuming, frustrating, and expensive to treat.

Symptoms

Active TrPs produce a clinical complaint (usually pain) that the patient recognizes when the TrP is digitally compressed. **Latent TrPs** can produce the other effects characteristic of a TrP including increased muscle tension and muscle shortening (but do not produce spontaneous pain). Both active and latent TrPs can cause significant motor dysfunction. It appears that the same factors which are responsible for the development of an active TrP, to a lesser degree, can cause a latent TrP. An active **key TrP** in one muscle can induce an active **satellite TrP** in another muscle. Inactivation of the key TrP often also inactivates its satellite TrP without treatment of the satellite TrP itself.

Onset. The activation of a TrP is usually associated with some degree of mechanical abuse of the muscle in the form of *muscle overload,* which may be acute, sustained, and/or repetitive. In addition, leaving the muscle in *shortened position* can convert a latent TrP to an active TrP and this process is greatly aggravated if the muscle is contracted while in the shortened position. In paraspinal (and very likely other) muscles, a degree of *nerve compression* that causes identifiable neuropathic electromyographic changes is associated with an increase in the numbers of active TrPs.[37] These TrPs may be activated by disturbed microtubule communication between the neuron and the endplate since the motor endplate is the peripheral core TrP pathophysiology.

The patient is aware of the pain caused by an active TrP but may or may not be aware of the dysfunction it causes. Latent TrPs characteristically cause some increased muscle tension and limitation of stretch range of motion, which often escapes the patient's attention or is simply accepted. The patient becomes aware of pain originating from a latent TrP only when pressure is applied to it. Spontaneous referred pain appears with increased irritability of the TrP, and it then is identified as active.[125]

The patient usually presents with complaints due to the most recently activated TrP. When this TrP has been successfully

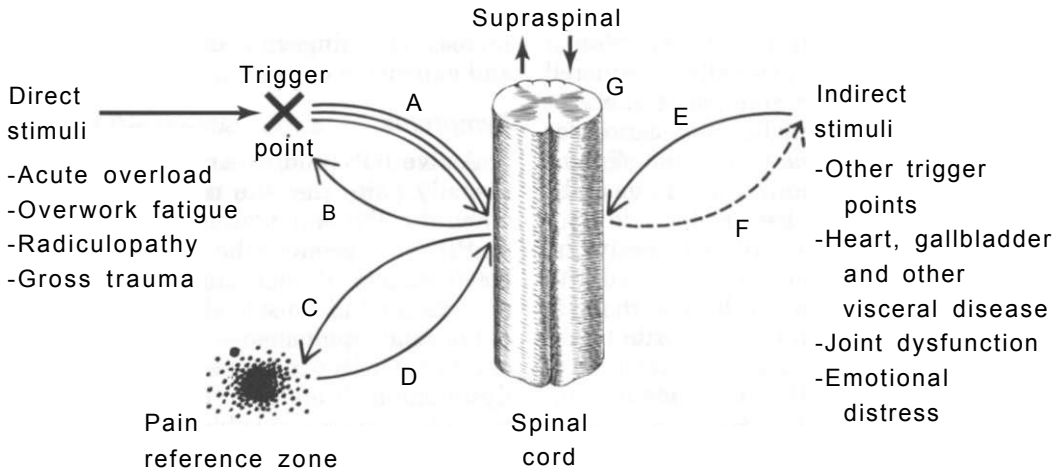


Figure 2.1. Schematic of central nervous system interactions with a trigger point (X). *Triple arrow A* running from the trigger point to the spinal cord represents sensory, autonomic and motor effects. *Arrow B* from the spinal cord to the trigger point includes autonomic modulation of the intensity of trigger point activation. *Arrow C* to the pain reference zone represents the appearance of referred pain and tenderness at distant sites that may be several neurological segments removed from the trigger point. *Arrow D* indicates the influence of the vapocoolant spray in the region of the reference zone that facilitates release of the trigger point. *Arrow E* signifies the activating effect of indirect stimuli on the trigger point; *dashed arrow F* denotes effects of trigger points on visceral function. *Thick arrows* G identify trigger point interactions at the supraspinal level. (Figure adapted from Travell JG. Myofascial trigger points: clinical view. In: Bonica JJ, Albe-Fessard D, eds. *Advances in Pain Research and Therapy*, Vol. 1. New York: Raven Press, 919-926, 1976.)

eliminated, the pain pattern may shift to that of an earlier, key TrP which also must be inactivated. If the key TrP is inactivated first, the patient may recover without further treatment.

The intensity and extent of the referred pain pattern depends on the degree of irritability of the TrP, not on the size of the muscle. Myofascial TrPs in small, obscure, or variable muscles can be as troublesome to the patient as TrPs in large familiar muscles.

As illustrated in Figure 2.1, trigger points are activated directly by acute overload, overwork fatigue, direct impact trauma, and by radiculopathy.

Trigger points can be activated indirectly by other existing TrPs, visceral disease, arthritic joints, joint dysfunctions, and by emotional distress. Satellite TrPs are prone to develop in muscles that lie within the pain reference zone of key myofascial TrPs, or within the zone of pain referred from a diseased viscus, such as the pain of myocardial infarction, peptic ulcer, cholelithiasis, or renal colic. A perpetuating factor *(see* Chapter **4)** increases the likelihood of overload stress converting a latent TrP to an active TrP.

With adequate rest, and in the absence of perpetuating factors, an active TrP may revert spontaneously to a latent state. Pain symptoms disappear, but occasional reactivation of the TrP by exceeding that muscle's stress tolerance can account for a history of recurrent episodes of the same pain over a period of years.

Pain Complaint. Patients with active myofascial TrPs usually complain of poorly localized, regional, aching pain in subcutaneous tissues, including muscles and joints. They rarely complain of sharp, clearly-localized cutaneous-type pain. The myofascial pain is often referred to a distance from the TrP in a pattern that is characteristic for each muscle. Sometimes the patient is aware of numbness or paresthesia rather than pain.

Infants have been observed with point tenderness of the rectus abdominis muscle and colic, both of which were relieved by

sweeping a stream of vapocoolant over the muscle, which helps to inactivate myofascial TrPs.

When children with musculoskeletal pain complaints were examined for myofascial TrPs, the TrPs were found to be a common source of their pain.[12] It is our impression that the likelihood of developing pain-producing ***active*** TrPs increases with age into the most active, middle years. As activity becomes less strenuous in later years, individuals are more likely to be aware of the stiffness and restricted motion resulting from ***latent*** TrPs.

Sola[259] found that laborers who exercise their muscles heavily every day, are less likely to develop active TrPs than are sedentary workers who are prone to intermittent orgies of vigorous physical activity. Our clinical experience has been similar.

Active TrPs are found commonly in postural muscles of the neck, shoulder and pelvic girdles, and in the masticatory muscles. In addition, the upper trapezius, scalene, sternocleidomastoid, levator scapulae and quadratus lumborum muscles are very commonly involved.

Dysfunctions. In addition to the clinical symptoms produced by the sensory disturbances of referred pain, dysesthesias, and hypesthesias, patients also can experience clinically important disturbances of autonomic and motor functions.

Disturbances of **autonomic functions** caused by TrPs include abnormal sweating, persistent lacrimation, persistent coryza, excessive salivation, and pilomotor activities. Related proprioceptive disturbances caused by TrPs include imbalance, dizziness, tinnitus, and distorted weight perception of lifted objects.

Disturbances of **motor functions** caused by TrPs include spasm of other muscles, weakness of the involved muscle function, loss of coordination by the involved muscle, and decreased work tolerance of the involved muscle. The weakness and loss of work tolerance are often interpreted as an indication for increased exercise, but if this is attempted without inactivating the responsible TrPs, the exercise is likely to encourage and further ingrain substitution by other muscles with further weakening and deconditioning of the involved muscle. The combination of weakness in the hands and loss of forearm muscle coordination makes grasp unreliable. Objects sometimes slip unexpectedly from the patient's grasp. The weakness results from reflex motor inhibition and characteristically occurs without atrophy of the affected muscle. The patient is prone to substitute intuitively without realizing that, for instance, he or she is carrying the grocery bag in the nondominant but now-stronger arm.

The motor effects of TrPs on the muscle in which the TrPs are located are considered in detail below under *Surface EMG.*

Sleep Disturbances. Disturbance of sleep can be a problem for patients with a painful TrP syndrome. Moldofsky[196] has shown in a series of studies that many sensory disturbances, including pain, can seriously disturb sleep. This sleep disturbance can, in turn, increase pain sensitivity the next day. Active myofascial TrPs become more painful when the muscle is held in the shortened position for long periods of time and also if body weight is compressing the TrP. Thus, for patients with active TrPs, sleep positioning can be critical to avoid unnecessarily disturbing their sleep.

Physical Findings

A muscle harboring a TrP is prevented by pain from reaching its full stretch range of motion, and is also restricted in its strength and/or endurance. Clinically, the TrP is identified as a localized spot of tenderness in a nodule in a palpable taut band of muscle fibers. Restricted stretch range of motion and palpable increase in muscle tenseness (decreased compliance) are more severe in more active TrPs. Active TrPs are identified when patients *recognize* the pain that is induced by applying pressure to a TrP as "their" pain.[94] The taut band fibers usually respond with an LTR when the taut band is accessible and when the TrP is stimulated by *properly applied* snapping palpation. The taut band fibers respond consistently with a twitch response when the TrP is penetrated by a needle.

Taut Band. By gently rubbing across the direction of the muscle fibers of a superficial muscle, the examiner can feel a nodule at the TrP and a rope-like induration that extends from this nodule to the attachment of the taut muscle fibers at each end of the muscle. The taut band can be

snapped or rolled under the finger in accessible muscles. With effective inactivation of the TrP, this palpable sign becomes less tense and often (but not always) disappears, sometimes immediately.

Tender Nodule. Palpation along the taut band reveals a nodule exhibiting a highly localized, exquisitely tender spot that is characteristic of a TrP. When the tender spot was tested for tenderness, displacement of the algometer by 2 cm produced a statistically significant decrement in pain threshold algometer readings.[212] Clinically, displacement of the application of pressure by 1-2 mm at a TrP can result in a markedly reduced pain response.

This strong localization of tenderness in the vicinity of a TrP corresponds to the localized sensitivity of the experimental muscle for eliciting LTRs as demonstrated in rabbit experiments.[128] A 5 mm displacement to either side of the trigger spot (at right angles to the taut band) resulted in almost total loss of response. However, the response faded out more slowly when stimulated over a range of several centimeters from the trigger spot along the taut band. *See* Local Twitch Response in section D at the end of this chapter.

Recognition. Application of digital pressure on either an active or latent TrP can elicit a referred pain pattern characteristic of that muscle. However, if the patient "recognizes" the elicited sensation as a familiar experience, this establishes the TrP as being active and is one of the most important diagnostic criteria available when the palpable findings are also present.[94,242] Similar recognition is frequently observed when a needle penetrates the TrP and encounters an active locus.[123-249]

Referred Sensory Signs. In addition to referring pain to the reference zone, TrPs may refer other sensory changes such as tenderness and dysesthesias. This referred tenderness has been measured in experiments by Vecchiet, *et* a/.[283]

Local Twitch Response. Snapping palpation of the TrP frequently evokes a transient twitch response of the taut band fibers. This is fully described in Section 9 of Chapter 3. Its pathophysiological nature is considered in Section D of this chapter. Twitch responses can be elicited both from active and latent TrPs. In one study, no difference was noted in twitch responses whether elicited by snapping palpation or by needle penetration.[246]

Limited Range of Motion. Muscles with active myofascial TrPs have a restricted passive (stretch) range of motion because of pain, as demonstrated by Macdonald.[177] An attempt to passively stretch the muscle beyond this limit produces increasingly severe pain because the involved muscle fibers are already under substantially increased tension at rest length. The limitation of stretch due to pain is not quite as great with active movement as with passive lengthening of the muscle, at least partly due to reciprocal inhibition. When the TrP is inactivated and the taut band is released, range of motion returns to normal. The degree of limitation produced by TrPs is much more marked in some muscles (e.g., subscapularis) than others (e.g., latissimus dorsi).

Painful Contraction. When a muscle with an active TrP is strongly contracted against fixed resistance, the patient feels pain.[177] This effect is most marked when an attempt is made to contract the muscle in a shortened position.

Weakness. Although weakness is generally characteristic of a muscle with active myofascial TrPs, the magnitude is variable from muscle to muscle and from subject to subject. Electromyographic studies indicate that, in muscles with active TrPs, the muscle starts out fatigued, it fatigues more rapidly, and it becomes exhausted sooner than normal muscles.[116-118] These changes are presented in more detail under surface electromyography in Section B of this chapter. The weakness may reflect reflex inhibition of the muscle by the TrPs.

Testing

No laboratory test or imaging technique has been generally established as diagnostic of TrPs. However, three measurable phenomena help to substantiate objectively the presence of characteristic TrP phenomena and all are valuable as research tools. Two of them, surface EMG and ultrasound, also have much potential for clinical application in the diagnosis and treatment of TrPs.

Needle Electromyography. In 1957, Weeks and Travell[288] anticipated the 1993 Hubbard and Berkoff[133] report of finding

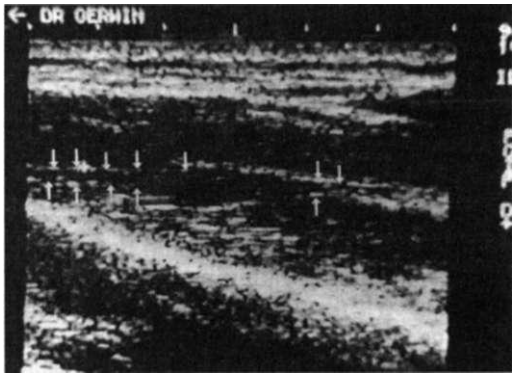


Figure 2.2. High-resolution ultrasound image of a local twitch response in the taut band fibers of a trigger point. The twitch was elicited by needle penetration of the trigger point in a taut band of a right infraspinatus muscle. The band across the middle of the figure that was seen by ultrasound imaging to contract is identified by *white arrows.* The transient contraction coincided with the patient's verbal report that he felt his typical pain and experienced the referred pain to his shoulder and arm. (Reprinted with permission from Gerwin RD, Duranleau D. Ultrasound identification of the myofascial trigger point [Letter]. *Muscle Nerve* 20:767-768, 1997.)

EMG activity identified as specific to myofascial TrPs. Subsequent rabbit and human studies[248,250,252] have confirmed the presence of spontaneous low-voltage motor endplate "noise" activity as well as high voltage spike activity that is highly characteristic of myofascial TrPs but not pathognomonic. The source of the associated high-voltage spikes can be ambiguous. When the endplate noise activity is observed, it is a strongly confirmatory finding and an invaluable research tool. A detailed consideration of this phenomenon appears in section D of this chapter.

Ultrasound Imaging. Visualization of an LTR using ultrasound was first noted by Michael Margolis, M.D.[181] This observation was followed up by Gerwin and Duranleau.[91, 92] The published finding is illustrated in Figure 2.2. This imaging procedure not only provides a second way, in addition to EMG recording, of substantiating and studying the LTR, but also has a strong potential for providing a much needed available imaging technique that could be widely used to objectively substantiate the clinical diagnosis of TrPs. This test, however, would require the examiner to use the skill-demanding snapping palpation technique, or to insert a needle into the TrP, in order to elicit the twitch response.

Surface Electromyography. Trigger points cause distortion or disruption of normal muscle function. Functionally, the muscle with the TrP evidences a three-fold problem: it exhibits *increased responsiveness, delayed relaxation,* and *increased fatigability,* which, together, increase overload and reduce work tolerance. In addition, the TrP can produce *referred spasm* and *referred inhibition* in other muscles. With the recent appearance of online computer analysis of EMG amplitude and mean power spectral frequency, a few pioneer investigators have reported the effects of TrPs on muscle activity.[56,116] The reports indicate that TrPs can influence motor function of the muscle in which they occur, **and** that their influence can be transmitted through the central nervous system to other muscles. To date, there has not been a sufficient number of well-controlled studies to establish the clinical reliability and application of these observations, but the few reports of these TrP effects are very promising.

On one hand, the strong clinical effects of TrPs on sensation, as evidenced by TrP tenderness and referred pain, have been well-documented in this volume. It is well known that strong cutaneous stimuli (e.g., electric shocks) can cause reflex motor effects (e.g., flexion reflex).[114] If skin can modulate motor activity and TrPs can modulate sensory activity, it should be no surprise that TrPs can also strongly affect motor activity. In fact, the motor effects of TrPs may be the most important influence they exert, because the motor dysfunction they produce may result in overload of other muscles and spread the TrP problem from muscle to muscle. Accumulating evidence now indicates that the muscles targeted for referred spasm from TrPs also usually have TrPs themselves. These motor phenomena of TrPs deserve serious competent research investigation.

An **increased responsiveness** of some affected muscles is indicated by abnormally high amplitude of EMG activity when the muscle is voluntarily contracted and loaded. Clinical evidence suggests that some muscles tend to be shortened and abnormally excitable, while others appear to be weak and inhibited.[142,170] The upper trapezius is identified as an excitable muscle and EMG studies showed that, although the muscle showed no abnormal motor unit activity at rest, when it harbored TrPs it tended to "overreact" when voluntarily contracted.[56] During flexion/extension movements of the head, the upper trapezius and/or sternocleidomastoid muscles with TrPs presented surface EMG amplitudes over 20% greater than asymptomatic muscles in 80% of cases.[56] Headley[117] demonstrated a similar, marked augmentation of EMG activity in upper trapezius muscles harboring TrPs as compared to the uninvolved muscle on the contralateral side when the patient attempted to shrug both shoulders equally.

Preliminary studies indicate that TrPs can refer inhibition or excitation to functionally related muscles, especially if the target muscles also have TrPs. In several instances referred motor effects were observed electromyographically from latent TrPs, indicating that these motor effects may be produced by a TrP independent of its pain-producing characteristics. This apparently specific motor effect of TrPs is an unexplored but fertile field for TrP research. The presence of a TrP may characteristically induce excitation in some muscles and inhibition in others. If so, the presence of TrPs may help to explain why some muscles frequently develop the clinical picture of being inhibited, and others become excessively responsive to clinical activation.

Hagberg and Kvarnstrom[115] demonstrated **accelerated fatiguability** electromyographically and in terms of work tolerance of the trapezius muscle that had myofascial TrPs as compared to a contralateral muscle that was pain-free. The EMG amplitude increased and median power frequency decreased significantly in the involved muscle as compared to the uninvolved muscle. Both of these changes are characteristic of initial fatigue. Mannion and Dolan[179] showed, during fatiguing exercise, a nearly linear relationship between the decline in median power frequency and the decline in strength of maximum voluntary contraction, tested intermittently. The increasing fatigue of the muscle was demonstrable as increasing weakness.

There is general acceptance of median power frequency as a valid criterion of muscle fatigue. Headley[118] reported **delayed recovery** following fatiguing exercise in 55 patients with muscle-related cumulative trauma disorder (CTD). Myofascial TrPs were very common in the involved muscles in this group. Median power spectral analysis of sEMG activity of bilateral lower trapezius muscles was monitored pre- and post-exercise and after a 7 minute rest. There was a statistically significant difference between pre- and postexercise mean power spectral values. The postexercise values of affected muscles showed minimal recovery in seven minutes whereas normal muscles recover 70-90% within 1 minute.

Delayed relaxation is commonly seen in muscle-overload work situations.[118] This failure to relax is a common surface EMG finding during repetitive exercises of muscles with myofascial TrPs. Headley[118] emphasized the importance of the brief surface EMG gaps observed in normal records of repetitive movements. Loss of these gaps can contribute significantly to muscle fatigue. G. Ivanichev[138] demonstrated delayed relaxation (loss of clean gaps with loss of muscle coordination) in a study of hand extensor muscles with TrPs or flexor muscles with TrPs when subjects were doing rapid alternating movements of extension and flexion at the wrist. The presence of a sustained low-level EMG activity when the muscle could and should be relaxed is sometimes referred to as a static load. Delayed or missing relaxation accelerates fatigue of the muscle.

Figure 2.3 illustrates schematically the EMG changes observed in muscles with TrPs. The involved muscle shows a fatigue pattern at the beginning of a repetitive task and then accelerated fatiguability with delayed recovery.[118] These features apparently are hallmarks of the motor dysfunction of muscles containing myofascial TrPs.

In addition, the TrP can also induce motor activity **(referred spasm)** in other muscles. Headley[116] illustrated an example of

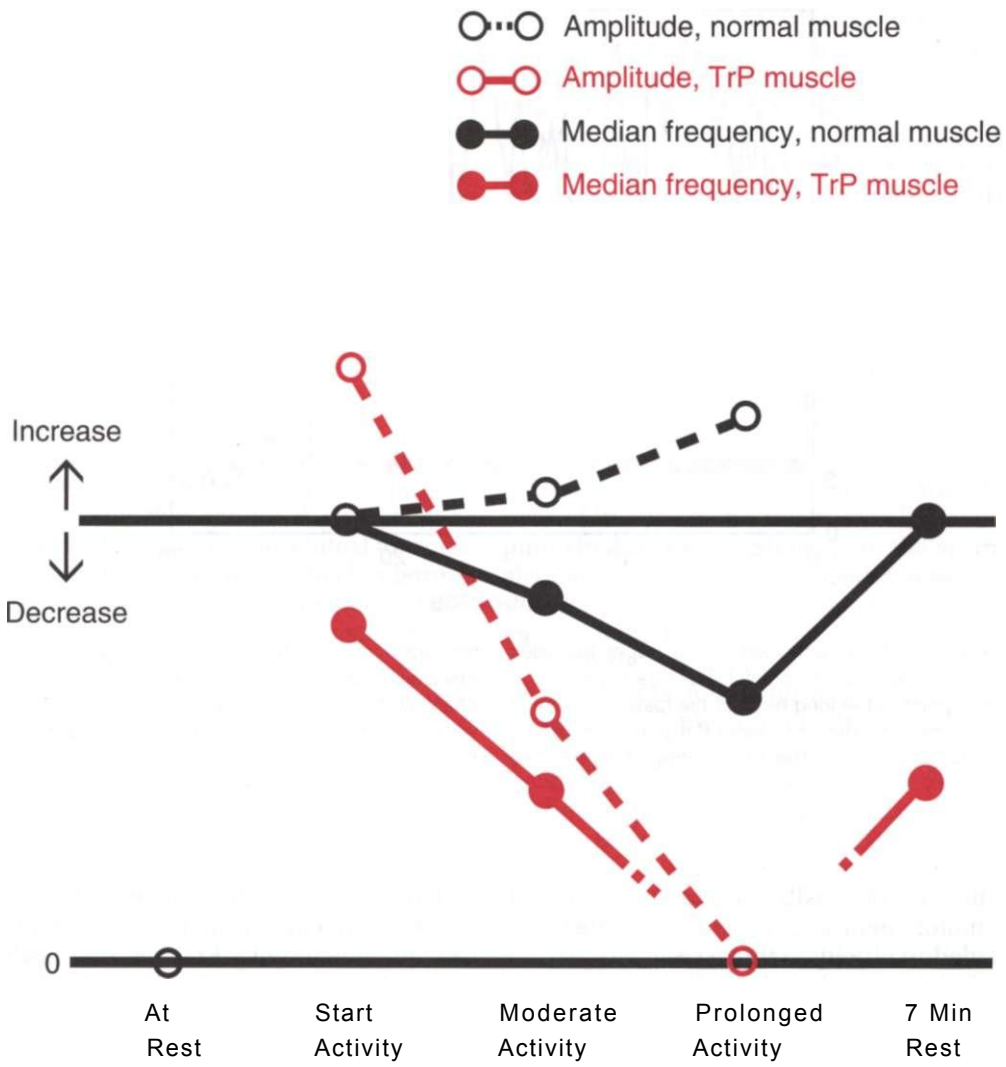


Figure 2.3. Comparison of surface electromyographic response to fatiguing exercise of normal muscle *(black lines)* and muscle with active myofascial trigger points *(red lines).* The averaged amplitude *(open circles)* and mean power frequency *(solid circles)* of the electromyographic record from the muscle with trigger points start out as if the muscle is already fatigued and show that the muscle reaches exhaustion more quickly (and is slower to recover) than normal muscle. These changes are accompanied by accelerated fatigue and weakness of the muscle with trigger points.

this phenomenon where pressure on a TrP in a right soleus muscle induced a strong spasm response in the right lumbar paraspinal muscles. Figure 2.4 illustrates a similar response with pressure applied to a TrP in the long head of the triceps brachii muscle inducing a strong motor unit response (spasm) in the ipsilateral upper trapezius muscle only during the 20 seconds that pressure was being applied. This response failed to occur following inactivation of the triceps TrP. The upper trapezius muscle in this case also had TrPs, and its response fits with the impression that muscles with TrPs are more readily activated (and therefore are more likely to become target muscles for referred spasm) than muscles free of TrPs. This may be another

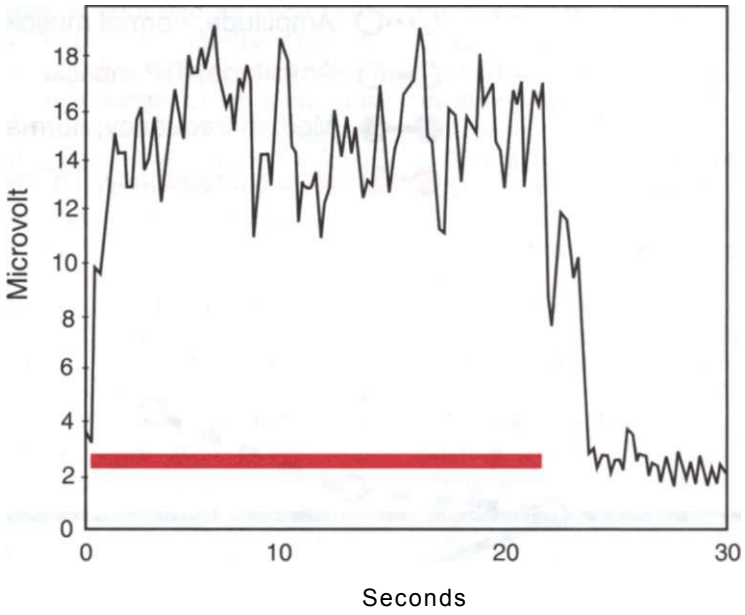


Figure 2.4. Motor activation of the upper trapezius muscle in response to painful pressure applied to a trigger point in the long head of the ipsilateral triceps brachii muscle. The bar marks the period of painful pressure applied to the triceps trigger point. The marked increase in surface electromyographic activity (referred spasm) corresponds to the period of mechanical stimulation of the trigger point. (Redrawn with permission from the data of Barbara J. Headley, PT.)

indication of sensitization of TrP-involved *a* motor neurons. Research studies are needed to elucidate this issue.

Certain muscles tend to be targets of referred spasm so TrPs in a number of distant muscles can accentuate EMG activity and irritability of a target muscle. The upper trapezius, masseter, posterior cervicals, and lumbar paraspinal muscles appear to be common target muscles. These are also muscles that are prone to develop tightness, according to Janda.[142]

Carlson, *et al.*[32] demonstrated the TrP-target muscle relationship for referred spasm between the upper trapezius and the ipsilateral masseter muscle. Following TrP injection of the trapezius muscle, there was a significant reduction in pain intensity ratings and EMG activity in the masseter muscle. Every one of the patients in the study had localized TrP tenderness in the masseter TrP, location, reinforcing the suspicion that target muscles characteristically develop TrPs, but not necessarily active TrPs.

These examples are analogous to the activated segment concept described in an osteopathic study by Korr, *et al.*[15i] In their study, the spasm was demonstrated by the paraspinal muscles acting as target muscles at the level of a vertebra showing pressure sensitivity that was considered indicative of an articular dysfunction. A previous report noted that the spasm response was most marked when pressure was applied to a pressure-sensitive vertebra.[50]

Spasm may be referred by TrPs independent of pain referral. Headley[116] noted that some distant TrPs which referred spasm to the paraspinal muscles were not prone to refer pain and were rated as only mildly painful on application of pressure. She reported that inactivation of these spasm-inducing TrPs resulted in marked reduction of low back pain. Although these "latent" TrPs were not themselves referring pain, they apparently were inducing algogenic activity in the back muscles.

The capacity of TrPs to refer **inhibition** can cause major disruption of normal mus-

cle function. Headley[117] illustrated two clear examples of movement-specific inhibition where the muscle worked well during a test movement but did not contract at all during a movement for which it would normally serve as prime or assistive mover. A frequently-seen example of referred inhibition is an anterior deltoid muscle that is strongly inhibited during shoulder flexion but is recruited essentially normally during shoulder abduction. In these cases, the normal functional pattern returned with inactivation of the problematic TrP in the infraspinatus muscle (Headley, personal communication, 1996).

Another reported example of referred inhibition[117] was an active TrP in the quadratus lumborum that inhibited gluteal muscles. Normal function of the gluteal muscles was restored when the quadratus lumborum TrP was inactivated. The immediate restoration of normal strength and normal median power spectral frequency during repetitive activity strongly suggests that the recruited muscle was not lacking strength before the test, but was probably neurologically inhibited by the quadratus lumborum TrP. With sufficient repetitions in a work situation, these abnormal patterns appear to become well "learned" when the muscle no longer returns immediately to a normal pattern with inactivation of the TrP. Now it becomes necessary to retrain the muscle to a normal pattern *after* inactivation of the responsible TrPs. Surface EMG biofeedback from the inhibited muscle(s) can facilitate retraining.

All of these motor phenomena and their complexity suggest that the motor dysfunctions caused by TrPs are as complicated and important as the sensory story emphasized in the first edition of the *Trigger Point Manual.* These motor dysfunctions alone could constitute a book. However, an enormous amount of competent, thoughtful surface EMG research will be needed before that book can be written.

Algometry. Sensitivity to pain in patients with TrPs has been measured as the pain threshold to electrical stimulation[283,284] or to applied pressure. Pressure algometry has been most commonly reported. Pressure algometry involves induction of a specific pain level in response to a measured force applied perpendicularly to the skin. Three endpoints are reported: the onset of local pain (pressure pain threshold), the onset of referred pain (referred pain threshold), and intolerable pressure (pain tolerance). Most commonly, the pressure required to reach pain threshold is measured directly from a spring scale calibrated in kilograms, Newtons, or pounds. Since the pressure is applied through a circular foot plate, its diameter is a factor and the actual measurement being made is stress (Kg/cm^2) applied to skin. Since one of the most common algometers has a foot plate area of 1 cm^2, the meter reading in Kg is numerically the same as Kg/cm^2, so no numerical conversion is needed.

A convenient hand-held spring algometer that is commercially available was described in 1986,[72] and standard values were published in 1987.[73] Since then, the spring algometer has been widely used in research. This device is useful for making a measurement of pain pressure threshold at a TrP site so the initial tenderness can be compared to measurements following a therapeutic or experimental intervention. It is relatively objective, since the subject need not see the meter display, but the reading does depend on the subject's report of a subjective sensation. It is very useful for research studies and helpful in many clinical situations, but the user must be aware of three kinds of limitations when applying it to TrPs.

First, the measurement, per se, indicates absolutely nothing about the source or cause of the tenderness being measured. The tenderness may be due to myofascial TrPs, to tender points of fibromyalgia, to bursitis, to severe spasm, etc. Therefore, by itself, tenderness cannot serve as a diagnostic criterion. The cause of the tenderness must be determined by other diagnostic observations.

Second, the absolute value obtained at any one site can be strongly influenced by variations in the thickness and compliance of subcutaneous tissues from subject to subject and by inherent differences in the sensitivity of different muscles.[73]

Third, the relatively high degree of skill required to use this instrument effectively, and the exquisite specificity of the location of the TrP being measured are generally underrated. The precise location of maximum tenderness of that TrP must first be established by palpation and with the subject's

cooperation. Since the tenderness of the *nodule* in a *taut band* is being measured, the foot plate must be centered over the point of maximum tenderness in the nodule, and pressure must be aimed precisely in the direction of maximum tenderness. The foot plate *must remain* in this position throughout the measurement. If the foot plate slips off the nodule and compresses the tissue adjacent to the nodule (which it is very prone to do), an entirely different and erroneously high reading is obtained. For these reasons, errors in measuring TrP tenderness are nearly always underestimations, not overestimations. By placing a finger on each side of the nodule or taut band and positioning the foot plate between the fingers, the fingers can serve as a guide to maintain the footplate position over the point of maximum tenderness. These difficulties can be at least partly ameliorated by averaging the lowest two of three readings if they are in reasonable agreement.

What constitutes appropriate interpretation of results from algometry of TrPs was recently greatly clarified by Hong, *et al.*[125] The authors examined three sites associated with latent and with active TrPs in the middle finger extensor of the extensor digitorum communis muscle by algometry. The three sites were on the TrP, on the taut band 2 cm distal to the TrP, and a control (normal muscle) site 1 cm further distal to the taut-band site and 1 cm lateral to the taut band site. At each site, three kinds of thresholds were measured: onset of (local) pain, onset of referred pain, and intolerable pain. The results are presented graphically in Figure 2 of their paper.

The authors[125] showed convincingly that eliciting referred pain in the expected pattern for that muscle is **not** a specific finding of TrPs. Instead, its presence is primarily dependent on the amount of pressure applied to the site. In all 25 examinations, referred pain was elicited from both the active TrP site and its taut band site (2 cm removed from the TrP). At the control site of patients with active TrPs, referred pain was elicited in **half** of the examinations before reaching pain tolerance. In the subjects with latent TrPs, characteristic referred pain was elicited from control sites in **one-quarter** of the examinations. These findings agree with those of Scudds, *et al.*[232] that referred pain frequently can be elicited from normal muscle with the application of sufficient pressure in subjects with no pain complaint. The presence of local tenderness at these apparently normal-muscle sites is more likely in subjects who are suffering from TrP pain and is to be expected in patients with fibromyalgia.

Hong, *et al.*[125] found that referred pain could be elicited from every active TrP site, but from only 47% of the latent TrP sites. Stated another way, it took less pressure to elicit referred pain from an active TrP than from a latent TrP. As would be expected, all three kinds of thresholds were significantly lower ($P < 0.01$) at active TrPs than at latent TrPs. The more irritable the TrP, the lower its pain threshold. However, there was considerable overlap between values obtained from active and latent TrPs, so threshold measurements alone were not sufficient to distinguish active from latent TrPs. This study demonstrates that pressure algometry can be a powerful research tool and useful clinical tool.

An incomplete, single-subject, illustrated report[77] indicates that pressure pain thresholds measured at intervals along the taut band are lowest at the TrP and that both TrP and taut band thresholds increase considerably following needling and injection of the TrP. Systematic controlled studies of pressure thresholds throughout the length of the taut band including the attachment are needed. The essentials for such a research study have been identified.[243]

Another form of pressure algometer is an electronic pressure-sensitive film that can be placed on the finger tip. Such a device was described as a palpometer.[16] All of the versions tried so far had a problem with adequate sensitivity and linearity of instrumental response at small pressure values, where resolution and accuracy are most important. Since some degree of sensation as to what is being palpated is transmitted through the film to the finger tip, a properly engineered device may have a significant advantage over the spring scale system. The palpometer approach has the advantage that it is electronic and that the results can be recorded readily and the data entered directly into a computer for analysis and storage.

Thermography. Thermograms can be recorded by infrared radiometry or with films of liquid crystal. Recording infrared radiation (electronic thermography) with computer analysis provides a powerful tool for the accurate rapid visualization of skin temperature changes over large areas of the body. This technique can demonstrate cutaneous reflex phenomena characteristic of myofascial TrPs. The less expensive contact sheets of liquid crystal have limitations that make reliable interpretation of the findings considerably more difficult.

Each of these thermographic techniques measures the skin surface temperature to a depth of only a few millimeters. The temperature changes correspond to changes in the circulation within, but not beneath, the skin. The endogenous cause of these temperature changes is usually sympathetic nervous system activity. Thermographic changes in skin temperature, therefore, are comparable in meaning to changes in skin resistance or changes in sweat production. However, electronic infrared thermography is superior to these other two measures in convenience and in spatial as well as temporal resolution.

In summary, the following research studies indicate that just finding a hot spot on the thermogram is NOT sufficient to identify a TrP beneath it. A similar temperature change can be expected from radiculopathy, an articular dysfunction, enthesopathy, or due to a local subcutaneous inflammation. The thermographic hot spot of a TrP is described as a discoid region 5 to 10 cm in diameter, displaced slightly from directly over the TrP.[71] Five studies reported a region of hyperthermia over the TrP (a total of 170 TrPs);[52,53,74,158-161] none reported a finding of hypothermia. No such agreement exists with regard to skin temperature changes in the region of referred pain. However, available data suggest an interesting possibility. Undisturbed TrPs referring spontaneous autonomic cutaneous effects may tend to induce hyperthermia in a limited area of the skin overlying the TrP, whereas mechanical stimulation of the TrP that causes additional pain induces a "reflex" hypothermia that is dependent on the stimulus. This reflex hypothermia phenomenon may be a far more discriminating criterion of a TrP than the hyperthermia over the TrP. However, research studies are needed to investigate whether this reflex hypothermia is distinguishable from that which may occur when painful pressure is applied to a tender articular dysfunction, area of bursitis, or an area of enthesopathy.

A thermographic hot spot was used by Kruse and Christiansen[161] as an initial identifier of the likely location of a TrP. Then, the presence of the TrP was confirmed by physical examination. This procedure eliminated from consideration TrPs that might not be thermographically active.

Fischer and Chang[79] examined the gluteal region of 14 consecutive low back pain patients for thermographic hot spots. Hot spots were examined for spot tenderness in 13 muscles and 1 ligament. Reduced pressure threshold readings were significantly correlated ($P < 0.01$) with hot spots compared to contralateral control sites. Hot spots were likely to be tender sites, but the report left open the question of whether these sites were tender because of TrPs, fibromyalgia tender points, or other causes.

Swerdlow and Dieter[265] examined 165 patients who suffered whiplash injury and found 139 of them had TrPs in the upper, middle, or lower trapezius muscles. Using Fischer's thermographic criteria,[74] they found 40% false-positives and 20% false-negatives among these patients, which is unacceptable as a diagnostic criterion.

Scudds, *et al.*[231a] examined the backs of 49 fibromyalgia patients and 19 myofascial pain patients using infrared thermography under resting conditions in conjunction with a dolorimeter study of referred pain. They found that the average skin temperature of the myofascial pain patients was 0.65%C warmer than the fibromyalgia patients. Apparently this study identified TrPs only by spot tenderness and referral of pain, which another study showed can also occur in normal subjects.[232] All TrPs caused referred pain, and half of the most tender spots in fibromyalgia patients also referred pain. This result may mean that half of the fibromyalgia patients also had TrPs, which is consistent with the finding of another

investigator who looked for that possibility,[90] or it may mean that some tender points that are not TrPs may also refer pain. These studies do suggest that patients selected primarily for myofascial TrPs are more likely to exhibit hyperthermia than patients with fibromyalgia. Apparently, the active loci responsible for TrPs not only can cause referred pain, but they also can refer local cutaneous hyperthermia. A thermographic research study is needed of TrPs identified by adequate diagnostic criteria (see Section B of this chapter), and of tender points that are not TrPs in fibromyalgia patients.

Diakow[53] conducted a study to see if active TrPs exhibited a region of hyperthermia extending toward the pain reference zone beyond the usual hot spot as compared to latent TrPs, which were assumed not to do so. In addition, he analyzed a subgroup who showed evidence of articular dysfunction that would be likely to cause hyperthermia in the same region to which a TrP might refer hyperthermia. By eliminating this subgroup of 25 patients (leaving 104), the discrimination of active versus latent TrPs on the basis of Cohen's Kappa statistic improved from 0.44 to 0.55 (bad to poor) and specificity improved from 0.70 to 0.82 (fair to good). These results suggest that articular dysfunction can be an additional source of hot spots, which fits with Korr's studies of facilitated segments.[154]

Two studies indicated that when referred pain is produced by compressing the TrP, the reference zone becomes hypothermic. Travell examined one patient who showed this very clearly.[279]

Kruse and Christiansen[161] did a well-controlled study of temperature change in the reference zone of TrPs in response to pressure stimulation of middle trapezius TrPs. The criteria used for diagnosis of TrPs were not specifically stated but given only by a general reference to the first edition of Volume 1 of this Manual. Infrared thermograms were obtained bilaterally from 5 prescribed upper extremity locations of 11 student volunteers with symptomatic TrPs in the middle trapezius muscle and from 11 asymptomatic controls. Initially thermograms were used to locate thermally active TrPs which were confirmed as TrPs by palpation. Pressure threshold of the TrP and corresponding control sites was determined by algometry. Pressure was then applied to the TrP until the subject felt referred pain, and it was maintained for 1 minute while thermograms were recorded every 15 seconds.

Initially, the region of the TrP site always showed increased temperature compared to its control site. The referred-pain zone, initially, often showed a lesser increase.[161] With compression of the TrP, the areas of thermal response (in the direction of referred pain) showed a statistically significant reduction in temperature, whereas corresponding control sites showed a nonsignificant increase in temperature. The region of thermal response was remarkably more extensive than the region of referred pain. The pressure threshold values at TrP sites were significantly ($P < 0.001$) lower (reflecting more tenderness) than at control sites.

The literature to date fails to address a number of critical questions concerning thermographic changes associated with TrPs. Since many acupuncture practitioners use a skin-resistance point finder to locate the appropriate place to insert the needle for inactivating a TrP (or for treating a pain-type acupuncture point), it would be of considerable interest to explore in a blinded research study, the region of a hot spot for a point of low resistance and determine to what extent a point of low resistance is located within the hot spot and how consistently a low-resistance point has a TrP (active or latent) nearby, beneath it. The presence of a TrP should be determined by adequate diagnostic criteria applied by examiners tested for good interrater reliability. Since several research studies show that the dysfunction characteristic of TrPs is modulated by sympathetic nervous system activity,[33"-167-186] research studies of the effects of TrPs on sympathetic control of skin perfusion should improve our understanding of the functional relationships between myofascial TrPs and the autonomic nervous system.

Treatment

Effective treatment of a myofascial pain syndrome caused by TrPs usually involves more than simply applying a procedure to

the TrPs. It is often necessary to consider and deal with the cause that activated the TrPs, to identify and correct any perpetuating factors (which often are different than what activated the TrPs), and to help the patient to restore and maintain normal muscle function.

This volume includes a number of release and injection techniques, many of which were not considered in the previous edition. These techniques are considered in detail in Chapter 3, Section 12 of this volume. These treatment approaches include the use of simple muscle stretch, augmented muscle stretch, postisometric relaxation, reciprocal inhibition, slow exhalation, eye movement, TrP pressure release, massage, range of motion, heat, ultrasound, high-voltage galvanic stimulation, drug treatment, biofeedback, and new injection techniques.

There are a number of common **misconceptions** about the treatment of TrPs.

1. *Simply treating the TrP should be sufficient.* Occasionally this may be true IF the stress that activated the TrP is not recurrent and IF there are no perpetuating factors present. Otherwise, the TrP is likely to be reactivated again by the same stress. Ignoring perpetuating factors invites recurrence. After the TrPs have persisted for some time, failure to retrain the muscle to normal function or failure to reestablish its full-stretch range of motion results in a degree of persistent motor dysfunction.
2. *The pain cannot be as severe as the patient says and must be largely psychogenic.* The patients are trying to communicate their suffering. Believe them. It feels severe to them. Patients in a general medical practice rated their pain as severe as, or more severe than pain from other causes such as pharyngitis, cystitis, angina, and herpes zoster.[257] In addition, an appreciable amount of the pain reported by many patients with fibromyalgia comes from their TrPs. The pain of fibromyalgia rates fully as severe as the pain of rheumatoid arthritis. It is severe enough to cause central nervous system changes characteristic of chronic pain. Because of their chronic TrP and fibromyalgia pain, these patients often develop pain behaviors which tend to reinforce dysfunction and suffering. Many patients have suffered grievously and needlessly because a series of clinicians unacquainted with myofascial TrPs erroneously applied the psychogenic label to them covertly if not overtly.
3. *Myofascial pain syndromes are self-limiting and will cure themselves.* An acute uncomplicated TrP activated by an unusual activity or muscle overload can revert spontaneously to a latent TrP within a week or two, IF the muscle is not overstressed (used within tolerance, which may be limited) and IF there are no perpetuating factors. Otherwise, if the acute syndrome is not properly managed, it evolves needlessly into a chronic myofascial pain syndrome.
4. *Relief of pain by treatment of skeletal muscles for myofascial TrPs rules out serious visceral disease.* Because of the referred pain nature of visceral pain, application of vapocoolant spray or infiltration of a local anesthetic into the somatic reference zone can temporarily relieve the pain of myocardial infarction, angina, and acute abdominal disease with no effect on the visceral pathology.[290]

Diagnostic Criteria

The lack of general agreement as to appropriate diagnostic criteria for examining trigger points has been an increasingly serious impediment to more widespread recognition of myofascial trigger points and to compatible studies of the effectiveness of treatment.

Interrater Reliability. Of four recent studies on interrater reliability of TrP examinations, the first three reported unsatisfactory to marginal interrater reliability. The fourth study showed why such results were obtained. It demonstrated convincingly the need for all examiners to be both experienced and trained in order to perform reproducible examinations. This section will summarize these studies and the lessons learned.

Four well-designed studies have recently evaluated the reliability of various myofascial TrP examinations. Results are summarized in Table 2.3. In 1992 Wolfe, et al.[293] reported a study, part of which involved the evaluation of 8 muscles in 8 patients by 4 physicians experienced in

Table 2.3. ***Interrater Reliability of Examinations for Trigger Point Characteristics, Kappa Values***

Examination	*Wolfe, et al., 1992*[293]	*Nice, et al., 1992*[95]	*Njoo, et al., 1994*[*°']	*Gen/i/in, et al., 1995*[92]	*Mean*
Spot Tenderness	0.61		0.66	0.84	0.70
Jump Sign			0.70		0.70
Pain Recognition	0.30		0.58	0.88	0.59
Palpable Band	0.29		0.49	0.85	0.54
Referred Pain	0.40	0.38	0.41	0.69	0.47
Twitch Response	0.16		0.09	0.44	0.23
Mean	0.35	0.38	0.49	0.74	

examining patients for TrPs. The muscles examined included the levator scapulae, supraspinatus, anterior scalene, upper trapezius, infraspinatus, pectoralis major, sternocleidomastoid, and the iliocostalis/longissimus muscles in the T_{10}-L_1 region. Each of the four examiners had many years of independent experience, but had no chance prior to this study to agree on a technique for examining the upper body TrPs (they were untrained, experienced examiners). The physicians examined each muscle for 5 findings characteristic of TrPs (Table 2.3). Since subsequent studies reported interrater reliability results in terms of the kappa statistic, two co-authors of this study [Simons and Skootsky] analyzed the original data for the kappa statistic, which corrects for chance agreement. The examiners achieved poor interrater reliability.

Nice, et al.[198] reported on the examination of three sites in the thoracolumbar paraspinal muscles of 50 patients with low back pain by 12 experienced full-time physical therapists who routinely treated patients with low back pain. "A practice session was held to allow the therapists to practice this method on each other until all physical therapists reported that they felt capable of using the method on patients."[198] This was inadequate training because there was no evaluation of uniformity of technique. Again, these were experienced but inadequately trained examiners and they also achieved poor interrater reliability.

Njoo and Van der Does[201] reported the examination of 2 muscles (quadratus lumborum and gluteus medius) in 61 patients with low back pain by 2 examiners picked from a pool of 1 physician in general practice and 4 medical students. Each medical student was well-trained by the physician over a 3 month period but was inexperienced. The average kappa values for the 6 examinations were essentially equal for the quadratus lumborum and gluteus medius muscles, indicating that for them, those muscles were about equally difficult to examine. Four of the five examiners were well-trained but inexperienced. Their interrater reliability was better than that in previous studies, but not good.

Gerwin, et al.[94] reported a double study in which 4 experienced physicians examined 5 muscles bilaterally in each of 10 subjects with myofascial TrPs. The first study was conducted with the assumption that the four experienced examiners employed essentially the same examination technique. They achieved the same poor interrater reliability of other experienced untrained examiners.

However, in a second study by the same four physicians, but following a three-hour training session, agreement among doctors was assessed statistically and found to be reliable before proceeding with the study. The study showed that examination of the extensor digitorum communis and latissimus dorsi muscles was most reliable. Examination of the sternocleidomastoid and upper trapezius mus-

cles was less reliable, and examination of the infraspinatus muscle was least reliable, which suggests that, of the five muscles tested, it is the most difficult to examine reliably.

The results of the four studies are summarized in Table 2.3, from which a number of inferences can be drawn. The table shows, across the bottom row, the mean kappa value of all examinations for each study. The examiners in these studies fell into 3 categories: experienced and untrained, trained and inexperienced, trained and experienced. Two studies, Wolfe, *et al.*[233] and Nice, *et al.*[198] tested experienced but untrained examiners and obtained unsatisfactory mean kappa values of 0.35 and 0.38, respectively. On the other hand, Njoo and Van der Does[201] tested well-trained but inexperienced examiners, who reached a barely satisfactory mean kappa value of 0.49. Gerwin, *et al.*[33] tested well-trained and experienced examiners who achieved a good mean kappa value of 0.74. In the subsequent publication of this study as a paper,[94] the fact that the Kappa statistic is inappropriate when all examiners report the same finding in a subject was considered. Avoiding that mistake showed that the reliability was actually good to excellent, considerably better than that reported in the initial abstract,[93] but the abstract data were used in Table 2.3 so that the Kappa statistics results of the four studies could be compared directly.

Clearly, a clinical or experimental research study of human myofascial trigger points, to obtain the most meaningful results, should employ both experienced and trained examiners who have been tested for inter-rater reliability *BEFORE* the study is conducted. The necessary skill can be learned. Fricton, in a diagnostic study of masticatory myofascial pain, likewise found that experienced raters were more reliable than inexperienced raters and also concluded that findings by palpation are technique-sensitive.[82]

Looking at Table 2.3 from another point of view, one can examine the mean kappa values of all four studies in terms of each examination technique tested *[see* right hand column of Table 2.3). In Table 2.4A, the difficulty of the examinations was ranked according to the mean kappa values derived from these four studies.

Diagnostic Value of Examinations. A second question must be considered, "What is the diagnostic value of the examination technique in terms of its specificity for identifying trigger points?" An estimate of the relative diagnostic value of each measure without regard to other findings is presented in the last column of Table 2.4A. These estimates are based on considerations presented below. However, they need confirmation or modification by experimental studies that examine the sensitivity and specificity of each examination, and combinations of examinations as controlled research studies.

An examination for **spot tenderness** or the **jump sign** is essentially the same test. The vigorousness of the jump sign is an indicator of the amount of pressure applied and the degree of spot tenderness. Either of these tenderness findings alone has limited diagnostic value because of ambiguity as to the cause of tenderness. The tenderness might

Table 2.4A. ***Comparative Reliability of Diagnostic Examinations for Trigger Points, Estimate of the Relative Difficulty Performing the Examinations, and Estimated Relative Diagnostic Value of each Examination by Itself, Regardless of Other Findings***

Presence of	*No. of Studies*	*Mean Kappa*	*Difficulty*	*Diagnostic Value Alone*
Spot Tenderness	3	0.70	+	+*
Pain Recognition	3	0.59	+ +	+ + +
Palpable Band	3	0.54	+ + +	+ +*
Referred Pain	4	0.47	+ + +	+
Twitch Response	3	0.23	+ + + +	+ + + +

***The combined presence of these two will likely have a high diagnostic value for sufficiently skilled examiners.**

be due to myofascial TrPs, fibromyalgia, enthesopathy, bursitis, tendinitis, etc. The response observed is strongly dependent on the amount of pressure applied.[125] For reliable results, the pressure must be quantitatively standardized in some way. If a quantitative estimate of spot tenderness is desired, properly administered pressure algometry[72,73] is superior to testing for the jump sign.

Pain recognition is a relatively reliable test, as long as patients understand that the examiner is asking them IF they recognize the pain as a familiar one that they have experienced recently. They are NOT to identify a referred pain that is new and not familiar to them. **If** the patient recognizes the pain generated by pressure on a TrP, then that tender spot can be considered a source (trigger) that is contributing to at least part of the patient's pain problem.

The finding of a **palpable taut band,** by itself, may be ambiguous because it can sometimes be observed in pain-free subjects without other clinical evidence of TrP phenomena.[201,293] The presence of a palpable nodule in the taut band has not been tested as a possible criterion of myofascial TrPs but some clinicians observe the phenomenon routinely and the nodule is to be expected based on the pathogenesis of TrPs. Normal palpable structures such as intramuscular septa should not be tender. The value of examining for a taut band alone is further limited by the inaccessibility of many muscles to satisfactory manual palpation. However, although never tested experimentally, the presence of spot tenderness combined with a palpable band and nodule should prove highly reliable, if the examiner is skillful at detecting these structures. Addition of a palpable nodule at the tender spot as a criterion may enhance diagnostic sensitivity. Historically, this has been a criterion for diagnosing fibrositis, Myogelosen, Muskelharten, and muscular rheumatism.

Recognized referred pain that reproduces the patient's pain complaint identifies an active TrP and adds greatly to the specificity of the diagnosis. An unrecognized referred pain that corresponds to the known referral zones of the TrP being examined is nonspecific.[125] No study is known that has examined under controlled conditions specifically how commonly this referred pain can be elicited from tender points of fibromyalgia that are not also TrPs. However, tender points of fibromyalgia *per se* should *not* have the other palpable trigger point characteristics.

Scudds, *et al.*[232] did a related study when they located tender muscular spots that referred sensation in 54% of healthy subjects, nearly half of whom described the sensation as pain. The authors did not examine the sensitive locations for other evidence of latent TrPs. The study by Sola, *et al.*[261] found a similar percentage of latent TrPs in an unselected population, suggesting that many of the pain-referring spots found by Scudds, *et al.* were latent TrPs. Hong, *et al.*[125] showed that the threshold to produce a referred pain pattern was reached with less pressure on an active TrP than on a latent one. The additional pressure required to progress from the pain threshold to the referred pain threshold was less at all three sites in muscles with active TrPs than in muscles with latent TrPs. However, there was no sharp line of distinction between active and latent TrPs with regard to the pressure needed to elicit unrecognized referred pain.

Although eliciting referred pain that is not recognized by the patient but conforms to the expected pain pattern for that muscle does not identify a latent TrP unambiguously, it can be very helpful diagnostically. The *spontaneous* referred pain pattern reported by the patient is a very helpful indicator of where to start looking for TrPs.

Twitch responses are strongly associated with the presence of TrPs and this finding is probably the most specific single clinical test of a TrP.[123] However, the extent to which twitch responses can be elicited from other parts of the muscle, particularly in an area of enthesopathy, has not been critically evaluated. Enthesopathy by definition is found only in the region of attachment at the ends of the muscle fibers, whereas TrPs are closely associated with endplates, which are located near the middle of muscle fibers. The clinical diagnostic usefulness of the twitch response is limited to those muscles in which it can be reliably identified visually, by palpation, or by ultrasound imaging. The local twitch response is the most difficult of the diagnostic signs to elicit reliably manually, and rela-

tively few examiners have developed the needed skill. On the other hand, it does seem to be highly specific and is readily elicited by needle penetration of the trigger point.

The addition of ultrasound imaging may greatly increase the importance of testing for an LTR. The LTR requires a high level of skill for reliable results, but, with ultrasound imaging, it also has the potential for providing a specific, objective, recordable, clinically available imaging test for myofascial TrPs. The ultrasound test can provide an objective measure of the relative skill of examiners.

Pain-restricted **range of motion** is a fundamental characteristic of TrPs that has not been subjected to testing for interrater reliability among examiners.

Recommendation. Clearly, there is no one diagnostic examination that alone is a satisfactory criterion for routine clinical identification of a trigger point. Based on experimental information now available,[94] *the combination of spot tenderness in a palpable band and subject recognition of the pain are minimum acceptable criteria.* The criteria currently recommended for diagnosing a myofascial TrP are listed in Table 2.4B. **Most important:** at present, every author reporting a study of myofascial TrPs should identify in the methods section specifically which TrP examinations were used as diagnostic criteria and should describe *in detail* exactly how they were performed. A consensus document that establishes official diagnostic criteria is an urgent need.

Differential Diagnosis and Confusions

Three possible sources of musculoskeletal pain are common and are commonly overlooked: myofascial TrPs, fibromyalgia, and articular dysfunction that requires manual mobilization. These three conditions often interact with one another, require different diagnostic examination techniques, and need significantly different treatment approaches.

One current source of confusion is use of the term *myofascial pain syndrome* for two different concepts.[241] Sometimes, myofascial pain syndrome is used in a general sense that applies to a regional muscle pain syndrome of any soft tissue origin.[108, i6o, 194.207,298,299] Historically, the term myofascial pain syndrome has been used in the restricted sense of that syndrome which is caused by TrPs within a muscle belly (not scar, ligamentous, or periosteal TrPs).[88,255' 260,278,279] g m c e the *general usage* includes many conditions that cause muscle pain without reference to and in absence of TrPs, the use of that terminology is ambiguous and very confusing to those who think in terms of TrPs, which is only one of the conditions included in the general-usage term. For authors, one unambiguous approach is to specify myofascial pain syn-

Tabie 2.4B. ***Recommended Criteria for Identifying a Latent Trigger Point or an Active Trigger Point***

Essential Criteria

1. Taut band palpable (if muscle accessible).
2. Exquisite spot tenderness of a nodule in a taut band.
3. Patient's recognition of current pain complaint by pressure on the tender nodule (identifies an active trigger point).
4. Painful limit to full stretch range of motion.

Confirmatory Observations

1. Visual or tactile identification of local twitch response.
2. Imaging of a local twitch response induced by needle penetration of tender nodule.
3. Pain or altered sensation (in the distribution expected from a trigger point in that muscle) on compression of tender nodule.
4. Electromyographic demonstration of spontaneous electrical activity characteristic of active loci in the tender nodule of a taut band.

drome *due to TrPs* or use the term *regional muscle pain syndrome* to identify the more general usage. The unmodified, unspecified use of the term myofascial pain is discouraged.

This section begins with a listing of common diagnoses that often are made mistakenly without considering the possibility of TrPs. Patients are frequently referred to myofascial TrP experts with one of these diagnoses (and often a patient has been given several of them), but when the patient's pain problem was actually caused by unrecognized or inadequately treated myofascial TrPs.

Next, this section discusses other conditions that are closely related to myofascial TrPs. Commonly both conditions are present. This confusing situation makes it particularly important to draw a sharp diagnostic distinction when the two conditions require different treatment approaches. The related conditions considered include: fibromyalgia, articular dysfunctions, temporomandibular dysfunctions, occupational myalgia, nonmyofascial TrPs, and the posttraumatic hyperirritability syndrome. Also, the relation between acupuncture and myofascial TrPs is considered.

Myofascial Trigger Points Mistakenly Diagnosed as Other Conditions. Those clinicians who have become skilled at diagnosing and effectively managing myofascial TrPs frequently see patients who were referred to them by other practitioners as a last resort. These patients commonly arrive with a long list of diagnostic procedures and diagnoses, none of which satisfactorily explained the cause of, or relieved, the patient's pain. Table 2.5 lists examples of these diagnoses. Beside each diagnosis are listed likely TrP sources of that pain. This frustrating situation is understandable because very few medical schools or physical therapy schools teach myofascial TrPs as a regular part of the curriculum, so most physicians and therapists now in practice have received *at most* a hit-or-miss exposure to myofascial TrPs. For most clinicians, their understanding of, and competence achieved in, diagnosing myofascial TrPs must have been achieved through supplemental learning following graduation.

This list reminds us that every skeletal muscle of the body can develop TrPs, and many of them commonly do. Since myofascial TrP pain is so common and because patients are most likely to experience the pain at sites other than the TrP location, the clinician is prone to miss the diagnosis unless he or she considers the possibility of, and specifically searches for, the distant TrP culprit(s).

Fibromyalgia Syndrome. Two of the three most common muscle pain syndromes, fibromyalgia and myofascial pain due to TrPs, are now recognized as quite separate clinical[90,126] and etiological entities.[224,242] Since both conditions are likely to cause severe muscle pain and frequently co-exist but need a different treatment approach, it is of great importance for the patient's sake that any clinician dealing with a patient who has muscle pain be able to clearly distinguish these two conditions. For one who is interested in understanding what fibromyalgia is, what it means to the patient, and how best to manage it, the reader is referred to an authoritative, comprehensive, readable book for patients written by a nurse and a physician.[81] For one interested in a manual that similarly identifies the clinical nature of both fibromyalgia and chronic myofascial pain caused by TrPs, the reader is referred to the *Survival Manual* by Starlanyl and Copeland.[263] Dr. Starlanyl is a physician who herself has both conditions and has learned how she can deal with them. A third useful patient manual is focused on myofascial TrPs. It is written by a physical therapist who learned about TrPs through personal experience.[118]"

At the beginning of this decade, the American College of Rheumatology established official criteria for the classification of fibromyalgia[294] (Table 2.6). Anyone writing a paper that identifies subjects as having fibromyalgia should adhere closely to these criteria. Likewise, in examining patients for the possibility of fibromyalgia, these are the only criteria that determine officially whether or not that is the appropriate diagnosis. This is a clinical operational definition that makes no pretense at identifying an etiology. In fact, Simms, *et al.*[23]* studied tenderness in 75 anatomical sites comparing fibromyalgia patients and normal control subjects. Simms, *et al.* concluded that of the previously proposed 18 tender points, only 2 were included in

Table 2.5. ***Common Referral Diagnoses Received When Overlooked TrPs were Actually the Cause of Patients Symptoms***

Initial Diagnosis	*Some Likely Trigger Point Sources*	*Trigger Point Manual Chap. # (Volume 1)*
Angina Pectoris (atypical)	Pectoralis major	42
Appendicitis	Lower rectus abdominis	49
Atypical Angina	Pectoralis major	42
Atypical Facial Neuralgia[274]	Masseter	8
	Temporalis	9
	Sternal division of sternocleidomastoid	7
	Upper trapezius	6
Atypical Migraine	Sternocleidomastoid	7
	Temporalis	9
	Posterior cervical	16
Back Pain, Middle	Upper rectus abdominis	49
	Thoracic paraspinals	48
Back Pain, Low[255]	Lower rectus abdominis	49
	Thoracolumbar paraspinals see Volume 2 muscles	48
(Bicipital) Tendinitis	Long head of biceps brachii	30
Chronic Abdominal Wall Pain[106]	Abdominal muscles	49
Dysmenorrhea	Lower rectus abdominis	49
Earache (enigmatic)	Deep masseter	8
Epicondylitis	Wrist extensors	34
	Supinator	36
	Triceps brachii	32
Frozen Shoulder	Subscapularis	26
Myofascial Pain Dysfunction	Masticatory muscles	8-11
Occipital Headache[103]	Posterior cervicals	16
Postherpetic Neuralgia	Serratus anterior	46
	Intercostals	45
Radiculopathy, C_6	Pectoralis minor	43
	Scalenes	20
Scapulocostal Syndrome	Scalenes	20
	Middle trapezius	6
	Levator scapulae	19
Subacromial Bursitis	Middle deltoid	28
Temporomandibular Joint Disorder	Masseter	8
	Lateral pterygoid	11
Tennis Elbow	Finger extensors	35
	Supinator	36
Tension Headache[140]	Sternocleidomastoid	7
	Masticatory muscles	8-11
	Posterior cervicals	16
	Suboccipital muscles	17
	Upper trapezius	6
Thoracic Outlet Syndrome[127]	Scalenes	20
	Subscapularis	26
	Pectoralis minor and major	43,42
	Latissimus dorsi	24
	Teres major	25
Tietze's Syndrome	Pectoralis major enthesopathy	42
	Internal intercostals	45

Table 2.6. ***The American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia****

1. History of widespread pain.
   Definition. Pain is considered widespread when all of the following are present: pain in the left side of the body, pain in the right side of the body, pain above the waist, and pain below the waist. In addition, axial skeletal pain (cervical spine or anterior chest or thoracic spine or low back) must be present. In this definition, shoulder and buttock pain are considered as pain for each involved side. "Low back" pain is considered lower segment pain.
2. Pain in 11 of 18 tender point sites on digital palpation.
   Definition. Pain on digital palpation must be present in at least 11 of the following 18 tender point sites:
   Occiput: bilateral, at the suboccipital muscle insertions.
   Low cervical: bilateral, at the anterior aspects of the intertransverse spaces at C5-C7.
   Trapezius: bilateral, at the midpoint of the upper border.
   Supraspinatus: bilateral, at origins, above the scapular spine near the medial border.
   Second rib: bilateral, at the second costochondral junctions, just lateral to the junctions on upper surfaces.
   Lateral epicondyle: bilateral, 2 cm distal to the epicondyles.
   Gluteal: bilateral, in upper outer quadrants of buttocks in anterior fold of muscle.
   Greater trochanter: bilateral, posterior to the trochanteric prominence.
   Knee: bilateral, at the medial fat pad proximal to the joint line.
   Digital palpation should be performed with an approximate force of 4 kg.
   For a tender point to be considered "positive" the subject must state that the palpation was painful. "Tender" is not to be considered "painful."
   Note: For classification purposes, patients are said to have fibromyalgia if both criteria are satisfied. Widespread pain must have been present for at least 3 months. The presence of a second clinical disorder does not exclude the diagnosis of fibromyalgia.

•Reprinted by permission from Wolfe F, Smythe HA, Yunus MB, *et al.* The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: Report of the Multicenter Criteria Committee. *Arthritis Rheum* 1990; 33:160-170.

what they found to be the 19 most discriminating points. The tender sites selected as diagnostic criteria are quite arbitrary, but adequately representative of the patient's total-body, physiologically enhanced sensitivity to pain.

Fibromyalgia can be thought of as a set of core features and two types of ancillary features. The core features are generalized pain and tenderness over 11 of 18 prescribed anatomical sites. Characteristic ancillary features occur in over three-quarters of individuals: fatigue, nonrestorative sleep, and morning stiffness. Less common findings, in perhaps 25% of cases, include: irritable bowel syndrome, Raynaud's phenomenon, headache, subjective swelling, nondermatomal paresthesia, psychological stress, and marked functional disability. Patients with fibromyalgia experience at least as much pain as those with other painful disease states.[183] Even though fibromyalgia was at first thought to originate in skeletal muscles, a careful histological and ultrastructural study has shown no abnormality of skeletal muscles that was sufficiently common for that to be considered the cause of fibromyalgia.[18'224]

On the other hand, the etiology of myofascial TrPs is clearly a focal muscular dysfunction which can exert a strong influence on all major parts of the nervous system, and can lead to spinal level neuroplastic changes that help to convert an acute pain problem into a chronic one.

There is strong research support for a systemic, metabolic/neurochemical pathogenesis of fibromyalgia. Fibromyalgia is considered an upward modulation of pain sensitivity throughout the body. Extensive research in recent years has led to the "serotonin deficiency hypothesis"[224] that involves measurable disturbance in nociception, including serotonin regulation of

the hypothalamic pituitary axis and the pituitary adrenal axis, and substance P. There is a close relationship between substance P and calcitonin gene-related peptide that also appears to be involved.[224] Experimental evidence also indicates that N-methyl-**D**-aspartate receptors of the central nervous system are involved in the pain mechanisms of fibromyalgia.[262] A specific and often hard-to-detect thyroid dysfunction may be a commonly overlooked, but treatable, factor in fibromyalgia.[176] Muscle nociceptive input may contribute to the pathogenesis or severity of fibromyalgia.[18]

Many studies show that a considerable number of fibromyalgia patients also have myofascial TrPs. In three studies, the percentages of fibromyalgia patients who also have TrPs were reported as 100% to 68%.[70, 104] A study of 22 fibromyalgia patients[119] found that 40% needed TrP injections, and 89% of those injected reported relief. One early author considered the presence of myofascial TrPs an essential feature of primary fibromyalgia.[45] layson[144] considered injection of TrPs an important part of treating fibromyalgia syndrome. Others[217, 230] emphasized the clinical importance of clearly distinguishing fibromyalgia and myofascial TrPs.

Distinguishing myofascial TrPs and fibromyalgia is relatively simple when the myofascial TrPs are acute, but can be much more difficult when the myofascial TrPs have evolved into a chronic pain syndrome through neglect or inappropriate treatment. Fibromyalgia, by definition, is a chronic pain syndrome. Table 2.7 lists a number of clinical features that distinguish myofascial pain due to TrPs from fibromyalgia. The following comments relate to this table.

Trigger points occur with nearly equal prevalence in male and female subjects,[261] whereas usually between 4 to 9 times as many females as males are observed to have fibromyalgia,[182] depending on the population studied.

Since fibromyalgia is by definition characterized by widespread, generalized pain and tenderness, this provides a basic distinction from a myofascial TrP, which causes a specific localized pain and tenderness pattern originating from a lesion in a muscle.

When examined, muscles harboring TrPs feel tense because of the contraction knots and taut bands, whereas muscles of a patient with fibromyalgia feel softer and more doughy, unless the fibromyalgia patient also has TrPs in the muscle being examined. The muscles of fibromyalgia patients show increased compliance.

Restricted range of motion is characteristic of TrPs, whereas hypermobility is relatively common in children[86] and in adults[292] who have fibromyalgia.

Patients with myofascial pain are examined for myofascial TrPs as described in this volume, whereas fibromyalgia patients are examined for tender points. Myofascial TrPs and fibromyalgia tender points are equally tender at the cutaneous, subcutaneous, and intramuscular levels. However, the two conditions are sharply distinguished by the fact that locations other than tender point sites in fibromyalgia patients are as tender at all three depths of tissue as are their tender point sites,[284] whereas non-TrP sites in myofascial pain

Table 2.7. ***Clinical Features Distinguishing Myofascial Pain due to Trigger Points (TrPs) from Fibromyalgia***

Myofascial Pain (TrPs)	*Fibromyalgia*
1 female : 1 male	4-9 females : 1 male
Local or Regional pain	Widespread, general pain
Focal tenderness	Widespread tenderness
Muscle feels tense (taut bands)	Muscle feels soft and doughy
Restricted range of motion	Hypermobile
Examine for trigger points	Examine for tender points
Immediate response to injection of TrPs	Delayed and poorer response to injection of TrPs
20% also have fibromyalgia[90]	72% also have active TrPs[90]

patients have been shown to measure the same high pain thresholds as corresponding sites in normal subjects.[284] Fibromyalgia patients are abnormally tender almost everywhere. Myofascial pain patients are abnormally tender only at sharply circumscribed TrP sites and specific sites of referred tenderness.

Recently, tender points have been designated as fibrositic tender points.[183] This is an unfortunate misnomer since there is no palpable or pathological evidence that muscular fibrosis is involved in fibromyalgia. It is equally inappropriate as a name for TrPs with rare exceptions.

TrPs injected in myofascial pain patients who also have fibromyalgia showed a delayed and poorer response than TrPs injected in patients who have myofascial pain syndrome without fibromyalgia.[125]

Articular Dysfunctions. Articular dysfunctions that require manual mobilization make up one of the three major categories of musculoskeletal pain syndromes that are often overlooked. The pain in these syndromes is commonly caused by TrPs. Traditional medical physicians pioneered an understanding of TrPs while osteopathic physicians, chiropractors, and practitioners of orthopedic medicine have developed and promoted manual medicine techniques. Until recently, the two have, for the most part, followed separate paths. The osteopathic pioneer F. Mitchell for many years taught, and eventually published, his insight into the close relationship between articular dysfunctions and the muscular system.[195] However, his writings show no evidence that he was aware of the important role of myofascial TrPs.

Currently, at least one college of osteopathy emphasizes the importance of the close relation between TrPs and articular dysfunction. Rarely do medical schools teach mobilization of joints. Physical therapy curricula are more likely to include the diagnosis and treatment of articular dysfunctions than that of myofascial pain due to TrPs.

An outstanding osteopathic pioneer in the establishment of physiological dysfunctions associated with articular dysfunction, Irvin Korr, explored and promoted the concept of the facilitated segment. In the segmental vicinity of an "osteopathic lesion" (vertebra with evidence of articular dysfunction), Korr and associates demonstrated decreased pain thresholds, increased sympathetic activity (decreased skin resistance), and facilitation of motor pathways.[154] With other coworkers,[50] Korr demonstrated a muscular component to the facilitated segment. They reported a marked increase in paraspinal muscle activity associated with dysfunctional articular segments. However, they apparently were unaware of myofascial TrPs and how they related to the muscle tenderness that the authors associated closely with the articular dysfunction.

There is a remarkable analogy between this concept of a facilitated segment that can strongly influence the three components of the nervous system—motor, sensory, and autonomic—and the nervous system effects that can be caused by myofascial TrPs. The important relationship between the muscles and articular dysfunction is well recognized by many clinicians, but has been badly neglected as a subject for serious research investigation.

Karel Lewit[168,169] published observations and studies from his extensive experience as a neurologist practicing manual medicine and described the close relationship between articular dysfunction and myofascial TrPs. He emphasizes the importance of addressing therapeutically the muscle-dysfunction component *and* the articular-dysfunction component of musculoskeletal pain syndromes when both are present.[170] The increased tension of TrP taut bands and their facilitation of motor activity can maintain displacement stress on the joint while abnormal sensory input from the dysfunctional joint can reflexly activate the TrP dysfunction. The two conditions can aggravate each other.

Since publication of the *Trigger Point Manual* in 1983, the chiropractic profession has become increasingly interested in myofascial TrPs as such. One of their members has presented the only published report[175] of which we are aware that looked specifically at the relationship between articular dysfunction and TrPs. In this preliminary test, he examined the relative amount of EMG activity that appeared in paraspinal muscles of normal, slightly involved, and severely involved segments in response to pressure on a distant TrP. He found that induction of additional pain by pressure on a distant ac-

tive TrP markedly augmented the EMG activity in muscles of severely subluxed segments as compared to normal segments. This finding indicates that articular dysfunction can effectively increase the responsiveness of motor neurons of adjacent muscles to nociceptive input from distant TrPs.

Occupational Myalgias. The subject of occupational myalgias has attracted increasing interest in recent years. A MEDLINE search from 1990 through 1995 recovered 56 abstracted articles on the subject. The 11 different terms used by the authors fell into 3 groups: cumulative trauma, repetitive strain, and overuse. Twenty of the papers dealt with cumulative trauma, of which 18 used the term "cumulative trauma disorder." In the repetitive strain group, only 12 of the 28 papers used the term "repetitive strain injury." Others used "repetitive motion injuries" and "repetitive motion studies." Seven of the 8 in the overuse group were labeled "overuse syndrome." This is another example of many authors using different terms to identify essentially the same muscle pain syndrome. All authors had one root concern-patients developed musculoskeletal pain symptoms as a result of work activity. Many authors expressed frustration at the lack of a satisfactory explanation for the cause of the pain itself.

A cardinal feature of myofascial TrPs is that they are activated either by an acute overload or repeated overuse. The one common denominator of all 56 articles is the association of musculoskeletal pain with overload and/or overuse of the muscle. Placing a muscle in an awkward position that requires sustained contraction of specific muscles in order to maintain that posture is one of the most common examples of overuse. Headley[118] emphasized how commonly the symptoms of patients with cumulative trauma disorder are caused by myofascial TrPs. She demonstrated electromyographically abnormal function of muscles caused by the TrPs in these patients. This study supports the clinical experience of the authors and practicing clinicians.[178282]

Remarkably, **NOT ONE** of these 56 occupational myalgia abstracts indicated that the author(s) had considered the possibility that myofascial TrPs may be contributing to the workers' or patients' problems. This is a serious oversight for all concerned. By consistently overlooking a major treatable cause of muscle pain, confusion and frustration are bound to follow. One study[55] queried doctors as to their understanding of the term "repetitive strain injury" and found that, because diagnostic criteria are so variable among papers, the term is effectively meaningless. Half of the doctors who responded were of the opinion that there was no genuine organic condition corresponding to their assessment of what that term means. The other half thought it was a genuine disease entity but showed little agreement as to what they thought was wrong. One likely possibility is that a TrP origin of the pain is being overlooked by most practitioners concerned with this condition.

Fortunately, most authors approached resolution of occupational myalgias by reducing the overload and/or overuse whenever possible. This way, the mechanical perpetuating factors that could have been aggravating TrPs were ameliorated or eliminated, allowing the muscle to partially, or occasionally completely, recover normal function.

However, if the source of pain and dysfunction of occupational myalgias were specifically related to TrPs in the muscle being overused, local TrP management of that muscle would expedite return to normal function. The employees or patients could be trained to recognize activities that abused the involved muscles and to tailor routine activities and stretching exercises to maintain normal function of those muscles, which would greatly reduce the likelihood of reactivation. Rosen[219,220] emphasizes the importance of the awareness of TrPs in the management of painful muscles that are used beyond their "critical load," especially among performing artists.

Trigger Points and Acupuncture. The distinction between TrPs and acupuncture points *for the relief of pain* is blurred for a number of good reasons. First, the mechanisms responsible for the pain relief associated with the two concepts have until very recently been enigmatic or controversial. Second, as reported by Melzack, *et al.*,[109] there is a high degree of correspondence (71% based on their analysis) between published locations of TrPs and classical acupuncture points *for the relief of pain.* Third, a number of studies report similar

results when needling TrPs using acupuncture needles as when using hypodermic needles with injected solution.[110,123,141]

The evidence that TrP phenomena originate in the vicinity of dysfunctional endplates is presented later in this chapter. Classical acupuncture points are identified as prescribed points along meridians defined by ancient Chinese documents. As Melzack, *et al.*[187] showed, the ancient Chinese clinicians were astute enough to recognize the importance of many common TrP locations and to include them in their charts of acupuncture points for pain.

Currently, there are a number of practitioners of acupuncture who use a modified definition of acupuncture points which would selectively identify TrP locations. Belgrade[13] states that "tender points are acupuncture points and can often be chosen for therapy." If one defines an acupuncture point for treatment of pain as a tender spot, one is using a cardinal definition of TrPs as a criterion for an acupuncture point, which would increase the likelihood of treating a TrP and calling it an acupuncture point. Supporting this concept, Loh, *et al.*[174] compared acupuncture therapy with medical treatment for migraine and muscle tension headaches. They found that benefit from acupuncture was more likely to occur when the subject was treated at local tender muscular points. However, some classical acupuncture points for pain cannot be myofascial TrPs, such as those in the ear. Central myofascial TrPs occur in the midfiber region of a muscle belly.

It is now well-established that pain relief experienced from classical acupuncture points is associated with an endorphin response in the central nervous system.[13] However, the reduction of pain by inactivating a TrP is produced by eliminating the nociceptive focus in a muscle that is responsible for the pain. The fact that nociceptive input from the TrP can cause some central modulation of endorphins[69] tends to confuse the issue but does not change the primary muscular site of the TrP mechanism.

One student of acupuncture, Pomeranz,[208] emphasized the importance of the Deqi phenomenon for identifying an acupuncture point. The Deqi phenomenon is described as a sensation of fullness, distension, and pins and needles when the inserted needle encounters the acupuncture point. However, essentially the same sensory phenomenon is frequently observed when injecting a TrP and the local twitch response is observed.[123] In a study of the analgesia obtained by electroacupuncture, the authors[210] concluded that the effect may be the result of intense stimulation of TrPs.

Another version of "acupuncture" used for the treatment of TrPs involved insertion of the needle to only a depth of approximately 4 mm into the skin and subcutaneous tissue overlying the TrP.[7, 8] Compared to needle penetration of the TrP, this insertion technique must involve an entirely different mechanism that depends on nervous system modulation of TrP activity.[247] This technique requires a controlled clinical study to confirm its efficacy for TrPs and, if effective, needs further research to identify its mechanism.

Ward[286] examined 12 acupuncture sites that were also common TrP sites in either a trapezius or infraspinatus muscle for the electrical activity characteristic of an active locus in a TrP (see Section D, Nature of Trigger Points). Characteristic endplate spike activity was observed in every case.

In conclusion, frequently the acupuncture point selected for the treatment of pain is actually a TrP. Sometimes, it is not a TrP. Because of the fundamental differences in mechanism, approach to management, and prognostic implications, it is important that clinicians identify TrPs as such so they can institute an appropriate home program and correct perpetuating factors, if present.

Nonmyofascial Trigger Points. Trigger points that refer pain also may be observed in what appears to be normal skin, in scar tissue, fascia, ligaments, and the periosteum. The reason for sensitization of nociceptors at these sites needs to be clarified, but must be different from the central TrP mechanism that is closely associated with motor endplates.

Sinclair[256] reported **skin TrPs** in 8 of 30 healthy young adults. He found sharply circumscribed TrP areas while exploring the body by pinching the skin between the finger and thumb. He studied 18 skin TrPs intensively in 4 of these subjects and performed a skin biopsy. Generally, a sharp, stinging, moderately severe pain was referred either locally or remotely to the skin from a cutaneous TrP. The area of referred

pain also showed modulation of sensation (referred tenderness or referred dysesthesia) by stimulation of the TrP. Some reference zones were within the same segmental distribution, but others had no segmental relation to their skin TrPs.

Trommer and Gellman[281] reported seven patients in whom skin TrPs referred pain or numbness to other skin areas that were often nearby, sometimes remote. The skin TrPs were found by pricking the skin with a needle, exploring for a sensitive spot that reproduced the patient's symptoms. In every case, the symptoms were relieved by repeated *intracutaneous* injections, but only if they were made precisely at the skin TrP.

These studies do not suggest a constancy in the referred pain patterns of cutaneous TrPs like that observed for myofascial TrPs. Also, there was no indication in these reports, nor in our observations, that the reference zones of skin TrPs bear any relation to the reference zones of TrPs in underlying muscles.

In our experience, **scar TrPs** (in skin or mucous membranes) refer burning, prickling, or lightning-like jabs of pain. Defalque[49] reported using alcohol injection to treat TrPs in postoperative scars of 69 patients, and 91% of the patients experienced permanent cure or marked improvement. Such scar TrPs can often be inactivated by precise intracutaneous injection with 0.5% procaine solution. In refractory cases, the addition of a soluble steroid to the local anesthetic solution used for injection of the scar TrP can be effective. Bourne[22] injected the scar TrPs with triamcinolone acetonide and lidocaine hydrochloride. Travell similarly used dexamethasone sodium phosphate with 0.5% procaine, injecting a few tenths of a milliliter at any one location.

Nonmyofascial TrPs may also be found in **fascia, ligaments,** and **joint capsules.** Kellgren[149] demonstrated experimentally that fascial epimysium of the gluteus medius muscle referred pain several centimeters distally when injected with 0.1 ml of 6% saline solution, and that a tender spot in the tendon of the tibialis anterior, similarly injected, referred pain to the medial aspect of the ankle and instep.

Travell[269] reported that an acute sprain of the ankle was accompanied by the development of four TrPs in the joint capsule, each of which referred pain to the ankle and foot. Myofascial TrPs resulting from acute sprains of the knee, ankle, wrist and metacarpophalangeal joint of the thumb have been reported to cause referred pain, which was at first elicited and then permanently relieved by injection of each TrP with physiologic saline.[268 277] Leriche[166] identified ligamentous TrPs following fracture or sprain; the TrPs responded completely to 5 or 6 injections of a local anesthetic. Gorrell[100] reviewed the anatomy of the ankle ligaments and described a technique for the identification and injection of ligamentous TrPs at this joint.

Kraus[159] briefly reviewed the literature on ligamentous TrPs and noted that they are easily localized for injection, which often gives immediate pain relief and a postinjection soreness lasting up to 10 days. Hackett[113] illustrated patterns of pain referred from the iliolumbar, sacroiliac, sacrospinal, and sacrotuberous ligaments; he recommended injection of a sclerosing agent, which was not widely accepted because his technique caused too many complications. Dittrich[54] found TrPs in the aponeurosis of the latissimus dorsi muscle where it joins the lumbodorsal fascia; the TrPs referred pain to the shoulder region. Two authors, de Valera and Raftery[51] reported trigger areas in three pelvic ligaments, the sacroiliac, sacrospinous and sacrotuberous, which, when strained, become tender to palpation, refer pain, and respond to injection with a local anesthetic.

Tenderness at a musculotendinous junction may be enthesopathy secondary to taut-band tension of a TrP in the muscle belly or may be a local tendinous TrP. Weiser[289] described point tenderness at the insertion of the semimembranosus muscle in 98 patients who complained of spontaneous pain at the medial aspect of the knee. The pain was reproduced by local pressure or tension at that insertion site. Symptoms were relieved by injecting 2% lidocaine hydrochloride with triamcinolone into the tender spot. Unless the clinician also examines the muscle for taut bands and TrPs, it is not clear what is the cause of the tenderness and therefore how to prevent its recurrence.

Kellgren[150] established an experimental basis for **periosteal TrPs** by demonstrating that the periosteum also can refer pain in response to injection of hypertonic saline,

just as the muscles do. Among 160 experiments designed to determine the nature of referred pain originating from deep tissues, Inman and Saunders[134] reported that noxious stimulation of the periosteum by scratching it with a needle, by injecting it with 6% salt solution, or by applying a measured pressure elicited severe referred pain that sometimes radiated for considerable distances. Tenderness was referred to the muscles and bony prominences within the pain reference zone, as also happens with myofascial TrPs. Repeated stimulation of the same periosteal or ligamentous attachments consistently referred pain in the same direction, but the extent of radiation varied with the intensity of the stimulus. Unfortunately, the authors did not report the distribution of these specific periosteal referred pain patterns. Autonomic reactions to the stimulation, such as sweating, blanching, and nausea were frequently observed in the subjects.

Clinically, the periosteum can be a potent source of referred pain.[107] Relief of this referred pain may be obtained by injecting periosteal TrPs, analogous to the relief obtained by injecting myofascial or cutaneous TrPs.[165]

Posttraumatic Hyperirritability Syndrome. The term "posttraumatic hyperirritability syndrome" was introduced[135,237] to identify a limited number of patients with myofascial pain who exhibit marked hyperirritability of the sensory nervous system and of existing TrPs. A similar syndrome was described earlier by Margoles as the stress *neuromyelopathic pain syndrome.*[180] These patients may be sometimes identified as suffering from severe sudden-onset fibromyalgia that is associated with physical trauma and myofascial TrPs. This syndrome follows a major trauma, such as an automobile accident, a fall, or a severe blow to the body that is apparently sufficient to injure the sensory modulation mechanisms of the spinal cord or brain stem. The patient has constant pain, which may be exacerbated by the vibration of a moving vehicle, by the slamming of a door, by a loud noise (a firecracker at close range), by jarring (bumping into something or being jostled), by mild thumps (a pat on the back), by severe pain (a TrP injection), by prolonged physical activity, and by emotional stress (such as anger). Recovery from such stimulation is slow. Even with mild exacerbations, it may take the patients many minutes or hours to return to the baseline pain level. Severe exacerbation of pain may require days, weeks, or longer to return to baseline. These patients may have multiple TrPs which are not the primary cause of their condition, but which do contribute to their misery and require special consideration in treatment because of the adverse consequences of strong sensory stimulation, especially pain.

Patients with posttraumatic hyperirritability syndrome almost always give a history of having coped well in life prior to the injury, having paid no more attention to pain than did their friends and family. They were no more sensitive to ordinary stimuli than other persons. From the moment of the initial trauma, however, pain suddenly became the focus of life. They must pay close attention to the avoidance of strong sensory stimuli; they must limit activity because even mild to moderate muscular stress or fatigue intensifies the pain. Efforts to increase exercise tolerance may be self-defeating. Such patients, who suffer greatly, are poorly understood and, through no fault of their own, are difficult to help. It may be worth considering an unconventional but knowledgeable approach, such as that of Goldstein,[98] for these patients.

In patients with posttraumatic hyperirritability, the sensory nervous system behaves much as the motor system does when the spinal cord has lost supraspinal inhibition. With increased motor responsiveness, a strong sensory input of almost any kind can activate nonspecific motor activity (spasm) for an extended period of time. Similarly in patients with hyperirritability syndrome, a strong sensory input can increase the excitability of the nociceptive system for long periods. In addition, these patients may show lability of the autonomic nervous system with skin temperature changes and swelling that resolve with inactivation of regional TrPs. Since *routine* medical examination of hyperirritability syndrome patients fails to show any organic cause for their symptoms, they are often disposed of by assigning them a psychological or behavioral diagnosis.

Any additional fall or motor vehicle accident that would ordinarily be considered

minor can severely exacerbate the hyperirritability syndrome for years. Unfortunately, with successive traumas, the individual may become increasingly vulnerable to subsequent trauma. A common finding is a series of relatively minor motor vehicle accidents or falls over a period of several years that cumulatively became severely incapacitating.

Similar phenomena were subsequently described as the *cumulative trauma disorder*[30] and the *jolt syndrome.*[01]

C. MUSCLE STRUCTURE AND FUNCTION

To understand the nature of myofascial TrPs, it is necessary to understand several aspects of basic muscle structure and function that are not usually emphasized. In addition to the material presented here, this subject is also treated in detail in Chapter 8 of a companion text.[191]

Muscle Structure and Contractile Mechanism

A striated (skeletal) muscle is an assembly of fascicles, each of which is a bundle of roughly 100 muscle fibers (Fig. 2.5, fiber in upper panel). Each muscle fiber (a muscle cell) encloses approximately 1000-2000 myofibrils in most skeletal muscles. A myofibril consists of a chain of sarcomeres connected serially, end-to-end. The basic contractile unit of skeletal muscle is the sarcomere. Sarcomeres are connected to each other by their Z lines (or bands) like links in a chain. Each sarcomere contains an array of filaments that consist of actin and myosin molecules which interact to produce the contractile force. The middle panel of Figure 2.5 shows a resting-length sarcomere with complete overlap of actin and myosin filaments (maximum contractile force). During *maximum shortening* the myosin molecules impinge against the "Z" band blocking further contraction (not shown). The lower panel shows a nearly fully stretched sarcomere with incomplete overlap of actin and myosin molecules (reduced contractile force).

The myosin heads of a myosin filament are a form of the enzyme adenosine triphosphatase that contacts and interacts with the actin to produce a contractile force. These contacts are seen through the electron microscope as cross bridges between the actin and myosin filaments. Ionized calcium triggers the interaction between the filaments, and adenosine triphosphate (ATP) provides the energy. The ATP releases a myosin head from the actin after one power "stroke" and immediately "recocks" it for another cycle. The ATP is converted to adenosine diphosphate (ADP) in the process. The presence of calcium immediately triggers another cycle. Many such power "strokes" are needed to produce the random rowing motion that is required of many myosin heads of many filaments to accomplish one smooth twitch contraction.

In the presence of both free calcium and ATP,[197] the actin and myosin continue to interact, expending energy and exerting force to shorten the sarcomere. This interaction of actin and myosin, that produces tension and consumes energy, cannot happen if the sarcomeres are lengthened (the muscle stretched) until no overlap remains between the actin and the myosin heads. This has started to happen in the lower panel of Figure 2.5, where the actin filaments are beyond the reach of half of the myosin heads (cross bridges). The contractile force that any one sarcomere can exert on activation depends strongly on its length. The force drops off rapidly as the sarcomere approaches maximum or minimum length (fully stretched or fully shortened). Therefore, each sarcomere of a muscle can generate maximum force only in the midrange of its length but it can expend energy in the fully shortened position trying to shorten further.

The calcium is normally sequestered in the tubular network of the sarcoplasmic reticulum (Fig. 2.5, upper panel, and Fig. 2.6) that surrounds each myofibril. Calcium is released from the sarcoplasmic reticulum that surrounds each myofibril when a propagated action potential reaches it from the surface of the cell through "T" tubules (Fig. 2.6). Normally, after it has been released, the free calcium is quickly pumped back into the sarcoplasmic reticulum. The absence of free calcium terminates the contractile activity of the sarcomeres. In the absence of ATP, the myosin heads remain firmly attached (failure to "recock") and the muscle becomes stiff as in rigor mortis.

A well illustrated, more detailed description of the entire contractile mechanism is available.[3]

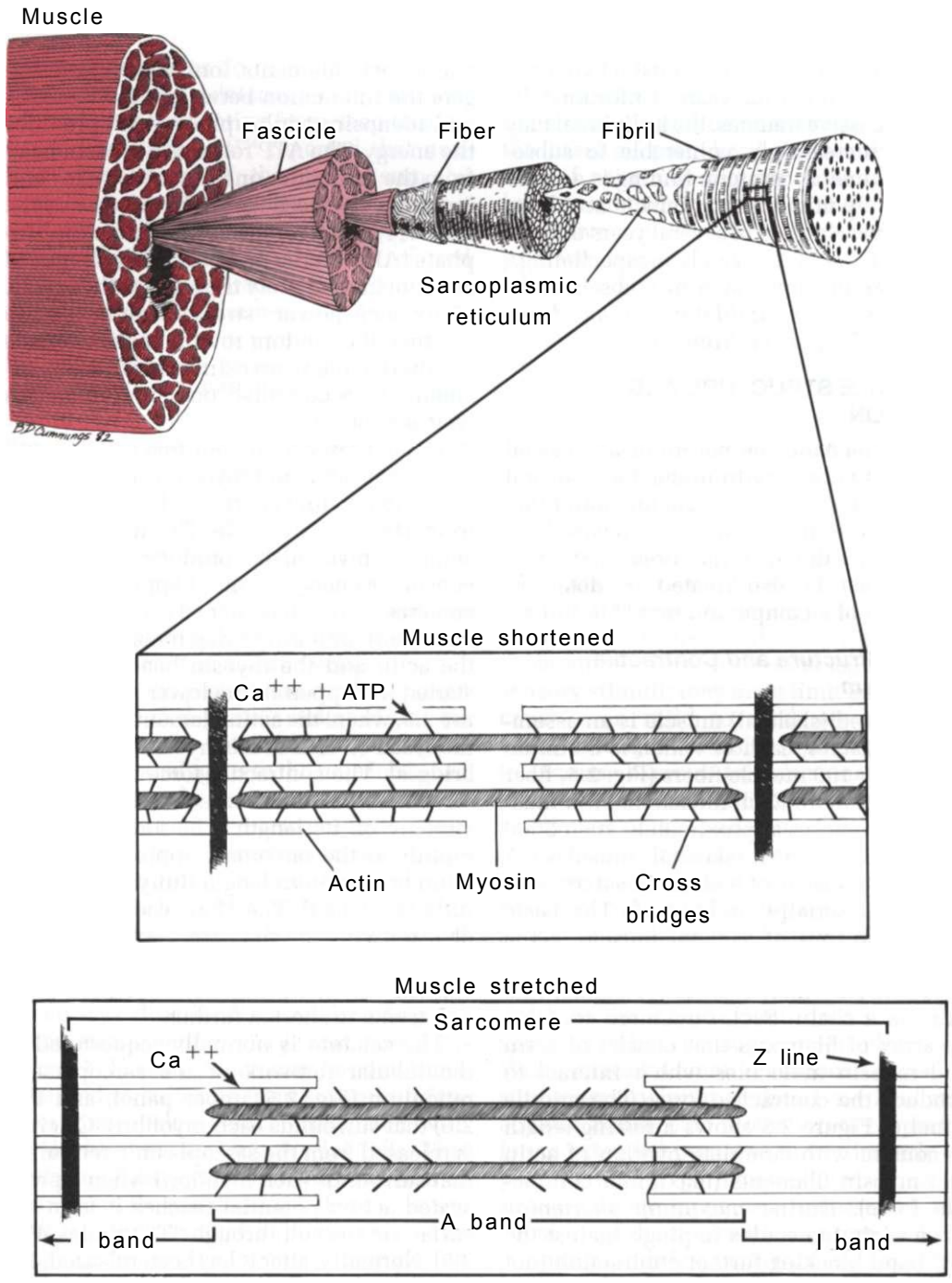


Figure 2.5. Structure and contractile mechanism of normal skeletal muscle. The muscle is a bundle of fascicles (*light red),* each of which consists of striated muscle cells or fibers *(fiber).* One fiber contains on the order of 1000 myofibrils *(fibril).* The myofibril is surrounded by a network of saclike structure, the sarcoplasmic reticulum *(sarcoplasmic reticulum). Insets:* adenosine triphosphate *(ATP)* and free calcium (Ca^{++}) activate the cross bridges of the myosin *(shaded rods)* to tug on the actin filaments *(open rods).* This pull brings the Z *lines* together and shortens the *sarcomere,* which is the source of the contractile force that shortens the muscle. The portions of the actin filaments in two sarcomeres that are adjacent to a Z *line* and are free of myosin filaments form an / *band.* The presence of myosin filaments determines the extent of the A band. The presence of only an A band in the absence of an I band indicates maximum shortening (complete overlap of filaments).

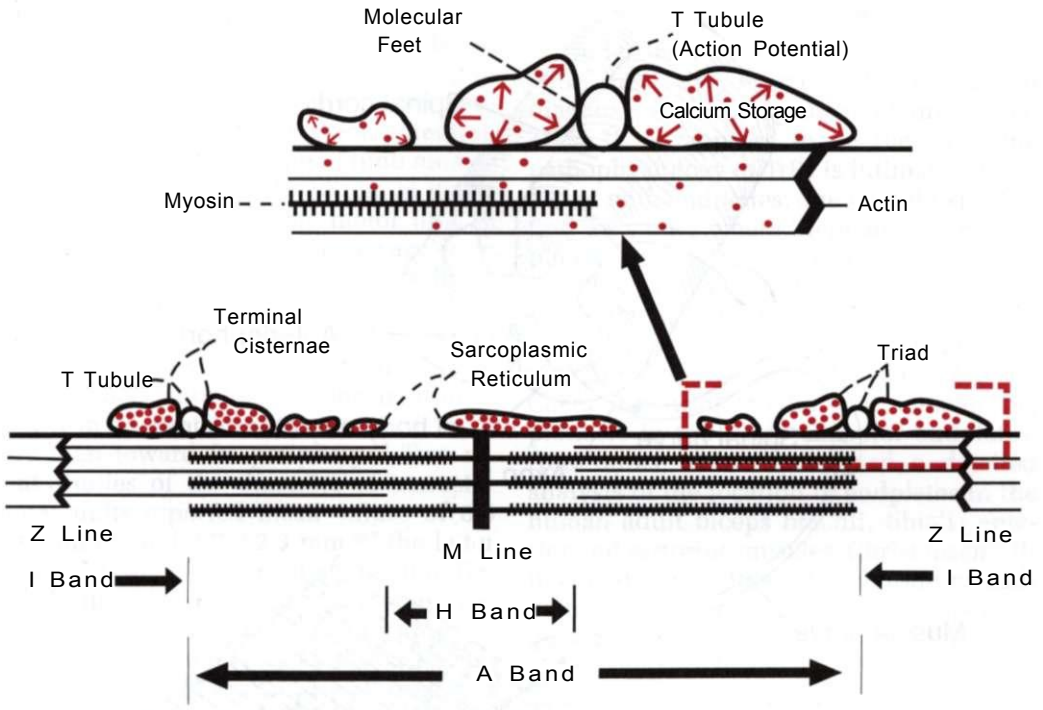


Figure 2.6. One sarcomere shown schematically in longitudinal section that also shows a triad and sarcoplasmic reticulum in cross section *(see* Fig. 2.5 for orientation). The human sarcoplasmic reticulum is a tubular network that surrounds the myofibrils in a muscle fiber of skeletal muscle. It is the reservoir for calcium, which is normally released by action potentials that are propagated along the surface of the muscle cell (sarcolemma) and along the T-tubules *(open circles),* which are invaginations of the sarcolemmal membrane. The **lower schematic** portrays one sarcomere (the functional unit of skeletal muscle) that extends from one Z-line to the next Z-line. The Z-line is where sarcomeres join to form a chain of interlaced links. The A band is the region occupied by myosin molecules *(brush-like structures)* and their projecting myosin heads. The I band includes a central Z-line where the actin molecular filaments *(thin lines)* attach to the Z-line and the I band includes as much of the actin filaments as are free of myosin cross bridges. The M line is produced by the overlapping interweaving tails of the myosin molecules, which head away from the M line in both directions.

One triad (two terminal cisternae and one T tubule seen in *red box)* is shown in more detail in the **upper schematic.** The depolarization (that is caused by the action potential propagated along the T-tubule) is transferred through molecular feet to induce calcium release *(red arrows)* from the sarcoplasmic reticulum. Calcium *(red dots)* interacts with the contractile elements to induce contractile activity, which continues until the calcium is pumped back into the sarcoplasmic reticulum or until the ATP energy supply becomes depleted.

The Motor Unit

Motor units are the final common pathway through which the central nervous system controls voluntary muscular activity. Figure 2.7 schematically illustrates a motor unit, which consists of the cell body of an a-motoneuron in the anterior horn of the spinal cord, its axon, (which passes through the spinal nerve and then through the motor nerve and enters the muscle where it branches to many muscle fibers), and the multiple motor endplates where each nerve branch terminates on one muscle fiber (cell). The motor unit includes all of those muscle fibers innervated by one motoneuron. In summary, a motor unit includes one a-motoneuron and all of the muscle fibers that it supplies. Any one muscle fiber normally receives its nerve supply from only one motor endplate and therefore only one motoneuron. The motoneuron determines the fiber type of all of

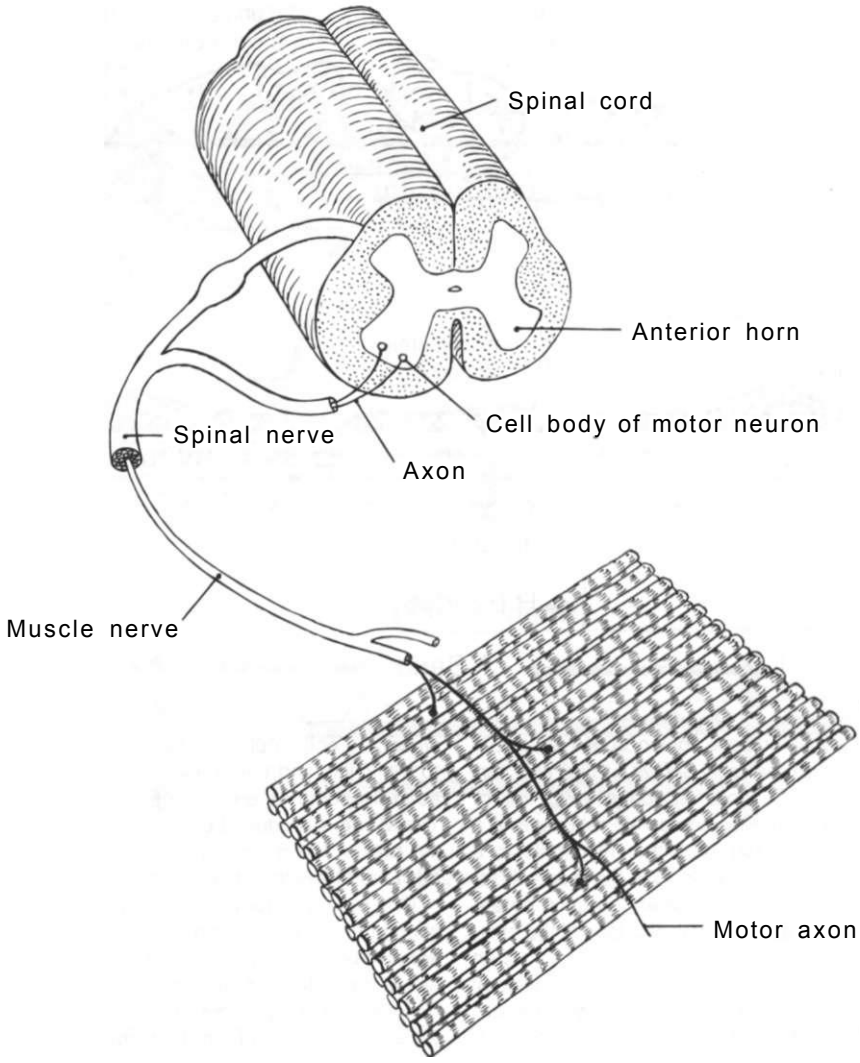


Figure 2.7. Schematic of a motor unit. The motor unit includes the cell body of a motor neuron, its axon, all its arborizations, and the muscle fibers that it supplies (usually about 500). In human skeletal muscle, each final arborization terminates at one motor endplate *(solid black circle).* Approximately 10 motor units interdigitate at any one location so that one axon sends a branch to approximately every tenth muscle fiber.

the muscle fibers that it supplies. In postural and limb muscles, one motor unit supplies between 300 and 1500 muscle fibers. The smaller the number of fibers that are controlled by individual motoneurons of a muscle (smaller motor units), the finer is the motor control in that muscle.

When the cell body of a motor neuron in the anterior horn initiates an action potential, the potential propagates along the nerve fiber (axon) through each of its arborizations to the specialized nerve terminal that helps to form the neuromuscular junction (motor endplate) on each muscle fiber. On arrival at the nerve terminal, the electrical action potential is relayed chemically across the synaptic cleft of the neuromuscular junction to the postjunctional membrane of the muscle fiber. There the message again becomes an action potential

that propagates in both directions to the ends of the muscle fiber, causing the fiber to contract. The nearly synchronous firing of all the muscle fibers innervated by one neuron produces a motor unit action potential.

One motor unit of a human limb muscle usually spans a territory 5-10 mm in diameter.[29] The diameter of one motor unit in the biceps brachii muscle can vary from 2-15 mm. This allows space for the intermingling of the fibers of approximately 15-30 motor units. Both EMG and glycogen-depletion studies show that the density of muscle fibers supplied by one neuron is greater in the center of the motor unit territory than toward its periphery.[29] Two recent studies of the diameter of masseter motor units reported mean values of 8.8 ±3.4 mm[185] and 3.7 ±2.3 mm,[267] the latter ranging between 0.4 mm and 13.1 mm. Detailed three-dimensional analysis of the distribution of fibers in five motor units of cat tibialis anterior muscles showed some marked variations in diameter throughout the length of a motor unit.[222] Thus, the size of a taut band, if it were produced by only one motor unit, could vary greatly and could have more or less sharply defined borders depending on the uniformity of muscle fiber density within that motor unit. A similar variability could result from the involvement of selected muscle fibers of several interdigitating motor units.

The Motor Endplate Zone

The motor endplate is the structure that links a terminal nerve fiber of the motoneuron to a muscle fiber. It contains the synapse where the electrical signal of the nerve fiber is converted to a chemical messenger (acetylcholine [ACh]) which in turn initiates another electrical signal in the cell membrane (sarcolemma) of the muscle fiber.

The endplate zone is the region where motor endplates innervate the fibers of the muscle. This region is now known as the motor point.[153] The motor point is identified clinically as the area where a visible or palpable muscle twitch can be elicited in response to minimal surface electrical stimulation. Originally the motor point was erroneously thought to represent the hilar region where the motor nerve enters the muscle.[4]

Location of Motor Endplates

Understanding the location of motor endplates is very important for the clinical diagnosis and management of myofascial TrPs. If, as appears to be the case, the pathophysiology of TrPs is intimately associated with endplates, one would expect to find TrPs only where there are motor endplates. Endplates in nearly all skeletal muscles are located near the middle of each fiber, midway between its attachments. This principle in human muscles was illustrated schematically (Fig. 2.8) by Coers and Woolf,[44] who were outstanding pioneers in the study of motor endplates. Aquilonius, *et al.*[5] presented a detailed analysis of the location of endplates in the human adult biceps brachii, tibialis anterior and sartorius muscles. Christensen[36] illustrated the midfiber distribution of endplates in stillborn infants in the opponens pollicis, brachioradialis, semitendinosus (two transverse bands of endplates), biceps brachii, gracilis (two distinct transverse bands), sartorius (scattered endplates), triceps brachii, gastrocnemius, tibialis anterior, opponens digiti quinti, rectus femoris, extensor digitorum brevis, cricothyroid, and deltoid muscles.

As the above illustrations show, the principle applies regardless of the fiber arrangement of the muscle. For that reason, knowledge of the arrangement of fibers in a muscle is essential to understanding the arrangement of the endplates within that muscle and, therefore, where one can expect to find TrPs. Fiber arrangements of muscles include: parallel, parallel with tendinous insertions, fusiform, fusiform with two bellies, unipennate, bipennate, multipennate, and spiral (Fig. 2.9).

Among skeletal muscles, there are at least four kinds of exceptions to the general guideline that there is one endplate zone located in the midbelly region of the muscle.

1. Several human muscles, including the rectus abdominis, the semispinalis capitis, and the semitendinosus have inscriptions dividing the muscle into serial segments each of which has its own endplate zone, as illustrated in murine muscles in Figure 2.10A, B, **C,** and **E.** In comparison, Figure 2.10D and **F** show the usual endplate arrangement.

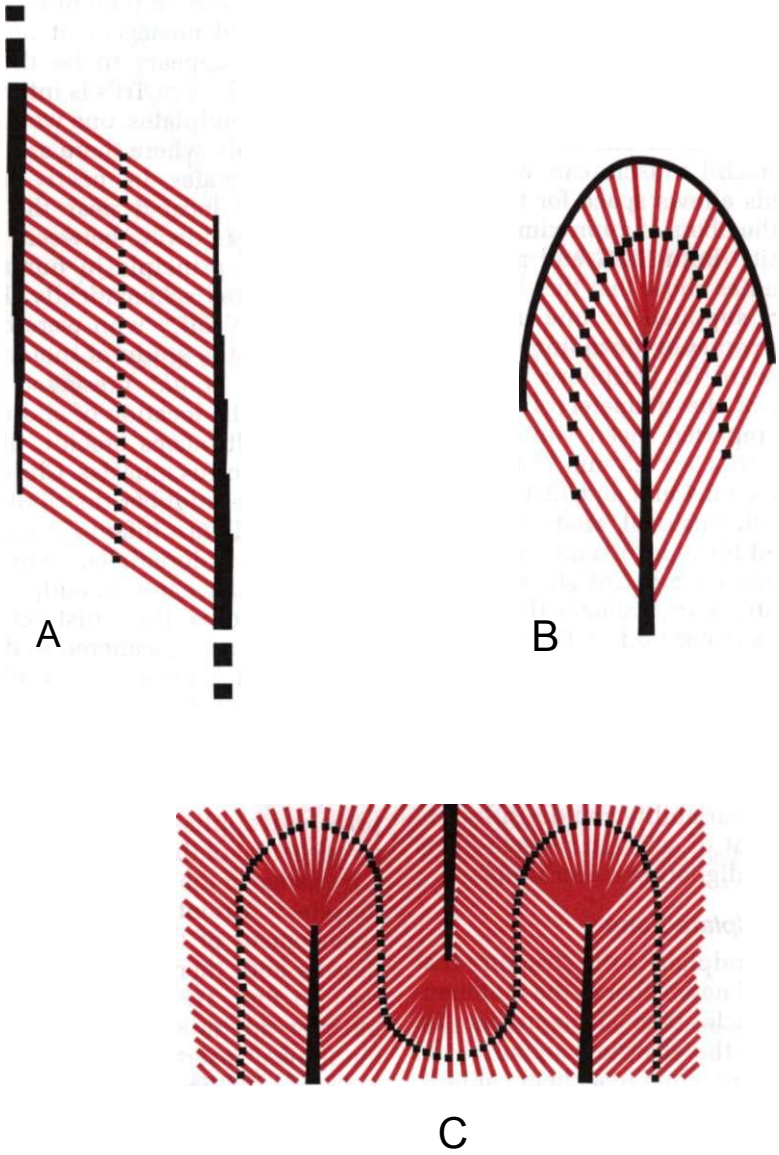
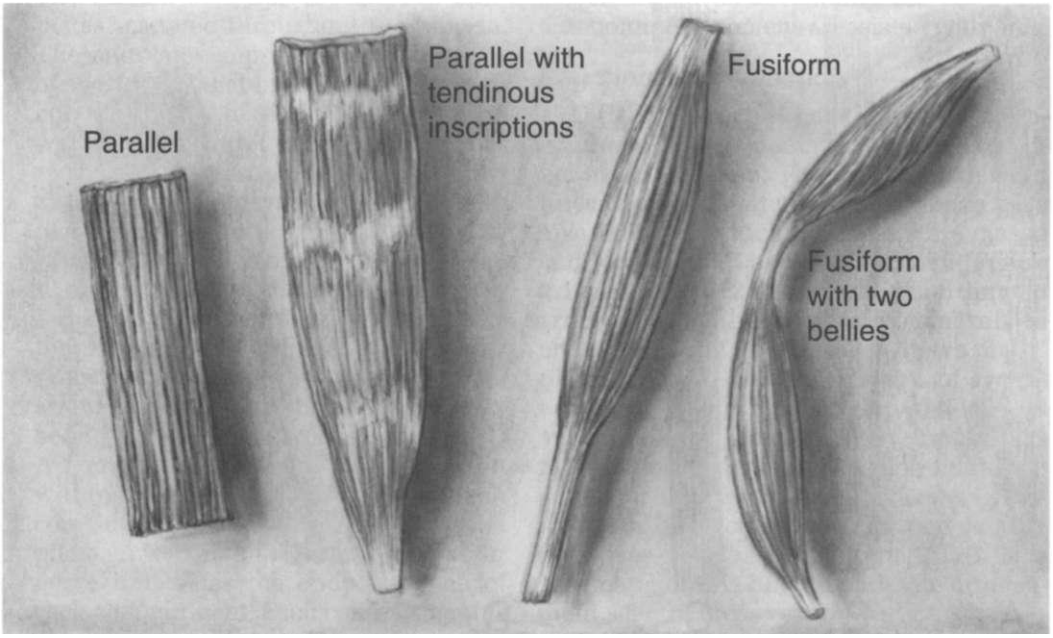


Figure 2.8. Location of endplates in human skeletal muscles of different structure. The *red lines* represent muscle fibers; the *black dots* represent motor endplates of those fibers and the *black lines* represent aponeurotic attachments. Endplates are consistently found in the midregion of each muscle fiber. **A,** linear endplates in muscle with short fibers that are arranged between parallel aponeuroses, as seen in the gastrocnemius muscle. **B,** loop arrangement of endplates in circumpennate (feather-like) arrangement of muscle fibers in the flexor carpi radialis, and palmaris longus. C, sinuous arrangement of endplates in middle deltoid muscle fibers that have a complex pennate configuration. (Adapted with permission from Coers C. Contribution a l'etude de la jonction neuromusculaire. II. Topographie zonale de l'innervation motrice terminale dans les muscles stries. *Arch Biol Paris* 64.-495-505, 1953.)[42]



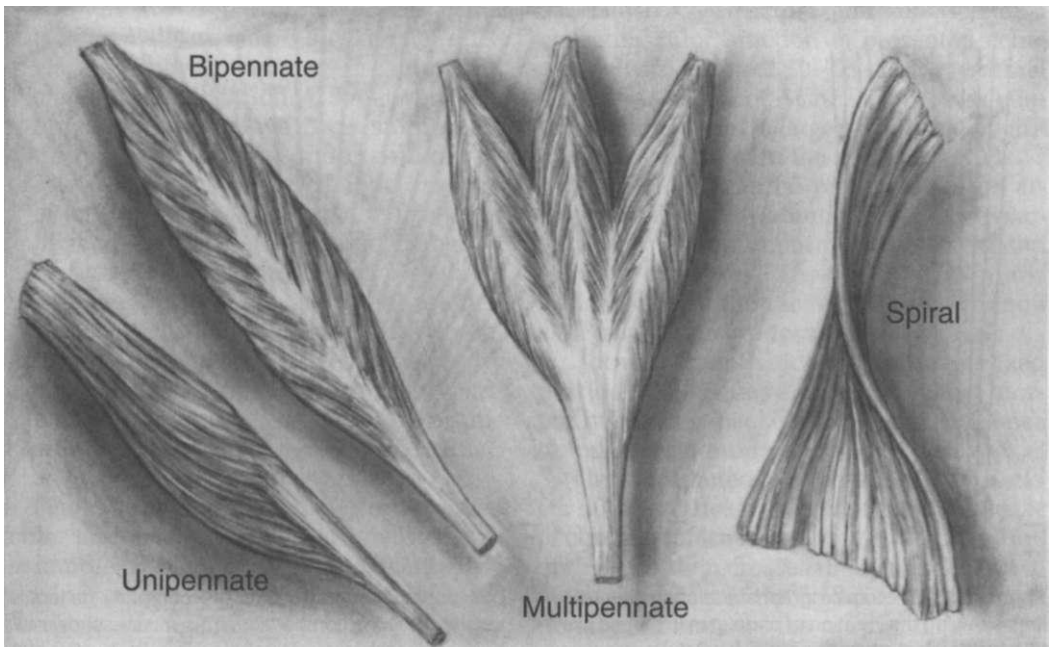


Figure 2.9. Parallel and fusiform fiber arrangements provide greater length change at the expense of force, Pennate arrangements provide more force at the expense of length change. Note that the attachments of muscle fibers in each muscle provide nearly equal length for all of its fibers. See Figure 2.8 to see how the location of motor endplates would relate to these various fiber arrangements. (Adapted with permission from Clemente CD. *Gray's Anatomy of the Human Body.* 30th ed. Philadelphia: Lea & Febiger, 1985:429.)

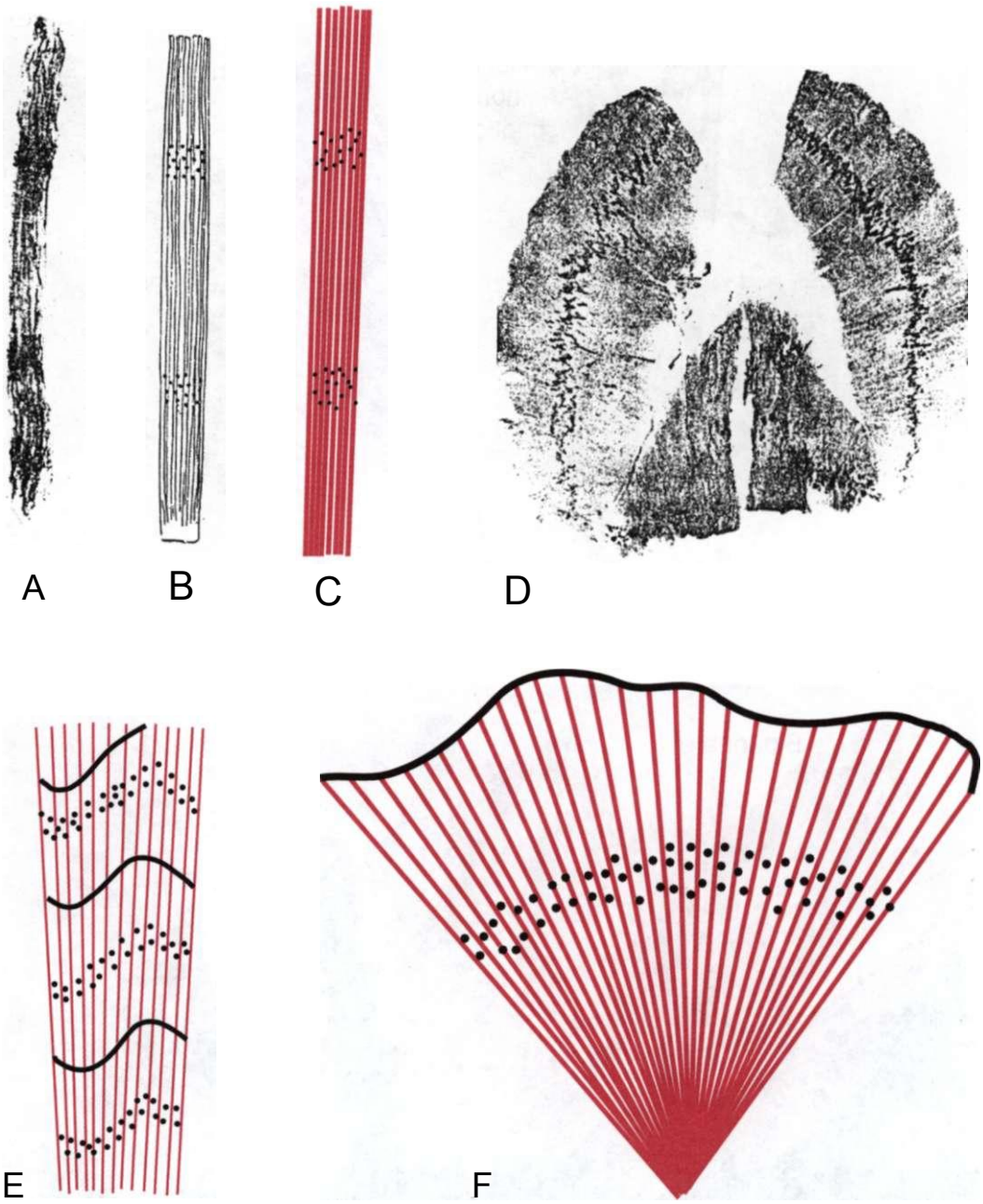


Figure 2.10. Photomicrographs and schematic drawings showing the location of endplates in mouse skeletal muscles based on the study by Schwarzacher using the cholinesterase stain technique of Koelle modified by Coers to emphasize motor endplates.[231] In the computer-generated schematics **(C, E, F),** *red lines* represent muscle fibers; *black dots* represent motor endplates of those fibers and *black lines* represent muscle fiber attachments either directly to bone or to an aponeurosis. **A** is a photomicrograph and **B** is the published schematic drawing made from it of the gracilis posterior muscle. **C** is the computer-generated version of **B** for comparison. This muscle shows two bands of endplates. **D,** photomicrograph of the diaphragm showing the endplate zone running midway between the ends of the muscle fibers. **E,** schematic of endplate arrangement in the semitendinosus muscle and **F,** in the gluteus maximus muscle. (Photomicrographs reproduced with permission from Schwarzacher VH. Zurlage der motorischen endplatten in den skeletmuskeln. *Acta Anat* 30:758-774, 1957. Schematics were derived from the same source.)

2. The human sartorius muscle has endplates scattered throughout the muscle. The endplates supply parallel bundles of short fibers that interdigitate throughout the length of the muscle with no well defined endplate zone.[44] The human gracilis is described by one author[36] as having two transverse endplate zones like the semitendinosus, but as having multiple interdigitating fibers with a scattered endplate distribution like the sartorius by others.[44] This interdigitating configuration is unusual in human skeletal muscles and the endplate arrangement in these two muscles may be highly variable among individuals.
3. A review of compartmentalization within a muscle[62] emphasized that each compartment is isolated by a fascial plane. A separate branch of the motor nerve innervates the endplate zone of each compartment. Each compartment is also functionally distinct. Examples given are the proximal and distal partitions of the extensor carpi radialis longus and the distal partitions of the flexor carpi radialis muscle. The masseter muscle also shows evidence of motor unit compartmentalization.[185] Relatively few human muscles have been studied for this feature. It may be quite common.
4. The gastrocnemius muscle is an example of the arrangement of muscle fibers that increases strength by reducing range of motion. The fibers are strongly angulated so that one individual fiber is only a small percent of the total muscle length. Consequently the endplate zone runs centrally down most of the length of each compartment of the muscle. An example of this arrangement is shown in Figure 2.8A.

Figure 2.11 schematically portrays two motor endplates and the small neurovascular bundles that cross the muscle fibers as the terminal axons supply motor endplates.[60] The linear arrangement of endplates that follows the path of such a neurovascular bundle is oriented across the direction of the muscle fibers.[544] The neurovascular bundle includes nociceptor sensory nerves and autonomic nerves that are closely associated with these blood vessels. The close proximity of these structures to motor endplates is important for understanding the pain and autonomic phenomena associated with TrPs.

Neuromuscular Junction

Different species have different topographical arrangements of the nerve terminal at an endplate. The frog has extended linear synaptic gutters. Rats and mice have a variation in which the gutters are curled and convoluted as illustrated in Figure 2.11. Figure 2.12 shows the usual human arrangement. Cholinesterase stain of an endplate (Fig. 2.12A) clearly shows multiple more or less separate groups of synaptic clefts. With sufficient separation, this arrangement might effectively function as multiple small synapses, which could account for multiple sets of spikes originating from one active locus in one muscle fiber *(see* Section D). Figure 2.12B is a schematic of this human endplate arrangement seen in cross section.

The neuromuscular junction is a synapse which, like many in the central nervous system, depends on ACh as the neurotransmitter. The basic structure and function of a neuromuscular junction is presented schematically in Figure 2.13. The nerve terminal produces packets of ACh. This process consumes energy that is largely supplied by mitochondria located in the nerve terminal.

The nerve terminal responds to the arrival of an action potential from the a-motoneuron by the opening of voltage-gated calcium channels. These channels allow ionized calcium to move from the synaptic cleft into the nerve terminal. The channels are located on both sides of the specialized portion of the nerve membrane that normally releases packets of ACh in response to ionized calcium.

The simultaneous release of many packets of ACh quickly overwhelms the barrier of cholinesterase in the synaptic cleft. Much of the ACh then crosses the synaptic cleft to reach the crests of the folds of the postjunctional membrane of the muscle fiber where the ACh receptors are located (Fig. 2.13). However, the cholinesterase soon decomposes any remaining ACh, limiting its time of action. The synapse can now respond promptly to another action potential.

The normal random release of individual packets of ACh from a nerve terminal

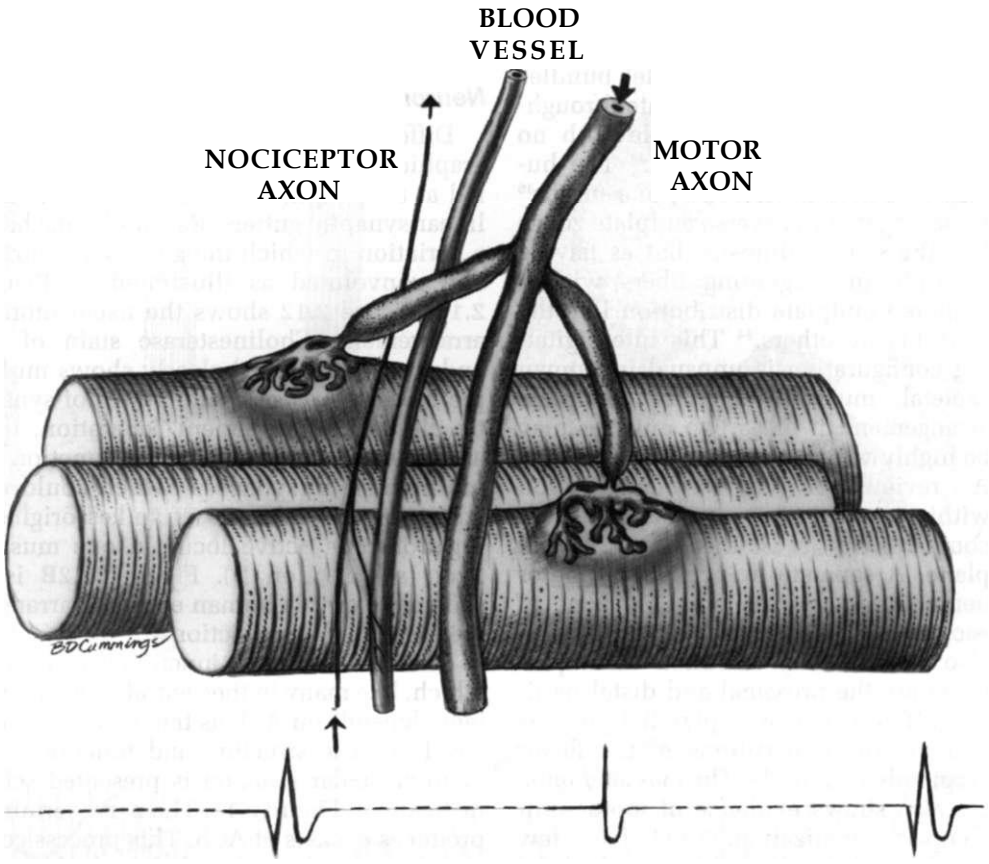


Figure 2.11. Sketch overview of two mammalian motor endplates and the neurovascular bundle associated with them. The nerve terminals of a motor axon are twisted into a compact neuromuscular junction that is imbedded into the slight elevation of the endplate region on the muscle fiber. The motor nerve fibers are accompanied by sensory nerve fibers and blood vessels. Autonomic nerves are found in close association with these small blood vessels in muscle tissue. Action potentials recorded at the endplate region of a muscle fiber show an initially negative deflection. Beyond a very short distance to either side of the endplate on the right, the action potentials of that fiber have a positive-first deflection. This is one way of localizing motor endplates electromyographically. The action potential configurations at the bottom of the figure correspond to the waveforms that would be recorded at various locations along the foreground muscle fiber. (Adapted with permission from Figure 5 of Salpeter MM. Vertebrate neuromuscular junctions: General morphology, molecular organization, and functional consequences. In: Salpeter MM, ed. ***The Vertebrate Neuromuscular Junction.*** New York: Alan R. Liss, Inc., 1987:1-54.)[225]

produces well separated individual miniature endplate potentials. These individual miniature endplate potentials are not propagated and die out quickly. On the other hand, the mass release of ACh from numerous vesicles in response to an action potential arriving at the nerve terminal depolarizes the postjunctional membrane enough for it to reach its threshold for excitation. This event initiates an action potential that is propagated by the surface membrane (sarcolemma) throughout the muscle fiber.

Muscle Pain

The current understanding of the neurophysiology of muscle pain is a subject that requires a separate book for adequate coverage.[191] The subject was summarized in 1993,[188 189] and updated.[1908240]

In brief outline, several endogenous substances are known to sensitize muscle no-

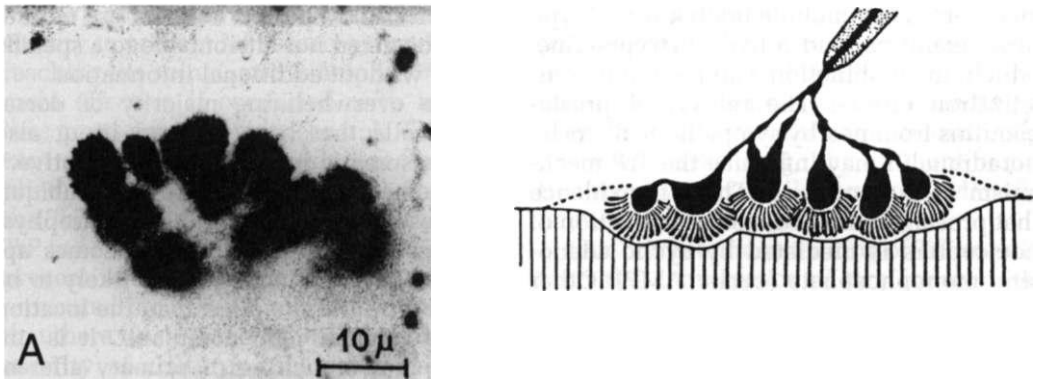


Figure 2.12. Structure of a motor endplate. Photomicrograph of the subneural apparatus and a schematic cross section of the terminal arrangement in human muscle. **A,** Photomicrograph of human endplate region, stained by a modified Koelle's method to reveal cholinesterase, shows the multiple groups of discrete synaptic clefts of the subneural apparatus. This terminal motor nerve ending of one endplate shows 11 distinct round or oval couplets. This structural form is distinctly different than the tortuous and plexiform terminals in rats and mice. (Reproduced with permission from Coers C. Structural organization of the motor nerve endings in mammalian muscle spindles and other striated muscle fibers. In: Bouman HD, Woolf AL, eds. *The Innervation of Muscle.* Baltimore: Williams & Wilkins, 1960:40-49.)[43] **B,** Schematic of cross section through the motor endplate region. This unmyelinated terminal nerve ends in six terminal expansions *(black globules).* Each terminal expansion has its own synaptic gutter and system of postsynaptic folds. The *dotted lines* represent the Schwann cell extension that attaches to the sarcolemmal membrane of the muscle cell and isolates the content of the synaptic cleft from the extracellular milieu. The *vertical parallel lines* represent the striations (Z lines) of the muscle fiber. (Reproduced with permission from Coers C Contribution a l'etude de la jonction neuromusculaire. Donnees nouvelles concernant la structure de l'arborisation terminale et de l'appareil sousneural chez l'homme. *Arch Biol Paris* 64:133-147, 1953.)[41]

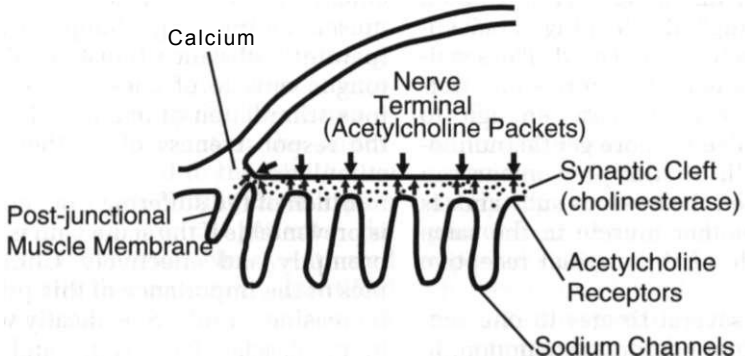


Figure 2.13. Schematic cross section of part of a neuromuscular junction, which transmits a nerve action potential across the synapse via a chemical messenger so it becomes a muscle action potential. In response to an action potential propagated down the motor nerve, the synaptic membrane of the nerve terminal opens voltage-gated calcium channels, allowing an influx of calcium from the synaptic cleft *(small red up arrows).* This calcium causes the release of many packets of acetylcholine (ACh) into the synaptic cleft *(larger down arrows).* Receptors specific for ACh depolarize the postsynaptic membrane of the muscle fiber sufficiently to open sodium channels deep in the folds of the postjunctional membrane. Sufficient depolarization of these sodium channels initiates a propagated action potential in the muscle fiber.

ciceptors. They include bradykinin, E-type prostaglandins, and 5-hydroxytryptamine, which, in combination, can potentiate sensitization effects. The release of prostaglandins from nearby sympathetic fibers by noradrenalin may influence the TrP mechanism at the endplate. There is evidence that prostaglandin-induced sensitization of nociceptors is mediated by cyclic adenosine monophosphate (cyclic AMP). Other factors known to enhance sensitization locally are increases in hydrogen ion concentration (Ph decreased to 6.1), and substance P.[188] Peripheral sensitization of nociceptors would be responsible for local tenderness to pressure and most likely also for referred pain. Which of these, or other substances, are responsible for sensitizing nociceptors in the region of active loci of TrPs is not known at this time, but this issue offers a fertile field for research investigation that may involve drugs.

Several phenomena occurring at the spinal cord level can be related to referred pain. Experiments monitoring the response of a dorsal horn cell to deep-tissue (including muscle) stimulation can establish the location and extent of the receptive field(s) of that neuron. Injection of a pain-inducing substance into the muscular receptive field of a nociceptor neuron can result in the appearance of additional receptive fields in that limb.[121] This phenomenon is attributed to the "awakening" of "sleeping" nociceptive pathways in the spinal cord. The sensitivity of the original nociceptive-only dorsal horn cells can increase enough to become responsive to more gentle, nonnociceptive stimuli. Similar phenomena can be observed when the noxious substance is injected into another muscle in the same limb but outside of the original receptive field.[121190]

Inputs from several tissues to one sensory lumbar spinal neuron are common. In a study of cats,[95] most of the 188 units studied (77%) were hyperconvergent and responded to nociceptive input from two or more deep tissues: facet joints, periosteum, ligaments, intervertebral disc, spinal dura, low back/hip/proximal leg muscles and tendons. Most of these units (93%) also had a cutaneous nociceptive site.[95] This finding corresponds to the clinical experience that low back and referred leg pain are neither well localized nor attributable to a specific tissue without additional information.

The overwhelming majority of dorsal horn cells that have visceral input also have a somatic input that is nociceptive.[33] As one becomes more aware of the ubiquitousness of referred pain, both neurophysiologically and clinically, it becomes apparent that a patient's pain is likely to be referred from a site other than the location of the original pain complaint. It is unlikely that branching of primary afferent fibers is responsible for referred pain except in rare instances.[190]

An awareness of neuroplastic changes[296] in the central nervous system is a relatively new and fundamental development with profound clinical implications. An acute nociceptive input can induce prolonged changes in the processing of nociceptive signals in the central nervous system that involves both functional and structural changes. Neurophysiological evidence of the "wind-up" of neuronal activity has recently been summarized by Yaksh and Abram.[295] More prolonged nociceptive input can induce more long-lasting changes that may *not* be reversible with time alone.

Yu and colleagues[296] have shown neuroplastic sensitization of sensory nerves that increased responsiveness to stimulation in cutaneous and in deep receptive fields of muscle by injecting a temporarily noxious (painful) substance (mustard oil) into the tongue muscle of anesthetized rats. Noxious stimulation of one muscle influenced the responsiveness of another muscle to stimulation.

Much of the suffering from chronic pain is preventable if the acute pain is controlled promptly and effectively. Clinical examples of the importance of this principle are increasing rapidly. Specifically with regard to myofascial TrPs, Hong and Simons[127] demonstrated that the length of treatment required for patients who had developed a pectoralis myofascial TrP syndrome as the result of whiplash injury was directly related to the length of time between the accident and the beginning of TrP therapy. With longer initial delay, more treatments were required and the likelihood of complete symptom relief decreased.

The use of local analgesia at the time of surgery to prevent nociceptive signals from reaching the spinal cord is helpful,[295] but is more effective if combined with meticulous post- surgical pain control. The concept of preventive analgesia has been applied successfully by blocking pain from the TrP with preinjection blocks **prior** to a TrP injection.[76,78] Katz, *et al.*[78] showed that preventing acute surgical pain, in turn, prevented progression to chronic pain and that there was a direct relation between the severity of acute postoperative pain and the severity of subsequent chronic postoperative pain.

Recent investigations show that different areas of the brain become activated in response to an experimentally induced *acute* pain as compared to *chronic neuropathic* pain.[130] Neuropathic pain shows by positron emission tomography a striking preferential activation of the right anterior cingulate cortex (Brodman area 24), regardless of the side of the painful mononeuropathy. Activation of this region of the brain is associated with emotional distress (suffering). Acute pain activates both motor and sensory portions of the cortex producing a cognitive and motor behavioral experience rather than an emotional experience. These findings emphasize the importance of the affective-motivational dimension in chronic ongoing neuropathic pain that is not involved in acute pain. Chronic pain causes suffering that is processed differently in the brain than is the experience of acute pain. These neurophysiological facts emphasize the importance to the patient and to the health care delivery system of **preventing** chronic pain and properly interpreting patients' descriptions and behavior. Newly-activated myofascial TrPs that are poorly identified and poorly managed can become a major unnecessary cause of expensive, misery-producing chronic pain.

D. NATURE OF TRIGGER POINTS

Trigger points have been difficult to understand because there has been no method of studying them electrophysiologically, and those investigating pathology were looking for characteristic histological changes distributed uniformly throughout the TrP or palpable nodule. Adding to the problem, differences in terminology often made it difficult to know whether or not different investigators were examining patients with basically the same medical condition but identifying it by different names that emphasized similar but somewhat different diagnostic aspects.

Our current understanding of TrPs results from the convergence of two independent lines of investigation, one electrodiagnostic and the other histopathological. Fitting together the lessons from each leads to an *Integrated Hypothesis* that appears to explain the nature of TrPs. It is now becoming clear that the region we are accustomed to calling a TrP or a tender nodule is a cluster of numerous microscopic loci of intense abnormality that are scattered throughout the nodule. The TrP is like a nest of hornets that contains multiple minute sources of intense trouble. The critical TrP abnormality now appears to be a neuromuscular dysfunction at the motor endplate of an extrafusal skeletal muscle fiber, in which case myofascial pain caused by TrPs would be a neuromuscular disease. This section reviews the research data that provides the basis for this concept.

Electrodiagnostic Characteristics of Trigger Points

The basis for the electrodiagnostic approach to the study of TrPs was anticipated by Weeks and Travell in 1957[288] when they reported and illustrated that TrPs in the resting trapezius muscle exhibited a series of high frequency spike-shaped discharges while at the same time adjacent sites in this muscle were electrically silent. Unfortunately, this observation was not effectively pursued. In 1993, Hubbard and Berkoff[133] reported similar electrical activity as being characteristic of myofascial TrPs. Their paper, like the previous 1957 report, called attention only to high-amplitude (>100 u.V) spike potentials as being characteristic of TrPs. Hubbard and Berkoff hypothesized that the source of the electrical activity was abnormal muscle spindles and rejected the possibility that the potentials might be coming from extrafusal motor endplates.

When Simons, Hong, and Simons started to investigate the electrical activity in TrPs described by Hubbard and Berkoff,[133] they

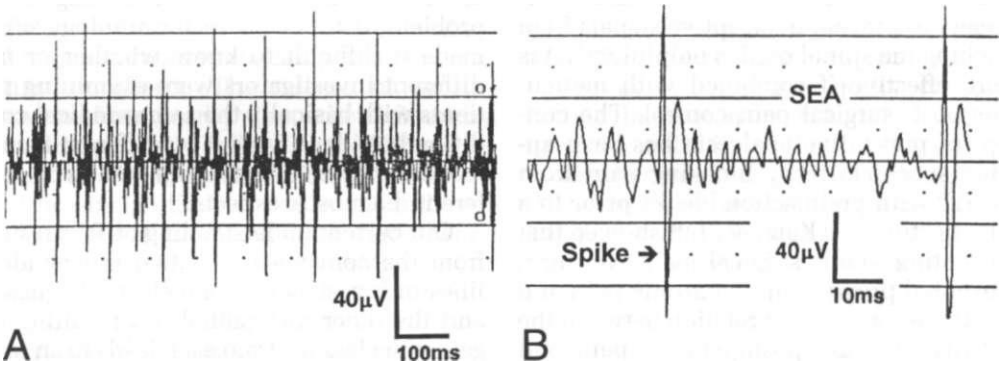


Figure 2.14. Typical recording of the spontaneous electrical activity (SEA) and spikes recorded from an active locus of a trigger point at two different sweep speeds. **A,** recording at the same slow sweep speed of 100 msec/div used by Hubbard and Berkoff[133] to report this electrical activity. Only spikes of unknown initial polarity are identifiable. **B,** a similar amplification but a ten-times higher sweep speed of 10 msec per division that was used in subsequent studies by others[248,250] who also have observed the low amplitude noise component as well as the polarity of initial deflection of the spikes from active loci. This additional information is of critical importance for understanding the source and nature of these potentials.

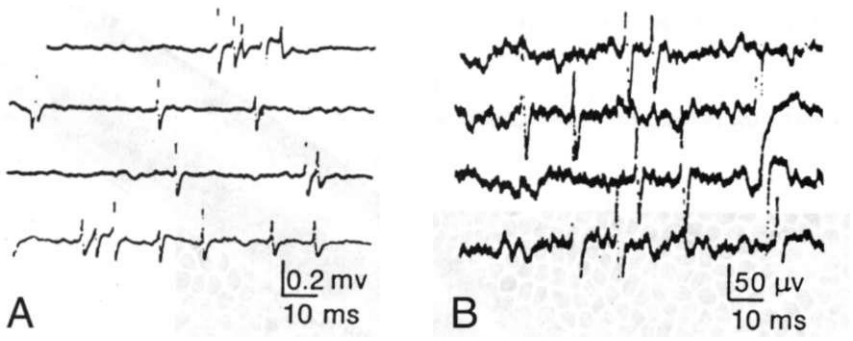
employed a five-fold higher amplification and ten- fold increase in sweep speed for their recordings. It was immediately apparent that there were two significant components to the electrical activity. In addition to the intermittent and variable high-amplitude spike potentials, there was a consistently present, lower amplitude (maximum of about 60 u.V) noise-like component.

Figure 2.14A shows the electrical activity that Simons, Hong, and Simons[248] recorded at the same slow speed that Hubbard and Berkoff reported. Only spikes are distinguishable in this record and the polarity of their onset is not identifiable. Figure 2.14B presents similar electrical activity recorded at the same amplification but with a ten-fold increase in sweep speed. In this record, the noise-like low-amplitude potentials are clearly apparent and distinguishable from the spike activity, and the negative polarity of the initial deflection of each spike potential is clearly evident.

To deal with the potential terminology confusion inherent in this situation, the three investigators adopted the noncommittal term **spontaneous electrical activity** (SEA) to identify this noise-like component.[248] Since spikes only, SEA only, or both components might appear from one of these minute needle sites, the neutral term **active locus** was adopted to identify such a site of electrical activity. These three authors used the same kind of needle and the same slow insertion technique reported by Hubbard and Berkoff.

In time, it became increasingly apparent to the three investigators with the help of S. Mense, that the potentials found at the active loci of TrPs corresponded completely to the potentials that are recognized by electromyographers as **normal** motor endplate potentials. However, they did not conform to the miniature endplate potentials described by physiologists. Electromyographers identify the low-amplitude component (like SEA of TrPs) as endplate noise and the high-amplitude spike component as endplate spikes.[153] The similarity can be seen by comparing Figures 2**.14B** and 2**.15B.** The endplate potentials in Figure 2**.15** are presented as normal endplate activity in a current electrodiagnostic textbook.[153] This interpretation is based on the study reported by Wiederholt.[291] At this point, it became necessary for the three investigators to resolve what appeared to be the incompatible "facts" that the SEA and spikes characteristic of active loci in symptom-producing TrPs were generally considered to be normal endplate activity.

Spontaneous Electrical Activity. To reliably identify SEA of TrPs electromyo-



cal potentials identified as normal endplate activity of the tibialis anterior muscle and published in a current textbook of electrodiagnosis.[153] Recordings are at the higher sweep speed of 10 ms per division. **A,** endplate spikes recorded at low amplification; the relatively low-amplitude noise-like component is barely apparent endplate activity showing both the continuous endplate noise and occasional spikes. (Reprinted with permission from Kimura J. ***Electrodiagnosis in Diseases of Nerve and Muscle,*** Vol. 2. Philadelphia: F.A. Davis, 1989.)

graphically, it is necessary to use a relatively high amplification (20 **U.** V/division) and fast sweep speed (10 msec/division). If the needle examination is conducted using the thrust technique normally employed by electromyographers, the examiner is likely to pass an active locus without recognizing it or to elicit a local twitch response instead of finding SEA. A very gentle insertion technique is required that includes back and forth rotation of the needle between the thumb and finger as it is slowly advanced. On these higher-amplification records, the peak amplitudes of spikes are often off scale but their presence is unmistakable and the polarity of their initial deflection from the baseline is observable in detail.

The SEA presented here was recorded with the commonly used, disposable, teflon-coated, monopolar EMG needle. The exposed tip of this needle is relatively large compared to the diameter of a muscle fiber or of the endplate region of a muscle fiber. Figure 2.16 shows the relative size of the needle and muscle fibers. The exposed tip of a needle was approximately 0.45 mm (450 u,m) long. The mean diameter of normal muscle fibers varies with fiber type ranging from 41 to 59 u,m.[57] Therefore, either side of the exposed tip would contact approximately 9 muscle fibers of 50 lxm diameter.

One should not expect to record normal miniature endplate potentials with such a large needle. However, the SEA of an active locus is a different matter. Individual miniature endplate potentials have been very difficult to detect extracellularly using a microelectrode[67] because of the minute source and because their potentials are propagated such a short distance along the outer surface of the postjunctional membrane, and because the potentials reappear so infrequently at any one location.

On the other hand, if excessive ACh release induces greatly increased and continuous electrical activity that produces a contraction knot *(see* p. 69, Fig. 2.24), the resultant higher-voltage endplate potentials would be more readily detectable with the relatively large needle electrode, and much of the endplate region would likely be active continuously (not active intermittently at a few isolated minute locations). The double-size, contraction knot region would increase the target size that could now be 100 u,m or more in diameter.[214, 253]

Evidence indicates that the SEA may be present spontaneously regardless of the presence of the EMG needle. Since the needle is carefully advanced slowly and smoothly, it usually evokes very few, small insertion potentials. As the needle slowly

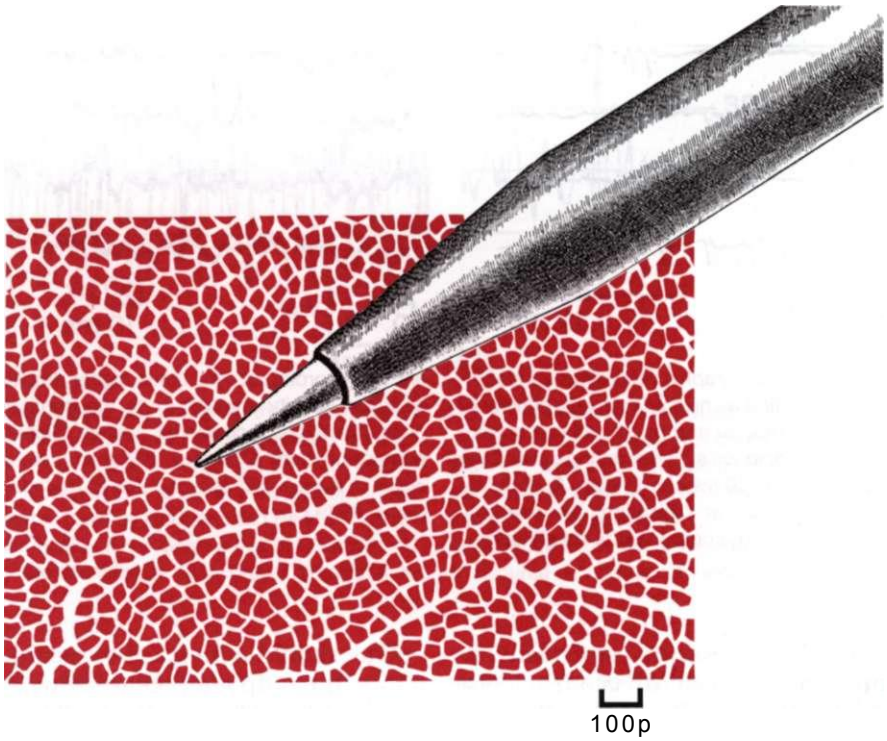


Figure 2.16. Schematic that shows the relative size of the exposed tip of a standard teflon-coated electromyographic needle as compared to normal muscle fibers. Muscle fibers are generally about 50 u,m in diameter. The exposed needle tip (without the teflon coating) can extend about 450 u,m and therefore could be in contact with approximately 18 muscle fibers, counting both sides of the needle.

advances through the TrP region in this electrically quiet background, the examiner occasionally hears a distant rumble of noise that swells to full SEA dimensions as the needle continues to advance. This "acquisition" of SEA at an active locus in a TrP is illustrated in Figure **2.17A** and presents a record of the needle approaching the immediate vicinity of the SEA. The transition represents a fraction of a millimeter of needle displacement. Sometimes the SEA can be increased or decreased by simply applying gentle side pressure to the hub of the EMG needle. The distance of the needle from the discrete source of the electrical activity can be that critical.

Early in the study of the electrical activity found at active loci,[248,250] the investigators needed to test whether or not active loci were located at motor endplates. Figure **2.17B** shows a recording of a voluntary motor unit action potential and illustrates one strong indication that SEA originated at a motor endplate. Buchthal, *et al.*[27] showed that a biphasic motor unit potential with an initial negative deflection followed by a rapid rate of rise indicates that the recording needle is close to (within 1 mm of] the origin of the action potential (a motor endplate). The trace in the lower box of Figure 2.17B shows the regular firing pattern of one voluntarily recruited motor unit. The upper trace in the upper box of Figure 2.17B presents in detail the action potential that is shown between the + marks in the lower trace. It has the intitial negative deflection followed by rapid rise to peak negative voltage and the biphasic waveform characteristic of a motor unit action potential recorded at its origin, the motor endplate.[27] This potential was recorded at the site of a trigger point active locus. The lower trace of the upper box in Figure 2.17B was recorded from an adjacent control site about 1 cm away. Its waveform (a triphasic potential without a sharp spike) shows that the recording needle was not located at the origin of that electrical

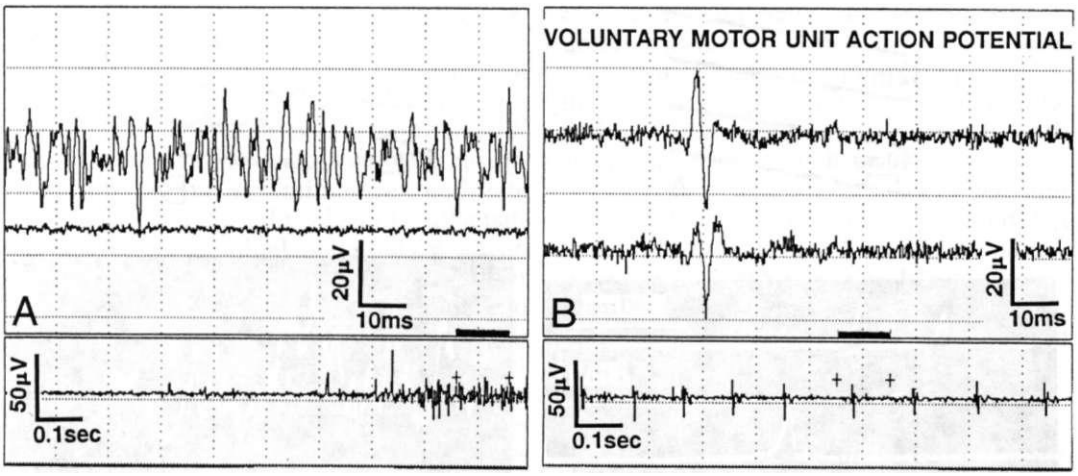


Figure 2.17. Two examples of electrical activity recorded at active loci in trigger points. **A,** The slow sweep-speed, 1 second recording in the *lower box* obtained as the needle approached an active locus shows the quiet baseline becoming increasingly active due to endplate noise (spontaneous electrical activity) at the same time that the investigators heard a corresponding development of a noise-like (seashell) sound. The last 0.1 sec of that record from the search needle is displayed at increased amplification and ten times the sweep speed in the *upper trace* of the *upper box* in **A.** It shows typical spontaneous electrical activity of approximately 20 uV amplitude. The *lower trace* of the upper box displays the quiet baseline present throughout this record that was simultaneously recorded from a control needle at a site near to, but outside of, the trigger point.

B, The 1 second recording in the *lower box* of **B** displays repetitive discharges of 1 motor unit recorded from an endplate location found by the appearance of spontaneous electrical activity of a trigger point active locus. The motor unit activity is in response to the subject performing (on request) a minimal voluntary contraction of the muscle. The *upper trace* in the *upper box* of **B** displays in detail (at 2.5 times the amplitude and 10 times the sweep speed) the sixth action potential in the lower box. The abrupt initially negative diphasic spike of this upper trace indicates that the recorded potential originated within a few micrometers of the search needle, which means it had to be that close to the motor endplate. The *lower trace* of the *upper box* of **B** was recorded from an adjacent control site in the endplate zone but out of the trigger point and shows a triphasic, rounded, initially negative deflection of longer duration from different muscle fibers of the same motor unit. This potential did not originate at a motor endplate. That both potentials came from the same motor unit was confirmed by a constant time relationship in all nine repetitions of them throughout the 1 second record. This experiment illustrates how one can establish independently the presence of an endplate in the absence or presence of spontaneous electrical activity. The finding supports the concept that the spontaneous electrical activity observed in a trigger point active locus arises in the immediate vicinity of, or from, a motor endplate.

activity. However, it is a simultaneous recording from another fiber (or fibers) of the same motor unit. The potential in the upper trace was recorded from the site of an active locus originating within a millimeter or less from a motor endplate. This kind of single-fiber potential, voluntarily recruited and originating at an active locus, was a consistent finding. Frequently, when the subjects initiated a gentle voluntary contraction, they first recruited only the same motor unit that included the muscle fiber that was exhibiting SEA. This indication of selective recruitment needs to be studied quantitatively in a controlled research experiment. A confirmatory result would indicate that the motor neurons with dysfunctional endplates are more excitable than others.

The issue of whether the endplate potentials now recognized by electromyographers as endplate noise arise from normal or abnormal endplates is critical and questions conventional belief. Figure 2.18 illustrates the difference between normal miniature endplate potentials (Fig. 2.18A and C) and abnormal endplate noise (Fig. 2.18B and D) which corresponds to the SEA of active loci in TrPs.

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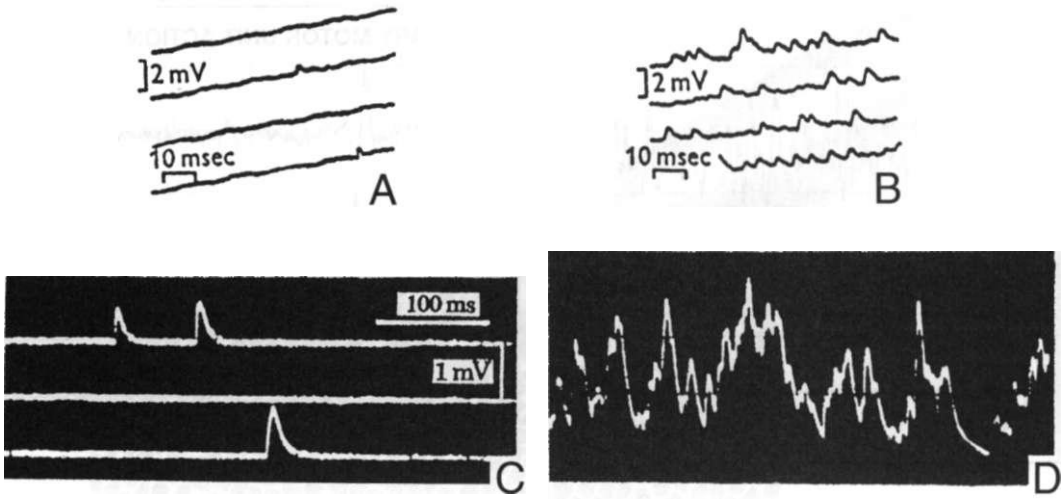


Figure 2.18. Physiological studies of the endplate potentials characteristic of normal endplates **(A** and **C)** and of dysfunctional endplates **(B** and **D)** under resting conditions. **A** and **B,** are early intracellular recordings published in 1956.[171] **A,** two **normal** (isolated, monophasic, low amplitude) miniature endplate potentials. **B,** a continuous series of overlapping, superimposed, noise-like, higher-amplitude **abnormal** potentials produced by almost any mechanical disturbance of the endplate region. (*A* and **B** reproduced with permission from Liley AW. An investigation of spontaneous activity at the neuromuscular junction of the rat. / ***Physiol*** 332:650-666, 1956)

C and **D** are slower-speed physiological research recordings made in 1974 with greater amplification.[137] **C,** normal, infrequent, individual, monophasic miniature endplate potentials. **D,** response to exposure of the endplate region to incompatible blood serum. This continuous noise-like (abnormal) discharge appears the same as the so-called normal endplate noise component of motor endplate potentials as usually described by electromyographers and the same as the spontaneous electrical activity observed in trigger points. This noise-like electrical discharge was caused by a nearly 1000-fold increase in the rate of acetylcholine release from the resting nerve terminal. **(C** and **D** reproduced with permission from Ito Y, Miledi R, Vincent A. Transmitter release induced by a "factor" in rabbit serum. ***Proc R Soc Lond B*** 387:235-241, 1974.)

Wiederholt in 1970,[291] electromyographers have accepted his apparently mistaken conclusion that potentials similar to what we now identify as SEA represent *normal* miniature endplate potentials. Electromyographers commonly identify the low-amplitude potentials as "seashell" noise.[153] Wiederholt was correct in concluding that the low-amplitude potentials arose from endplates, and illustrated one recording of a few discrete monophasic potentials having the configuration of normal miniature endplate potentials as described by physiologists. However, the continuous noise-like endplate potentials that he also illustrated and that we observe from active loci have an entirely different configuration and have an abnormal origin.

Three studies by physiologists, two of which appeared following Wiederholt's study, indicate that the SEA (endplate noise) arises from a functionally disturbed endplate. In 1956, Liley[171] observed that even a relatively minor mechanical disturbance applied to the endplate region could greatly increase the frequency of the postjunctional membrane potentials, from a normal maximum of 118/sec to as high as 1,000/sec (an increase of one order of magnitude). Minor mechanical stimuli (minor traumas) that produced this effect included pulling gently on the motor nerve, vibration of the endplate region, and visible dimpling of the surface of the muscle fiber by touching it with an electrode. These mechanical stimuli converted the discharge pattern from normal to abnormal, and once converted, the pattern remained abnormal (Fig. 2.18B).[171]

Two decades later, studies by Miledi and coworkers identified excessive release of acetylcholine packets as the cause of the increased electrical activity. These studies

were published several years after Wiederholt's seminal paper.[291] In 1971 Heuser and Miledi[120] demonstrated that exposure of the endplate region to lanthanum ions produced a 10,000-fold (four orders of magnitude) increase in the release of ACh resulting in so many miniature endplate potentials that it produced a noise-like pattern where individual potentials were no longer discernable. In a subsequent study,[137] exposure of the endplate region to a foreign serum produced a similar result that is illustrated in Figure 2.18D. If a similarly disturbed nerve terminal extends the length of a TrP contraction knot *[see* page 69), then the entire postjunctional membrane covered by the nerve terminal could be expected to evidence the endplate noise (SEA). The discharge of ACh into the synaptic cleft is illustrated schematically in Figure 2.13.

Recently, Ertekin, *et al.*[m] reported a marked increase in the number of miniature endplate potentials during an attack of hypokalemic periodic paralysis. This indicates that low serum potassium can also lead to abnormally increased (but much less severe and also reversible) release of ACh under resting conditions.

This "acetylcholine noise," as Miledi and associates called it in their papers, looks remarkably like the potentials produced by Liley,[171] the endplate noise of electromyographers, and the SEA found in TrPs. Their findings suggest that the SEA which identifies active loci in TrPs is produced by grossly increased release of ACh due to a serious disturbance of normal endplate function and that the endplate noise identified by electromyographers is the signature of a dysfunctional endplate. Endplate dysfunction can be caused by a number of conditions.

Investigators in one recent study concluded that there were no abnormal EMG findings in TrPs.[59] The examiners apparently used the standard clinical EMG insertion technique, which is less likely than a slow gentle needle movement to reveal the SEA of TrPs. The relatively low gain of 50 uV/division that was reported by the authors might have revealed the SEA of active loci, but the investigators would have needed to be looking for such a low-amplitude phenomenon. They made no mention of seeing endplate noise and spikes. Even if they had identified SEA, they would have had no reason to mention it, because the authors might have considered it to be normal endplate potentials that are to be expected in the endplate zone and not worthy of mention. Previous investigators had been similarly misled.

Based on his clinical experience and early studies of SEA, Hong[122] proposed that the clinically identified TrP consists of multiple discrete sensitive spots. It now appears that those sensitive spots are abnormal endplates evidencing SEA and are scattered among uninvolved normal endplates. This configuration based purely on electrophysiological evidence, is illustrated schematically as a cross section of the muscle fibers of a TrP in Figure 2.19 (also see Fig. 2.21).

Subsequent reports[19,37,248250] concluded that the electrical activity which is characteristically found in TrPs is the same as the motor endplate potentials recognized as normal by electromyographers.[153,291] The dysfunction seen in TrPs is NOT normal.

Spikes. It is now recognized by electromyographers that spikes which originate in the endplate region are action potentials of the skeletal muscle fiber supplied by that endplate.[153] To confirm this concept and to eliminate the possibility that SEA originates in intrafusal fibers of a dysfunctional muscle spindle, the taut band was monitored as far as 2.6 cm from the endplate for the same action potentials that originated at the endplate as spikes. The same potentials were observed at both locations.[251] These distant potentials must have been propagated by extrafusal rather than intrafusal fibers since that distance was more than twice the total length of an intrafusal muscle fiber.[132]

Contrary to the experience with SEA during needle exploration of TrPs, spikes were not recognized or anticipated at a distance, but appeared suddenly, often simultaneously with SEA. Since spikes are often 10 times the voltage of SEA, when they occur with SEA and the SEA was apparent from a distance, the spikes should have been equally apparent when the needle was more than three times (square root of 10) as far from the source of the voltage. Repeatedly, very light side pressure on the hub of the EMG needle terminated the spike potentials, while release of the pressure or added pressure in the other direction restored them. These observations left

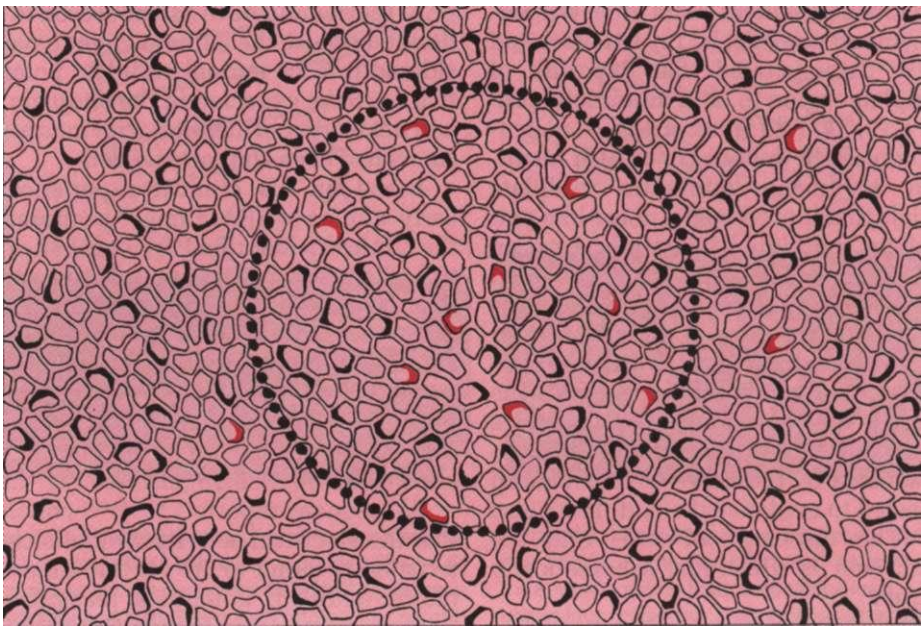


Figure 2.19. Schematic drawing of a cross sectional view through a trigger point *{dotted circle).* This schematic gives an indication of the relative frequency and distribution of active loci. It identifies muscle fibers that did not have endplates included in this section *(clear forms),* fibers with normal endplates *(forms with black crescents),* and fibers with active loci showing spontaneous electrical activity *(forms with red crescents).* The locations and frequency of normal endplates *(black crescents* bordering muscle fibers) were identified by initially negative motor unit potentials produced by a minimal voluntary contraction. See text for more explanation. Drawing based on published data.[242,248,249]

the impression that the presence or absence of spikes in only moderately active (irritable) TrPs depends significantly on the mechanical disturbance (stimulus) introduced by the needle at active loci of the TrP.[251]

When numerous spikes were present, it was not uncommon to see three or four different trains of spikes each of which had its own waveform characteristics and repetition rate. This observation suggested three or four different sites of origin within one endplate or, less likely, individual sites of origin from a cluster of involved endplates. If multiple trains of spikes originate from one muscle fiber, the multiple pockets of synaptic folds illustrated in Figures 2.12A and **B** may account for this phenomenon, provided that a train of spike potentials originated independently from the individual synaptic pockets. If the multiple trains of spikes originate in a cluster of endplates, each source would be propagated in a different but nearby muscle fiber. Determination of which mechanism is operating is an important issue that needs to be resolved by research experiments.

Available data indicate that spikes occur when a sufficient number of ACh packets are released to depolarize the postjunctional membrane to the threshold for excitation of the Na^+ channel receptors located in the depths of the synaptic folds (Fig. 2.13). Opening of these channels then initiates a propagated action potential in that muscle fiber. The mechanical pressure exerted by the needle or related mechanical disturbances apparently facilitates ACh release sufficiently to produce spikes in moderately dysfunctional endplates. Severely dysfunctional endplates of very active TrPs produce spikes spontaneously without stimulation. This clinical impression needs to be clarified by carefully designed experiments.

One must be aware of the danger of assuming that spikes alone observed in a TrP originate at an active locus when no SEA is

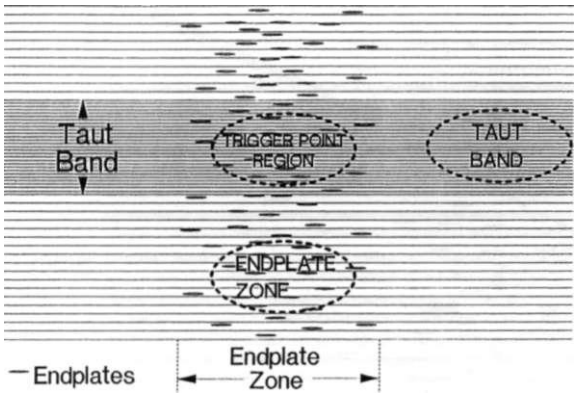


Figure 2.20. Schematic showing the three locations that were explored for active loci. One was a trigger point site selected as a clinically identified trigger point in a taut band. Another was an endplate zone site that was in the independently and electrically identified endplate zone, but was outside of any clinically identifiable trigger points. The third was a taut band site that was beyond the endplate zone and also not at a trigger point. All of the trigger points were found to be located in the endplate zone. The distribution of endplates ***(thin ovals)*** determines the extent of the endplate zone. The taut band was identified by palpation.

identified. We define an active locus only as a TrP site where SEA occurs, or where SEA occurs with spikes. It can be difficult to distinguish spikes originating at a dysfunctional endplate from a series of motor unit action potentials originating at the same endplate.

Distribution of Active Loci in a Muscle. A recent study[249] examined the location of active loci in different parts of a muscle with a TrP. The trigger point was always found to be located within the endplate zone, the boundaries of which had been determined independently. This study examined three test sites (Fig. 2.20) for active loci: in the TrP, in the endplate zone outside of a TrP, and in the taut band associated with that TrP but outside the TrP and outside of the endplate zone. A fourth location (control) was monitored in the same muscle, but outside each of the three test sites. Each of the three sites was explored systematically (Fig. 2.21) by inserting the needle sequentially into three divergent tracks, stopping eight times in each track. A recording was made whenever observing SEA alone, spikes alone, SEA with spikes, a local twitch response, and also whenever the needle had advanced approximately 1.5 mm and no activity had been located. After each advance very gentle side pressure was applied to the hub of the teflon monopolar EMG needle to see if activity appeared or changed. Needle advancement was very slow with gentle rotation of the needle back and forth to facilitate its smooth entry through the muscle tissue.

Using the presence of SEA with or without spikes as the criterion of an active locus, 11 muscles (a total of 264 needle advances) were examined (Table 2.8). The study showed active loci to be four times more common in TrPs than in the endplate zone outside of a TrP (35:9).[252] No active loci were observed in the taut band outside of the endplate zone. Clearly, the SEA (noise) type of endplate electrical activity is significantly related to myofascial TrPs. This same SEA was significantly related to trigger spots of rabbits (similar to human TrPs) as compared to adjacent nontaut band sites.[248] However, it is also clear that the isolated observation of SEA alone does not assure one that the needle is located in a clinically identifiable TrP. It may represent a site of mechanical stress on the synaptic connection, or an immune system reaction. It might also be too small a group of active loci to be clinically detectable.

The question arose, "If the SEA and spike potentials that we are observing arise from dysfunctional endplates, then why don't we also see the normal configuration of individual miniature endplate potentials observed by physiologists and occasionally by electromyographers?"[28, 63, 291] Those normal miniature endplate potentials that have been observed were recorded using coaxial needle electrodes, which characteristically have a smaller exposed surface (0.03 mm^2)[28] compared to the tip of a monopolar needle (0.08 mm^2). The coaxial configuration also makes the needle more directional in sensitivity. Both of these factors could be important consider-

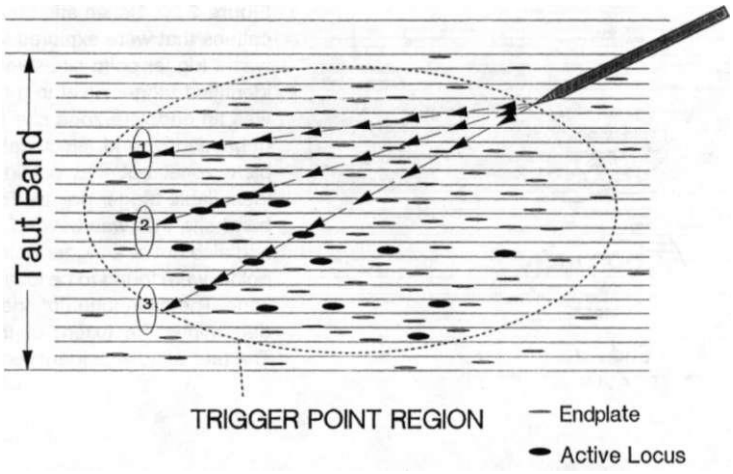


Figure 2.21. Schematic of search pattern at one experimental site. The large ***dotted oval*** represents the region of the clinically identified trigger point. The ***thin filled ovals*** represent active loci. The ***thin open ovals*** represent normal endplate locations that show no spontaneous electrical activity. The electromyographic needle was very slowly advanced eight times in each of three diverging tracks (labeled *1, 2,* and **3**). Each needle advance was approximately 1.5 mm.

Table 2.8. ***Prevalence of Spontaneous Electrical Activity (SEA) with or without Spikes at 3 Sites, Based on 264 Needle Advances at Each Site***

	Trigger point Site	*Endplate Zone*	*Taut Band*
SEA Only	21	7*	
SEA with Spikes	14		0**
SEA Present (with or without spikes)	35	9ns	0ns

***P* values compared to TrP site: * = 0.024; ** < 0.005; *** < 0.001; ns > 0.05**

ing the minute area of extracellular endplate membrane from which a normal endplate potential can be recorded.[67] The first two reports[28-63] illustrated both the endplate noise pattern and the lower-amplitude individual miniature endplate pattern, which is what would be expected if some recordings came from dysfunctional endplates and others from normal endplates. Figure 2.16 illustrates the relative size of a monopolar teflon-coated EMG needle and the diameter of a muscle fiber, which is also the approximate size of the endplate that surrounds it.

In studies of active loci,[249-250-252] it became important to confirm the presence of normal endplates in addition to the apparently abnormal ones that were generating SEA at the TrP. One can confirm the presence of a functional motor endplate by the presence of diphasic motor unit action potentials that have a sharp initial negative spike. In accordance with the volume conduction theory[58] and as observed by Buchthal, *et al.*,[27] this waveform occurs only when the potentials originate in the region of the needle tip. Figure 2.11 illustrates how the waveform changes when it is recorded at its endplate point of origin and after it has propagated a short distance in either direction along the muscle fiber. Figure 2.17B illustrates the differences in waveform when action potentials of the same motor unit are recorded simultaneously at the origin of propagation at the endplate of one muscle fiber (Fig 2.17B)

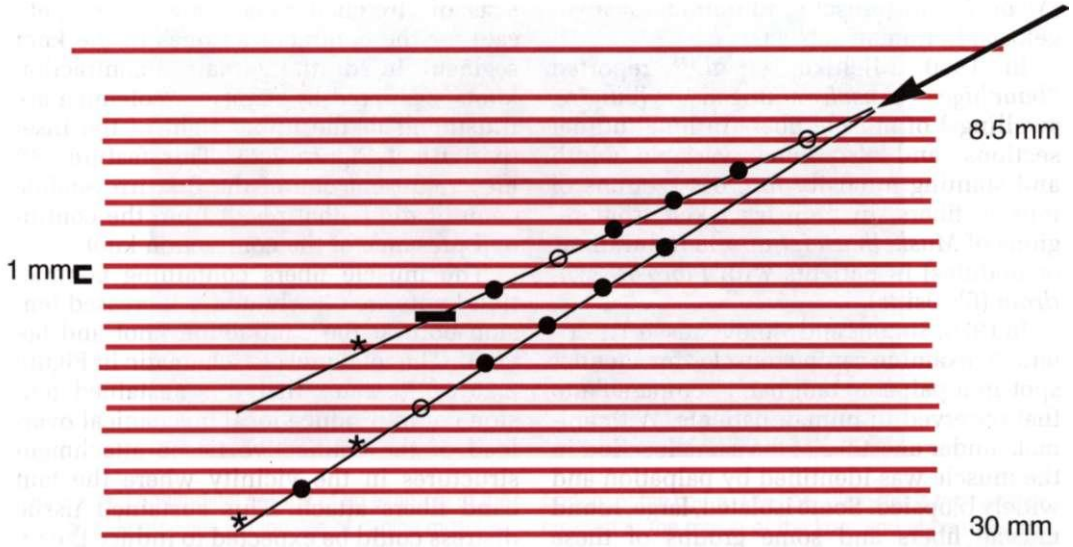


Figure 2.22. Distribution of active loci (that showed spontaneous electrical activity and endplate response to voluntary contraction) and of endplate locations without active loci in a trigger point. Endplates were recognized by the origin-waveform produced by gentle voluntary contraction. A total of 18 positions were tested along two needle tracks. Nine positions *(solid circles)* were identified as endplates without spontaneous electrical activity. All 3 positions *(asterisks)* where an electrically active locus was found were also located at an endplate. Three positions *(open circles)* showed quiet baseline without evidence of an endplate. One record *(horizontal bar)* was not counted because it was ambiguous as to the presence of an endplate. These findings indicate that the "abnormal" endplate potentials of trigger point active loci are found scattered among "normal" endplates that do not evidence spontaneous electrical activity and that the SEA abnormality is located at an endplate.

and from other fibers of the same motor unit at a site away from their endplates.

Using the technique described above to locate SEA, we examined several TrPs for the presence of SEA and for normal (SEA-free) motor endplate locations by sampling 8 locations in each of two tracks in a TrP. The subject was asked to make a minimal voluntary contraction at each location. Figure 2.22 graphically presents the results. Of the 16 locations tested in the TrP (which was in the endplate zone), three locations were active loci (SEA appeared and also negative voluntary spikes), nine were at an endplate (negative voluntary spikes without SEA), and four were at neither an endplate nor an active locus (no evidence of electrical activity beyond background). This is consistent with the concept that a group of dysfunctional motor endplates are at the heart of the TrP mechanism and that the dysfunctional endplates are a minority located among normal endplates.

If spikes originate at an active locus and are propagated action potentials in just that one muscle fiber, and if the taut band represents taut muscle fibers passing through the TrP, then it should be possible to record a train of spikes simultaneously from the active locus and from the taut band some distance from the TrP. This was observed in several human subjects and in several rabbits.[251] In one human subject the distance between the TrP and the recording needle in the taut band was 2.6 cm, twice the total length of an intrafusal muscle fiber.

Histopathological Characteristics of Trigger Points

Contraction knots, a characteristic histopathologic finding in TrPs and in tender palpable nodules, have been repeatedly noted but their significance not appreciated. In 1951, Glogowski and Wallraff[96] reported finding numerous *"knotenformig gequollene Muskelfasern "* (knot-like swollen muscle fibers) in biopsies of *Muskelharten*

(Myogelosen) (muscle indurations or myogelosis) in human subjects.

In 1960, Miehlke, *et al.*[193] reported *"bauchige Anschwellungen"* (bulging swellings) of muscle fibers in longitudinal sections, and also much variable width and staining intensity in cross sections of muscle fibers, in biopsies taken from regions of *Muskelharten* (muscle indurations or nodules) in patients with *Fibrositissyndrom* (fibrositis).

In 1976, Simons and Stolov[253] used TrP criteria to examine canine muscles for a tender spot in a palpable taut band comparable to that observed in human patients. With animals under anesthesia, the same location in the muscle was identified by palpation and widely biopsied. Some isolated, large, round muscle fibers and some groups of these darkly staining, enlarged, round muscle fibers appeared in cross sections (Fig. 2.23). In longitudinal sections, the corresponding feature was a number of contraction knots. An individual knot appeared as a segment of muscle fiber with extremely contracted sarcomeres. This contractured segment showed a corresponding increase in diameter of the muscle fiber, as illustrated in Figure 2.24.

The structural features of contraction knots, one of which is illustrated in Figure 2.24, are portrayed schematically in the lower half of Figure 2.25. This figure presents a likely explanation for the palpable nodules and the taut bands associated with TrPs. The inset below in Figure 2.25B shows three single contraction knots scattered among normal muscle fibers. Figures 2.24 and 2.25B illustrate that beyond the thickened segment of contractured muscle fiber at the contraction knot, the muscle fiber becomes markedly thinned and consists of stretched sarcomeres to compensate for the contractured ones in the knot segment. In addition, a pair of contraction knots separated by empty sarcolemma are illustrated in the upper right of the inset (part **B)** of Figure 2.25. This feature[96,253] may represent one of the first irreversible complications that result from the continued presence of the contraction knot.

The muscle fibers containing contraction knots are clearly under increased tension both at the contraction knot and beyond. The total muscle schematic in Figure 2.25A illustrates that this sustained tension could produce local mechanical overload of the connective tissue attachment structures in the vicinity where the taut band fibers attach. This sustained tissue distress could be expected to induce the release of sensitizing agents that would sensitize local nociceptors, producing local tenderness and the characteristics of an attachment TrP

In 1996, Reitinger, *et al.*[21i] biopsied in fresh cadavers the still-palpable nodules of myogelosis that were located in the gluteus medius muscle where trigger point 1 and trigger point 2 are found as described by Travell and Simons.[280] Cross sections showed the previously described, large, rounded, darkly staining muscle fibers and a statistically significant increase in the average diameter of muscle fibers in the myogelosis biopsies compared to nonmyogelotic control biopsies from the same muscle. Electron microscopic cross sections showed an excess of the A-Band and lack of the I-Band configuration. Exclusive presence of A-Band in the absence of I-Band occurs only in fully contracted sarcomeres.[15] It is highly likely that this fully-

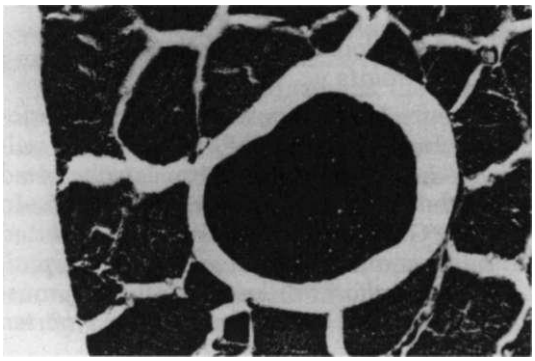


Figure 2.23. Giant round muscle fiber in the center of the figure is surrounded by open space that may have resulted from a local severe energy crisis. This space may contain substances that could sensitize adjacent nociceptive nerve fibers. In addition to the normal-size irregularly shaped muscle fibers surrounding the giant fiber, there are four abnormally small fibers, two above to the right, and two below to the left, that may be the segments of muscle fibers which are narrowed because of a contraction knot elsewhere in that fiber.

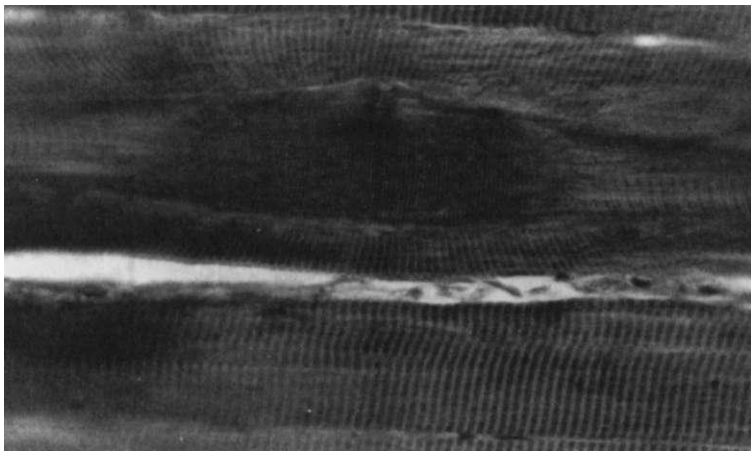


Figure 2.24. Longitudinal section of an example of the contraction knots seen in biopsies of canine muscles, in this case the gracilis. An exquisitely tender spot in a taut band of the muscle was selected as the biopsy site. These are two essential trigger point criteria. The striations (corresponding to sarcomere length) indicate severe contracture of the approximately 100 sarcomeres in the knot section of the muscle fiber. The sarcomeres on both sides of the knot show compensatory elongation compared to the normally spaced sarcomeres in the muscle fibers running across the bottom of the figure. The fiber diameter is markedly increased in the region of the knot and abnormally decreased on either side of it. The irregularity of the sarcolemma along the upper border of the fiber (in the center of the contraction knot) may represent an endplate. The distortion of the sarcomere alignment in adjacent muscle fibers represents sheer stresses in those fibers that may, in time, play a part in the propagation of this dysfunction to neighboring muscle fibers.

contracted electron microscopic pattern seen in cross sections and the large round fibers correspond to the (fully contractured) contraction knots seen in longitudinal sections under light microscopy.

Two features of Figure 2.24 suggest that the SEA does originate at a contraction knot and that the contraction knot may be caused by a dysfunctional endplate. First, this figure illustrates a longitudinal section of a contraction knot, which, in this case, is a segment of muscle fiber that includes about 100 maximally contractured sarcomeres. Normally sarcomeres range in length from about 0.6 u,m when fully shortened to about 1.3 |xm when fully extended, which is a full 1:2 length ratio.[15] Based on a minimum sarcomere length of 0.6 u,m, the 100 sarcomeres of the contraction knot would extend 60 urn. This is within the 20 to 80 u>m range in the length of normal motor endplates, depending on the muscle.[225] Second, although one cannot be sure of this in the absence of cholinesterase stain, the irregularity of the upper border in the middle of the contraction knot in Figure 2.24 fits the appearance one would expect if the motor endplate for that muscle fiber was centered over and extended the length of the contraction knot. A definitive experiment to confirm this impression is described under *Confirmation* of the **Integrated Trigger Point Hypothesis** that follows.

Integrated Trigger Point Hypothesis

This section includes several diagnostic categories that have German names, which are explained in the historical review part of section A of this chapter. It is presented from the point of view that TrPs are fundamentally the same disease process as other diagnoses based on tender nodules which are responsible for the patient's pain, diagnoses that may emphasize some aspects, and that have different names which are often in other languages.

The integrated hypothesis combines information from electrophysiological and histopathological sources. The energy crisis part of the hypothesis began to take

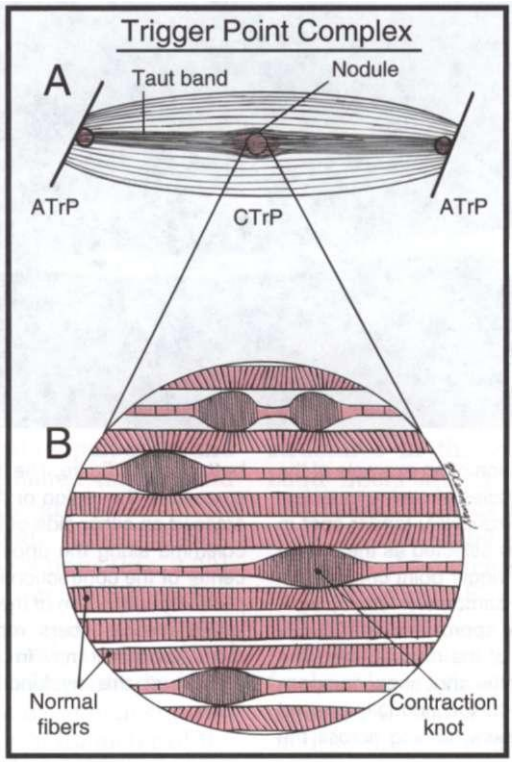


Figure 2.25. Schematic of a trigger point complex of a muscle in longitudinal section. The schematic identifies three regions that can exhibit abnormal tenderness *{red).* It also illustrates contraction knots that most likely: make a trigger point feel nodular, cause the taut band, and mark the site of an active locus. **A,** the **central trigger point** *{CTrP)* which is found in the endplate zone, contains numerous electrically active loci, and contains numerous contraction knots. The local tenderness of the CTrP is identified by a *red oval.* A *taut band* of muscle fibers extends from the trigger point to the attachment at each end of the involved fibers. The sustained tension that the taut band exerts on the attachment tissues can induce a localized enthesopathy that is identified as an **attachment trigger point** *{ATrP).* The local tenderness of the enthesopathy at the ATrP is identified by a red *circle with a black border.*

B, this enlarged view of part of the central trigger point shows the distribution of five contraction knots and is based on **Figures 2.23** and **2.24.** The vertical lines in each muscle fiber identify the relative spacing of its striations. The space between two striations corresponds to the length of one sarcomere. Each contraction knot identifies a segment of muscle fiber experiencing maximal contracture of its sarcomeres. The sarcomeres within one of these enlarged segments (contraction knot) of a muscle fiber are markedly shorter and wider than the sarcomeres of the neighboring normal muscle fibers which are free of contraction knots. In fibers with these contraction knots (note the lower three individual knots), the sarcomeres in the part of the muscle fiber that extends beyond both ends of the contraction knot are elongated and narrow compared to normal sarcomeres. At the top of this enlarged view is a pair of contraction knots separated by an interval of empty sarcolemma between them that is devoid of contractile elements. This configuration suggests that the sustained maximal tension of the contractile elements in an individual contraction knot could have caused mechanical failure of the contractile elements in the middle of the knot. If that happened, the two halves would retract, leaving an interval of empty sarcolemma between them. In patients, the CTrP would feel nodular as compared to the adjacent muscle tissue, because it contains numerous "swollen" contraction knots that take up additional space and are much more firm and tense than uninvolved muscle fibers.

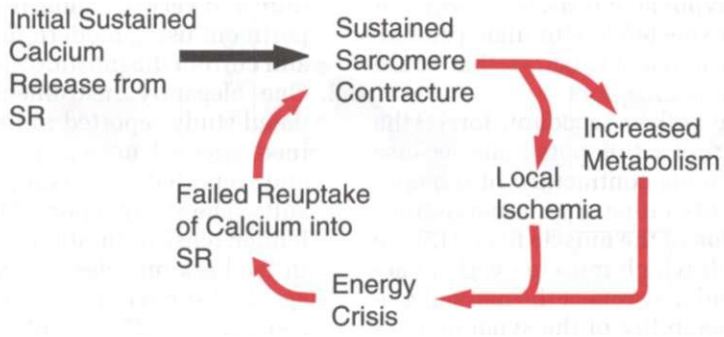


Figure 2.26. Schematic of the energy crisis hypothesis which postulates a vicious cycle *(red arrows)* of events that appears to contribute significantly to myofascial trigger points. The function of the sarcoplasmic reticulum (SR) is to store and release ionized calcium that induces activity of the contractile elements, which causes sarcomere shortening. An initiating event such as trauma or a marked increase in the endplate release of acetylcholine can result in excessive release of calcium from the SR *(black arrow).* This calcium produces maximal contracture of a segment of muscle which creates a maximal energy demand and chokes off local circulation. The ischemia interrupts energy supply which causes failure of the calcium pump of the sarcoplasmic reticulum, completing the cycle.

form about 20 years ago and has been evolving ever since. The energy crisis concept is compatible with recent electrodiagnostic findings, both of which fit the newly recognized histopathological picture.

Energy Crisis Component. This concept developed from efforts to identify a pathophysiological process that could account for: (1) the absence of motor unit action potentials in the palpable taut band of the TrP when the muscle was at rest, (2) the fact that TrPs are often activated by muscle overload, (3) the sensitization of nociceptors in the TrP, and (4) the effectiveness of almost any therapeutic technique that restores the muscle's full stretch length. The energy crisis concept was introduced in 1981[254] and was recently updated.[190,239]

Figure 2.26 shows the basic concept of the energy crisis hypothesis. It postulated an increase of the calcium concentration outside of the sarcoplasmic reticulum possibly due to mechanical rupture of either the sarcoplasmic reticulum[239] or of the muscle cell membrane (sarcolemma).[17] A sufficient increase in calcium would maximally activate actin and myosin contractile activity. However, if the damage were repairable, the abnormality would be temporary. It is now apparent that a more likely mechanism for sustained contractile activity is abnormal depolarization of the postjunctional membrane that could continue indefinitely based on continuing excessive ACh release from a dysfunctional nerve terminal. In this way, maximum contracture of the muscle fibers in the vicinity of the motor endplate could persist indefinitely without motor unit action potentials.

The sustained contractile activity of the sarcomeres would markedly increase metabolic demands and would squeeze shut the rich network of capillaries that supply the nutritional and oxygen needs of that region. Circulation in a muscle fails during a sustained contraction that is more than 30% to 50% of maximum effort. This combination of increased metabolic demand and impaired metabolic supply could produce a severe but local energy crisis. This functional component of the energy crisis should be reversible in a short period of time.

The Ca^{++} pump that returns the calcium into the sarcoplasmic reticulum is dependent on an adequate supply of adenosine triphosphate (ATP) and appears to be more sensitive to low ATP levels than the contractile mechanism. Thus an impaired uptake of calcium into the sarcoplasmic reticulum would expose the contractile elements to a further increase in calcium concentration and contractile activity. This completes a vicious cycle. In addition, the

severe local hypoxia and tissue energy crisis would be expected to stimulate production of vasoreactive substances that could sensitize local nociceptors.

Thus, the hypothesis accounts for: (1) the lack of motor unit action potentials because of the endogenous contracture of the contractile elements rather than a nerve-initiated contraction of the muscle fibers; (2) the frequency with which muscle overload activates TrPs and may reflect the marked mechanical vulnerability of the synaptic cleft region of an endplate; (3) the release of substances that could sensitize nociceptors in the region of the dysfunctional endplate of the TrP as a result of tissue distress caused by the energy crisis; and (4) the effectiveness of essentially any technique that elongates the TrP portion of the muscle to its *full* stretch length even briefly, which could break the cycle that includes energy-consuming contractile activity.

This fourth point can be explained by the fact that the continued activity of the actin-myosin interaction depends on physical contact between the actin and myosin molecules, which occurs fully when the sarcomere is approximately midlength or less. The molecules lose overlap contact at full length. This principle is illustrated in the lower part of Figure 2.5. With cessation of contractile activity because of actin-myosin separation, both the energy consumption and compression of capillaries would be relieved. This opportunity to restore energy reserves could help to block two critical steps in the energy-crisis cycle.

Based on this hypothesis, the TrP region should have three demonstrable characteristics: (1) be higher in temperature than surrounding muscle tissue because of increased energy expenditure with impaired circulation to remove heat, (2) be a region of significant hypoxia because of ischemia, and (3) have shortened sarcomeres.

1. The only two published reports that specifically measured intramuscular TrP temperature were an early report by Travell in 1954[270] and another described very briefly in Russian in 1976 by Popelianskii, *et al.*[209] Both recorded a focal increase in temperature in the region of the TrP. It would be a relatively simple procedure and very desirable to repeat this experiment using modern instrumentation and current diagnostic criteria of a TrP.
2. One elegantly instrumented and validated study reported in German[26] examined affected muscle for focal hypoxia and reported remarkably positive results. The study reported the findings in tender, tense indurations (Muskelharten) in the back muscles of three patients diagnosed as having *Myogelosen* (myogelosis). Figure 2.27 presents the graphic results of the three patients examined in this way. The first 5 to 8 mm of sensor advancement shows the normal random variation of tissue oxygen tension with successive 0.7 mm steps of advancement as the oxygen sensor approached the tender induration [TrP]. As the probe approached the palpable border of the tender induration, the tissue oxygen tension increased as if there were a compensatory hyperemia surrounding the region of hypoxia. After reaching a peak, the tissue oxygen tension fell abruptly to nearly (but not quite) zero, indicating profound hypoxia in the central region of the induration. It is noteworthy that the volume of the region of increased oxygen tension which surrounded the central region of oxygen deficit was at least as large as the volume of hypoxic tissue.
3. The contraction knots and electronmicroscopic findings described above confirm the presence of contractured sarcomeres.

In addition, the tendinous attachment of many of the fibers with these shortened segments would be likely to develop enthesitis because of the abnormally increased, sustained tension exerted by the double source of tension in each involved muscle fiber.

Although no experimental investigation of the development of enthesitis, where taut bands attach at the ends of the muscle, has been reported to date, its frequent clinical occurrence is illustrated repeatedly throughout this volume and confirmed by clinicians who look for it.

Integrated Trigger Point Hypothesis. When combined, the electrophysiological and histological lines of evidence indicate that a TrP is essentially a region of many dysfunctional endplates, and that

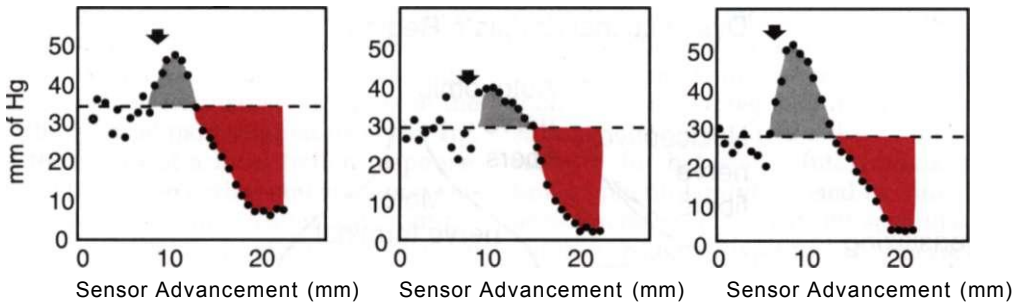


Figure 2.27. Tissue oxygen saturation values recorded by an oxygen probe that progressed in 0.7 mm steps through normal muscle and then into a tender, tense induration-Muskelharten (another name for a TrP)-in three patients with Myogelosis. *Arrow* marks the palpable border of the induration. The dashed line indicates the mean oxygen saturation of adjacent normal muscle. The area marked in *red* identifies the severe oxygen deficiency recorded as the probe approached the center of the induration. Note the comparable region of increased oxygen saturation surrounding the central region of hypoxia. (Data reproduced with permission from Bruckle W, Suckfull M, Fleckenstein W, et al. Gewebe-p02-Messung in der verspannten Ruckenmuskulatur [m. erector spinae]. *Zeitschrift fur Rheumatologie* 49:208-216,

each dysfunctional endplate is associated with a section of muscle fiber that is maximally contracted (a contraction knot).

The spontaneous electrical activity and spikes that characterize active loci within TrPs are currently recognized by electromyographers as 'normal" endplate potentials. However, physiological experiments have shown that these potentials are not normal, but are the result of a grossly abnormal increase in ACh release by the nerve terminal. It appears very likely that a contraction knot is located at an endplate and that it is caused by this endplate dysfunction. The following hypothesis proposes a likely relationship between the dysfunctional endplate and the contraction knot. The hypothesis provides a model that can be used to design critical experiments with which to verify, refine, or refute the hypothesis.

Figure 2.28 presents the integrated TrP hypothesis schematically. The hypothesis is based on continuous excessive ACh release from a dysfunctional motor nerve terminal into its synaptic cleft. Impaired cholinesterase function would potentiate the effect. The excessive ACh activates ACh receptors in the postjunctional membrane to produce greatly increased numbers of miniature endplate potentials. These potentials are so numerous that they superimpose to produce endplate noise or SEA, and a sustained partial depolarization of the postjunctional membrane. The excessive demand for production of ACh packets in the motor nerve terminal would increase its energy demand (evidenced by abnormal mitochondria in the nerve terminal). The increased activity of the postjunctional membrane and sustained depolarization would impose an additional local energy demand. Increased numbers of subsarcolemmal mitochondria and abnormal mitochondria have been noted repeatedly in past studies. This mechanism may be responsible for the presence of many ragged red fibers in muscles with characteristics that are compatible with the presence of myofascial TrPs.

The calcium channels that trigger release of calcium from the sarcoplasmic reticulum are voltage gated, normally by depolarization of the T tubule at the triad where the T tubule communicates with the sarcoplasmic reticulum. The T tubule is part of the same sarcolemmal membrane that forms the postjunctional membrane. The sustained depolarization of this membrane is one mechanism that might account for a tonic increase in the release of calcium from the sarcoplasmic reticulum to

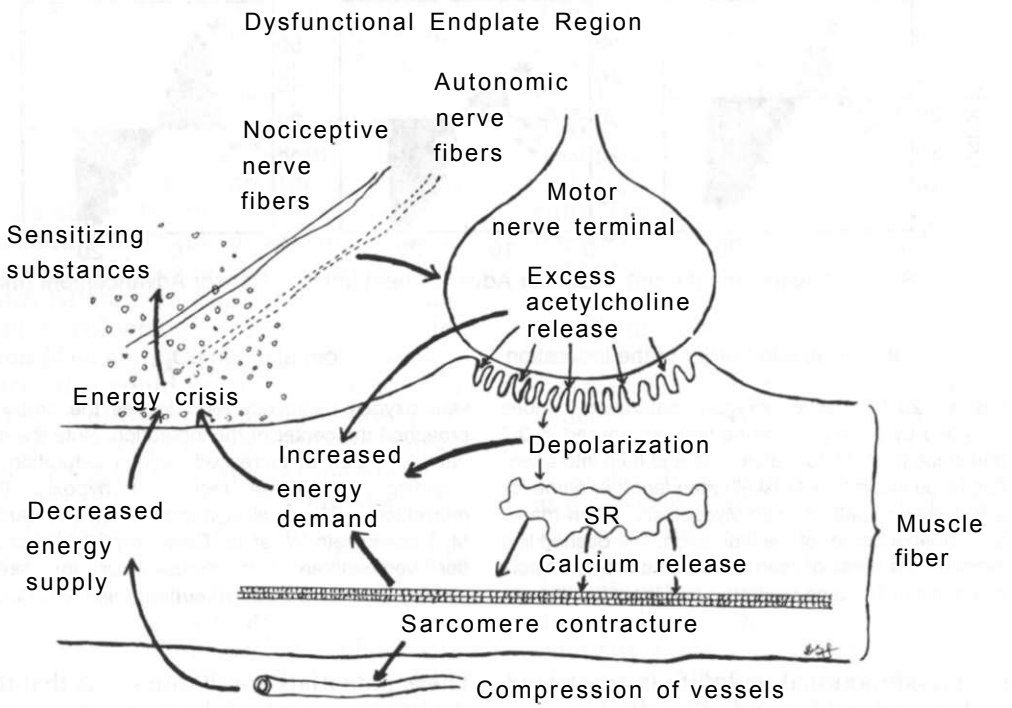


Figure 2.28. Integrated hypothesis. The primary dysfunction hypothesized here is an abnormal increase (by several orders of magnitude) in the **production and release of acetylcholine** packets from the motor nerve terminal under resting conditions. The greatly increased number of miniature endplate potentials produces endplate noise and **sustained depolarization of the postjunctional membrane** of the muscle fiber. This sustained depolarization could cause a **continuous release and uptake of calcium ions from local sarcoplasmic reticulum** *(SR)* and produce **sustained shortening (contracture) of sarcomeres.** Each of these four highlighted changes would increase energy demand. The sustained muscle fiber shortening compresses local blood vessels, thereby reducing the nutrient and oxygen supplies that normally meet the energy demands of this region. The increased energy demand in the face of an impaired energy supply would produce a local energy crisis, which leads to release of sensitizing substances that could interact with autonomic and sensory (some nociceptive) nerves traversing that region. Subsequent release of neuroactive substances could in turn contribute to excessive acetylcholine release from the nerve terminal, completing what then becomes a self sustaining vicious cycle.

produce the local sarcomere contracture of the contraction knots. The increased volume occupied by the contraction knots would also explain why clinicians describe palpating a nodule at the TrP along with the narrower taut band. This contracture process appears to occur in the immediate vicinity of an endplate. A sustained release of calcium from the sarcoplasmic reticulum would increase the energy demand of the calcium pumps in the sarcoplasmic membrane that return the calcium into the sarcoplasmic reticulum. The sustained contracture of the sarcomeres in the contraction knot would greatly increase the local energy and oxygen demand.

The concept of sustained contracture of sarcomeres in the muscle fiber supplied by the affected endplate is compatible with the previously proposed energy crisis hypothesis reviewed in detail above. The severe energy crisis in the vicinity of the endplate can be expected to release neuroactive substances that sensitize and modify the function of any sensory and autonomic nerves in that region. As noted in Section C above, small blood vessels, sensory nerves, and autonomic nerves normally are

part of the same neurovascular bundle or complex that includes the motor nerve.

Sensitization of local nociceptors could account for the exquisite tenderness of the TrP, the referred pain originating at the TrP, and the origin of a local twitch response. Several lines of experimental evidence suggest that autonomic (especially sympathetic) nervous system activity can strongly modulate the abnormal release of acetylcholine from the nerve terminal.

The clinical effectiveness of Botulinum A toxin injection for the treatment of myofascial TrPs[1,34,297] helps to substantiate dysfunctional endplates as an essential part of the pathophysiology of TrPs. This toxin specifically acts **only** on the neuromuscular junction, effectively denervating that muscle cell.

Studies by Gevirtz and associates support indications that the autonomic nervous system can modulate spike activity (and therefore the rate of release of ACh packets) at a motor endplate. Trigger point EMG activity was increased by psychological stressors both in normal subjects[186] and in patients with tension-type headache.[167] These two reports did not specify whether the TrP EMG activity being measured was SEA or spikes or some combination of both.

More recently, Hubbard[132] published additional experimental data indicating that the amount of electrical activity is strongly influenced by the autonomic nervous system. All intramuscular injections employed EMG guidance to place the injected solution close to the source of the TrP EMG potentials. Four patients were injected with phentolamine intramuscularly and in two patients phentolamine was injected intravenously. In all six studies, the TrP EMG activity subsided for the duration of the phentolamine effect. Phentolamine is a competitive a-adrenergic blocker.[132] In a series of uncontrolled studies, a total of 108 patients received EMG-guided TrP injections of phenoxybenzamine, which is a long-lasting adrenergic, noncompetitive a-receptor blocking agent that can produce a chemical sympathectomy with no effect on the parasympathetic system. It has an intravenous half-life of 24 hours. Between one-half and two-thirds of the patients experienced at least 25% pain relief within 1 month following treatment and relief generally lasted for 4 months. Apparently very few subjects realized complete relief. The phentolamine study is more convincing than the phenoxybenzamine study and is strongly supported by a subsequent rabbit study.[33a] In that study, intravenous injection of phentolamine caused as much as a 68% decrease in SEA in 80 seconds. Apparently, roughly two-thirds of the ACh release was dependent on local sympathetic nervous system effects.

In addition, in conjunction with a human study of active loci in TrPs,[249] the investigators confirmed a previous observation[131] that in many subjects spike activity associated with SEA in the upper trapezius muscle was clearly increased by normal resting inhalation and was inhibited by exhalation. Exaggerated respiratory efforts increased the response. They[249] also noted an increase in the amplitude of SEA during inhalation.

The possibility that the presence of excess calcium in the vicinity of the contractile elements is due to an excess of calcium release compared to calcium uptake into the sarcoplasmic reticulum is supported by a case report.[233] Two patients prone to trigger points in the right gluteus medius muscle experienced a flare and became refractory to the usually successful injection after taking a calcium channel blocker, alodipine besylate, for hypertension. Treatment became effective again in the absence of alodipine. This calcium-channel blocker inhibits the reuptake of calcium into the sarcoplasmic reticulum of vascular smooth muscle and cardiac muscle. If this is also true of skeletal muscle, the resultant increase in calcium to stimulate contracture of sarcomeres in the TrP region would aggravate the vicious cycle of Figure 2.28.

Clinical Correlations. If multiple active loci are part of the same pathophysiological process as multiple contraction knots, and if this relationship applies equally to TrPs and to tender nodules, it would represent a major step forward in our understanding of enigmatic myogenic pain. Based on the integrated hypothesis just described, many of the clinical features of this clinical condition can now be explained.

Two aspects of Figure 2.24 suggests that, in fact, the SEA does originate at a contraction knot and that the contraction knot may be caused by a dysfunctional endplate. Assuming this pathophysiological interpretation is correct, it explains a number of clinical features that apply to both TrPs and myogelosis, although in both cases, some features commonly have been overlooked.

The *taut band* of a TrP would be caused by the increased tension of involved muscle fibers both because of the tension produced by the maximally shortened sarcomeres in the contraction knot and also because of the increased (elastic) tension produced by all the remaining elongated (and therefore thin) sarcomeres. Ordinarily a muscle fiber runs from its musculotendinous attachment at one end of a muscle to its musculotendinous attachment at the other end; in fusiform muscles, that is nearly the full length of the muscle.

Figure 2.24 shows clearly the abnormally shortened and abnormally lengthened sarcomeres of the muscle fiber that contains the contraction knot (in the center of the figure). These abnormal lengths contrast to the normal resting length of sarcomeres in the uninvolved muscle fibers running across the lower part of the figure. With the involvement of a sufficient number of muscle fibers within several fascicles, the increased tension of the involved muscle fibers should be palpable as a taut band running the length of the muscle. This full-length description applies if the muscle fibers run nearly parallel to the long axis of the muscle, and the muscle has no inscriptions.

The *palpable nodule* of TrP-related diagnoses, such as fibrositis and myogelosis, can be explained by the presence of multiple contraction knots (Fig. 2.25). Since a sarcomere must maintain a nearly constant volume, it becomes broader as it shortens. The sarcomeres in a contraction knot appear at least twice the diameter of the distant sarcomeres beyond the contraction knot in the same fiber. The nodule feels larger than surrounding tissue because of the greater volume occupied by the contraction knots and it feels firmer because of the highly condensed state of the contractile elements in each knot. The region of contraction knots feels larger than the rest of the taut band because the normal fibers and stretched-thin fibers in the taut band extend beyond the nodule unchanged. The contraction knots represent additional volume (Fig. 2.25).

The *spot tenderness* of both TrPs and nodules would be the result of sensitized nociceptors. The nociceptors are most likely sensitized by substances released as a result of the local energy crisis and tissue distress which is associated with these histopathological changes and endplate dysfunction.

The *enthesopathy* (tenderness at the muscle attachment where the taut band terminates) is explained by the inability of the muscle attachment structures to withstand the unrelieved sustained tension produced by the taut band. In response, these tissues develop degenerative changes that are likely to produce substances which could sensitize local nociceptors. Fassbender and Wegner[66] presented histological evidence in fibrositis (nonarticular rheumatism) patients for the kind of degenerative changes to be expected in regions of TrP-induced enthesopathy.

The *myoglobin response to massage* of fibrositic nodules can be explained on the basis of the observed histopathological changes in nodules. Repeated deep massage of the fibrositic nodules (TrPs) produced transient episodes of myoglobinuria that were not produced by similar massage of normal muscle.[47,48] The intensity of myoglobin response, the degree of tenderness, and firmness of the nodule progressively faded out with repeated treatments (Fig. 2.29). The distended sarcoplasm of these contraction knots could well be more vulnerable to rupture by mechanical trauma and external pressure than normal fibers. If massage applied by the therapist resulted in cell rupture, the cell would spill myoglobin and most likely destroy the involved neuromuscular junction as a functional structure, thus effectively terminating the contracture and associated energy crisis. As more and more contraction knots within the nodule were eliminated, the patient would experience increasing relief of symptoms.

The development of *histopathological complications* that could contribute to chronicity and make treatment more difficult is suggested by two observations. First, Figure 2.24 clearly illustrates marked distortion of the striations (sarcomere arrangement) in adjacent muscle fibers for some

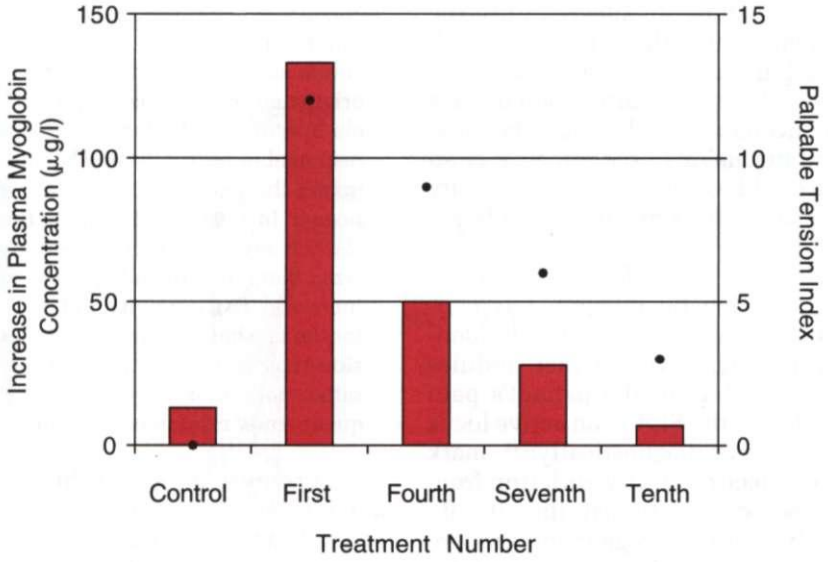


Figure 2.29. Median values resulting from repeated deep massage of the same fibrositic nodules (TrPs) in 13 patients. Plasma myoglobin levels increased 10-fold following the first treatment. By the tenth treatment, the response had declined to the same low level observed in normal muscle. The "fibrositic" palpable tension index was proportional to the extent of muscle tension before a treatment. This index ***(small black closed circles)*** declined progressively to the tenth treatment reaching one-fourth of the value that it had at the time of the first treatment. Graph based on data in Table I of Danneskiold-Samsoe, et al., 1983.[47]

distance beyond the contraction knot. This would produce unnatural shear forces between fibers that could seriously (and chronically) stress the sarcolemma of the adjacent muscle fibers. If the membrane were stressed to the point that it became pervious to the relatively high concentration of calcium in the extracellular space, it could induce massive contracture that would compound the shear forces. Bennett[17] described this mechanism clearly and how it could lead to severe local contracture of the muscle contractile elements. This mechanism might account for the "keulenformige gequollene Muskelfasern" (club shaped swollen muscle fibers) described by Glogowski and Wallraff,[96] which look like elongated versions of a contraction knot. If this happens, it might occur anywhere along a muscle fiber where it has been affected by an adjacent contraction knot. This could explain the tendency for clumping of giant fibers mixed with unusually small fibers (segments of stretched sarcomeres) that is seen in cross sections. This tendency for clumping was illustrated by Simons and Stolov[253] in their Figure 9 and by Reitinger, *et al.*[214] in their *Abb.* 3c.

Second, the occasional finding of a segment of empty sarcolemmal tube between two contraction knots (Fig. 2.25) may represent an additional irreversible complication of a contraction knot. Miehlke, *et al.*[193] described "Entleerung einzelner Sarkolemmschlauche" (emptying of individual sarcolemmal tubes). Reitinger, *et al.*[211] described "Muskelfasern mit optisch leerem, zystichen Innenraum (Myofibrillenverlust?)" [muscle fibers with an optically empty, cystic interior (loss of myofibrills?)]. Simons and Stolov,[253] in their Figure 13, illustrated and described the complete emptying of the sarcolemmal tube between two contraction knots (Fig. 2.25). This configuration appears as if the sustained maximal tension of the contractile elements in a contraction knot caused mechanical failure of the contractile elements in the middle of the knot. This allowed the two halves to retract, leaving an interval of empty sarcolemma between them. Electronmicroscopic illustrations by

Fassbender[64,66] show disintegration of the actin filaments where they attach to the Z-line suggesting that this is the location in the chronically contractured sarcomeres where the mechanical failure may begin.

These additional histopathological complications could contribute to chronicity and may relate to the transition from latent to active TrPs.

Confirmation. A relatively simple study could validate the integrated hypothesis. The investigators would need to identify myofascial TrPs with tender nodules that are responsible for the patient's pain complaint; locate the SEA of an active locus in the TrP electrodiagnostically;[242] mark that location electrolytically with iron from the EMG needle;[147,291] biopsy the site; fix the biopsy by liquid nitrogen; and prepare *longitudinal* sections that are stained for iron,[147,291] for acetylcholinesterase,[291] and a base stain such as one of the trichromes.[214] If the iron-stained regions include contraction knots with motor endplates attached to them, it would greatly advance understanding of, and the acceptance of the diagnoses of TrPs and TrP-related conditions that are characterized by tender nodules and/or taut bands. Descriptions of this critical experiment, and the rationale for it have been published.[244,245]

Other Hypotheses

Pain-Spasm-Pain Cycle. The old concept of a pain-spasm-pain cycle does not stand up to experimental verification either from a physiological[191] point of view or from a clinical[105] point of view.

> **Physiological studies show that muscle pain tends to inhibit, not facilitate, reflex contractile activity of the same muscle.[191] Walsh[285] explained clearly how this misconception has been strongly reinforced by a misunderstanding of normal human motor reflexes based on spinalized cat experiments and how the misconception has persisted throughout the 20th century.**
>
> **In 1989, Ernest Johnson,[146] editor of the *American Journal of Physical Medicine* summarized overwhelming evidence that the common perception of muscle pain being closely related to muscle spasm is a myth and that the myth has been strongly encouraged by commercial interests.[148] The term tension headache is a good example of this myth in action. The term originated with the assumption that muscle spasm (involuntary contraction) was responsible for the headache and that relaxing the pericranial muscles would relieve it. In 1991, an editorial in the journal *PAIN*[202] reviewed this issue and emphasized that it was unambiguously clear that increased EMG activity did not account for the muscle tenderness and pain of tension-type headache. The author had no satisfactory alternative solution. A subsequent study reinforced this conclusion.[145]**

A current variation of this pain-spasm-pain concept, the stress-hyperactivity-pain theory,[38] seems equally invalid for the same reasons.

Muscle Spindle Hypothesis. In their initial communication, Hubbard and Berkoff,[133] and again Hubbard in his more recent report,[132] concluded that the source of EMG activity in TrPs was a dysfunctional muscle spindle. They[133] gave three reasons for dismissing the possibility that these potentials might arise from motor endplates: (1) the activity is not localized enough to be generated in the endplate, (2) the activity does not have the expected location, and (3) the activity does not have the expected waveform morphology.

Existing literature and our experimental findings contradict these three assertions.

1. The degree of localization that is described under the headings Active Loci and Spikes above corresponds closely to that previously described in the classical paper on the source of motor endplate potentials.[291]
2. Recent studies[248,249,252] explicitly examined the distribution within the muscle of the electrically active loci and found that they are chiefly in a TrP, to some extent also in the endplate zone, but were not found outside of the endplate zone. Muscle spindles are scattered throughout a muscle as shown in Figure 2.30[35] and in Figure 2.31.[211] Muscle spindles clearly are not concentrated just in the endplate zone where TrPs are found. The studies associated with Figures 2.17 and

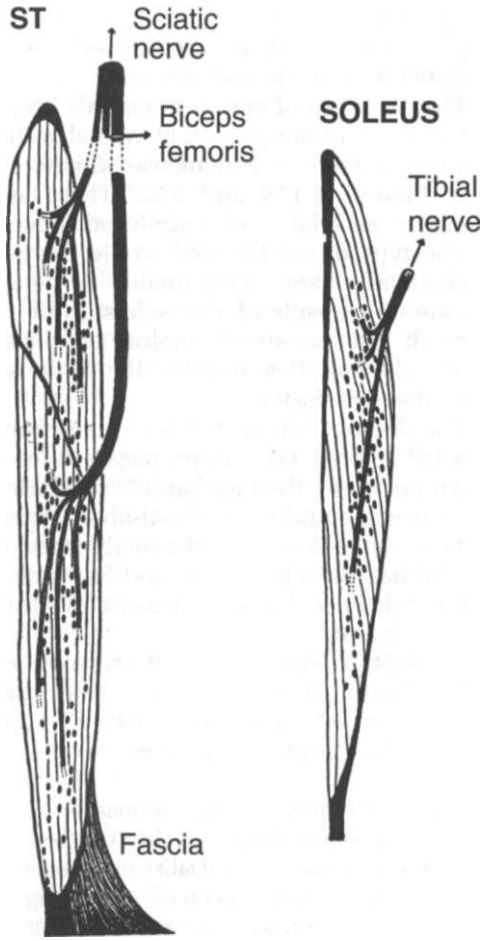


Figure 2.30. Distribution of muscle spindles *{small black ovals)* in the semitendinosus (ST) and *soleus* muscles of the cat. The semitendinosus muscle is divided into two segments that are separately innervated. However, the muscle spindles are uniformly distributed throughout the length of both segments and not concentrated in the endplate zone as are motor endplates. (Reproduced with permission from Chin NK, Cope M, Pang M. Number and distribution of spindle capsules in seven hindlimb muscles of the cat. In: Barker D, ed. *Symposium on Muscle Receptors.* Hong Kong: University Press, 1962:241-248.)

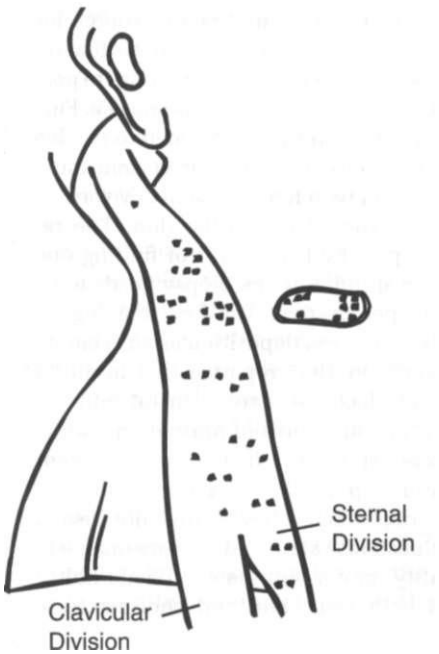


Figure 2.31. An example of the distribution of muscle spindles in the sternocleidomastoid muscle of a 14 weeks old human fetus. The spindles are distributed rather uniformly throughout the muscle and not clustered in the midbelly region of the muscle as are the motor endplates. (Reproduced with permission from Radziemski A, Kedzia A, Jakubowicz M. Number and localization of the muscle spindles in the human fetal sternocleidomastoid muscle. *Folia Morphol 50(1/2)65-70,* 1991.)

2.22 demonstrate that active loci occur at motor endplates.

3. The reader can judge with regard to the waveform morphology by comparing the spikes with SEA in our recordings from an active locus (Fig. 2.14B) with the endplate potentials illustrated in a current electromyography text (Fig. 2.15). The amplitude and sweep speed with which a recording is made can produce great differences in waveform appearance, which can be very misleading (Fig. 2.14A). At similar sweep speeds the SEA and endplate potentials have the same waveform morphology.

Other authors agree that these spikes and spontaneous electrical activity found in TrPs arise from motor endplates.[19, 37] Brown and Varkey[24] also attributed the spontaneous electrical activity to potentials of the endplate zone and attributed spikes to postsynaptic muscle-fiber action potentials that were presynaptically activated by mechanical irritation, with which we agree.

There are four additional reasons why one must very seriously question the validity of the hypothesis that the heart of the TrP dysfunction lies in dysfunctional muscle spindles rather than in dysfunctional motor endplates.

1. If the conclusion that these potentials arise from dysfunctional muscle spindles is correct, then Wiederholt's comprehensive EMG, histological and pharmacological study[291] reached an erroneous conclusion and electromyographers ever since have been misled. It may be difficult to convince the electromyographic community that what they have identified as endplate potentials are really muscle-spindle potentials. If the potentials described by Hubbard are other than endplate potentials, then where in his studies are the endplate potentials identified by electromyographers? Because most of the studies by *physiologists* describing abnormal endplate potentials corresponding to SEA did not appear in print until after his paper, Wiederholt did not realize that the potentials commonly identified as endplate noise must be distinguished from true miniature endplate potentials, which are considerably more difficult to locate and record.
2. The presence of action potentials originating at an endplate that was also the site of a TrP active locus was illustrated in Figures 2.17B and 2.22. These are motor endplates of extrafused fibers. The type of needle used would be mechanically (Fig. 2.16) unable to penetrate the capsule of a muscle spindle to reach an intrafusal motor endplate. Muscle spindles usually lie in loose connective tissue.
3. The demonstration that the spikes from a TrP active locus can propagate at least 2.6 cm along the taut band[251] precludes a muscle-spindle intrafusal-fiber origin. This distance is twice the total length of a human muscle spindle and four times the half-fiber distance measured in this experiment.
4. In addition, the clinical effectiveness of Botulinum A toxin injection for the treatment of myofascial TrPs[1,34,297] supports the endplate hypothesis.

If muscle-spindles were the location of TrPs, it would not help to explain the close relation between TrPs and taut bands, since propagated action potentials originating from motor neurons are not responsible for the tension of the band. It is true that a muscle-spindle is an attractive source for the afferent limb of the local twitch response. However, it is not necessary to postulate a dysfunctional muscle spindle. Further research studies are needed to resolve whether muscle spindles ever contribute to the local twitch response in any way.

Two issues need clarification. The recent report by Hubbard[132] of finding one muscle spindle in one biopsy needs to be put in perspective. The first histological study using iron deposition as an accurate marker[147] in 1955 reported that in all 28 sites of electrical activity in rat muscles "no other structures of muscle, including muscle spindles, had any consistent relationship to the area containing the iron deposits." They[147] did not use a cholinesterase stain and so were unable to identify motor endplates. Wiederholt[291] used both iron stain and cholinesterase

stain when he strongly associated the source of the electrical activity with endplates. He made no mention of muscle spindles, although it would be no surprise if a muscle spindle appeared in a few of his sections since they are widely distributed in the muscle, including the endplate region. The methylene blue injection used by Hubbard to localize the site for evaluation is well known for its tendency to diffuse along the fascial planes where muscle spindles are located. As the author noted, this one biopsy, which contradicts previous studies, is not conclusive.

The report[132] that in two subjects EMG-guided intramuscular TrP injections of curare had no effect on either the amplitude or frequency of the TrP-EMG activity would seem to be convincing evidence that the EMG activity did not come from motor endplate activity. However, in several pilot tests using intravenous injection of curare in the rabbit (Hong, Simons, Simons, unpublished data) the investigators learned that unless one establishes, by some independent means such as motor nerve stimulation, that the motor endplates are effectively blocked by the curare, one cannot draw any conclusions with confidence concerning its effect on the electrical activity of active loci. This confirmation was lacking in the Hubbard study. To be seriously considered, this experiment needs to be repeated with proper controls.

One other study[205] suggested that spikes arise from intrafusal muscle fibers. Those authors discussed why spikes are not ectopic discharges of motor axons but did not consider the possibility that spikes are the result of mechanically induced release of abnormal amounts of acetylcholine at the neuromuscular junction of an extrafusal fiber. However, all of their data were consistent with this latter mechanism of spike generation. Muscle spindles may, at times, contribute to TrP phenomena, but it seems extremely unlikely that muscle spindles are the primary site of the TrP mechanism.

Neuropathic Hypothesis. In 1980, Gunn[109] proposed that the cause of TrP hypersensitivity is neuropathy of the nerve serving the affected muscle. Recently, Chu[37] has presented extensive EMG evidence that neuropathic changes are significantly related to the presence of TrPs in the paraspinal musculature. There is much clinical evidence that compression of motor nerves can activate and perpetuate the primary TrP dysfunction at the motor endplate.

Fibrotic Scar Tissue Hypothesis. The concept that the palpable firmness of the tissues at the TrP represents *fibrotic (scar) tissue* is based on the assumption that damaged muscle tissue has healed by scar formation.[75] This concept derives from histological findings in a few most severely involved subjects in studies of Muskelharten, Myogelosen, Fibrositis, and Weichteilrheumatismus reported in the German literature throughout this century. Patients with myofascial TrPs would have been included under the diagnostic criteria used for these studies but so would almost any other muscular affliction with tender indurations.

Only two studies have reported biopsies of TrPs, one on dogs[253] and one on human subjects.[214] Both studies presented strong evidence for the presence of contraction knots and neither found fibrosis. In addition, the recently discovered endplate dysfunction described in this chapter and taut bands caused by sarcomere contraction fully account for the clinical findings of patients with myofascial TrPs without invoking fibrosis as part of the process. The rapid resolution of the palpable taut band with specific TrP treatment argues against the fibrosis explanation. A review by Simons[235] of all biopsies of tender nodules reported for much of this century found that the authors consistently reported little or no scar tissue and, when present, it was observed only in a relatively few clinically severe cases.

It is possible that if the endplate dysfunction is allowed to persist for an extended period of time, it may eventually lead to chronic fibrotic changes. How quickly and under what circumstances this might occur must be resolved with appropriate research studies. The increasing refractoriness to local TrP therapy with longer periods before effective treatment is started[127] can just as well be attributed to plastic changes of the central nervous system when subjected to prolonged nociceptive input as to fibrotic changes in the mus-

cle. This central mechanism is now well documented experimentally.

Local Twitch Response

The local twitch response (LTR) is a brisk transient contraction of the palpable taut band of muscle fibers elicited by mechanical stimulation of the TrP in that taut band. Mechanical stimulation may be produced by needle penetration of the TrP,[246] by mechanical impact applied directly to the muscle[128] (or applied through the skin over the TrP), or by snapping palpation of the TrP.[246]

Clinically, the response is most valuable as a confirmatory sign. When injecting a TrP, an LTR signals that the needle has reached a part of the TrP that will be therapeutically effective.[123] It is often not practical to include the LTR as a primary diagnostic criterion of a TrP because an LTR can be prohibitively painful to the patient when it is elicited, it is often inaccessible to manual palpation because of overlying fat and/or muscle, and the LTR requires a particularly high degree of manual skill for reliable detection.[94] However, when it does occur in the course of examination of a tender nodule or taut band, the LTR is strong evidence for the presence of a TrP. The rabbit localized twitch response has proven to be a valuable research tool for investigating the nature of twitch responses.[128,129]

Topographic Extent of the Local Twitch Response. To date, most experimental investigations of the local twitch response (LTR) examined the localized twitch response which is the rabbit counterpart of the LTR. The pioneering study by Hong and Torigoe in 1994[128] identified a trigger spot (comparable to the human TrP) in the rabbit biceps femoris muscle by locating a taut band using pincer palpation and testing along its length for a maximum twitch response to snapping palpation. This location was designated the trigger spot. Mechanical stimulation was standardized by using a solenoid-driven rod to impact the surface of the muscle at selected locations. The response was recorded electromyographically with a monopolar teflon-coated EMG needle placed in the taut band several centimeters distal to the trigger spot.

Figure 2.32**A** from this study compares the vigor of the twitch response to taps on the trigger spot and to taps applied short distances away from it. Responses were unobtainable 5 mm to either side of the trigger spot, were greatly attenuated when applied in the taut band 1 cm from the trigger spot toward the recording needle, and were vestigial in the taut band 3 cm from the trigger spot. The vigor of the twitch response was very sensitive to small displacements of only a few *millimeters* when the stimulus was applied to muscle fibers adjacent to the trigger spot, and was similarly attenuated by displacement a few *centimeters* along the same fibers that pass through the trigger spot. These findings correspond to the location of tenderness at TrPs in human patients. Responsiveness to snapping palpation is greater at the nodule or TrP as compared to a distance from it along the taut band. The findings also correspond to the meticulous accuracy with which one must stimulate the sensitive locations in the taut band and not adjacent tissue in order to evoke the LTR.

Figure 2.32B examines the effect of tapping the trigger spot and recording the twitch response with a needle in the taut band and with the needle placed 5 mm to either side of the taut band. The latter positions showed vestigial twitch responses. The action potentials of the twitch response were propagated in just those fibers passing through the trigger spot and did not involve adjacent muscle fibers. The twitch response was highly localized to the trigger spot and to the taut band passing through it.

Origin and Propagation of the Local Twitch Response. No studies to determine the specific structure(s) responsible for the origin of the LTR are known to date. Clinically, the strong relation between the appearance of LTRs during successful needling of a TrP[123] and the severe pain frequently experienced by the subject when a twitch response occurs suggests that it can originate from stimulation of sensitized nociceptors in the region of the TrP.

The a-motoneurons with endplates suffering from excessive ACh release appear to be preferentially responsive to the strong sensory spinal input from these sensitized nociceptors. This possibility is reinforced by the observation that snapping palpation of one TrP resulted in simultaneous LTRs in the taut band of that TrP and in a taut band of another nearby muscle. It is possible that adequate mechanical stimulation of any

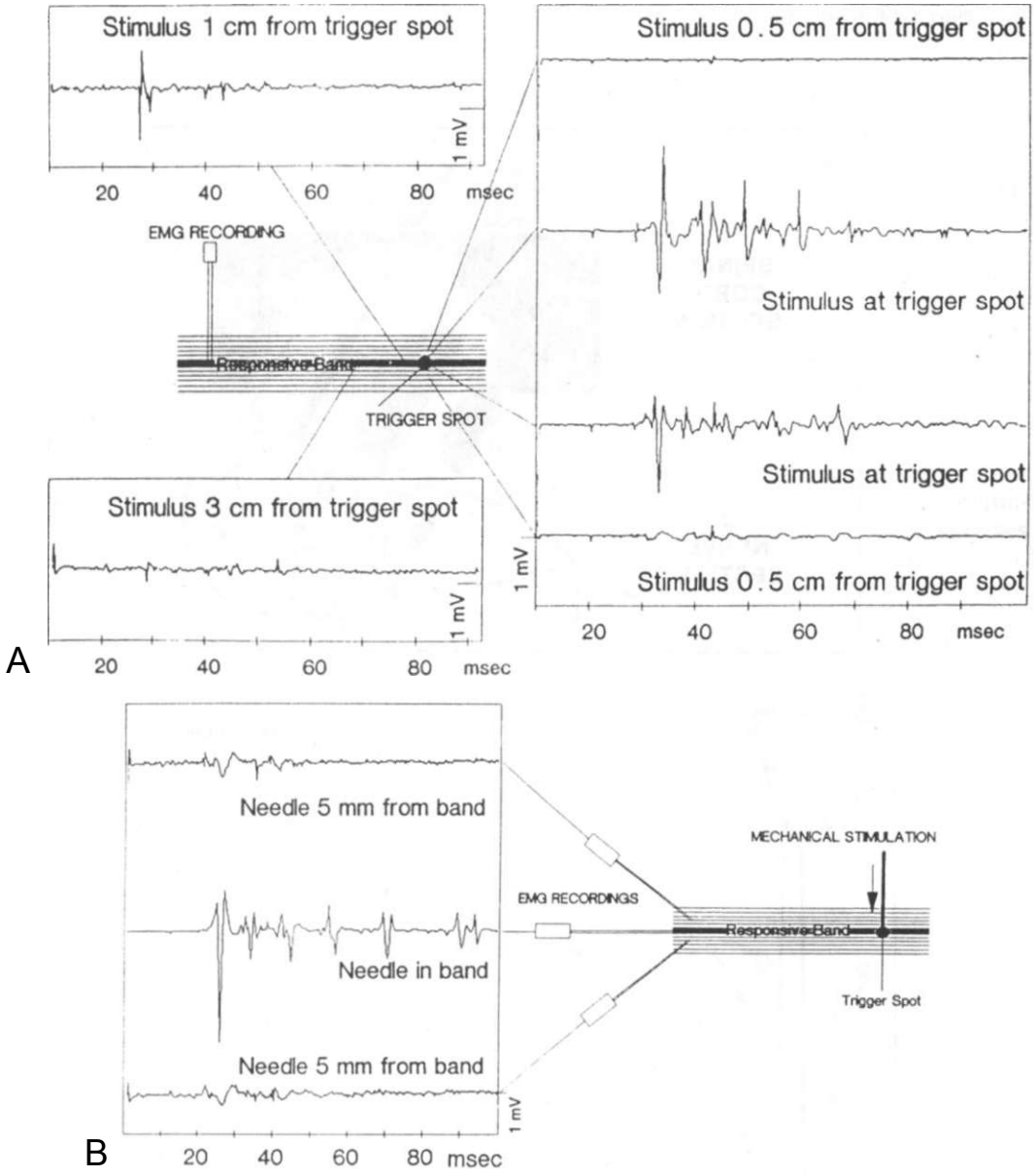


Figure 2.32. Electromyographic recordings demonstrating the spatial specificity of the rabbit localized twitch response with regard to the region of the trigger spot that was stimulated mechanically to elicit the response and with regard to the region of the taut band from which the response could be recorded. The ***solid black line*** represents the taut band (marked ***responsive band)*** that was selected for testing by manual palpation.

A, specificity of the point of stimulation in the region of the trigger spot in the taut band. Electromyographic recordings of twitch responses were obtained from a needle inserted in the taut band distant from the trigger spot. Stimuli were delivered directly on the trigger spot, to either side of it, and along the taut band toward the recording needle, as indicated by labels on the recordings and location of the label lines. The most vigorous response was observed at the trigger spot, almost none to either side, and progressively less as the point of stimulation moved farther from the trigger spot.

B, The arrow indicates the point of mechanical stimulation by a tap delivered with a solenoid-driven thin rod. The three electromyographic tracings were obtained in, and 5 mm to either side of, the taut band. The recordings near but not in the taut band show only distant waveforms.

These observations substantiate the clinical impression that the local twitch response is specific to mechanical stimulation of the trigger spot (point) region and is ordinarily propagated only by the taut band fibers passing through the trigger spot. (Reproduced with permission from Hong CZ, Torigoe Y. Electrophysiological characteristics of localized twitch responses in responsive taut bands of rabbit skeletal muscle. / ***Musculoske Pain*** *2(2)^* 7-43, 1994.)

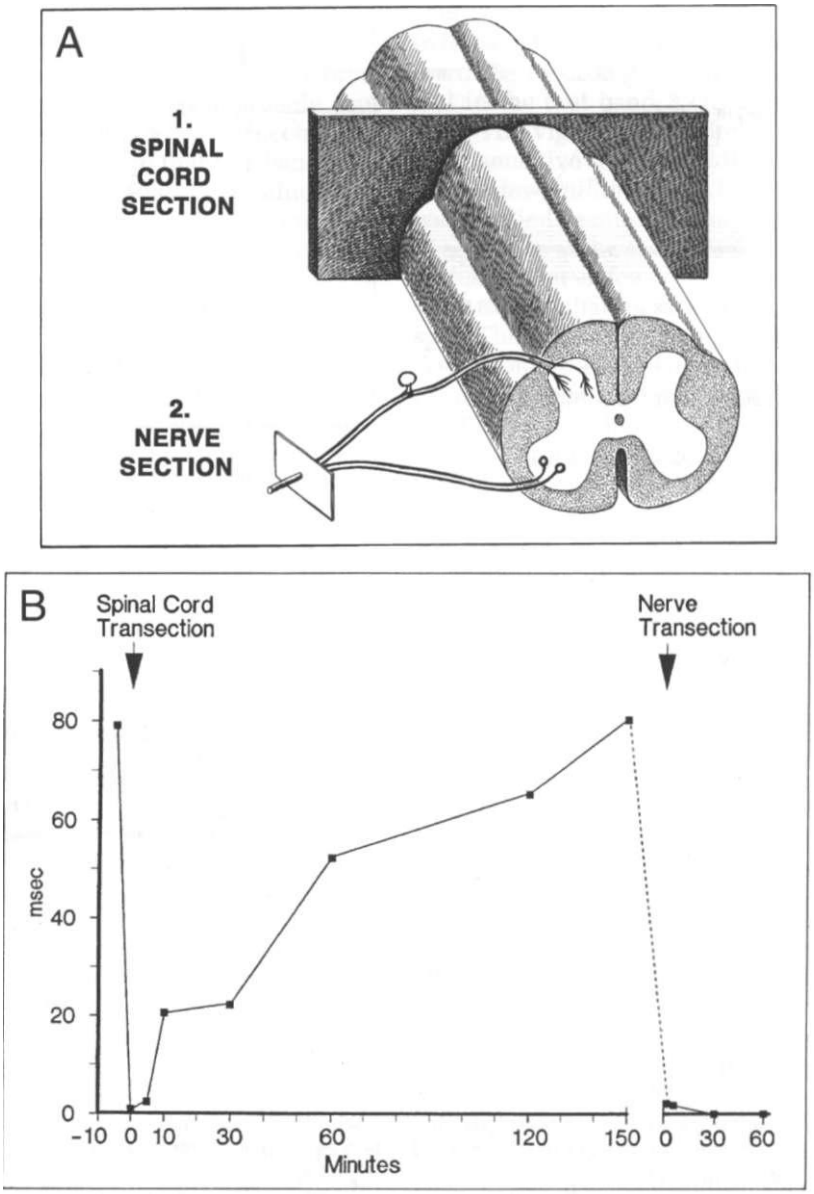


Figure 2.33. Evidence that the local twitch response is a spinal reflex not dependent on higher centers. A, schematic of procedure for a localized twitch response experiment.[129] First, the spinal cord of the fully anesthetized rabbit was completely severed. Later, the motor nerve was severed. **B,** results of the localized twitch response experiment. Abscissa: time elapsed in minutes; Ordinate: mean duration of localized twitch responses in milliseconds. As soon as the spinal cord was severed, the localized twitch response disappeared due to spinal shock. As the animal recovered from spinal shock, the localized twitch response slowly returned. However, after the motor nerve was severed, localized twitch responses became unobtainable and remained that way. Based on published data.[129]

nidus of sensitization in the muscle, including bursitis or enthesopathy in the region where the muscle attaches, may be able to initiate an LTR. Although LTRs were significantly more likely to occur at a TrP site than out of a TrP,[248] the fact that responses did occur as the result of needling two other sites supports the possibility of less specific sites of origin for this response than just active loci at motor endplates.

Hong and coworkers reported several studies that examined propagation of the rabbit localized twitch response. The initial rabbit study[128] reported that vigorous twitch responses to mechanical stimulation with a solenoid device were terminated by anesthetizing the muscle nerve supplying the muscle or by severing it with scissors. A subsequent study[129] of five rabbits examined the effect on the twitch response by first transecting the spinal cord at the T_4, T_5, or T_6 level and later cutting the sciatic nerve, as illustrated in Figure 2.33A. Figure 2.33B presents the duration of localized twitch responses recorded before and repeatedly after each procedure. Immediately following spinal cord transection rostral to segments supplying the biceps femoris muscle, no twitch response was obtainable. As the spinal cord recovered from spinal shock caused by the spinal surgery, the duration of twitch responses recovered to their presurgical level. Following sectioning of the sciatic nerve, the duration of twitch responses again fell to zero and remained there until the end of the experiment an hour later. These results indicate that the rabbit localized twitch response is propagated essentially as a spinal reflex that is not dependent on supraspinal influences.

A human study[124] followed changes in the local twitch response during the recovery phase after a brachial plexus injury that resulted in complete loss of nerve conduction. The EMG activity of twitch responses recovered in parallel with the recovery of nerve conduction. This result is consistent with the other evidence that the twitch response is largely if not completely a spinal reflex. The reflex pathway is illustrated schematically in Figure 2.34.

In a study[266] of the motor innervation of the cat gastrocnemius muscle, the authors described and illustrated what portion of the muscle contracted in response to electrical stimulation of one fascicle of the motor

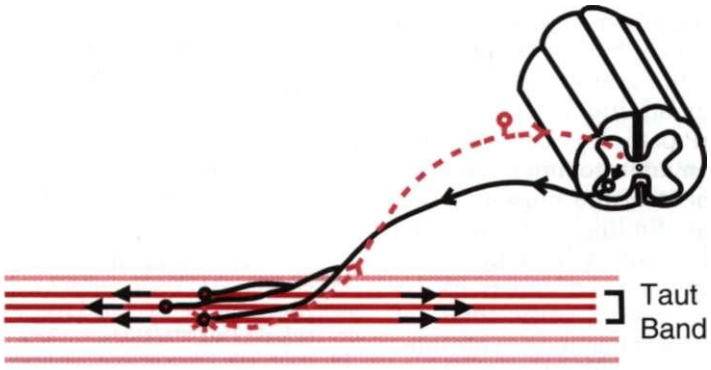


Figure 2.34. Schematic of the most likely reflex pathway followed by a local twitch response originating in a myofascial trigger point. The *open black circle with red rays* identifies the active locus and its associated sensitized nociceptor fibers of a myofascial trigger point. The *dotted red line* represents the nociceptor pathway to the dorsal horn neuron *(solid red circle)* which connects through internuncial neurons to ventral horn cells. The *open black circle* in the ventral horn locates a motor neuron. The *curved black line* represents one motor unit of the return limb of the reflex. The apparent increase in responsiveness of *a*-motoneurons whose endplates exhibit the spontaneous electrical activity of active loci would account for a preferential response of an involved motor unit (or units) *(dark red lines)* which are present in the taut band. The muscle fibers of uninvolved motor units are *light red lines.* The local twitch is the motor response resulting from the activation of the involved motor unit(s) of the taut band. The *arrows* show the direction of action potentials in the nerve and in the muscle fibers.

nerve. The size of the contraction that they described fit nicely the appearance of twitch responses in rabbit muscle.[128] This is compatible with the other evidence suggesting that an LTR is the contraction of as many muscle fibers as belong to one or possibly several interdigitating motor units.

Taut Band. In muscles accessible to palpation, a myofascial TrP is consistently found within a palpable taut band. Theoretically and clinically, the taut band is a basic diagnostic criterion of a TrP.[94] However, by itself, it is an ambiguous finding. Taut bands are found in asymptomatic subjects with no evidence of tender nodules or TrPs.[293] Other muscle structures such as intermuscular and intramuscular septa can feel deceptively similar.

The source of increased tension palpable in the taut band is identified by the mechanism illustrated in Figure 2.25. Other hypotheses fail to explain how the tension can be relieved within seconds or minutes after inactivation of the TrP.

From a research point of view, the taut band remains one of the more neglected phenomena that are associated with musculoskeletal pain. It is difficult to measure with accuracy, specificity, and reliability. Studies indicate that palpable taut bands can be present in normal muscles without any other indication of abnormality such as tenderness or pain.[206,293] This observation suggests that the symptoms of a clinical TrP represent additional spread and propagation of TrP pathology beyond a few simple contraction knots to much more extensive involvement of the muscle fibers. The pathological findings of club-like swellings, double knots with empty sarcoplasm between them, and areas of degenerating fibers may identify some of those additional complications. The integrated trigger point hypothesis provides a basis for histopathological research to clarify the origin of taut bands. Such studies could examine the development of taut bands following the experimental induction of contraction knots.

REFERENCES

1. Acquadro MA, Borodic GE: Treatment of myofascial pain with botulinum A toxin [Letter], *Anesthesiology 80(3):705-706,* 1994.
2. Adler I: Muscular rheumatism. *Med Rec* 57:529-535, 1900.
3. Aidley DJ: *The Physiology of Excitable Cells.* The University Press, Cambridge, 1971.
4. Althaus J: *Elektricitat in der Medizin: Mit besonderer Riicksicht auf Physiologie, Diagnostik und Therapie.* Druck und Verlag von Georg Reimer, Berlin, 1860 (p. 118).
5. Aquilonius SM, Askmark H, Gillberg PG, *et al.:* Topographical localization of motor endplates in cryosections of whole human muscles. *Muscle Nerve* 7:287-293, 1984.
6. Arat A: *Neck Sprains as Muscle Injury, Tension Headache and Related Conditions.* Ed. 2. Guynes Printing Company.El Paso, Texas, 1973 (pp. 134,136).
7. Baldry P: *Acupuncture, Trigger Points, and Musculoskeletal Pain.* Churchill Livingstone, New York, 1989.
8. Baldry P: Superficial dry needling at myofascial trigger point sites. /*Musculoske Pain* 3(3j;117-126,1995.
9. Bardeen CR: The musculature. Section 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (p. 355).
10. Bateman JE: *The Shoulder and Neck.* W.B. Saunders, Philadelphia, 1972 (pp. 182).
11. Bates T: Myofascial pain. Chapter 14. In: *Ambulatory Pediatrics II.* Edited by Green M, Haggerty RJ. W.B. Saunders, Philadelphia, 1977 (pp. 147, 148).
12. Bates T, Grunwaldt E: Myofascial pain in childhood. *JPediatr* 53:198-209, 1958.
13. Belgrade M: Two decades after ping-pong diplomacy: is there a role for acupuncture in American pain medicine? *APS J 3(2):73-S3,* 1994.
14. Bell WE: *Temporomandibular Disorders: Classification, Diagnosis, Management.* Year Book Medical Publishers, Chicago, 1990.
15. Bendall JR: *Muscles, Molecules, and Movement: An Essay in the Contraction of Muscles.* American Elsevier Publishing Company, Inc., New York, 1969.
16. Bendtsen L, Jensen R, Jensen NK, et al.: Muscle palpation with controlled finger pressure: new equipment for the study of tender myofascial tissues. *Pain* 59:235-239, 1994.
17. Bennett RM: Myofascial pain syndromes and the fibromyalgia syndrome: a comparative analysis, Ch. 2. In: *Myofascial Pain and Fibromyalgia,* Vol. 17 of *Advances in Pain Research and Therapy.* Edited by Fricton JR, Awad EA. Raven Press, New York, 1990 (pp. 43-65).
18. Bennett RM: The contribution of muscle to the generation of fibromyalgia symptomatology. *J Musculoske Pain* 4(J/2j:35-59, 1996.
19. Bohr, TW: Fibromyalgia syndrome and myofascial pain syndrome: do they exist? *Neurolog Clin* J3'2j:365-384, 1995.
20. Bonica JJ: Myofascial syndromes with trigger mechanism. In: *The Management of Pain.* Edited by Bonica JJ. Lea & Febiger, Philadelphia, 1953 (pp. 1150-1151).
21. Bonica JJ: Preface. In: *Advances in Neurology,* Vol. 4. Edited by Bonica JJ. Raven Press, New York, 1974 (p. vii).
22. Bourne IH: Treatment of painful conditions of the abdominal wall with local injection. *Practitioner* 224:921-925, 1980.
23. Brown BR: Diagnosis and therapy of common myofascial syndromes. *JAMA* 239:646- 648, 1978.

24. Brown WF, Varkey GP: The origin of spontaneous electrical activity at the end-plate zone. *Ann Neurol* 20:557-60, 1981.
25. Bruce E: Myofascial pain syndrome: early recognition and comprehensive management. *AAOHN J* 43(9j:469-474, 1995.
26. Briickle W, Suckfull M, Fleckenstein W, *et al.:* Gewebe-p02-Messung in der verspannten Riickenmuskulatur (m. erector spinae). *Zeitschrift fur Rheumatologie* 49:208-216, 1990.
27. Buchthal F, Guld C, Rosenfalck P: Innervation zone and propagation velocity in human muscle. *Acta Physiologica Scand* 35:175-190, 1955.
28. Buchthal F, Rosenfalck P: Spontaneous electrical activity of human muscle. *Electroenceph Clin Neurophysiol* 20:321-336, 1966.
29. Buchthal F, Schmalbruch H: Motor unit of mammalian muscle. *Physiol Rev* 60:90-142, 1980.
30. Burnette JT, Ayoub MA: Cumulative trauma disorders. Part I. The problem. Pain *Management* 2:196-209, 1989.
31. Cailliet R: *Low Back Pain Syndrome.* Ed. 3. F. A. Davis, Philadelphia, 1981 (pp. 86-87).
32. Carlson CR, Okeson JP, Falace DA, *et al.:* Reduction of pain and EMG activity in the masseter region by trapezius trigger point injection. *Pain* 55:397-400, 1993.
33. Cervero F: Visceral nociception: peripheral and central aspects of visceral nociceptive systems. *Trans R Soc Lon [B]* 308:325-327, 1985.
33a. Chen JT, Chen SU, Kuan TS, et al. Effect of phentolamine on spontaneous electrical activity of active loci in a myofascial trigger spot of rabbit skeletal muscle. *Arch Phys Med Rehabil* (In Press)
34. Cheshire WP, Abashian SW, Mann JD: Botulinum toxin in the treatment of myofascial pain syndrome. *Pain* 59:65-69, 1994.
35. Chin NK, Cope M, Pang M: Number and distribution of spindle capsules in seven hindlimb muscles of the cat. In: *Symposium on Muscle Receptors,* edited by Barker D. Hong Kong University Press, 1962, pp. 241-248.
36. Christensen E: Topography of terminal motor innervation in striated muscles from stillborn infants. In: *The Innervation of Muscle.* Edited by Bouman HD, Woolf AL. Williams & Wilkins, Baltimore, 1960 (pp. 17-26).
37. Chu J: Dry needling (intramuscular stimulation) in myofascial pain related to lumbosacral radiculopathy. *Eur J Phys Med Rehabil* 5(4j:106-121, 1995.
38. Clark GT: A critique of the stress-hyperactivity-pain theory of myogenic pain. *Pain Forum 5(1):70-73,* 1996.
39. Clemente CD: *Gray's Anatomy of the Human Body,* American Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 429).
40. *Ibid.* (Fig. 6.2).
41. Coers C: Contribution a l'etude de la jonction neuromusculaire. Donnees nouvelles concernant la structure de l'arborisation terminale et de l'appareil sousneural chez l'homme. *Arch Biol, Paris,* 64:133-147, 1953.
42. Coers C: Contribution a l'etude de la jonction neuromusculaire. II. Topographie zonale de ['innervation motrice terminale dans les muscles strips. *Arch Biol, Paris,* 64.495-505, 1953.
43. Coers C: Structural organization of the motor nerve endings in mammalian muscle spindles and other striated muscle fibers. In: *The Innervation of Muscle.* Edited by Bouman HD, Woolf AL. Williams & Wilkins, Baltimore, 1960 (pp. 40-49).
44. Coers C, Woolf AL: *The Innervation of Muscle, A Biopsy Study.* Blackwell Scientific Publications, Oxford, 1959 (Figs. 9-15).
45. Coulehan JL: Primary fibromyalgia. *Am Fam Phys 32(3):170-177,* 1985.
46. D'ambrosia RH: *Musculoskeletal Disorders: Regional Examination and Differential Diagnosis.* J.B. Lippincott, Philadelphia, 1977 (p.332).
47. Danneskiold-Samsoe B, Christiansen E, Andersen RB: Regional muscle tension and pain ("Fibrositis"). *Scand J Rehabil Med* 25:17-20, 1983.
48. Danneskiold-Samsoe B, Christiansen E, Andersen RB: Myofascial pain and the role of myoglobin. *Scand J Rheumatol* 25:174-178, 1986.
49. Defalque RJ: Painful trigger points in surgical scars. *Anesth Analg* 62:518-520, 1982.
50. Denslow JS, Korr IM, Krems AD: Quantitative studies of chronic facilitation in human motoneuron pools. *Am J Physiol* 205:229-238, 1947.
51. de Valera E, Raftery H: Lower abdominal and pelvic pain in women. In: *Advances in Pain Research and Therapy,* Vol. 1. Edited by Bonica JJ, Albe-Fessard D. Raven Press, New York. 1976 (pp. 935-936J.
52. Diakow PRP: Thermographic imaging of myofascial trigger points. / *Manipulative Physiol Ther* 22:114-117, 1988.
53. Diakow PR: Differentiation of active and latent trigger points by thermography. / *Manipulative Physiol Ther* 25(7j:439-441,1992.
54. Dittrich RJ: Low back pain—referred pain from deep somatic structure of the back. *J Lancet 73:63-68,* 1963.
55. Diwaker HN, Stothard J: What do doctors mean by tenosynovitis and repetitive strain injury? *Occup Med* 45(2J:97-104, 1995.
56. Donaldson CCS, Skubick DL, Clasby RG, Cram JR: The evaluation of trigger-point activity using dynamic EMG techniques. *Am J Pain Manag* 4:118-122, 1994.
57. Dubowitz V, Brooke MH: *Muscle Biopsy: A Modern Approach.* W. B. Saunders Company Ltd, Philadelphia, 1973 (pp. 76, 77).
58. Dumitru D, DeLisa JA: AAEM minimonograph #10: Volume conduction. *MuscleNerve* 24:606-624,1991.
59. Durette MR, Rodriquez AA, Agre JC, *et al.:* Needle electromyographic evaluation of patients with myofascial or fibromyalgic pain. *Am] Phys Med Rehabil* 70'3j:154-156, 1991.
60. Dutta CR, Basmajian JV: Gross and histological structure of the pharyngeal constrictors in the rabbit. *AnatRec 137:127-134,* 1960.
61. Elson LM: The jolt syndrome. Muscle dysfunction following low-velocity impact. *Pain Management* 3:317-326, 1990.
62. English AW, Wolf SL, Segal RL: Compartmentalization of muscles and their motor nuclei: the partitioning hypothesis. *Phys Ther* 73f22j:857-867,1993.
63. Ertekin C, Arag N, Uludag B, *et al:* Enhancement of "end-plate monophasic waves" during an attack of hypokalemic periodic paralysis. Letter to the Editor. *Muscle Nerve* 29f6j:680-681, 1996.

64. Fassbender HG: Nonarticular rheumatism. In: *Pathology of Rheumatic Disease.* Springer- Verlag, New York, 1975:303-314.
65. Fassbender HG, Martens KD: [Critical considerations of the pathogenesis of "soft tissue rheumatism" (fibromyalgia) and its therapeutic consequences]. *Zeitschrift fur Orthopadie und Ihre Grenzgebiete 130(2):* 99-103, 1992.
66. Fassbender HG, Wegner K: Morphologie und Pathogenese des Weichteilrheumatismus. *Z Rheumaforsch* 32:355-374, 1973.
67. Fatt P, Katz B: Spontaneous subthreshold activity at motor nerve endings. / *Physiol* 237:109-128, 1952.
68. Fine PG: Myofascial trigger point pain in children. *JPediatr 111:* 547-548, 1987.
69. Fine PG, Milano R, Hare BD: The effects of myofascial trigger point injections are naloxone reversible. *Pain* 32:15-20, 1988.
70. Finestone DH, Willingham SG, Koffman GE, et *al.:* Physical and psychiatric impairment in patients with myofascial pain syndrome compared to patients with fibromyalgia [Abstract]. / *Musculoske Pain 3(Suppl l)m,* 1995.
71. Fischer AA: Diagnosis and management of chronic pain in physical medicine and rehabilitation, Chapter 8. In: *Current Therapy in Physiatry.* Edited by Ruskin AP. W.B. Saunders, Philadelphia, 1984 (pp. 123-154).
72. Fischer AA: Pressure threshold meter: its use for quantification of tender spots. *Arch Phys Med Rehabil* 67:836-838, 1986.
73. Fischer AA: Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. *Pain* 30:115-126, 1987.
74. Fischer AA: Documentation of myofascial trigger points. *Arch Phys Med Rehabil* 69:286-91, 1988.
75. Fischer AA: Trigger point injection. In: *Physiatric Procedures in Clinical Practice.* Edited by Lennard TA. Hanley & Belfus, Philadelphia, 1995 (pp. 28-35).
76. Fischer AA: Trigger point injections can be performed painfree using preinjection block (PIB) [Abstract], *f Musculoske Pain 3(Suppl 1* J.140,1995.
77. Fischer AA: New developments in diagnosis of myofascial pain and fibromyalgia. *Phys Med Rehabil Clin North Am 8(1):1-21,* 1997.
78. Fischer AA: New approaches in treatment of myofascial pain. *Phys Med Rehabil Clin North Am* 8(2;:153-169, 1997b.
79. Fischer AA, Chang CH: Temperature and pressure threshold measurements in trigger points. *Thermology* 2:212-215, 1986.
80. Fishbain DA, Goldberg M, Meagher BR, *et al:* Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. *Pain* 26:181-197, 1986.
81. Fransen J, Russell IJ: *The Fibromyalgia Help Book.* Smith House Press, St. Paul, 1996.
82. Fricton JR: Myofascial Pain, Chapter 9. In: *Bail-Here's Clinical Rheumatology: Fibromyalgia and Myofascial Pain Syndromes,* Vol. 8, No. 4. Edited by Masi AT. Bailliere Tindall (Saunders), Philadelphia, 1994, (pp. 857-880).
83. Fricton JR, Kroening R, Haley D, *et al:* Myofascial pain syndrome of the head and neck: A review of clinical characteristics of 164 patients. *Oral Surg* 60:615-623, 1985.
84. Frohlich D, Frohlich R: Das Piriformissyndrom: eine haufige Differentialdiagnose des lumboglutaalen Schmerzes (Piriformis syndrome: a frequent item in the differential diagnosis of lumbogluteal pain). *Manuelle Medizin 33:7-10,* 1995.
85. Froriep: *Ein Beitrag zur Pathologie und Therapie des Rheumatismus.* Weimar, 1843.
86. Gedalia A, Press J, Klein M, Buskila D: Joint hypermobility and fibromyalgia in schoolchildren. *Ann Rheum Dis* 52f7j:494-496, 1993.
87. Gerwin RD: Myofascial pain. The future of pain management: the perspective of a specialist in myofascial pain. *Am J Pain Manage 1(1):9-10,* 1991.
88. Gerwin RD: The management of myofascial pain syndromes. /*Musculoske Pain* 2(3/4j:83-94, 1993.
89. Gerwin RD: Neurobiology of the Myofascial Trigger Point, Chap. 3. In: *Baillere's Clinical Rheumatology: Fibromyalgia and Myofascial Pain Syndromes,* Vol. 8, No. 4. Edited by Masi AT. Bailliere Tindall, London, 1994, (pp. 747-762).
90. Gerwin RD: A study of 96 subjects examined both for fibromyalgia and myofascial pain [Abstract]. / *Musculoske Pain 3(Suppl* 2J.121, 1995.
91. Gerwin RD: Personal communication, 1996.
92. Gerwin RD, Duranleau D: Ultrasound identification of the myofascial trigger point [Letter]. *Muscle Nerve* 20:767-768, 1997.
93. Gerwin RD, Shannon S, Hong C-Z, Hubbard D, Gevirtz R: Identification of myofascial trigger points: inter-rater agreement and effect of training [Abstract]. /*Musculoske Pain 3(Suppl* 3j:55, 1995.
94. Gerwin RD, Shannon S, Hong CZ, Hubbard D, Gevirtz R: Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
95. Gillette RG, Kramis RC, Roberts WJ: Characterization of spinal somatosensory neurons having receptive fields in lumbar tissues of cats. *Pain* 54:85-98,1993.
96. Glogowski G, Wallraff J: Ein Beitrag zur Klinik und Histologie der Muskelharten (Myogelosen). Z *Orthop* 80:237-268, 1951.
97. Goldberg M, Murray TG: Analgesic-associated nephropathy. *N Engl J Med* 299:716-717, 1978.
98. Goldstein JA: *Betrayal by the Brain: The Neurological Basis of Chronic Fatigue Syndrome, Fibromyalgia Syndrome, and Related Neural Network Disorders.* Haworth Medical Press, New York, 1996.
99. Good MG: Die primare Rolle der Muskulatur in der Pathogenese der rheumatischen Krankheit und die therapeutische Losung des Rheumaproblems. *Medizinische (Stuttgart)* 33:450-454, 1957.
100. Gorrell RL: Troublesome ankle disorders and what to do about them. *Consultant* 36:64- 69, 1976.
101. Gowers WR: Lumbago: its lesions and analogues. *Br Med f* 3:117-121, 1904.
102. Graff-Radford B: Myofascial trigger points: their importance and diagnosis in the dental office. / *Dent Assoc S Afr* 39:237-240, 1984.
103. Graff-Radford S, Jaeger B, Reeves JL: Myofascial pain may present clinically as occipital neuralgia. *Neurosurgery* 39(4J:610-613, 1986.
104. Granges G, Littlejohn G: Prevalence of myofascial pain syndrome in fibromyalgia syndrome and regional pain syndrome: a comparative study. *J Musculoske Pain* 3/2J.19-35, 1993.
105. Graven-Nielsen T, Svensson P, Arendt-Nielsen L: Effects of experimental muscle pain on muscle ac-

tivity and coordination during static and dynamic motor function. *Electwenceph Clin Neurophysiol* 205(2j:156-164, 1997.

106. Greenbaum DS, Greenbaum RB, Joseph JG, *et al.:* Chronic abdominal wall pain: Diagnostic validity and costs. *Dig Dis Sci* 39f9j:1935-1941, 1994.
107. Gross D: *Therapeutische Lokalanasthesie.* Hippokrates Verlag, Stuttgart, 1972 (p. 142).
108. Grzesiak RC: Psychological considerations in myofascial pain, fibromyalgia, and related musculoskeletal pain, Chap. 4. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 61-90.
109. Gunn CC: Prespondylosis and some pain syndromes following denervation supersensitivity. *Spine 5(2j:W5,* 1980.
110. Gunn CC: *The Gunn Approach to theTreatment of Chronic Pain, Intramuscular Stimulation for Myofascial Pain of Radiculopathic Origin.* Ed. 2. Churchill Livingston, New York, 1996.
111. Gunn CC: Transcutaneous Neural Stimulation, Needle Acupuncture & 'Teh Ch'i' Phenomenon. *Am J Acupuncture* 4(4j:317-322, 1976.
112. Gutstein M: Diagnosis and treatment of muscular rheumatism. *Br f Phys Med* 3:302-321, 1938.
113. Hackett GS: *Ligament and Tendon Relaxation Treated by Prolotherapy,* Ed. 3. Charles C Thomas, Springfield, 111., 1958 (pp. 27-36).
114. Hagbarth KE, Finer B: The plasticity of human withdrawal reflexes to noxious skin stimuli in lower limbs. Progr *Brain Res (Amst)* 2:65-78, 1963.
115. Hagberg H, Kvarnstrom S: Muscular endurance and electromyographic fatigue in myofascial shoulder pain. *Arch Phys Med Rehabil* 65:522-525,1984.
116. Headley BJ: Evaluation and treatment of myofascial pain syndrome utilizing biofeedback. In: *Clinical EMG for Surface Recordings,* Vol. 2. Edited by Cram JR. Clinical Resources, Nevada City, 1990 (pp. 235-254).
117. Headley BJ: The use of biofeedback in pain management. *Physical Therapy Practice* 2(2j:29-40,1993.
118. Headley BJ: Physiologic risk factors. In: *Management of Cumulative Trauma Disorders.* Edited by Sanders M. Butterworth-Heineman, London, 1997 (pp. 107-127).

118.a Headley B: *When Movement Hurts: A Self-help Manual for Treating Trigger Points.* Innovative Systems for Rehabilitation, Boulder, Colorado, 1997.

119. Hess MJ, Borg-Stein J, Goldenberg DL: Role of rehabilitation in the management of fibromyalgia. *Arch Phys Med Rehabil* 76:1049, 1995. [Abstract]
120. Heuser J, Miledi R: Effect of lanthanum ions on function and structure of frog neuromuscular junctions. *Proc R Soc Lond B* 279:247-260, 1971.
121. Hoheisel U, Mense S, Simons DG, *et al:* Appearance of new receptive fields in rat dorsal horn neurons following noxious stimulation of skeletal muscle: a model for referred muscle pain? *NeurosciLett* 253:9-12, 1993.
122. Hong CZ: Myofascial trigger point injection. *Crit Rev Phys Med Rehabil* 5:203-217, 1993.
123. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am f Phys Med Rehabil* 73:256-263, 1994.
124. Hong CZ: Persistence of local twitch response with loss of conduction to and from the spinal cord. *Arch Phys Med Rehabil* 75:12-16, 1994.
125. Hong CZ, Chen YN, Twehous DA, *et al.:* Pressure threshold for referred pain by compression on the trigger point and adjacent areas. *J Musculoske Pain 4(3)m-79,* 1996.
126. Hong CZ, Hsueh TC: Difference in pain relief after trigger point injections in myofascial pain patients with and without fibromyalgia. Arch Phys Med Rehabil 77(11):1161-1166, 1996.
127. Hong CZ, Simons DG: Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. / *Musculoske Pain* 2f2j:89-131, 1993.
128. Hong CZ, Torigoe Y: Electrophysiological characteristics of localized twitch responses in responsive taut bands of rabbit skeletal muscle, *f Musculoske Pain 2(2):17-43,* 1994.
129. Hong CZ, Torigoe Y, Yu J: The localized twitch responses in responsive taut bands of rabbit skeletal muscle fibers are related to the reflexes at spinal cord level. *J Musculoske Pain* 3(2j;15-34, 1995.
130. Hsieh JC, Belfrage M, Stone-Elander S, *et al.:* Central representation of chronic ongoing neuropathic pain studied by positron emission tomography. *Pain* 63:225-236, 1995.
131. Hubbard DR: Personal communication, 1994.
132. Hubbard DR: Chronic and recurrent muscle pain: pathophysiology and treatment, and review of pharmacologic studies. / *Musculoske Pain* 4(2/2j:124-143, 1996.
133. Hubbard DR, Berkoff GM: Myofascial trigger points show spontaneous needle EMG activity. *Spine* 28:1803-1807, 1993.
134. Inman VT, Saunders JB: Referred Pain from skeletal structures. / *Nerv Ment Dis* 99:660- 667, 1944.
135. Institute of Medicine: *Pain and Disability: Clinical Behavioral and Public Policy Perspectives.* National Academy Press, Washington, D.C., May 1987.
136. International Anatomical Nomenclature Committee: *Nomina Anatomica.* Excerpta Medical Foundation, Amsterdam, 1966 (pp. 38-43).
137. Ito Y, Miledi R, Vincent A: Transmitter release induced by a "factor" in rabbit serum. *Proc R Soc LondB* 287:235-241, 1974.
138. Ivanichev GA: *[Painful Muscle Hypertonus].* Russian. Kazan University Press, Kazan, 1990.
139. Jacob AT: Myofascial pain. In: *Physical Medicine and Rehabilitation: State of the Art Reviews,* Volume 5/Number 3. Edited by Schwab CD. Hanley & Belfus, Inc., Philadelphia, 1991, pp.573-583.
140. Jaeger B: Differential diagnosis and management of craniofacial pain, Chap. 11. In: *Endodontics,* Ed. 4. Edited by Ingle JI, Bakland LK. Williams & Wilkins, Baltimore, 1994 (pp. 550-607).
141. Jaeger B, Skootsky SA: Double blind, controlled study of different myofascial trigger point injection techniques. *Pain 4(Suppl):S292{Absti.)* 1987.
142. Janda V: Evaluation of muscular imbalance, Ch. 6. In *Rehabilitation of the Spine: A Practitioner's Manual.* Edited by C. Liebenson. Williams & Wilkins, Baltimore, 1996 (pp. 97-112).
143. Janssens LA: Trigger points in 48 dogs with myofascial pain syndromes. *Vet Surg* 20:274-278,1991.
144. Jayson MI: Fibromyalgia and trigger point injections. *Bull Hosp Joint Dis 55(4):\76-177,* 1996.
145. Jensen R: Mechanisms of spontaneous tension-

type headaches: an analysis of tenderness, pain thresholds and EMG. *Pain* 64:251-256, 1995.
146. Johnson EW: The myth of skeletal muscle spasm [Editorial]. *Am J Phys Med 68(1*1989.
147. Jones RV Jr, Lambert EH, Sayre GP: Source of a type of "insertion activity" in electromyography with evaluation of a histologic method of localization. *Arch Phys Med Rehabil* 36:301-310, 1955.
148. Katz J, Jackson M, Kavanagh BP, Sandler AN: Acute pain after thoracic surgery predicts long-term post-thoracotomy pain. *Clin J Pain* 22:50-55, 1996.
149. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3.175-190, 1938.
150. Kellgren JH: Deep pain sensibility. *Lancet* 2:943-949, 1949.
151. Kelly M: The treatment of fibrositis and allied disorders by local anesthesia. *Med JAust 1*:294-298,1941.
152. Kelly M; The relief of facial pain by procaine (novocain) injections. *J Am Geriatr Soc* 2 2:586-596,1963.
153. Kimura J: *Electrodiagnosis in Diseases of Nerve and Muscle,* Vol. 2. F.A. Davis, Philadelphia, 1989.
154. Korr IM, Thomas PE, Wright HM: Clinical significance of the facilitated state. *JAOA* 54:277-282,1955.
155. Kraft GH, Johnson EW, LaBan MM: The fibrositis syndrome. *ArchPhys Med Rehabil* 49:155-162,1968.
156. Kraus H: Behandlung akuter Muskelharten. *Wien Klin Wochenschr* 50:1356-1357, 1937.
157. Kraus H: Diagnosis and treatment of low back pain. *GP* 5(4j:55-60, 1952.
158. Kraus H: Evaluation and treatment of muscle function in athletic injury. *Am J Surg* 98:353-361,1959.
159. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (pp.95, 107).
160. Kraus H, Fischer AA: Diagnosis and treatment of myofascial pain. *Mt Sinai J Med* 58:235-249, 1991.
161. Kruse RA Jr, Christiansen JA: Thermographic Imaging of myofascial trigger points: a follow-up study. *Arch Phys Med Rehabil* 73:819-823, 1992.
162. Lange F: Die Muskelharten der Beinmuskein. *Munch Med Wochenschr* 72:1626-1629, 1925.
163. Lange F, Eversbusch G: Die Bedeutung der Muskelharten fur die allgemeine Praxis. *Munch Med Wochenschr* 68:418-420, 1921.
164. Lange M: *Die Muskelharten (Myogelosen).* Miinchen, J. F. Lehmann's Verlag, 1931.
165. Lawrence RM: Osteopuncture: theory and practice. Presented at the annual meeting of the North American Academy of Manipulative Medicine, 1977.
166. Leriche R: Des effetes de l'anesthesie a la novocaine des ligament et des insertions tendineuses peri-articulaires dans certaines maladies articulaires et dans vices de position fonctionnels des articulations. *Gazette des Hopitaux* 203:1294,1930.
167. Lewis C, Gevirtz R, Hubbard D, ef *al.:* Needle trigger point and surface frontal EMG measurements of psychophysiological responses in tension-type headache patients. *Biofeedback Self Regul 19(3):274-275,* 1994.
168. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Butterworth, London, 1985.
169. Lewit K: Chain reactions in disturbed function of the motor system. *Manual Medicine* 3:27-29, 1987.
170. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System,* 2nd Ed., Butterworth Heinemann, Oxford, 1991.
171. Liley AW: An investigation of spontaneous activity at the neuromuscular junction of the rat. / *Physiol* 232:650-666, 1956.
172. Llewellyn LJ, Jones AB: *Fibrositis.* London, Heinemann, 1915.
173. Lockhart RD, Hamilton GF, Fyfe FW: *Anatomy of the Human Body,* Ed. 2. J.B. Lippincott, Philadelphia, 1969 (p. 144).
174. Loh L, Nathan PW, Schott GD, *et al.:* Acupuncture versus medical treatment for migraine and muscle tension headaches. / *Neurol Neurosurg Psych* 47:333-337, 1984.
175. Lowe JC: The subluxation and the trigger point: measuring how they interact. *Chiropractic Journal 8(10):32* & 35, 1993.
176. Lowe JC, Cullum ME, Graf LH Jr, *et al.:* Mutations in the c-erbA beta 1 gene: do they underlie euthyroid fibromyalgia? *Medical Hypothesis 48(2):125-*135, 1997.
177. Macdonald AJ: Abnormally tender muscle regions and associated painful movements. *Pain* 8:197-205, 1980.
178. Maloney M: Personal communication, 1976.
179. Mannion AF, Dolan P: Relationship between mechanical and electromyographic manifestations of fatigue in the quadriceps femoris muscle of humans. *Muscle Nerve 4(Suppl):S46,* 1996.
180. Margoles MS: Stress neuromyelopathic pain syndrome (SNPS). / *Neurol Orthop Surg* 4:317-322, 1983.
181. Margolis M: Personal communication, 1996.
182. Masi AT: Review of the epidemiology and criteria of fibromyalgia and myofascial pain syndromes: Concepts of illness in populations as applied to dysfunctional syndromes. / *Musculoske Pain* 2(3/4j:113-157, 1993.
183. McCain GA: A clinical overview of the fibromyalgia syndrome. *(Musculoske Pain 4(1/2)^-34,*1996.
184. McClaflin RR: Myofascial pain syndrome. Primary care strategies for early intervention. *Postgrad Med* 96f2j:56-59, 63-66, 69-70, 1994.
185. McMillan AS, Hannam AG: Motor-unit territory in the human masseter muscle. *Arch Oral Biol 36(6):435-441,* 1991.
186. McNulty WH, Gevirtz RN, Hubbard DR, *et al.:* Needle electromyographic evaluation of trigger point response to a psychological stressor. *Psychophysiology* 32'3j:313-316, 1994.
187. Melzack R, Stillwell DM, Fox EJ: Trigger points and acupuncture points for pain: correlations and implications. *Pain* 3:3-23, 1977.
188. Mense S: Nociception from skeletal muscle in relation to clinical muscle pain. *Pain* 54:241-289, 1993.
189. Mense S: Peripheral mechanisms of muscle nociception and local muscle pain. / *Musculoske Pain 1(1):133-170,* 1993.
190. Mense S: Referral of muscle pain: new aspects. *Am Pain Soc J* 3.1-9, 1994.
190a. Mense S: Pathophysiologic basis of muscle pain syndromes. Phys Med Rehabil Clin N Amer 8:23-53, 1997.
191. Mense S, Simons DG: *Muscle Pain.* Williams & Wilkins, Baltimore (in press).
192. Michele AA, Davies JJ, Krueger FJ, *et al.:* Scapulocostal syndrome (fatigue-postural paradox). *NY State J Med* 50:1353-1356, 1950.
193. Miehlke K, Schulze G, Eger W: Klinische und ex-

perimentelle Untersuchungen zum Fibrositissyndrom. *Z Rheumaforsch* 29:310-330, 1960.

194. Miller B: Manual therapy treatment of myofascial pain and dysfunction, Chap. 13. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 415-454).
195. Mitchell FL Jr, Moran PF, Pruzzo NA: *An Evaluation and Treatment Manual of Osteopathic Muscle Energy Procedures.* Mitchell, Moran and Pruzzo, Associates. Valley Park, MO, 1979.
196. Moldofsky H: The contribution of sleep-wake physiology to fibromyalgia. Chapter 13. In: *Advances in Pain Research and Therapy. Vol. 17: Myofascial Pain and Fibromyalgia.* Edited by Fricton JR, Awad EA. Raven Press, New York, 1990 (pp.227-240).
197. Needham DM: Biochemistry of muscle, Chapter 8. In: *The Structure and Function of Muscle,* Ed. 2, Vol. 3. Edited by Bourne GH. Academic Press, New York, 1973 (p.377).
198. Nice DA, Riddle DL, Lamb RL, *et al:* Intertester reliability of judgments of the presence of trigger points in patients. *Arch Phys Med Rehabil* 73:893-898, 1992.
199. Nielsen AJ: Spray and stretch for myofascial pain. *Phys Ther* 58:567-569, 1978.
200. Nielsen AJ: Case study: myofascial pain of the posterior shoulder relieved by spray and stretch. / *Orthop Sports Phys Ther* 3:21-26, 1981.
201. Njoo KH, Van der Does E: The occurrence and interrater reliability of myofascial trigger points in the quadratus lumborum and gluteus medius: a prospective study in non-specific low back pain patients and controls in general practice. *Pain* 58:317-323,1994.
202. Olesen J, Jensen R: Getting away from simple muscle contraction as a mechanism of tension-type headache [editorial]. *Pain* 46:123-124, 1991.
203. Ormandy L: Scapulocostal Syndrome. *Va Med Q Spring* 222(2J;105-108, 1994.
204. Pace JB: Commonly overlooked pain syndromes responsive to simple therapy. *Postgrad Med* 58:107-113, 1975.
205. Partanen JV, Nousiainen U: End-plate spikes in electromyography are fusimotor unit potentials. *Neurology* 33.1039-1043, 1983.
206. Pellegrino MJ, Waylonis GW, Sommer A: Familial occurrence of primary fibromyalgia. *Arch Phys Med Rehabil* 70:61-63, 1989.
207. Perry F, Heller PH, Kamiya J, ef *al.:* Altered autonomic function in patients with arthritis or with chronic myofascial pain. *Pain* 39:77-84, 1989.
208. Pomeranz BH: Acupuncture in America: a commentary. *APS Journal* 3'2j:96-100, 1994.
209. Popelianskii II, Zaslavskii ES, Veselovskii VP: [Medicosocial significance, etiology, pathogenesis, and diagnosis of nonarticular disease of soft tissues of the limbs and back.] (Russian) *Vopr Revm* 3:38-43, 1976.
210. Price DD, Rafii A, Watkins LR, *et al.: A* psychophysical analysis of acupuncture analgesia. *Pain* 29:27-42, 1984.
211. Radziemski A, Kedzia A, Jakubowicz M: Number and localization of the muscle spindles in the human fetal sternocleidomastoid muscle. *Folia Morphol 50(l/2):65-70,* 1991.
212. Reeves JL, Jaeger B, Graff-Radford S: Reliability of the pressure algometer as a measure of myofascial trigger point sensitivity. *Pain* 24:313-321, 1986.
213. Reiter RC, Gambone JC: Nongynecologic somatic pathology in women with chronic pelvic pain and negative laparoscope *JReprod Med* 36f4j:253-259, 1991.
214. Reitinger A, Radner H, Tilscher H, *et al:* Morphologische Untersuchung an Triggerpunkten [Morphologic study of trigger points]. *Manuelle Medizin* 34:256-262, 1996.
215. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981 (Table 2).
216. Reynolds MD: The development of the concept of fibrositis. *J Hist Med Allied Sci* 38:5- 35, 1983.
217. Rogers EJ, Rogers R: Fibromyalgia and myofascial pain: either, neither, or both? *Orthop Rev* 28f22j:1217-1224, 1989.
218. Romano TJ: Non-articular rheumatism. / *Musculoske Pain* 2'2J:133-143, 1993.
219. Rosen NB: Myofascial pain: the great mimicker and potentiator of other diseases in the performing artist. *Md Med J* 42f3j:261-266, 1993.
220. Rosen NB: The myofascial pain syndromes. *Phys Med Rehabil Clin North Am* 4(Febj:41-63, 1993.
221. Rosomoff HL, Fishbain DA, Goldberg M, ef *al.:* Physical findings in patients with chronic intractable benign pain of the neck and/or back. *Pain 37(3):279-287,* 1989.
222. Roy RR, Garfinkel A, Ounjian M, *et al.:* Three-dimensional structure of cat tibialis anterior motor units. *Muscle Nerve* 28:1187-1195, 1995.
223. Rubin D: Myofascial trigger point syndromes: an approach to management. *Arch Phys Med Rehabil* 62:107-110, 1981.
224. Russell IJ: Neurochemical pathogenesis of fibromyalgia syndrome. *J Musculoske Pain 4(l/2):61-92,*1996.
225. Salpeter MM: Vertebrate neuromuscular junctions: General morphology, molecular organization, and functional consequences, Chap. 1. In: Tiie *Vertebrate Neuromuscular Junction.* Edited by Salpeter MM. Alan R. Liss, Inc., New York, 1987 (pp. 1-54).
226. Schade H: Beitrage zur Umgrenzung und Klarung einer Lehre von der Erkaltung. *Z Ges Exp Med* 7:275-374, 1919.
227. Schade H: Untersuchungen in der Erkaltungstrage: III. Uber den Rheumatismus, insbesondereden Muskelrheumatismus (Myogelose). *Munch Med Wochenschr* 68:95-99, 1921.
228. Schiffman EL, Fricton JR, Haley DP, *et al:* The prevalence and treatment needs of subjects with temporomandibular disorders. / *Am Dent Assoc* 120:295-303, 1990.
229. Schmidt A: Zur Pathologie und Therapie des Muskelrheumatismus (Myalgie). *Munch Med Wochenschr* 63:593-595, 1916.
230. Schneider MJ: Tender points/fibromyalgia vs. trigger points/myofascial pain syndrome: a need for clarity in terminology and differential diagnosis. / *Manip Physiol Ther* 28(6j:398-406, 1996.
231. Schwarzacher VH: Zurlage der motorischen endplatten in den skeletmuskeln. *Acta Anat* 30:758-774, 1957.

231a. Scudds RA, Heck C, Delaney G, et al.: A comparison of referred pain, resting skin temperature and other signs in fibromyalgia (FM) and myofascial pain syndrome (MPS). *J Musculoske Pain* 3 (Suppl 1): 97, 1995.

232. Scudds RA, Landry M, Birmingham T, *et al.:* The frequency of referred signs from muscle pressure in normal healthy subjects. / *Musculoske Pain 3(Suppl* 3j:99, 1995 (Abstract).
233. Shenoi R, Nagler W: Trigger points related to calcium channel blockers. Letter to the Editor. *Muscle Nerve 19(2):256,* 1996.
234. Simms RW, Goldenberg DL, Felson DT, *et al.:* Tenderness in 75 anatomic sites distinguishing fibromyalgia patients from controls. *Arthritis Rheum* 31.183-187,1988.
235. Simons DG: Muscle pain syndromes - Parts I and II. *Am JPhysMed* 54:289-311,1975; 55:15-42. 1976.
236. Simons DG: Electrogenic nature of palpable bands and "Jump Sign" associated with myofascial trigger points. In: *Advances in Pain Research and Therapy,* Vol 1, edited by Bonica JJ, Albe-Fessard D. Raven Press, New York, 1976 (pp. 913-918).
237. Simons DG: Myofascial pain syndrome due to trigger points, Chapter 45. In: *Rehabilitation Medicine.* Edited by Goodgold J. C.V. Mosby Co., St. Louis, 1988 (pp. 686-723).
238. Simons D: Muscular Pain Syndromes, Chapter 1. In: *Myofascial Pain and Fibromyalgia, Advances in Pain Research and Therapy,* Vol. 17. Edited by Fricton JR, Awad EA. Raven Press, New York, 1990 (pp. 1-41).
239. Simons DG: Referred phenomena of myofascial trigger points, Chap. 28. In: *Pain Research and Clinical Management: New Trends in Referred Pain and Hyperalgesia,* Vol. 27. Edited by Vecchiet L, Albe-Fessard D, Lindblom U, et al. Elsevier Science Publishers, Amsterdam, 1993, (pp. 341-357).
240. Simons DG: Neurophysiological basis of pain caused by trigger points. *Am Pain Soc J* 3.17-19,1994.
241. Simons DG: Myofascial pain syndrome: one term but two concepts: a new understanding [Editorial]. *J Musculoske Pain 3(1):7-13,* 1995.
242. Simons DG: Clinical and etiological update of myofascial pain from trigger points. / *Musculoske Pain 4(1/2):* 97-125, 1996.
243. Simons DG: Taut band tenderness. / *Musculoske Pain* 4(3J:137-140, 1996.
244. Simons DG: Myofascial trigger points: the critical experiment. *J Musculoske Pain* 5f4j:113-118, 1997.
245. Simons DG: Triggerpunkte und Myogelose [trigger points and myogelosisl. Manuelle Medizin 35(6):190-294, 1997.
246. Simons DG, Dexter JR: Comparison of local twitch responses elicited by palpation and needling of myofascial trigger points. / *Musculoske Pain* 3flJ:49-61, 1995.
247. Simons DG, Hong CZ: Comment to Dr. Baldry's dry needling technique. / *Musculoske Pain* 3(4j:81-85, 1995.
248. Simons DG, Hong CZ, Simons LS: Prevalence of spontaneous electrical activity at trigger spots and control sites in rabbit muscle. / *Musculoske Pain* 3(lJ:35-48, 1995.
249. Simons DG, Hong CZ, Simons LS: Nature of myofascial trigger points, active loci. / *Musculoske Pain 3(Supplement 1)62,* 1995.(Abstract)
250. Simons DG, Hong CZ, Simons LS: Spontaneous electrical activity of trigger points. / *Musculoske Pain 3(Supplement* 17:124, 1995.(Abstract)
251. Simons DG, Hong CZ, Simons LS: Spike activity in trigger points. / *Musculoske Pain 3(Supplement* 1J.125, 1995.(Abstract)
252. Simons DG, Hong CZ, Simons LS: Presence of electrically active loci in human trigger points, endplate zones, and taut bands. (In Press).
253. Simons DG, Stolov WC: Microscopic features and transient contraction of palpable bands in canine muscle. *Am J Phys Med* 55:65-88, 1976.
254. Simons DG, Travell JG: Myofascial trigger points, a possible explanation. *Pain* 10:106- 109, 1981.
255. Simons DG, Travell JG: Myofascial origins of low back pain. Parts 1, 2, 3. *Postgrad Med* 73:66-108, 1983.
256. Sinclair DC: The remote reference of pain aroused in the skin. *Brain* 72:364-372, 1949.
257. Skootsky SA, Jaeger B, Oye RK: Prevalence of myofascial pain in general internal medicine practice. *West J Med* 251:157-160, 1989.
258. Smythe HA, Moldofsky H: Two contributions to understanding the "fibrositis syndrome." *Bull Rheum Dis* 28:928-931, 1977.
259. Sola AE: Personal communication, 1981.
260. Sola AE, Bonica JJ: Myofascial pain syndromes, Chap. 21. In: *The Management of Pain,* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990, (pp. 352-367).
261. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med* 34:585-590, 1955.
262. **Sorensen** J, Bengtsson A, Backman E, *et al.:* Pain analysis in patients with fibromyalgia. Effects of intravenous morphine, lidocaine, and ketamine. *Scand} Rheumatol 24(6):360-* 365, 1995.
263. Starlanyl D, Copeland ME: *Fibromyalgia &• Chronic Myofascial Pain Syndrome: A Survival Manual.* New Harbinger Publications, Oakland, 1996.
264. Stockman R: Chronic rheumatism, chronic muscular rheumatism, fibrositis, Ch. 2. In: *Rheumatism and Arthritis,.* Edited by Stockman R. W. Green & Son, Edinburgh, 1920 (pp. 41-56).
265. Swerdlow B, Dieter JN: An evaluation of the sensitivity and specificity of medical thermography for the documentation of myofascial trigger points. *Pain* 48:205-213, 1992.
266. Swett JE, Eldred E, Buchwald JS: Somatotopic cord-to-muscle relations in efferent innervation of cat gastrocnemius. *Am J Physiol 219(3):762-756,*1970.
267. Tonndorf ML, Hannam AL: Motor unit territory in relation to tendons in the human masseter muscle. *Muscle Nerve* 27:436-443, 1994.
268. Travell J: Basis for the multiple uses of local block of somatic trigger areas (procaine infiltration and ethyl chloride spray). *Miss Valley Med J* 71:13-22,1949.
269. Travell J: Pain mechanisms in connective tissue. In *Connective Tissues, Transactions of the Second Conference, 1951.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1952 (pp. 96-102, 105-109, 111).
270. Travell J: Introductory Comments. In *Connective Tissues, Transactions of the Fifth Conference, 1954.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1954 (pp. 12-22).
271. Travell J: Temporomandibular joint pain referred from muscles of the head and neck. / *Prosthet Dent* 10:745-763, 1960.

272. Travell J: Mechanical headache. *Headache* 7:23-29, 1967.
273. Travell J: Myofascial trigger points: clinical view. In: *Advances in Pain Research and Therapy,* Vol. 1. Edited by Bonica JJ, Albe-Fessard D. Raven Press, New York, 1976, pp. 919-926 (Fig. 10).
274. Travell J: Identification of myofascial trigger point syndromes: a case of atypical facial neuralgia. *Arch Phys Med Rehabil* 62:100-106, 1981.
275. Travell JG: Chronic Myofascial Pain Syndromes. Mysteries of the History, Chapter 6. In: *Advances in Pain Research and Therapy: Myofascial Pain and Fibromyalgia,* Vol. 17. Edited by Fricton JR, Awad EA. Raven Press, New York, 1990 (pp. 129-137).
276. Travell J, Rinzler S, Herman M: Pain and disability of the shoulder and arm: treatment by intramuscular infiltration with procaine hydrochloride. *JAMA* 320:417-422, 1942.
277. Travell J, Bobb AL: Mechanism of relief of pain in sprains by local injection techniques. *Fed Proc* 6:378, 1947.
278. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 3 3:425-434, 1952.
279. Travell JG, Simons DG: *Myofascial Pain and Dysfunction: The Trigger Point Manual,* Vol. 1. Williams & Wilkins, Baltimore, 1983.
280. Travell JG, Simons DG: *Myofascial Pain and Dysfunction: The Trigger Point Manual,* Vol 2. Williams & Wilkins, Baltimore, 1992.
281. Trammer PR, Gellman MB: Trigger point syndrome. *Rheumatism 867-72,* 1952.
282. Van Stolk I: Personal communication, 1997.
283. Vecchiet L, Galletti R, Giamberardino MA, *et al.:* Modifications of cutaneous, subcutaneous, and muscular sensory and pain thresholds after the induction of an experimental algogenic focus in the skeletal muscle. *Clin J Pain* 4:55-59, 1988.
284. Vecchiet L, Giamberardino MA, de Bigontina P, *et al:* Comparative sensory evaluation of parietal xtissues in painful and nonpainful areas in fibromyalgia and myofascial pain syndrome, Chapter 13. In: *Proceedings of the 7th World Congress on Pain: Progress in Pain Research and Management,* Vol. 2. Edited by Gebhart GF, Hammond DL, Jensen TS. IASP Press, Seattle, 1994, (pp. 177-249).
285. Walsh EG: *Muscles, Masses &• Motion. The Physiology of Normality, Hypotonicity, Spasticity &• Rigidity.* MacKeith Press, Distributed by Cambridge University Press, 1992. ISBN (UK) 0 901260 97 5, (USA) 0 521 43229 4.
286. Ward AA: Spontaneous electrical activity at combined acupuncture and myofascial trigger point sites. *Acupuncture Med 14(2):75-79,* 1996.
287. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. / *Am Osteopath Assoc* 72:697-710, 1973.
288. Weeks VD, Travell J: How to give painless injections. *AMA Scientific Exhibits 1957,* Grune & Stratton, New York, 1957 (pp. 318-322).
289. Weiser HI: Semimembranosus insertion syndrome: a treatable and frequent cause of persistent knee pain. *Arch Phys Med Rehabil* 60:317-319, 1979.
290. Weiss S, Davis D: The significance of the afferent impulses from the skin in the mechanism of visceral pain, skin infiltration as a useful therapeutic measure. *Am f Med Sci* 376:517-536, 1928.
291. Wiederholt WC: "End-plate noise" in electromyography. *Neurology* 20:214-224, 1970.
292. Wilkins JC, Meerschaert JR: Hypermobility syndrome-prevalence and manifestations. *Arch Phys Med Rehabil* 76:1047, 1995. [Abstract]
293. Wolfe F, Simons D, Fricton J, ef *al.:* The fibromyalgia and myofascial pain syndromes: a preliminary study of tender points and trigger points in persons with fibromyalgia, myofascial pain syndrome and no disease. *J Rheumatol* 39:944-951, 1992.
294. Wolfe F, Smythe HA, Yunus MB, ef *al.:* American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia: Report of the Multicenter Criteria Committee. *Arthritis Rheum* 33:160-172, 1990.
295. Yaksh TL, Abram SE: Focus Article: Preemptive analgesia: a popular misnomer, but a clinically relevant truth? *Am Pain Soc J* 2:116-121, 1993.
296. Yu XM, Sessle BJ, Hu JW: Differential effects of cutaneous and deep application of inflammatory irritant on mechanoreceptive field properties of trigeminal brain stem nociceptive neurons. / *Neurophysiol 70(4):1704-1707,* 1993.
297. Yue SK: Initial experience in the use of botulinum toxin A for the treatment of myofascial related muscle dysfunctions. *J Musculoske Pain 3(Supplement 1):22,* 1995 (Abstract).
298. Yunus MB: Research in fibromyalgia and myofascial pain syndrome: current status, problems and future decision. *J Musculoske Pain 1(1):23-41,*1993.
299. Yunus MB: Understanding myofascial pain syndromes: a reply. *J Musculoske Pain* 2(3J.-147-149, 1994.
300. Yunus M, Masi AT, Calabro JJ, ef *al.:* Primary fibromyalgia (fibrositis): clinical study of 50 patients with matched normal controls. *Semin Arthritis Rheum* 33.151-171, 1981.

CHAPTER 3
Apropos of All Muscles

HIGHLIGHTS: Considerations that apply generally to all the muscles are consolidated in this chapter. Detailed knowledge of the **REFERRED PAIN (AND TENDERNESS)** pattern is usually a valuable help in identifying which muscle(s) are responsible for myofascial trigger point (TrP) pain. The *precise* location of all of the patient's perceived pain is drawn on a body form to aid in diagnosis and for future reference. An area of referred tenderness corresponds approximately to the distribution of the referred pain described by the patient. Understanding the muscle's **ANATOMY** helps one to integrate knowledge of its chief action(s), functional relations to other muscles, how to find it for examination, how to stretch (lengthen) it, and the location of its TrPs for injection. The **FUNCTION** of a muscle reveals what movements and stress situations are likely to activate and perpetuate TrPs in it. The **FUNCTIONAL UNIT** identifies other muscles that are functionally closely related and, therefore, also likely to develop TrPs because of referred motor effects and interacting mechanical stresses. **SYMPTOMS** of myofascial pain and dysfunction that appear suddenly often begin after a clearly remembered movement or event at a specific time and place. In other cases, excessively prolonged or repetitive efforts insidiously activate TrPs in the abused muscles. The stressful movement or conditions responsible for **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in a particular muscle must be identified and eliminated or modified to prevent the same stresses from reactivating and perpetuating the TrPs following treatment. **PATIENT EXAMINATION** distinguishes between the primary effects of increased muscle tension and muscle shortening caused by the primary TrP pathophysiology and the secondary tension, reflex, and nerve sensitization effects. Examination begins with observation of the patient's posture, movements, and body structure and symmetry, and it includes screening movements that quickly identify which muscle groups have a reduced stretch range of motion (ROM). Pain is commonly caused in a muscle with TrPs by contracting it in the shorted position. **TRIGGER POINT EXAMINATION** of a muscle requires a knowledge of the location and direction of its fibers in relation to those of neighboring muscles. Objective confirmation of a TrP requires special examination techniques for locating its taut band, nodule, and spot tenderness by palpation. The diagnosis of an *active* trigger point is established by patient recognition of the pain elicited by pressure on the TrP and may be confirmed by eliciting a local twitch response (LTR) from it. Nerve **ENTRAPMENT** may occur because of pressure by the palpable bands of taut muscle fibers that are associated with myofascial TrPs, when the nerve passes through the muscle between taut bands, or when it is compressed between such a band and bone. The cause of the neurological symptoms and signs of neurapraxia that result is easily misinterpreted if this mechanism of entrapment is not recognized. **DIFFERENTIAL DIAGNOSIS** must consider symptoms which appear to come from trigger points but are caused by another diagnosis and also other diagnoses which the patient has received because of symptoms that are caused by trigger points. **TRIGGER POINT RELEASE** can be accomplished using spray and stretch or using voluntary contraction and release techniques that include postisometric relaxation, reciprocal inhibition, contract-relax, and muscle energy technique. Direct manual techniques include trigger point pressure release, deep stroking massage, and strumming. Indirect methods also can be used, and all of these methods can be supplemented with accessory techniques and modalities.

TRIGGER POINT INJECTIONS require first, accurate localization of the TrP by palpation and then, confirmation of precise placement of the needle based on needle-elicited pain and a local twitch response. Enough finger pressure is ap-

plied to insure hemostasis. After injection, the patient should perform three full cycles of slow active range of motion to reestablish normal function of the muscle. **CORRECTIVE ACTIONS** include both a stretch exercise program for use at home and the elimination of perpetuating factors (Chapter 4). Particularly among patients with chronic myofascial pain, these considerations usually determine the duration of relief experienced after treatment of the involved muscles.

1. REFERRED PAIN (AND TENDERNESS)

The patient's pattern of referred pain and tenderness is often the key to identifying the muscle(s) responsible for a myofascial pain syndrome. This section explains how to draw a representation of the patient's pain, and how to interpret the location of the pain. Identification of the areas of referred tenderness is covered under Section 8.

Patient's Pain History

Surprisingly, the patient is rarely aware of a trigger point (TrP) in the muscle that causes the myofascial pain; pain evoked by lying on an infraspinatus TrP at night is perceived in the shoulder, not at the guilty TrP in the muscle overlying the scapula. However, when the patient stretches or loads the involved muscle, he or she is likely to feel discomfort in the regions of

taut band attachments. The myofascial TrP pain patterns presented throughout this manual were described by patients as situated deep (subcutaneous and muscular) and aching in character, unless stated otherwise in our description.

Regardless of the mode of onset, whether abrupt or gradual, pain referred from myofascial TrPs is **characterized** as steady, deep, and aching, rarely as burning. It is to be distinguished from the prickling pain and numbness associated with paresthesias and dysesthesias of peripheral nerve entrapment or of nerve root irritation. However, two skin muscles, the platysma and palmaris longus, refer a needle-like prickling sensation superficially. Throbbing pain is more likely to be due to vascular disease or dysfunction. Occasionally, a myofascial TrP initiates sharp, lancinating, or lightning-like stabs of pain.

The patterns of pain referred from TrPs in a muscle are reproducible and predictable. Knowledge of these patterns is used to locate the muscles most likely to be causing the spontaneous pain, much as one suspects which viscus is diseased by its specific pattern of referred pain. The diagnostic value of the patient's pain patterns depends strongly on the accuracy and detail with which the exact location and extent of the pain are mapped.

Generally, the specificity of the pain patterns in this manual was established first by determining which muscle contained the TrP through observing the location of movement produced by a local twitch response (LTR), or (when the muscle was not accessible for eliciting an LTR) by noting other anatomical landmarks when injecting the TrP. Next, when injecting the TrP, the clinician asked the patient to note carefully the location of any associated pain when the needle produced an LTR in that TrP. The location of this pain was considered the referred pain pattern of a TrP in that muscle of that patient.

It would be helpful if there were a general rule that predicted the direction of the referred pain pattern of a TrP based on the location of the muscle. This possibility was investigated.[144] The direction of referral of the 147 pain patterns of volumes 1 and 2 of the *Trigger Point Manual* was classified as peripheral (away from the center of the body), mostly central (predominantly in the direction of the center of the body), and local (only in the immediate vicinity and surrounding the TrP). Examples illustrating these three kinds of pain patterns appear in Figure 3.1. Some TrPs produce pain patterns that are combinations of these three. Many patterns include the TrP and sometimes it is the most painful location. Other patterns do not include the TrP itself, which can be very misleading to the clinician and to the patient.

In general, referral at least partly in the peripheral direction is most common (85% of patterns). Half of all patterns (48%) refer *only* in the direction of the periphery. In addition, 20% of all patterns include both peripheral and central referral, and 17% of peripheral patterns include a strong local pattern. Just 10% of the patterns have only a local pattern and just 5% refer only in a central direction.

These data suggest that once it is established where the patient hurts, one is much more likely to find the TrP located toward the center of the body from the referred pain than to find the TrP located peripheral to the pain complaint. These data also warn that only a total of 27% of patterns include a substantial local component of TrP-generated pain. If a clinician depends on finding the TrP at the spot where a patient points when he or she shows where the pain is, the clinician is likely to be wrong nearly three quarters of the time. The *Trigger Point Wall Charts* and *Trigger Point Flip Charts* published by Williams & Wilkins are a great aid in this regard.

When the TrPs are more active, the extent of referred pain is greater, the pain is more intense, pain is more likely to persist at rest, the TrPs are more tender, the taut bands are more tense, and LTRs are more vigorous.[71]

In this volume, the *solid red area* in each drawing of referred pain and tenderness depicts the essential pain zone, which is present in nearly every patient when the identified TrP is active. Spillover pain zones, which may or may not be present, appear as *red stippling.* A black (or white) X published on pain pattern drawings

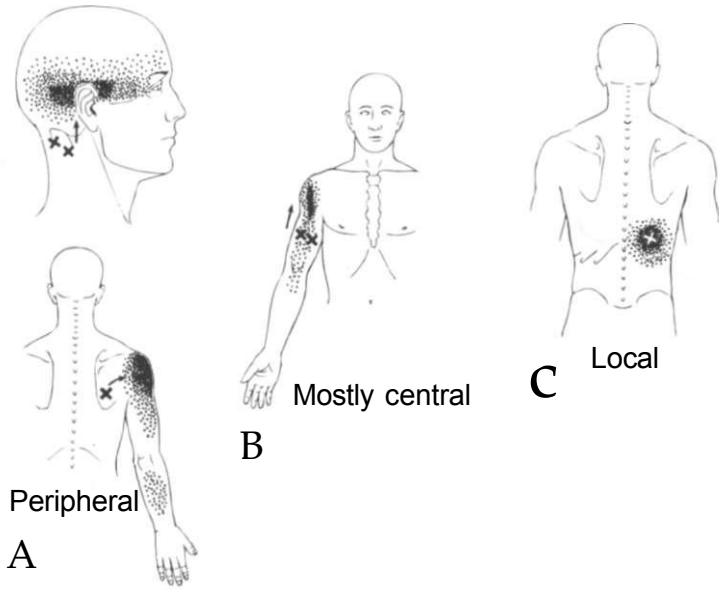


Figure 3.1. Examples of the three directions in which trigger points **(Xs)** may refer p a i n . **A,** peripheral projection of pain from suboccipital and infraspinatus trigger points. **B,** mostly central projection of pain from biceps brachii trigger points with some pain in the region of the distal tendinous attachment of the muscle. **C,** local pain from a trigger point in the serratus posterior inferior muscle.

identifies a common location of the TrP, or TrPs, in that muscle; this provides only a general guide. The TrPs may be located anywhere in the endplate zone(s) of the muscle. The location of the endplate zone(s) depends on the arrangement of fibers in that muscle (see Chapter 2, Section D).

Drawing the Pain Pattern

In addition to observing the patient's posture and examining for limitation in range of motion (see Section 8 in this chapter), a precise pictorial representation of the patient's pain is a valuable aid for locating TrPs causing myofascial pain. Verbal descriptions are often imprecise and misleading so a blank body form can be used routinely to record the patient's description of the pain. Figures 3.2, 3.3, and 3.4 are forms useful for this purpose. The same form also can be used to record the location and tenderness measure of the TrPs when they have been located. The form becomes a valuable medical record.

Communication concerning pain sensations is difficult, at best. When patients say, "My shoulder hurts," some will indicate pain in front of or behind the shoulder; one reaches back to the scapula; another grabs the entire shoulder indicating pain deep in the joint; and yet another rubs the upper arm. Therefore, the clinician needs to ask the *patient* to delineate the pain on his or her body using one finger; then the *practitioner* can draw its pattern on the blank form. The patient should then examine the drawing for *accuracy* and *completeness.* This procedure enhances the precision of the record, and improves communication. The locations of *all* the patient's separate pain patterns and the date of the first appearance of each are noted for future reference. Other authors also strongly endorse the use of pain drawings.[12-113,117] *Precise* delineation of the patient's pain areas is required to match them with the known pain patterns of individual muscles and to record progress.

It is common practice to give the patient a blank body form and ask him or her to indicate with symbols where the pain is felt. This is useful for identifying patients who have the widespread pain of fibromyalgia

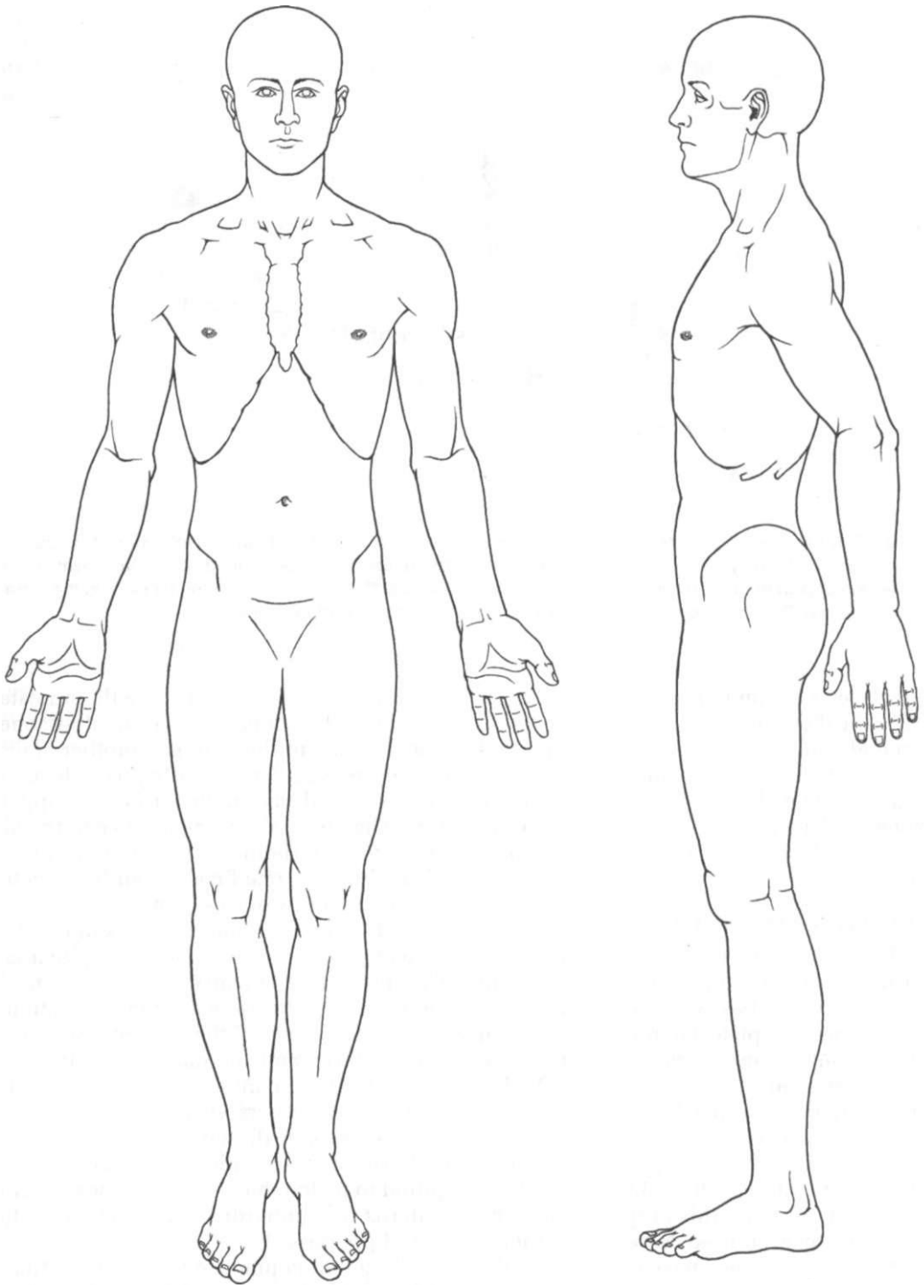


Figure 3.2. Body form: full figure, front and left side.

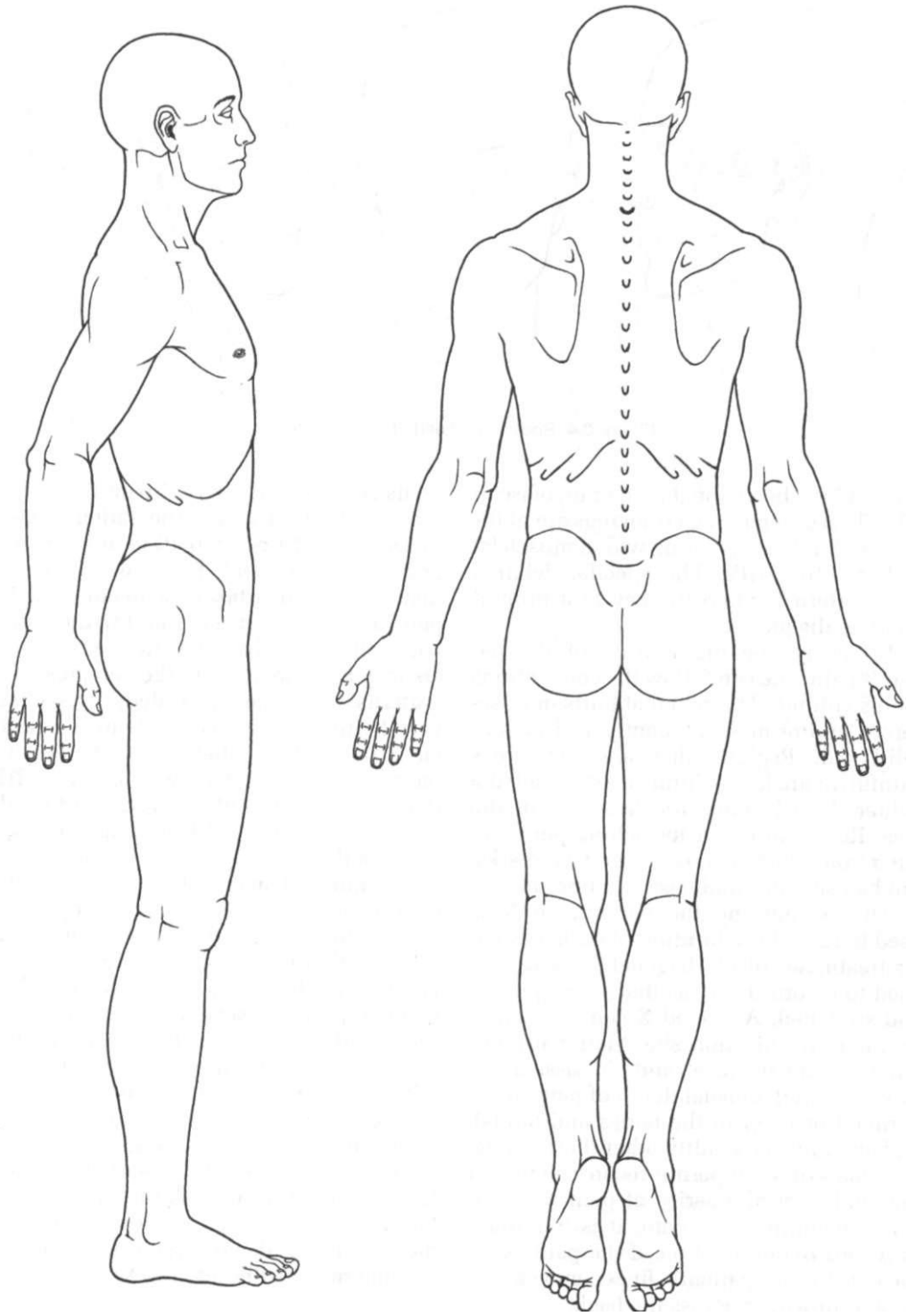


Figure 3.3. Body form: full figure, right side and back.

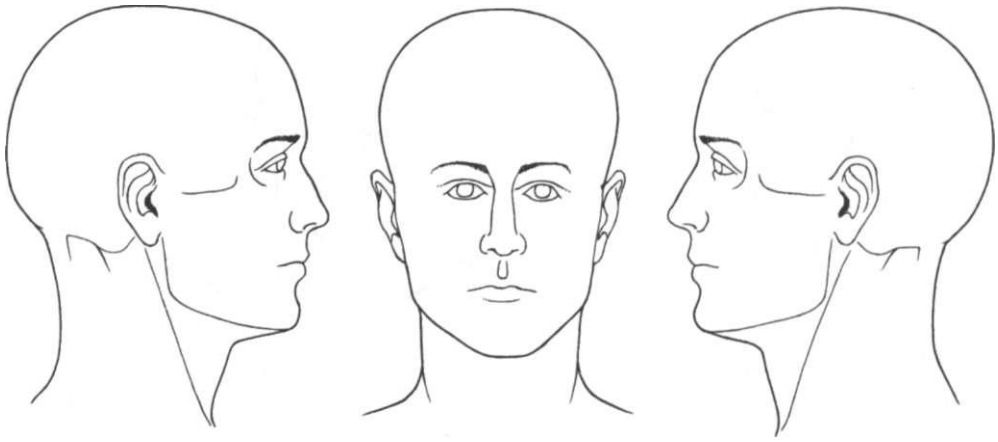


Figure 3.4. Body form: head, front and sides.

and not just the regional pain of myofascial TrPs. These generalized drawings are of little help for distinguishing which muscle(s) harbor active TrPs. The specific detailed pain pattern is often the key to a prompt accurate diagnosis.

To record the distribution of the patient's pain, one can follow the conventions in this volume. The area that hurts most severely, and/or most frequently, is drawn in solid red. Regions that are sometimes painful, or are less painful, are stippled; the lighter the stippling, the less painful the area. Red is reserved for aching pain; another color such as green, or check marks, can be used for numbness and tingling.

After examining the patient, an X is used to record the location of each TrP. After treatment, black diagonal lines can be used to record the areas that were sprayed and stretched. A circled X can be used to locate a TrP injection site. Marginal notes tell the date of onset and the associated event (if any), unusual depth of pain (if superficial or deep in the bones and joints), and any unusual quality other than aching. The dates of onset permit reconstruction of the evolution of a series of pain patterns. When mapping back pain, it is important to record the orientation of the pain as indicated by the patient's finger movement, up and down, or across the back.

Sometimes a patient will state, "I hurt all over." When asked if the nose hurts, the answer is almost always, "No." Nor do patients complain of referred pain in the fingernails. With this start, the patient begins to realize that discriminating answers are possible. The patients begin to understand that this examiner takes the details of their pain complaints seriously and will not discount their description of the pain as others may have done. After the patient's pain patterns have been recorded, it is often very helpful for the patient (and the clinician) to see the pattern of that patient's pain on a flip chart or wall chart of TrP Pain Patterns. Patients are relieved to realize that they have not been imagining the pain, as they ofttimes have been led to believe, and that many other patients have experienced the same pain. This opportunity to demonstrate the muscles that contain the TrPs which are causing their pain is especially helpful when the pain pattern is a composite of several TrPs. Details are important, such as which side of the limb hurts, and whether the pain skips across a joint or concentrates in the joint. It does injustice to the patient and to the diagnosis to depend on generalizations.

When a TrP is identified and its location has been marked by an X on the Pain-Pattern form, the TrP tenderness may be documented by a dated pressure algometry reading recorded beside the X.

Interpretation of Initial Pain Patterns

Is the drawing a simple, one-muscle, myofascial TrP pain pattern? Is it a com-

posite of several such patterns that are superimposed, or does it have a distribution that is foreign to TrP pain patterns and, therefore, most likely of non-myofascial origin? To answer these questions one needs to be familiar with the individual myofascial referred pain patterns, to know that myofascial pain caused by TrPs is rarely symmetrical, and to know that it rarely assumes a glove-and-stocking or hemialgia distribution.[175] The extent of a muscle's referred pain pattern tends to enlarge as the irritability (activity) of the TrP increases.

A referred pain pattern may be composite in two ways. A total pattern may comprise overlapping patterns from different muscles, so that the extent of the pattern exceeds that of one muscle.[80] Figure 3.5 illustrates how this can look when the patient complains of headache.

On the other hand, if TrPs in several muscles all refer pain to the same area (e.g., the shoulder), the area may be a little larger, but also will be more painful and hyperesthetic than if the symptoms came from only one muscle. Inactivation of only one of the contributing TrPs may produce little amelioration of the patient's pain; whereas, inactivation of all of them can achieve complete relief.

No two patients' problems are exactly alike. A few patients exhibit marked variations in the expected referred pain pattern, sometimes due to a genetic variation in central nervous system transmission, comparable to an anomalous muscle. Rarely are such variations in pattern symptoms of hysteria.

The history should indicate whether the pain pattern has been stable, or whether it has evolved over months or years. If the pattern is stable, the pain is likely to resolve promptly with specific myofascial TrP therapy. The progressive involvement of many muscles is a strong indication that perpetuating factors (Chapter 4) must be eliminated for lasting pain relief.

Interpretation of Pain Patterns on Return Visits

When the patient returns pain free with complete restoration of full range of motion and the prior TrP sites are no longer abnormally tender, the treatment was successful. However, if the patient returns claiming "no improvement," an accurate record of the previous pain pattern becomes critical. The earlier record is compared with a new drawing representing the patient's pain and algometer readings of TrP tenderness. If the patient has the same degree of tenderness in the same TrPs and the same pain pattern as before treatment, one must ask how long pain relief lasted following treatment. If pain relief was complete for some hours or days, one can assure the patient that a *muscular* cause of the pain is present, and that it can be relieved, at least temporarily. However, repeated treatment without first resolving the perpetuating factors that make the TrPs so hyperirritable is

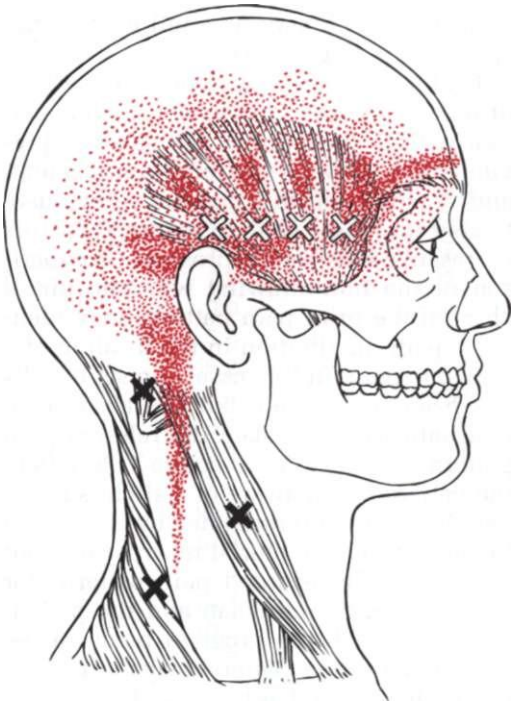


FIGURE 3.5. Common "tension type headache" pain pattern caused by overlapping referral patterns of pain *(red)* from trigger points in the temporalis *(white* **Xs),** suboccipital *(uppermost black* **X),** sternocleidomastoid *(middle black* **X),** and upper trapezius (fowesf *black* **X)** muscles. (Adapted with permission from Jaeger B: Differential diagnosis and management of craniofacial pain. Chapter 11. In: *Endodontics.* Ed. 4. Edited by Ingle JI, Bakland LK. Williams & Wilkins, Baltimore, 1994:550-607.)[80]

likely to be fruitless. A major effort should then focus on identifying and eliminating the perpetuating factors.

On the other hand, if careful comparison of a current "no improvement" pain pattern with the patterns of the patient's previous visit shows a distinct improvement, and if some of the muscles previously treated no longer contain tender TrPs, this represents satisfactory progress. Comparison of the new drawing with the initial chart of the prior pain patterns may identify a pain distribution in the location of a previous pain. In this case, one set of TrPs has been inactivated, but the absence of that pain has unmasked the referred pain pattern of the next most active TrPs. Often, the patient is not aware of a slight shift in pain location until reminded by comparing the old and new pictorial records. Without the accurately recorded pain patterns for comparison, the clinician and the patient might overlook the progress being made. Occasionally, the pattern may be new to that patient; a TrP has been newly activated and must be managed as any acute myofascial TrP syndrome.

2. ANATOMY

By knowing all of a muscle's attachments, one can deduce the major actions of the muscle, where to find it for palpation, and the direction of its fibers. The anatomical drawings of each muscle in this volume present the muscle alone with its bony attachments. The bones to which the muscle attaches are stippled more darkly than other bones. When necessary, additional drawings of regional anatomy show the muscle's relation to nearby muscles and structures. Anatomy textbooks were scoured for the needed views of muscles. When questions remained, dissections were studied in the anatomy laboratory. Although an anatomical variation may occur in only a small percent of the population, it is 100% to the patient who has the variation and to the clinician caring for the patient.

It has now become clear that knowledge of the location of the endplate zone(s) in a muscle is of fundamental importance to understanding where central TrPs can be found. Knowing the location of myotendinous junctions and tendo-osseous attachments is important for understanding where attachment TrPs may occur. Chapter 2 reviews the reasons for these phenomena. Some individual muscle chapters reflect this new understanding more fully than others.

Terminology

The names of the muscles come from *Nomina Anatomica.*[7] English usage follows the American edition of Gray's *Anatomy of the Human Body.*[33] In this manual, the words "origin" and "insertion" are avoided except in instances where the relation is unambiguous, as with finger attachments. Not uncommonly, the functions of the nominal origin and insertion become reversed, particularly during movement when muscles are likely to be strained and TrPs activated; use of the term "attachment" helps one to keep an open mind and to think of muscle functions in realistic terms, permitting interpretation of the specific stress situation described by the patient. To stretch a muscle therapeutically, it usually matters not which end is fixed and which end is moved.

Unless stated otherwise, descriptions of muscle attachments refer to the person in the upright position, standing straight, face forward, and the arms and forearms at the side with the forearm supinated (anatomical position, Fig. 3.2). Therefore, ***above*** is equivalent to cephalad, superior, or proximal, and ***below*** is equivalent to caudad, inferior, or distal.

Fiber Arrangement

A description of the fiber arrangement in muscles is commonly overlooked in anatomy texts, lost in the hiatus between gross and microscopic anatomy. It is rarely described adequately, except in a few older texts, such as Bardeen[6] and Eisler.[43] All the fibers of any one muscle are of nearly equal length, but usually with staggered attachments at the ends; muscle fibers usually attach to aponeuroses or to bone in a parallelogram arrangement. In long muscles with short fibers, such as the gastrocnemius, the aponeuroses overlap each other, or an aponeurosis at one end of the fibers overlaps a bony attachment at the other end.[22,27] Individual muscle fibers may be placed so diagonally, as in the soleus, that

Table 3.1 ***A Few of the Strongest Muscles Arranged in Their Order of Calculated Cross-sectional Area, Derived from Weber.***[184]

Muscle	*Cross-sectional Area*	*Mean Fiber Length*	*Total Muscle Weight*
	cm^2	cm	gm
External intercostal	79	1.5	126
Multifidus	68	2.9	210
Internal intercostal	47	1.5	77
Longissimus thoracis	32	7.2	223
Deltoid	32	9.0	305
Triceps brachii, short head	26	5.8	161
Subscapularis	25	6.2	164
Infraspinatus and teres minor	17	7.4	132
Biceps brachii, long head	16	9.7	168
Triceps brachii, long head	16	7.7	131
Internal abdominal oblique	14	7.0	107
Serratus anterior	13	13.7	186
Cucularis (trapezius)	13	10.9	146
Brachialis	13	8.4	117
Pectoralis major, sternal	12	14.7	187
External abdominal oblique	10	10.9	115
Flexor digitorum profundus	10	6.7	68

the fiber length is barely one-half the length of the whole muscle.

In 1851, Weber[184] studied the structure of muscle and its relation to function by measuring the weight and mean fiber length of each muscle in the body. Table 3.1 extracts data for a number of the larger muscles. The cross-sectional area of each muscle was calculated by the formula $S = P/pL$ where S = the cross-sectional area, in cm^2; P = the weight of the whole muscle, in grams; p = the specific gravity of muscle, 1.0583 gm/cm^3; and L = the mean length of the fibers in that muscle, in centimeters. This kind of measurement may vary greatly from person to person depending on body build, occupation, the degree and kind of physical activity, *etc.* Subsequent studies[22,181] have reported results comparable to those of Weber.

Assuming similar fiber diameters among muscles, the cross-sectional area is nearly proportional to the relative strength of each muscle, since this area also is proportional to the number of myofibrils contracting in parallel. This concept has been applied to the selection of muscles for transfer of tendon attachment.[22]

The relative length of individual fibers to total muscle length has important functional consequences.[105] Muscles like the quadriceps, scalenes, and gastrocnemius with relatively short fibers (low fiber length/muscle length ratios) are designed for force production. Muscles like the biceps, hamstrings, and tibialis anterior have high fiber length/muscle length ratios and are designed to produce high velocity movement. Muscles designed to produce force have endplate zones that tend to run the length of the muscle, whereas muscles designed for rapid movement have endplates zones that run relatively transverse to the muscle (depending on muscle structure), but always near the midpoint of the muscle fibers *(see* Chapter 2 section C).

Supplemental References

As a service to those who teach muscle anatomy and to those interested in different anatomical views or in a more detailed

understanding of a muscle, additional illustrations are listed at the end of Section 2 of each muscle chapter, under **Supplemental References.**

3. INNERVATION

The spinal and peripheral nerves that usually supply each muscle are identified in this section. In many muscles, there is considerable individual variation; rarely do anatomists agree completely on the segmental innervation of a muscle.

4. FUNCTION

Since the *actions* of a muscle that provide the *functions* for which it is used are so closely intertwined, these two issues are considered together under one heading in this edition.

Understanding the actions of muscles is valuable diagnostically and therapeutically. Diagnostically, an accurate description of the precise movement made by the patient at the time that the TrP was activated, together with a knowledge of which muscles are used to produce and to control that movement, helps to determine which muscles were likely to have been strained at the time. The strained muscles are then examined for restricted range of motion and tender nodules to see if they harbor active TrPs.

Therapeutically, a knowledge of the movements and activities that depend upon the muscles being treated is needed in order to explain proper body mechanics to the patient. The patient must understand precisely what movements and activities should be modified or avoided to prevent further muscular overload and perpetuation of the TrPs.

In this manual, actions of muscles are described as the movement *of a* segment *at* a joint; for example, the brachioradialis muscle flexes the forearm at the elbow. Terms describing directions of movement are defined in Chapter 1.

Four sources of information were used to summarize the actions of a muscle: (1) the actions listed in anatomy texts based on the attachments of the muscle; (2) the movements produced by stimulating the muscle electrically; (3) electromyographic studies that reported which movements or efforts generated motor action potentials in that muscle; and (4) the movements reported by patients that, when overloaded or repeated, produced TrPs in that muscle.

5. FUNCTIONAL UNIT

The functional unit to which a muscle belongs includes the muscle(s) that reinforce and counter its actions as well as the joint(s) which the muscle crosses. The interdependence of these structures functionally is reflected in the organization and neural connections of the sensorimotor cortex.

The functional unit is emphasized because the presence of an active TrP in one muscle of the unit increases the likelihood that other muscles of the unit also will develop TrPs. Dysfunction (weakness and shortening) of the affected muscle tends to overload other muscles of that functional unit. When inactivating TrPs in a muscle, one must be concerned about TrPs that may develop secondarily in muscles that are interdependent.

The physiological definition of a myotatic unit (the term used in the previous edition) includes the synergists, which help the prime mover (agonist), and the antagonists,[135] because these muscles are linked by interacting reflex pathways.[106,188] The term functional unit is used in this edition and includes the muscles noted above and also muscles that do not necessarily share common reflexes, but which have close functional relationships. An example of one relationship is muscles that extend the line of pull of the affected muscle during total body movements (e.g., the external abdominal oblique extends the line of pull of the serratus anterior muscle). Another example is found in stabilizing muscles, such as the upper trapezius and levator scapulae muscles when they help control the scapula during forceful lifting movements of the upper limb on that side.

6. SYMPTOMS

With a thorough knowledge of individual myofascial pain syndromes and of TrP referred pain patterns, one can often, with a careful history, not only identify the diagnosis of myofascial pain but also determine which muscles are most likely causing the pain. The chapters that follow note specific features of individual muscle syndromes. This section describes the features of the patient's history that help to identify

myofascial pain syndromes and to distinguish them from other painful conditions.

The myofascial TrPs may be activated acutely by an obvious cause of muscular strain or may become symptomatic insidiously due to less obvious chronic muscular overload. In either case, symptoms may continue for months or years if the myofascial TrP source of the pain is not recognized and treated. This situation often, but not always, leads to the syndrome of chronic pain, which is likely to become a way of life[156] and may require attention to learned pain behavior,[47] as well as the TrP origin of pain. This manual concentrates on the latter.

History

Travell emphasized the importance of taking a thorough and thoughtful history in patients with chronic musculoskeletal pain. The following material on the patient history is abstracted from a chapter written by Travell in 1990.[172]

Preliminary Review of Records. The completeness of the history is increased by a preliminary review of the patient's story and records. Before the first visit, the patient is requested to submit a chronology of life events, a chronology of medical events, and a complete list of current and recent medications including nutritional supplements.

The *chronology of life events* should give dates and places of residence, education, marriages, children living (age and where they live), sports activities, travel, and employment (what kind, where, for whom).

The *chronology of medical events* should include illnesses, infections, accidents (fractures, falls, etc.), surgical procedures, dental procedures, pregnancies and miscarriages, allergies (tests and hyposensitizations), and vaccinations. The patient may overlook a significant accident if no fracture occurred, but further interrogation will elicit the full history.

The patient is generally aware of inhalant allergies, but special care must be taken to check for food allergies and what foods cause symptoms. Myofascial TrPs are aggravated by high histamine levels and active allergies. Marking the skin to test for dermatographia is a simple way of identifying high histamine levels.

For inhalant allergies, reducing exposure by the use of electrostatic air cleaners is helpful. However, the fact that the patient has an electrostatic air cleaner may not be sufficient. One patient reported using it every night, but further inquiry revealed that she also opened her bedroom windows every night. She liked fresh air and did not realize that her air cleaner had no chance of eliminating the pollens that were coming in from outdoors.

The *list of medications* should include all medications currently being taken, including vitamin and mineral supplements. The patient is asked to bring a bottle of every medication so that the actual dosage can be established. This includes prescription and over-the-counter drugs, as well as nutritional supplements. A *list of the medications* taken in the past that caused side effects or **did not** relieve the pain is also important.

The patient is asked to send, in advance, a copy of all medical records in his or her possession and to request any others to be sent by any recent consulting physicians, especially orthopedic and neurological consultations. These records are carefully reviewed before the patient's initial visit.

Interview with Patient. While taking the history, patient comfort should be ensured by demonstrating the principles of good body mechanics to them. A footrest can be provided when the patient's legs are too short for the feet to rest firmly on the floor; additional armrest height can be supplied when the elbows do not reach the armrests of the chair; a butt-lift (ischial-lift) can be placed under the small hemipelvis when the patient's body is tilted because of this asymmetry; a small pillow positioned in the lumbar hollow helps maintain effortlessly a normal lumbar curve of the spine and helps the patient to sit erect rather than with the head and shoulders hunched forward. Patients are often amazed to discover the degree of immediate relief that can be obtained by relieving muscular strain due to these mechanical perpetuating factors. This relief helps the patient appreciate the strong impact that these factors can have on his or her pain.

A towel or scarf can be provided to protect the patient's shoulders when a chilling draft causes direct cooling of the muscles. If the hands and feet are cold, a dry heating pad placed on the abdomen warms the core of the body and sends more blood into

the limbs (reflex heat). Contrary to the patient's previous experience, with the needed postural and environmental corrections he or she may now be able to sit for one-half or three-quarters of an hour through the intensive medical history, as comfortable at the end as at the start.

To effectively understand the history, it is important to empathize but not to identify with the patient. Empathy is established by putting oneself in patients' shoes, objectively seeing their life problems from their point of view, understanding their jobs, their personal relationships, and their emotional stresses. Identification with the patient often results in emotional involvement that is destructive to the doctor-patient relationship and can be damaging to the doctor's own mental health.

Pain Distribution. If the pain is constant and in multiple locations, the patient is likely either to say, "I hurt all over," or to focus on the most intense pain, not mentioning other pains until the most severe pain is relieved.

Learning to discriminate where it really does hurt is essential. One patient said she had pain in her "TMJ." She had received temporomandibular joint arthrograms and multiple tests and treatments by many dentists and physicians for her "TMJ pain." When asked to point to where the pain was located, she put her finger on the mastoid process behind the ear. She never had any pain in the TMJ region. This lack of anatomical knowledge causes similar problems for the shoulder, buttock, low back, and other parts of the body.

When the patient complains of "pain all over," the doctor must ask, "Do you have pain in the nose? The earlobe? The knee?" When the patient says "no" to one or more of these questions, the patient realizes that the pain is not felt all over and that the clinician needs to know the precise distribution of pain. By mapping the specific pain patterns one can begin to identify the likely locations of the trigger points responsible for the pain complaints.

An accurate picture of all the areas of pain is very important. After completing the pain distribution on a body form that has each pain shaded in red (the same body form used for the pain diary between visits can be used for this purpose), the patient may be asked, "Are these all the areas where you have pain?"

"Yes."

"Do your feet hurt?"

"Why, yes! All my life."

"Why didn't you mention them?"

"Doesn't everyone's feet hurt?"

Another patient may fail to mention headaches, and then reply to a specific question, "They're normal. I've had them as long as I can remember."

Another helpful question is, "What do you do to get relief?" One woman, when asked how she relieved her backache (interscapular), confided that she lay on a warm iron and rubbed the pain away.

"Oh, dear, I never told anyone else that before. You will think I'm crazy."

"No, that is exactly what I would expect you to do to help relieve the pain from those muscles in your upper back."

It is important to convince the patient that whatever the pain history, it is believable to you.

Some patients are afraid of being labeled hypochondriacs or psychological cripples if they reveal all the places where they hurt. Some have been convinced by other practitioners that they really are crazy to think that they have so much pain.

Also, patients should be assured that you do not think they are "doctor shopping" because they have seen so many physicians for their long-standing severe pain problem. Rather, they are to be commended for their determination to get well and regain their normal function.

Review of Body Systems. A brief review of the major body systems helps to ensure that a significant medical problem is not overlooked. In reviewing the gastrointestinal tract, the history should be explored for diarrhea, constipation, nausea, heartburn, abdominal pain, hemorrhoids, blood in the stools, and the like. When a patient is low in folate, diarrhea is likely to occur intermittently with explosive, watery stools. Constipation often is associated with low thyroid function and/or vitamin B_1 inadequacy. Excessive flatus may be dietary or due to loss of normal intestinal bacterial flora.

Simple questionnaires are easily misleading. When one patient was asked if she had diarrhea, she answered, "Oh, no." As she was leaving the office, she asked for a

prescription for paregoric. When queried, she replied, "Oh, I'm going to the theater tonight and, if I didn't take the paregoric, during the performance I probably would have to rush out to the bathroom." She did not have diarrhea; she took paregoric regularly as a preventive.

Sleep. If patients report that they "sleep poorly," further questioning is in order. Is it because they cannot fall asleep or because sleep is interrupted repeatedly during the night? Do they wake up early and are unable to go back to sleep? Most important, what disturbs their sleep? In what position do they sleep? (There may be a mechanical cause of pain that interferes with sleep.) Do they have "restless legs" (folic acid deficiency)? Do they have a chronic urinary tract infection and nocturia or an enlarged prostate so that they have to get up at night to empty the bladder?

One patient, when asked if he had to get up at night to urinate, replied: "Oh, no."

"Was there ever a time when you did have to urinate at night?"

"Yes. Now, all the time, several times every night."

"But I thought you said you didn't have to get up at night."

"That's right, I don't. I use a bedside urinal."

Many times, the cause of sleep disturbance is specifically identifiable and correctable. A baby may cry at night because it doesn't have enough blankets and is cold. Body warmth is also important for myofascial pain patients. When the muscles become cool at night, they contract to generate heat, and this tension can activate latent trigger points. An electric blanket is most helpful, even during the summer in an air-conditioned, cool room. Often, only the spouse is aware of the painless jerking of "restless legs" at night. A supplement of folic acid, several milligrams daily, frequently resolves this source of sleep disturbance.

Diet. Questions regarding what foods the patient avoids may be as informative as those regarding what foods they eat. Patients may assure you that they eat a well-balanced, normal diet. When Dr. Travell questioned one man about his diet, he replied, "I have a wonderful appetite!" She repeated the question as to what he ate, and he smiled and said, "I'm always hungry." She changed her question: "Are there any foods that you avoid?"

"Oh, yes. I'm a complete vegetarian."

In his previous medical questionnaire, his doctor had marked his diet as normal. His myofascial pain had started insidiously soon after he stopped eating meat, fowl, fish, and dairy products. He took no vitamin or other nutritional supplements. He had a marked vitamin B_{12} deficiency.

The history should also determine whether meals are prepared ahead of time and placed on heated trays under fluorescent lighting, as in a doctors'/nurses' dining room, a home for the elderly, fast food stop, school cafeteria, or even at a first-class hotel buffet. This exposure of food to heat and fluorescent light causes rapid degradation and loss of vitamin C and some B vitamins.

The quality of the diet is determined not only by what the patient eats but by how this food is prepared. Are the potatoes fried or peeled and boiled? If boiled, are they cut into pieces to cook faster, which permits the water-soluble vitamins and minerals to leach out? If the raw spinach leaves are soaked in water to wash them well, this leaches out folic acid. Thus raw/green salads, fruits, milk, vegetables, and the like do not always provide an adequate, balanced diet. Some individuals have an unusually high requirement for specific vitamins.

Work Situation. A careful history of precisely what the patient ordinarily does at work (or at home) is fundamentally important. Many times, if the patient experiences intermittent pain, it is helpful for the patient to keep a written record of any onset of pain throughout the day and to relate it to activities at the time. The many sources of strain include an awkward positioning of a keyboard, documents, computer monitors, or of reading and writing material, visitors seated at one side that require the patient to turn the head and neck to face the individual with whom he or she is talking, holding a telephone receiver between chin and shoulder, or abuse of the muscles in housework.

An important source of overlooked muscle strain is a long-standing loss of range of motion in one arm, that requires the opposite afflicted extremity to be overworked. One patient, a dentist, had myofascial pain in the non-dominant arm and a painless middle finger of the dominant right hand

that he could not flex beyond 90 degrees. When asked why, the patient said, "I broke the finger when I was a youngster, 50 years ago, and the joint has been locked ever since."

While talking to the patient, gentle examination of the finger revealed that, indeed, it did bend. The middle finger's long extensor muscle harbored latent TrPs that restricted stretch but caused no pain. His muscles had learned to guard that part of the body. One brief application of the vapocoolant spray-passive stretch procedure promptly restored the full range of finger flexion. The dysfunction of the dominant right hand had caused compensatory overload and myofascial pain syndromes of the nondominant extremity.

Timing of Pain. Myofascial TrPs may cause constant pain, intermittent pain, or no pain complaint.[172] These differences in presentation affect diagnostic symptoms. Patients in **constant pain** caused by TrPs are usually unaware of activities that aggravate the pain. They already have such intense pain that they do not perceive an increase, and so cannot distinguish what makes it worse. Similarly, they may be aware of tenderness at the TrP, but may not distinguish a change in their referred pain when pressure is applied to the TrP, partly because the TrP is so hypersensitive that only a little pressure reaches local pain tolerance.

Most patients with active TrPs experience **intermittent** pain that is characteristically aggravated by specific movements and may be alleviated at least temporarily by a certain position. These patients may have some relatively pain-free days, especially if their pain is associated with muscle stress induced at work. They can usually identify what activities makes them worse, and what position or situation provides relief. The patient must learn not to be spartan and to avoid the "good sport" syndrome, and must learn how to protect the abused muscle(s) from unnecessary overload. This group of patients is ideal for patient education. They can learn to "listen" to their muscles and respond appropriately.

Latent TrPs give no primary pain clues, and must be identified by postural changes, muscle dysfunction, and physical examination.

Patients and clinicians need to understand that the onset of pain following activation of a TrP due to muscle overload can be delayed as long as 12 to 20 hours. Such a time delay makes it easy to overlook the cause of recurrence of the TrP pain. Usually, further activation of an existing latent TrP produces pain almost immediately. If the patient is subject to recurrence of severe episodic pain every few days, one should consider the possibility of episodic hypoglycemia. In this case, onset of pain should relate to eating and/or exercise and the patient can be tested for overreaction to a glucose tolerance test. The energy crisis at the TrP worsens when there is serious loss of its energy supply.

Pain patterns mapped on successive visits tell the story of progressive improvement with some pain areas disappearing and others diminishing in size. A new area of pain may mean that a less active TrP has been "unmasked" by eliminating a more severe pain from a TrP in the same functional unit.

About the same time Travell published the above clinical description,[172] other authors showed experimentally that patients with only latent TrPs can experience local TrP tenderness and TrP referred tenderness[177] with restricted range of motion that is limited by discomfort. However, patients avoid that degree of movement so they do not present with a pain complaint. Not only is the function of that muscle compromised, but the latent TrP also can refer motor dysfunction to other muscles without referring pain. The absence of pain can make it difficult to suspect and identify the latent TrPs responsible for the referred motor dysfunction. This situation is common among masticatory muscles.

Myofascial pain may start abruptly or gradually. With **abrupt onset,** the patient remembers clearly the first date of the pain and can usually describe in precise detail the exact event or movement, such as reaching back for something. Pain of **gradual onset** is usually due to chronic overload of muscles; myofascial pain may also appear during or after a period of viral infection, visceral disease, or psychogenic stress and may develop in association with radiculopathy of its nerve supply.[31,32]

Regardless of the mode of onset, whether abrupt or gradual, pain referred from myofascial TrPs is **characterized** as steady, deep, and aching, rarely as burning.

It is to be distinguished from the prickling pain and numbness associated with paresthesias and dysesthesias of peripheral nerve entrapment or of nerve root irritation. However, two skin muscles, the platysma and palmaris longus, refer a needle-like prickling sensation superficially. Throbbing pain is more likely to be due to vascular disease or dysfunction. Occasionally, a myofascial TrP initiates sharp, lancinating, or lightning-like stabs of pain.

When TrPs in several muscles refer pain to one target area, such as the shoulder, or to a naturally sensitive area like the nipple, the zone of referred tenderness may become intolerant of the lightest touch and exquisitely sensitive to pressure.

An essential part of the history is to determine in detail which activities and postures aggravate the pain and which ones relieve it.

Myofascial TrP pain is characteristically **aggravated:**

1. By strenuous use of the muscle, especially in the shortened position. Defining precisely the movement that increases the pain provides a major clue to the muscle that harbors the responsible TrPs.
2. By passively stretching the muscle. However, active stretch by voluntary contraction of the antagonist may only rarely cause pain because the patient subconsciously learns to limit this movement. The patient is aware of restricted range of motion and "weakness," but may not think of the affected muscle as painful.
3. By pressure on the TrP.
4. By placing the involved muscle in a shortened position for a prolonged period. Pain and stiffness are often at their worst when the patient gets out of bed in the morning, or when getting up from a chair after sitting immobile for a while.
5. By sustained or repeated contraction of the involved muscle.
6. By cold, damp weather, viral infections, and periods of marked nervous tension.
7. By exposure to a cold draft, especially when the muscle is fatigued.

Myofascial TrP pain is **decreased:**

1. By a *short* period of rest.
2. By slow, steady passive stretching of the involved muscles, particularly when the patient is seated under a warm shower or in a warm bath.
3. When moist heat is applied over the TrP. The pain is decreased much less when the heat is applied over the reference zone.
4. By *short* periods of light activity with movement (not by isometric contraction).
5. By specific myofascial therapy (Sections 12 and 13 of this chapter).

The development of a new pain during treatment must be diagnosed on its own merits and may not be myofascial in origin.

Limited Range of Motion

This is rarely the chief complaint, but it is a fundamental characteristic of TrPs that is readily identified by the pain that develops as the muscle approaches full stretch range of motion. Limitation of motion and increased stiffness are worse in the morning and recur after periods of overactivity or immobility during the day. This painful stiffness is apparently due to the abnormal tension of the palpable bands and to tension-induced sensitivity of the taut-band fiber attachments.

Weakness

Frequently, patients are aware of weakness of certain movements, as when pouring milk from a carton, turning a doorknob, or carrying groceries in one arm. This yields clues as to which muscles are involved. The muscle learns to limit the force of its contraction below the pain threshold of the central and attachment TrPs.

Weakness may be a reflection of inhibition referred from a TrP in another muscle (for example, inhibition of the anterior deltoid by a TrP in the infraspinatus muscle).[64]

Other Non-pain Symptoms

Patients may report excessive lacrimation, nasal secretion, pilomotor activity and occasionally changes in their sweat patterns, but TrP activity is rarely seriously considered as the source of these symptoms. An involved limb may feel cold as compared with the opposite one due to reflex vasoconstriction. The examiner should be alert for symptoms of postural

dizziness, spatial disorientation, and disturbed weight perception. All of these phenomena can be caused by myofascial TrPs; some are specific to particular muscles, others are not.

Depression

A major, well-recognized contributor to depression is chronic pain. On the other hand, depression may lower the pain threshold, intensify pain, and impair the response to specific myofascial therapy. Patients who have suffered myofascial pain for months or years are likely also to have developed secondary depression and sleep disturbances, and to have restricted their activity and exercise. The ensuing restriction of body movement and the increased psychic tension aggravate their TrPs, causing a vicious cycle. *All* contributory factors should be identified and corrective actions taken.

Depression must be recognized. If untreated, or undertreated, it blocks recovery from myofascial syndromes. It is diagnosed by a variety of clues. Physiologic clues are insomnia, anorexia and weight loss, impotence or decreased libido, or blurred vision. Mental-outlook symptoms include a sad mood, thoughts of suicide or death, and strong feelings of guilt. Other clinical changes are inability to concentrate, poor memory, indecisiveness, mumbled speech, and a negative reaction to suggestion. Socially, the patient exhibits a desire to be alone, disinterest in favorite activities, a drop in job performance, and neglect of personal appearance and hygiene.

Folic acid or pyridoxine deficiency and low thyroid function are potent contributors to depression, and may, in addition, increase neuromuscular irritability and TrP pain. An analysis of the problem should include, "What are the unique characteristics of this patient who has the pain?," not just, "What TrP involvement does this patient have?"

With developing depression, patients describe increasingly restricted movements and activity as their way to avoid pain. After a few weeks, most patients have discontinued their previous exercise program, and the unstretched muscles become increasingly deconditioned and irritable. This potentiates their tendency to develop TrPs and makes them less responsive to treatment.

Sleep Disturbances

A careful history identifies the seriousness and nature of sleep disturbance. Depressed patients tend to fall asleep readily, but awaken in the night and have trouble sleeping again. They arise in the morning feeling more tired than when they went to bed, suggesting fibromyalgia. Some patients are awakened by their myofascial pain, others by noises. Each is managed in terms of the cause.

Prognosis

Acute myofascial pain due to TrPs caused by a clearly identifiable strain of one muscle is, as a rule, able to be fully relieved and normal function restored. The longer the period between the acute onset of pain and the beginning of treatment, the greater the number of treatments that will be required over a longer period of time.[73]

Patients who have had a stable pattern of referred TrP pain for months or longer, without extension to other muscles, are likely to respond better to treatment than patients with progressively more severe symptoms. When the pain has spread and is gaining momentum with successively more muscles becoming involved, multiple perpetuating factors must be eliminated before specific myofascial therapy can provide sustained relief.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Acute events that precipitate a sudden onset of symptoms and the chronic stresses that are likely to produce a gradual onset of TrP symptoms are both considered here. One time traumatic occurrences can activate TrPs but are not responsible for perpetuating them. Other factors, such as those considered in Chapter 4, are responsible for maintaining their activity. Situations that cause repeated or chronic muscular overload can activate TrPs and then perpetuate them. In this latter case, the muscular stress is both an activating and a perpetuating factor. Obviously, from a clin-

ical management point of view, these two kinds of onset can result in quite different sets of problems that require different therapeutic considerations and approaches. Therefore, sudden onset and gradual onset are considered separately in this section.

Sudden Onset

When asked, "Do you remember the day your pain started?," most patients will respond either with a clear affirmative or a fuzzy negative. If affirmative, the details of posture and movement occurring at the time of onset permit estimation of the degree of stress that was imposed on various muscles. Sometimes, the pain was felt at the moment of stress; at other times, the patient remembers feeling "something happen" or hearing "a snap" at the moment of stress, but the pain developed gradually several hours later, reaching a maximum in 12-24 hours. Either is considered an acute single-event onset. The delayed onset can be a response to another soft tissue injury (as described under Low-Back Pain in Chapter 41) that causes reflex spasm and can induce secondary TrPs.

The mechanical stresses that tend to *activate* myofascial TrPs acutely include stresses such as a wrenching movement, automobile accidents, falls, fractures (including chip fractures), joint sprains, dislocations, or a direct blow to the muscle.[165] Acute onset also may be associated with an episode of excessive or unusual exercise, such as packing and handling boxes when moving.[165] Most of the time, myofascial TrPs due to such one-time gross trauma are easily inactivated as soon as any associated soft-tissue injury has healed; however, the TrPs may persist for years if untreated.

Intramuscular injection of medicinal substances given inadvertently at the site of a latent TrP may activate it.[163,166] The patient feels a local pain before the solution is injected when the needle tip reaches a TrP. If the injection is delayed for a few seconds, this pain can be distinguished from a second intense referred pain caused by activation of the TrP when a locally irritant medication is injected. It is wise to palpate for a non-tender area to insert the needle and to relocate the needle before injection, if its insertion encounters TrP tenderness. This activation of a latent TrP can be avoided by adding procaine to make a 0.5% solution. The procaine also reduces postinjection soreness compared to dry needling .[67]

Latent TrPs may be activated incidental to spray-and-stretch therapy. While one group of muscles is being passively stretched, their antagonists are shortening much more than usual. Fortunately, if latent TrPs in the antagonists are painfully activated in this way, they can be inactivated quickly by then spraying and stretching them.

During injection of an especially active TrP, the intense referred pain may activate latent TrPs in muscles in the reference zone. For instance, injection of scalene muscles has activated TrPs in the brachialis muscle, which entrapped the radial nerve and caused paresthesia and tingling of the thumb. Similarly, severe pain referred to a somatic area due to an acute visceral lesion, such as myocardial infarction or appendicitis, is likely to activate TrPs in the painful region of the chest wall or abdomen.[165]

Latent TrPs in a fatigued muscle, especially in the calf or neck and shoulders, may be activated by direct cooling of the overlying skin, as by a cold draft from air conditioning or an open car window.

Gradual Onset

Locating the cause of active TrPs that developed gradually due to chronic overload can be difficult, but it is important because the chronic strain, if continued, *perpetuates* and may intensify the TrPs. Typical causes of sustained postural overload are poor work habits, such as a slouched posture or a keyboard operator lifting the shoulders to reach an elevated keyboard. If the source of strain is not obvious, the patient must help to identify it. The patient should be instructed in the kind of movements that would overload the involved muscle, and then watch for daily activities that use that motion. The patient should also note any movement or activity that increases the referred pain, and then avoid it, or learn how to perform the activity (if essential) without overloading the muscles. Minutes spent tracking down precisely

what activated the TrPs can prevent recurrences and save hours of frustrating treatment time and treatment failure.

Synergistic muscles that axe overloaded by substituting for an involved muscle, or are in sustained contraction to protectively splint an involved muscle are themselves likely to develop secondary TrPs.

A muscle that is immobilized in the shortened position for prolonged periods tends to develop active TrPs.[165] This was demonstrated by the increased likelihood that patients with acute coronary thrombosis would develop a painful or frozen shoulder syndrome due to myofascial TrPs when they were kept flat on their backs in bed without regular, gentle, active motion of the upper extremities.[169]

Nerve compression, such as in the radiculopathy caused by a ruptured intervertebral disc, favors the development of TrPs in the muscles supplied by the compressed nerve root (postdisc syndrome).[165,190] Less severe radiculopathy also can activate TrPs.[31,32]

The "nervous tension" associated with emotional stress or psychological tension can induce TrPs.[80,96,165] The associated increased TrP activity[97,116] most likely is mediated by autonomic nervous system activity.

Muscle pain syndromes are commonly seen in patients with any of a number of viral diseases, including acute upper respiratory tract infections.[38]

8. PATIENT EXAMINATION

This section considers the examination of the patient for dysfunctions and phenomena that characteristically are produced by TrPs. It assumes that the clinician has taken or reviewed the patient's complete medical history, and that the patient has received a general medical examination that paid special attention to neurological function in order to distinguish symptoms of neurological origin from those of myofascial TrP origin. The examination of the muscle itself for evidence of TrPs is covered in Section 9, Trigger Point Examination. This patient examination section distinguishes between primary TrP effects that are the direct result of the TrP pathophysiology and secondary effects that are induced by the TrP activity. It is important to understand these basic principles because no two patients are alike. Only identical twins have identical genetic origins, and no two people have the same exposure to environmental influences during development. Even identical twins have different fingerprints. When it comes to musculoskeletal pain, there is no one-shot, cure-all silver bullet.

Patient Mobility and Posture

The patient's spontaneous posture and movements should be observed while he or she walks, sits, or removes articles of clothing *[see* Chapter 41, Section C for comments regarding posture and movement). People with painfully active TrPs tend to move slowly and protectively. They avoid, or explore gingerly, movements that might painfully stretch or load muscles with TrPs, and they may be compensating for muscles that are weak because the muscles are reflexly inhibited by TrPs in the same or other muscles. Some key observations: Does the patient use arms and hands bilaterally in their full range of motion? Does the body, rather than only the head, turn when the patient looks around? In the sitting position, is the spine crooked and one shoulder lower than the other? Is the face symmetrical? Does the patient perform spontaneous stretching movements for relief; if so, what muscles are being stretched?

Neuromuscular Functions

This heading includes examination for restricted stretch range of motion, weakness, distorted weight perception, and weak deep-tendon reflexes. Restriction of stretch is the primary effect of the increased muscle tension and shortening caused by the TrP mechanism. The restriction is augmented by pain arising secondarily from sensitized nociceptors in central TrPs and at attachment TrPs. On the other hand, weakness is caused secondarily by reflex motor inhibition induced by TrPs in the same muscle or in other muscles.

Some people have inherently poor muscular coordination; they move jerkily and quickly. Some individuals are tense and maintain a residual and unnecessary co-contraction of antagonist muscles. These are among the most difficult patients to treat because they keep misusing and abusing their muscles. On the other hand, the muscles of highly coordinated athletes

quickly learn to inhibit specific movements to avoid pain and thus develop weakness. With treatment, these athletic patients are likely to reestablish their normal function quickly.

Restriction of Movement. A muscle containing *active* TrPs is functionally shortened and somewhat weakened. Attempts to passively extend the muscle to its fully stretched length cause pain at less than normal range. This painful restriction of the passive stretch range of motion can be quickly detected by screening tests. Range of motion in the shortened position shows little or no restriction, but additional contraction effort in this position is likely to be painful. The characteristic painfulness to passive stretch in one direction and to active contraction in the other was reported specifically for 10 muscles by Macdonald.[109] Any movement, especially a quick maneuver, that markedly increases tension in the muscle, either stretching or contracting it, can cause pain.

In order to identify *active or latent* TrPs that may limit range of motion and thus influence dysfunction, as Boeve[18] suggested, the examiner should: (1) identify limited range of motion by performing specific range of motion testing for a particular segment; (2) take up slack to the point of tension before changing the position; (3) ask the patient *where* he or she feels the tension or where it hurts; (4) search (palpate) *there* for a taut band and TrP. Boeve[18] identified the TrPs that were located in this way as *relevant* TrPs. Such TrPs can produce dysfunction whether or not they produce pain.

As a screening test for normal range of head and neck muscles, the seated patient should be able to place the chin firmly on the chest, to look straight up at the ceiling, to turn the head at least 90% so that the chin points to the acromion, and to place the ear close to the shoulder without shrugging. For screening shoulder-girdle muscles with the Mouth-Wrap-around Test (Fig. 18.2), the hand should cover at least half of the mouth with the arm *behind* the head. When performing the Hand-to-shoulder-blade Test (Fig.22.3), the fingertips on the non-dominant side normally reach to the spine of the contralateral scapula. Reach with the dominant hand is usually 1 or 2 cm less than with the non-dominant hand. The Mouth Wrap-around Test is restricted to a greater degree by subscapularis TrPs than by TrPs in other shoulder muscles. The Hand-to-shoulder- blade Test range of motion is restricted the most by infraspinatus and anterior deltoid TrPs. Supination and pronation of the forearm are also tested because restricted range of these motions can overload the shoulder muscles as they attempt to compensate.

The stiffness and the relatively painless, but progressive, restriction of movement that characterize decrepitude of advancing age are often due largely to latent TrPs. These latent TrPs do not spontaneously refer pain. They cause muscle shortening and can restrict stretch range of motion without the patient being aware of the limitation because the muscles have learned to restrict movement to within the painless range. These latent TrPs respond as well to specific myofascial therapy and a regular stretching program as do active TrPs, relieving this decrepitude.

Although the range of motion of a muscle with TrPs does not test passively as being restricted at the *shortened* end of the range, such a muscle is intolerant of being left relaxed in the shortened position for a period of time, and quickly develops a cramp-like pain on voluntary contraction in the shortened position. This pain can be explained theoretically as the result of intensification of the shortening process at the contraction knots responsible for the TrP phenomena. Placing the muscle in a shortened position reduces the tension of the taut band, which could allow additional contraction of sarcomeres in the region of the contraction knot, thereby increasing the energy demand and intensifying its local energy crisis. This would increase sensitization of local nociceptors (refer to Chapter 2, Section D). However, gentle voluntary contraction with the muscle in the lengthened position should help to normalize sarcomere lengths throughout muscle fibers with contraction knots and contribute to recovery.

The Scalene-cramp Test *[see* Fig. 20.4) gives an example of cramping caused by contracting a muscle with TrPs in the shortened position. In addition to causing this cramping, TrPs in a scalene muscle can cause weakness and restricted range of motion in the extensor digitorum communis as evidenced by the Finger-flexion Test *{see*

Fig. 20.6). The weakness may be explained by reflex motor inhibition referred from the scalene TrPs to the extensor digitorum communis. The restricted range of motion is caused by satellite TrPs induced in the extensor digitorum communis by the key scalene TrPs. These motor referred phenomena are comparable to the pain referred from scalene TrPs to the same region.

Weakness. It is important to think beyond the obvious weak function of a muscle or muscle group and not to assume that all it needs is strengthening exercises. *The clinician must determine* **why** if *is weak and the* ***type*** *of weakness.* Myofascial TrPs contributing to or causing the weakness may be in the same muscle and/or in functionally related muscles.

Weakness resulting from TrPs may be detected by testing for static or dynamic muscle strength and the two methods can result in completely different answers. Static testing, as performed in conventional testing of muscle strength, attempts to produce a voluntary contraction of just the muscle being tested. This action depends strongly on direct cortical control of muscle contraction. Dynamic testing monitors muscle activity while the subject is performing functionally meaningful tasks that have been learned and that require muscle coordination. This activity is largely under cerebellar control and is much more susceptible to reflex inhibition. The monitoring of dynamic weakness may be done by palpation, but may be done quantitatively and more accurately with recordings using surface electromyography (EMG).

Sudden premature cessation of effort by the patient during static testing may be due to painful loading of distant stabilizing muscles, to painful loading of the muscle being tested, or to a sudden inhibition of effort just short of painful loading that has been "learned" by the muscle being tested. Determined effort by the subject can override at least some of this learned pain inhibition, and the amount and location of the pain associated with this kind of augmented strength testing can help to locate the inhibiting TrPs. Inactivation of these inhibiting TrPs may completely restore normal strength.

On the other hand, the reflexly induced weakness identified during dynamic testing is not under such direct cortical control. It requires inactivation of the responsible TrPs and often requires reeducation of the patient's motor control to "unlearn" the dysfunctional, poorly coordinated activity pattern caused by the TrP.

Distorted Weight Perception. Testing for the disturbance of weight appreciation caused by sternocleidomastoid TrPs is described in Chapter 7, Section 8. Loss of fine coordination among the muscles of mastication due to active TrPs in those muscles is described in Chapter 5.

Weak Deep-tendon Reflexes. Myofascial TrPs in a muscle can reduce the briskness of the deep-tendon reflex response elicited by tapping the tendon of that muscle. A weak or even absent ankle jerk due to active TrPs in the soleus muscle demonstrates this when, within minutes following inactivation of the TrPs, the previously weak ankle jerk equals that of the normal side.

Referred Tenderness

Referred pain and referred tenderness are closely related neurophysiologically. Most of the animal studies that are interpreted as relevant to the neurophysiological mechanisms responsible for referred pain are actually studies of referred tenderness.[120-145] One study in human subjects of active TrPs (pressure induced local and referred pain) and of latent TrPs (pressure produced local pain only) was reported by Vecchiet, *et al.*[177] They examined the tenderness to electrical stimulation in the TrP region, in the pain reference zone, and in contralateral control regions. Measures were taken of skin, subcutaneous, and intramuscular sensitivity to electrical stimulation at each site. Pain thresholds were significantly reduced intramuscularly at both sites (TrP region and pain reference zone) for both kinds of TrPs (active and latent), but thresholds were more markedly reduced by active TrPs and most markedly reduced at the TrP compared to the pain reference zone. The same pattern applied to subcutaneous thresholds except that they were lowered only in the more active TrPs. Pain thresholds were consistently lower in patients with more active TrPs. Pain sensitivity in the reference zone relates strongly to the irritability of the TrP.

A subsequent study[178] reported similar findings where pain thresholds to electri-

cal stimulation were significantly decreased at TrP sites in skin, subcutaneous and muscular tissues, as compared to an uninvolved control site in another muscle.

Trigger point referred tenderness must be distinguished from enthesopathy. Trigger point referred tenderness is distributed diffusely throughout the involved area and is not well localized. Enthesopathy presents a circumscribed tenderness specifically in the regions of muscle attachment. When enthesopathy is caused by central TrPs, the tenderness is localized where the taut bands (of the TrPs) attach.

Cutaneous and Subcutaneous Signs

Dermographia. Dermographia has been strongly identified with the fibrositis syndrome (the use of the term fibrositis was closely related to myofascial TrPs).[91] We find that dermographia in the skin overlying muscles with active myofascial TrPs occurs most often over muscles of the back of the neck, shoulders and torso, and less frequently over limb muscles. Regular use of an antihistamine may be indicated. No experimental investigation is known that has systematically explored the relation between myofascial TrPs and this phenomenon. One is needed.

Panniculosis. Despite the early use of the term panniculitis[5,114] and the subsequent interchangeable use of panniculosis and panniculitis to characterize *diffuse* subcutaneous induration,[21] panniculitis is now described in a current rheumatology text[126] as a nodular condition of the skin that is associated with erythema nodosum and with the termination of steroid therapy. This description of panniculitis does not fit the condition we identify here as panniculosis. In panniculosis, one finds a broad, flat thickening of the subcutaneous tissue with an increased consistency that feels coarsely granular.[21] It is not associated with inflammation. Panniculosis is usually identified by hypersensitivity of the skin and the resistance of the subcutaneous tissue to "skin rolling."

Skin rolling is accomplished by picking up a fold of skin and subcutaneous tissue between the fingers and the thumb, and moving the hand across the surface by rolling the fold forward, as clearly described and illustrated by Maigne.[111] The peculiar, mottled, dimpled appearance of the skin in panniculosis indicates a loss of normal elasticity of the subcutaneous tissue, apparently due to turgor and congestion.[114] This "peau de orange," or orange peel effect, and the persistent indentations of the "matchstick test," but without evidence of pitting edema, have been beautifully illustrated for the skin of the back under the term trophedema.[62] However, Dorland[3] defines trophedema as "a disease marked by permanent edema of the feet or legs, which is not what the authors[62] described.

Boos[21] observed that panniculosis is associated occasionally with the symptoms of "Muskelrheumatismus" (muscular rheumatism), "Muskelhartspann" (muscular firm tension), and "Myogelosen" (myogeloses or muscle gellings), because topographically the panniculosis is distributed similarly in all of those conditions. McKeag[114] considered panniculosis a form of fibrositis. All four of these diagnostic terms frequently were used to identify findings characteristic of myofascial TrPs. Boos[21] noted that freely mobile cutaneous tissue excludes panniculosis. We find panniculosis in a distribution and with a frequency similar to that of dermographia (above), but not necessarily in the same patients.

Panniculosis should be distinguished from adiposa dolorosa[21] and from fat herniations.[35, 114]

It is not known why some patients with myofascial TrPs show dermographia and/or panniculosis, and others do not. These conditions may be different forms of mild autoimmunity. In panniculosis, the subcutaneous tissue exhibits increased viscosity that responds to the application of barrier release pressure in a manner suggestive of thixotropy.[146, 183] This increased viscosity may be related to sympathetic nervous system activity and seems to have some channel of interaction with the TrP mechanism in underlying TrPs. Skin rolling applied as a series of treatments can normalize the panniculosis and can also relieve underlying TrP activity or make the TrPs more responsive to treatment. A well-designed study is needed that critically evaluates the relation between TrP activity and the presence of overlying panniculosis. The study could employ separate treatment of the TrPs and of the panniculosis, observing

what effect the treatment of one has on the other.

Compression Test

When a patient presents with myofascial pain felt only during movement (not at rest), manually compressing the muscle responsible for that movement (while the movement is being performed) sometimes prevents the referred pain. For example, when sternocleidomastoid TrPs cause pain on swallowing, firmly squeezing a roll of the skin overlying that sternocleidomastoid can block the pain and render swallowing temporarily pain-free. Section 8 of Chapter 34 describes the compression test for TrPs in the hand extensors that cause pain during handgrip. Painful abduction of the arm caused by a TrP in the upper trapezius is relieved by firm pressure on that muscle with the palm of the hand in the midscapular line during abduction.[89]

This Compression Test can be used to demonstrate to the patient the myofascial TrP origin of the pain without imposing additional pain. When patients have already heard numerous explanations for their pain from many doctors, they are naturally incredulous of yet another and unfamiliar explanation for their pain. First augmenting the patient's pain by pressure on the TrP, and then relieving it by the Compression Test, helps to convince the patient that the pain has a definite muscular source which responds to treatment. The neurological mechanism that makes the compression test effective may relate to the mechanism responsible for the effectiveness of vapocoolant spray. These mechanisms deserve experimental investigation.

Joint Play

Loss of joint play is a common cause of pain-producing joint dysfunction that commonly interacts strongly with myofascial TrPs. This joint dysfunction is considered an important component of osteopathic somatic dysfunction by Jacobs and Falls,[79] who state that, "The restoration of joint play appears to be the basis for the success of synovial joint mobilization using direct or indirect action treatment techniques in osteopathic manipulation." Joint play examination and treatment are frequently simple and full recovery can often be greatly expedited by examining and treating the patients promptly for restricted joint play, especially if inactivating the TrPs does not fully restore normal range of motion or if the TrPs recur promptly.

In 1964, Mennell[118] described how to examine for loss of joint play throughout the body and how to restore it. Since that time, joint play has become recognized and appreciated by the osteopathic profession,[59,79] and by many physical therapists, but is often neglected by others. The movement of joint play can not be induced by voluntary muscular effort or by passive movement of a joint through its functional range. It is normally a painless accessory movement that is essential to normal pain-free joint function and must be performed passively by an examiner. It is usually a movement of only a few millimeters that occurs roughly perpendicular to a major plane of voluntary movement at that joint. Lost joint play often can be restored quickly by a simple, gentle manipulation performed by one who is skilled in the appropriate technique for that joint.

9. TRIGGER POINT EXAMINATION

Limitations of stretch range of motion and records of referred pain patterns help to identify which muscles to examine for active TrPs; palpation and observation of TrP phenomena confirm which muscles are responsible for the myofascial pain.

This section deals with how to examine a muscle for TrPs. The anatomy drawing(s) in each chapter can assist the examiner in locating a specific muscle. To confirm its location, with one hand the examiner resists a voluntary movement by the patient that contracts the muscle, and with the other hand palpates for muscle contraction.

While the muscles are being examined for TrPs, the patient should be comfortable and warm. The muscle *must* be relaxed; otherwise, the distinction between tense bands and adjacent slack muscle fibers is diminished or lost.

Before the examiner attempts to palpate a muscle for TrPs, the examining digits *must* have the fingernails trimmed very short. This is especially critical during pincer palpation and when attempting to elicit digital LTRs. An appreciable length of fingernail not only causes the patient unnecessary (sometimes severe) pain, but the skin pain caused by long fingernails is

readily misinterpreted as TrP tenderness. One common reason for unreliable results when attempting to elicit LTRs is failure to use the finger tip because the fingernail of the palpating finger is inadequately trimmed. The skin pain from the fingernail prevents application of sufficient pressure to elicit the LTR, and the fingernail mechanically interferes with use of the tip of the finger to apply the pressure. Adequate trimming of fingernails is a fundamental requirement that is commonly ignored.

For those who have difficulty in recognizing TrPs by palpation, a dermometer, or similar device to measure skin conductance or skin resistance, is sometimes used to explore the skin surface for points of high conductance (low skin resistance), which apparently often overlie active TrPs. This device may be helpful, but has not been shown to be highly reliable. Use of the dermometer for identifying TrPs needs experimental evaluation of its reliability and, if reliable, investigation of the neurophysiological basis for its effectiveness.

Palpating TrPs can severely exacerbate the patient's referred pain activity for a day or two. For this reason, it is critically important to examine a muscle for TrPs *only* if the examiner then applies specific myofascial therapy such as spray and stretch followed by moist hot packs to muscles with TrPs. When the examiner neglects this caveat, patients with myofascial TrP pain come to dread a physical examination that includes palpating muscles for TrPs. The clinical rule is: palpate for TrPs in only those muscles that can be treated during the same visit. This consideration also should be incorporated into research protocols whenever practical.

Diagnostic Criteria

The reliability with which the physical features of TrPs could be determined was evaluated by four experienced physicians who, following a three hour training session immediately before the study, examined five pairs of muscles for five physical characteristics of TrPs in each of ten subjects.[52] The muscles examined were the infraspinatus, latissimus dorsi, upper trapezius, extensor digitorum, and sternocleidomastoid. Agreement among examiners was at least substantial and sometimes almost perfect for the detection of spot tenderness, a taut band, presence of referred pain, and reproduction of the subject's symptomatic pain. However, agreement on the presence or absence of an LTR was not as satisfactory for its use as a clinical diagnostic criterion of a TrP. The reliability for the identification of LTRs was poorest in the infraspinatus. The LTR is a difficult and relatively unreliable diagnostic test when the response is elicited manually. However, it is valuable as a strongly confirmatory diagnostic finding when elicited manually and is especially valuable when elicited during the needling of TrPs.

It now appears that the most reliable diagnostic criterion of TrPs on examination of the muscle is the **presence of exquisite tenderness at a nodule in a palpable taut band.** If, in addition, the patient recognizes pain that is elicited by digital pressure on (or needle penetration of) the TrP as his or her clinical pain complaint, the TrP is clinically **active,** not just latent. Associated phenomena, such as a characteristic pattern of referred pain or an LTR, are strongly supportive evidence. Other features, not critically evaluated but strongly characteristic of TrPs, are limited stretch range of motion and increased tension of the muscle observed during the patient examination.

Palpable Tender Nodule and Taut Band

Several other authors have recognized how critical the details of the palpation technique are for locating taut bands.[131,158] The optimal elongation of a muscle for palpating taut bands (which is usually the first step in palpating the TrP) is at a position that is slightly longer than the position of ease. In this case, the uninvolved muscle fibers are still slack, but the taut band fibers are placed under additional tension by lengthening the muscle to the point of a perceptible increase in resistance to movement. This places the taut band fibers under increased tension without tensing the uninvolved fibers (Fig. 3.6A) and produces the maximum palpable distinction between the normal tonus of the uninvolved fibers and the increased tension of the taut band fibers. This is also the optimal tension for eliciting LTRs and for making LTRs most visible. The stretch may be on the

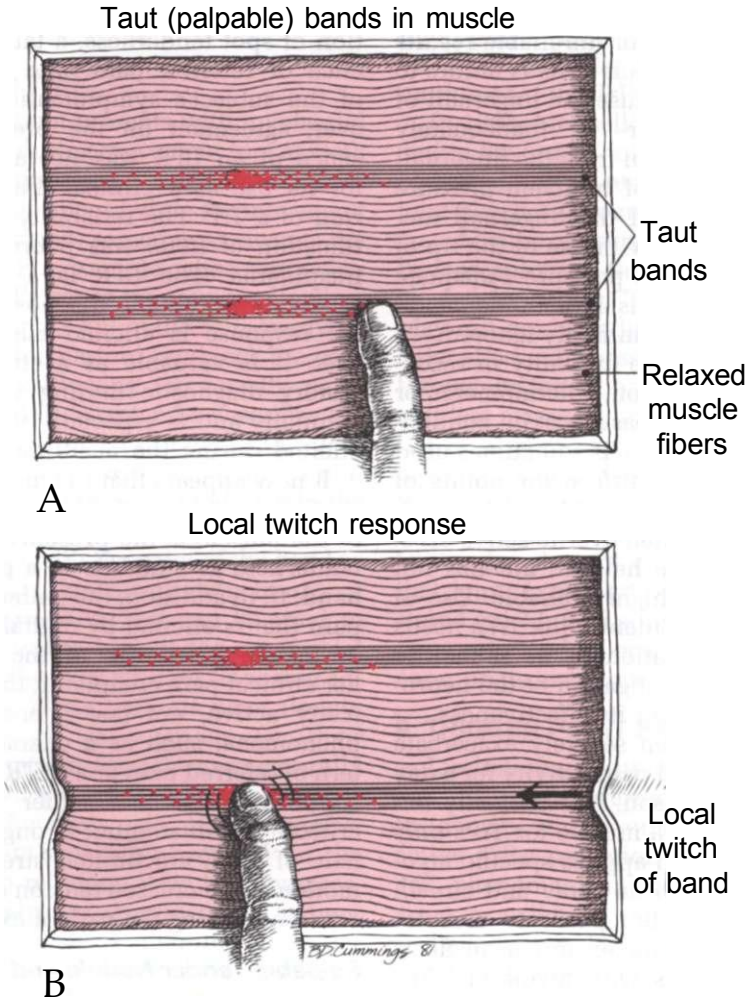


Figure 3.6. Longitudinal schematic drawing of taut bands, myofascial trigger points *(dark red spots),* and a local twitch response seen in longitudinal view of the muscle *(light red).* **A,** palpation of a taut band *(straight lines)* among normally slack, relaxed muscle fibers *(wavy lines).* The density of *red stippling* corresponds to the degree of tenderness of the taut band to pressure. The trigger point is the most tender spot in the band. **B,** rolling the band quickly under the fingertip (snapping palpation) at the trigger point often produces a local twitch response that usually is most clearly seen as skin movement between the trigger point and the attachment of the muscle fibers.

verge of causing pain, but should evoke, at most, only mild local discomfort. Optimal tension is usually about two-thirds of the muscle's normal stretch range of motion, but may be only one-third or less with very active TrPs. The reduction in range of motion varies greatly among muscles.

A taut band feels like a palpable cord of tense muscle fibers among the normally pliable fibers. Such palpable tense muscle fibers were described as "matted together" by Wilson.[189] The examiner should palpate along the taut band to locate the nodule that corresponds to a circumscribed slightly enlarged region of decreased compliance. This nodular region is also the location of maximum tenderness (the TrP).

Three kinds of palpation can be used: flat palpation, pincer palpation, and deep (probing) palpation. Flat palpation is used for relatively superficial muscles which have only one surface accessible for palpation (e.g., the extensor digitorum communis). Pincer palpation is used when oppo-

site sides of the muscle are accessible and the belly of the muscle can be grasped between the digits (e.g., the sternocleidomastoid, lateral border of the latissimus dorsi, biceps brachii, and part of the pectoralis major). Deep or probing palpation must be used for deep muscles with considerable tissue between them and the skin (e.g., the quadratus lumborum, gluteus minimus, and piriformis muscles).

In this manual, **flat palpation** refers to the use of a *fingertip* that employs the mobility of the subcutaneous tissue to slide the patient's skin across the muscle fibers. This movement permits detection of changes in the underlying structures (Fig. 3.7). First, the skin is pushed to one side of the area to be palpated (Fig. 3.7A) and the finger slides across the fibers to be examined (Fig. 3.7B), allowing the skin to bunch on the other side (Fig. 3.7C). Any ropy structure (taut band) within the muscle is felt as it rolls under the finger. A taut band feels like a cord that can range from 1 mm to 4 mm or more in diameter depending on the severity of the TrP. The sensation of applying snapping palpation across the taut band can be compared to what plucking a violin or guitar string imbedded in the muscle might feel like. In a muscle that has many TrPs, five or six such bands, or cords, may lie in such close proximity to one another that they seem to merge. If the examiner tips the palpating finger up on end to palpate with the end of the terminal phalanx, individual bands may be distinguishable. This technique *requires a* very short fingernail.

For examination of the abdomen, flat palpation using "fingertip" pressure locates *spot* tenderness in the abdominal wall, while "flathand" pressure using the flat part of the finger or hand is more likely to elicit tenderness of underlying viscera.[158] Static pressure with the finger flat can be expected to detect little more than underlying tenderness in any muscle.

The technique of **pincer palpation** is performed by grasping the belly of the muscle between thumb and fingers (Fig. 3.8A) and pressing the fibers between them with a back-and-forth rolling motion to locate taut bands (Fig. 3.8B). When a taut band is identified, it is explored along its length to locate the nodule and spot of maximum tenderness, which identifies a TrP.

When intervening tissue makes the muscle inaccessible to flat or pincer palpation, the examiner must use **deep palpation.** This means placing the fingertip over an area of skin that overlies the motor-point region or attachment of the muscle suspected of harboring TrPs. Localized tenderness that is elicited only when the finger pressure is directed in one specific direction is compatible with the diagnosis of either a central or attachment TrP if pressure elicits pain recognized by the patient as his or her pain complaint. Additional evidence, such as restricted stretch range of motion and characteristic referred pattern are helpful in making a provisional diagnosis when the usual palpable findings are inaccessible. Favorable response to specific myofascial TrP therapy helps to confirm the diagnosis.

Sufficient pressure on an active TrP almost always elicits at least withdrawal, wincing, or vocalization by the patient. In the past, if the withdrawal were sufficiently vigorous the response was identified as a "jump sign." This jerk response was noted by Good[55] in 1949 with regard to TrP characteristics that he called myalgic pain, and by Kraft *et* ai.[91] in 1968 with regard to TrP characteristics that they called fibrositis. Kraft later dubbed this response the "jump sign." This response served as a rough indication of the tenderness of the TrP that depended strongly on how much pressure was applied. Now the tenderness can be measured quantitatively using an algometer. The extreme sensitivity to applied pressure that elicits the jump sign is not by itself considered to be a sufficient diagnostic criterion of a TrP, but it is characteristic of an active TrP.

Referred Pain

The referred patterns that are characteristic of myofascial TrPs as presented in this Manual are not unique to just the TrP itself. Patterns that are similar or nearly identical may be elicited from other structures including zygapophyseal joints,[19] muscle tissue that is two centimeters removed from the TrP but still in the taut band,[71] and attachments of the muscle that exhibit enthesopathy.

Compression of either an active or latent central TrP *can* reproduce the typical pattern of referred pain of a given muscle, and

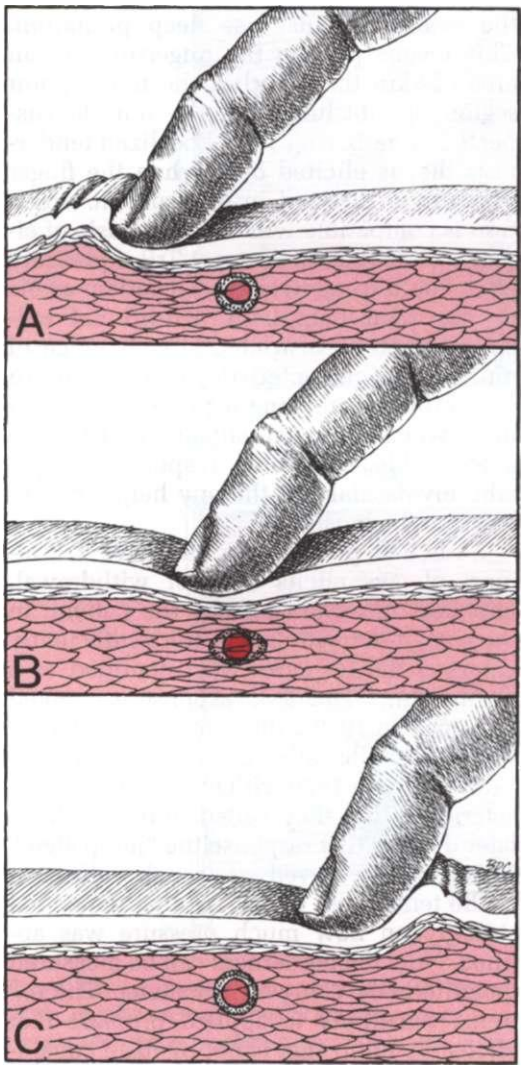


Figure 3.7. Cross-sectional schematic drawing showing flat palpation of a taut band *{black ring)* and its trigger point *(red spot).* Flat palpation is used for muscles *(light red)* that are accessible only from one side of the muscle, such as the infraspinatus. **A,** skin pushed to one side to begin palpation. **B, fingertip** slides across muscle fibers to feel the cord-like texture of the taut band rolling beneath it. **C,** skin pushed to other side at completion of movement. The same movement performed vigorously is snapping palpation.

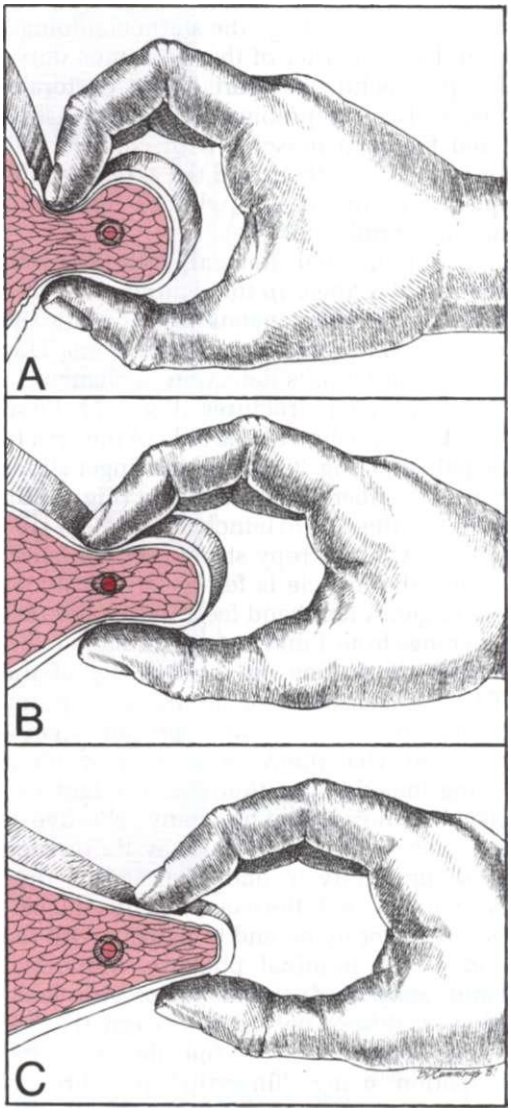


Figure 3.8. Cross-sectional schematic drawing showing pincer palpation of a taut band *(black ring)* at a trigger point *(red spot).* Pincer palpation is used for muscles *(light red)* that can be picked up between the digits, such as the sternocleidomastoid, pectoralis major and latissimus dorsi. **A,** muscle fibers surrounded by the thumb and fingers in a pincer grip. **B,** hardness of the taut band felt clearly as it is rolled between the digits. The change in the angle of the distal phalanges produces a rocking motion that improves discrimination of fine detail. **C,** the palpable edge of the taut band is sharply defined, as it escapes from between the **fingertips,** often with a local twitch response.

sometimes other sites of tenderness in muscle (such as enthesopathy) can also respond to pressure with similar referred pain patterns. Hypertonic saline injected into the muscle consistently produces these patterns. Eliciting a characteristic pain pattern at a muscle site is compatible with it being a TrP, but the finding by itself is not diagnostic of a TrP.

These characteristic referred pain patterns are most valuable as a preliminary guide as to which muscle(s) may harbor TrPs that are responsible for the patient's pain, and are useful for helping patients understand the source of their pain.

Gerwin *et al.*[52] in their interrater reliability study noted that the one criterion which distinguished an active TrP from a latent TrP was the patient's *recognition* of his or her pain complaint when the active TrP was compressed.

Local Twitch Response

In 1955, Travell[165, 166] and Weeks and Travell[185] reported a localized twitch of part of the muscle when the TrP was rolled under the fingers. The twitch could be vigorous enough to cause a perceptible jerk of the body part. Travell previously had observed this twitch response when a needle was inserted into a trigger area.[163] The EMG characteristics of LTRs were reported in 1976 by Simons,[143] but the LTR was then *misnamed* the "jump sign," which refers to a different phenomenon, as noted above.

The LTR is a transient contraction of essentially those muscle fibers in the tense band that are associated with a TrP (Fig. 3.6B). It may be *seen* as a twitch or dimpling of the skin near the terminal attachment of the fibers, or *palpated* through the skin with the examining hand. The response is elicited by a sudden change of pressure on the TrP, usually produced by transverse snapping palpation of the TrP across the direction of the muscle fibers (taut band), or by needle penetration into the TrP.[143'149] The optimal muscle length for eliciting an LTR by snapping palpation is the same as that for examining the muscle for taut bands as described above. The closer to the TrP that the taut band is stimulated by snapping, the more vigorous is the LTR.

The LTR is readily elicited and perceived in the muscles that permit pincer palpation. Other superficially placed muscles, such as the deltoid, gluteus maximus, vastus medialis, and the finger and wrist extensors, are likely to exhibit strong LTRs in response to snapping palpation with a fingertip. An LTR is not likely to be elicited by palpation of deep muscles, like the subscapularis or multifidi, but it can be elicited by needle contact with the TrP.

Most muscles exhibit a vigorous LTR only if they harbor active TrPs, but the middle finger extensor, in most adults, contains a latent TrP that responds with a readily visible LTR. In one study, the LTR was most reliably observed in this muscle as compared to four others[52] apparently because the response here is so accessible, so common, and so easily elicited. An LTR in this muscle extends the middle finger, which makes it obvious. For this test the *relaxed* arm rests on a table or armrest of a chair, and the wrist hangs over the edge. The tender spot is located in a palpable band of the middle finger extensor about 2 cm distal to the lateral epicondyle *[see* Fig. 35.1 A). With the forearm and hand to be tested fully relaxed, the TrP is rolled under the fingertip with rapid, strongly applied, snapping palpation *[see* Fig. 35.4), and the extensor response of the middle finger is observed.[143' 149]

The LTR elicited by snapping palpation or needle penetration has been studied electromyographically.[149] The LTR lasted from 12-76 msec in response to needle stimulation. Clinical evidence[69] and animal research studies[74,75] indicate that the LTR depends upon a spinal-level reflex mechanism.

In summary, Gerwin, *et al.*[52] showed that in many muscles it requires so much training and skill to elicit LTRs reliably by palpation, that for most clinicians an LTR is generally not a satisfactory criterion for making the diagnosis of myofascial pain caused by TrPs. When an LTR is elicited manually in the presence of other palpable indicators of a TRP, it is a strongly confirmatory finding. However, Hong[67] demonstrated that an LTR is a valuable indicator

of having found the mark whenever *needling* TrPs.

Central and Attachment Trigger Points

Elucidation of the pathophysiology responsible for central and attachment TrP phenomena, as illustrated in Figure 2.25 (and its associated text), makes it necessary to distinguish central TrPs located in the endplate zone of a muscle and attachment TrPs that occur in a region of attachment of the muscle. Fischer[46] recognized the importance of this distinction from a therapeutic point of view. The difference in pathophysiological mechanisms involved is also important.

The primary central TrP abnormality is associated with individual dysfunctional endplates in the endplate zone (or motor point). This dysfunction produces a local energy crisis that results in sensitization of local nociceptors. This dysfunction can produce contraction knots which then produce a nodule and a taut band of tense muscle fibers.

The attachment TrPs result from the sustained increased tension of these muscle fibers at the attachment point. This sustained tension can produce enthesopathy with swelling and tenderness where the muscle fibers attach to an aponeurosis, tendon, or bone. Some muscles have sufficient separation between the muscle fiber-to-tendon attachment and tendon-to-bone attachment that one end of the muscle may have two distinctly different attachment TrPs.

Figure 3.9 illustrates the location of a central TrP and of two corresponding attachment TrPs in the temporalis muscle. Sensitization of local nociceptors causes the pain in both kinds of TrPs, but the processes by which the sensitization develops are different in each. Table 3.2 lists the clinical findings characteristic of central TrPs as compared to attachment TrPs and includes the cause of each finding.

In the first edition of this volume, no distinction was drawn between central and attachment TrPs. Although the time available to incorporate this new understanding into all individual muscle chapters was limited, a major effort was made to identify the difference for the most obvious examples. Incorporation of an understanding of these two kinds of TrPs and the therapeutic ramifications should provide a major opportunity for advancement in the clinical practice of myofascial pain in the coming years.

Key and Satellite Trigger Points

A Key Myofascial TrP is one that is responsible for the activity of one or more satellite trigger points. Clinically, a key TrP becomes apparent when inactivating it also inactivates its satellite TrPs without direct treatment of the satellite TrPs themselves. This relationship was noted occasionally in the first edition of this volume. Many additional pairs of key and satellite TrPs are presented in Table 3.3, which is based largely on observations reported by Hong.[68] Figure 3.10 illustrates key TrPs in the upper trapezius and sternocleidomastoid muscles with corresponding satellite TrPs in the digastric, masseter, and temporalis muscles.

Key and satellite TrPs are *related* TrPs. Sometimes the "hierarchy" appears clear, but which TrP came first (or which is most important) is not always evident. What is clear is that TrPs in certain muscles are related to TrPs in certain other muscles; successfully treating one of these related TrPs may also inactivate the other. The role as to which muscle harbors the key TrP may sometimes reverse.[102] Knowledge of these relationships is used to examine for Key TrPs that might be overlooked when the patient complains chiefly of symptoms produced by the satellite TrP.

Whiteside[187] described an interesting example of a three-step satellite TrP phenomenon. A final-year physiotherapy student complained of a toothache that developed in her right upper jaw along with an ache in her right upper trapezius muscle when she studied for long periods of time. She had received extensive dental treatment including a root canal without relief. In response to firm pressure on a TrP in the right *lower* trapezius she said, "I am getting the dull ache in the *upper* trapezius that I get when I study." In response to pressure on an upper trapezius

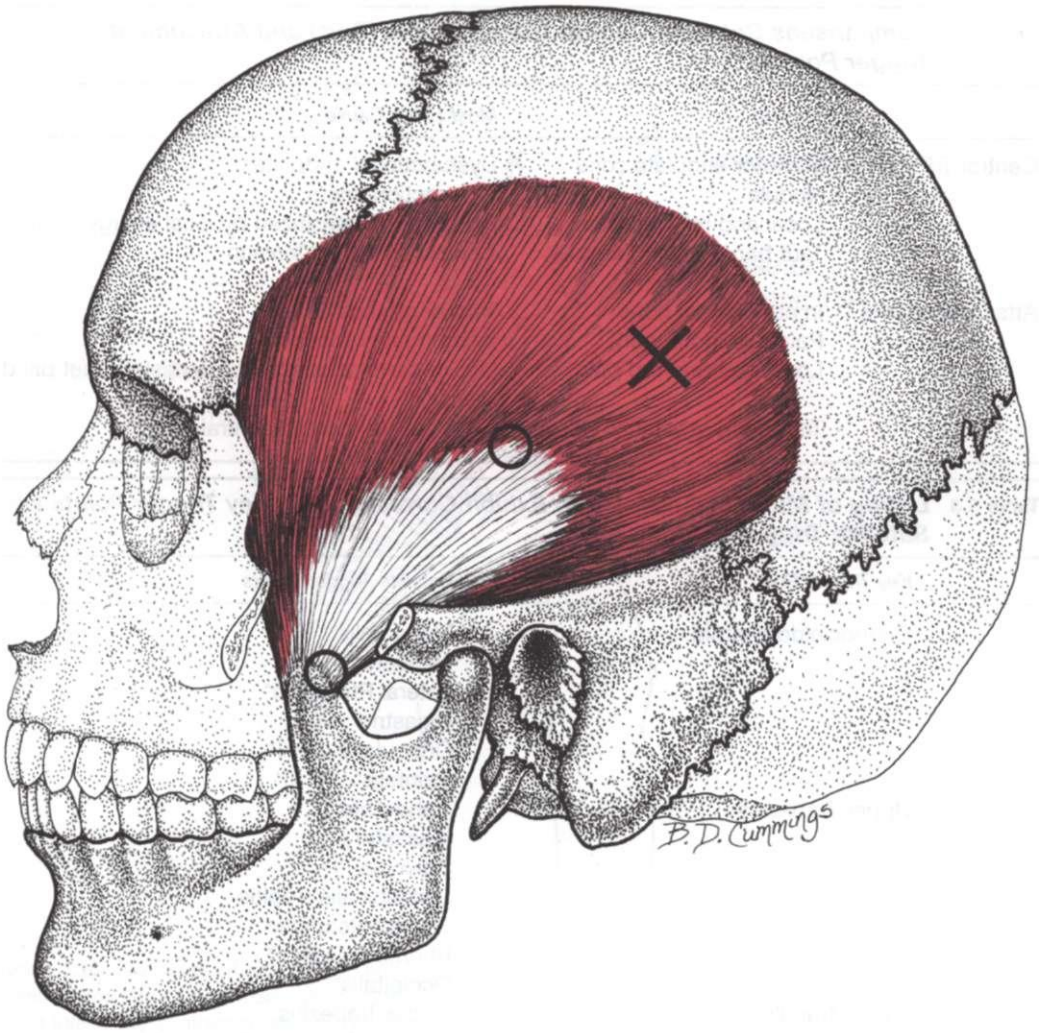


Figure 3.9. Example of a central trigger point *X* and two attachment trigger points *(black circles)* both of which correspond to regions of tension caused by the central trigger point. The uppermost attachment trigger point occurs at the musculotendinous junction, and the lower one occurs where the tendon attaches to bone. In this situation, an additional attachment TrP (not identified) could occur where the fibers of the taut band from the TrP attach superiorly directly to the skull.

TrP she said, "I am now getting pain in the right temporal region, but I've not had pain in that area before." In response to pressure on a right temporal TrP she responded, "Now I'm getting pain in the tooth that bothers me when I study."

10. ENTRAPMENT

When a nerve passes between taut bands in a muscle, or when a nerve lies between taut TrP bands and bone, the unrelenting pressure exerted on the nerve can produce neurapraxia (loss of nerve conduction) but only in the region of compression. Table 3.4 lists nerves that can be entrapped by a muscle in this manner. Occasionally, there is EMG evidence of some neurotmesis (axonal loss) in addition to neurapraxia.

The patient with one of these entrapments is likely to present with two kinds of symptoms: aching pain referred from

Table 3.2 ***Comparisons Between Central Trigger Points (TrPs) and Attachment Trigger Points***

	Finding	*Most Likely Cause*
Central TrPs	In motor endplate zone	Dysfunctional endplates
	Nodule	Contraction knots
	Local and referred pain	Nociceptors sensitized by local energy crisis
	Taut band beyond nodule	Contraction Knot Tension
Attachment TrPs	In attachment zone	Taut band tension
	Palpable induration	Inflammatory reaction
	Local and referred pain	Nociceptors sensitized by persistent taut band tension
	Taut band at attachment TrP	Contraction knots in central TrP

Table 3.3 ***Listing of Muscles Observed to Exhibit Corresponding Key Trigger Points and Satellite Trigger Points****

Key Trigger Point	*Satellite Trigger Points*
Sternocleidomastoid	Temporalis*
	Masseter*
	Lateral Pterygoid*
	Digastric
	Orbicularis Oculi*
	Frontalis*
Upper Trapezius	Temporalis*
	Masseter
	Splenius
	Semispinalis Capitis
	Levator Scapulae*
	Rhomboid Minor*
	Occipitalis*
Lower Trapezius	Upper Trapezius
Scaleni	Serratus Posterior Superior*
	Pectoralis Major* and Minor*
	Deltoid
	Extensor Digitorum Communis*
	Extensor Carpi Radialis and Ulnaris
	Long Head, Triceps Brachii*
Infraspinatus	Anterior Deltoid*
	Biceps Brachii
Latissimus Dorsi	Long Head, Triceps Brachii*
	Flexor Carpi Ulnaris

*From Hong CZ. Considerations and recommendations regarding myofascial trigger point injection. J Musculoskel Pain 2(1):29-59, 1994.

TrPs in the involved muscle, and the nerve compression effects of numbness and tingling, hypoesthesia, and sometimes hyperesthesia. Patients with nerve entrapment prefer cold packs on the neurogenically painful region; patients with pain of myofascial origin usually find their symptoms aggravated by chilling the muscle, and relieved by heat on the TrPs.

The signs and symptoms of partial neurapraxia may sometimes be relieved within

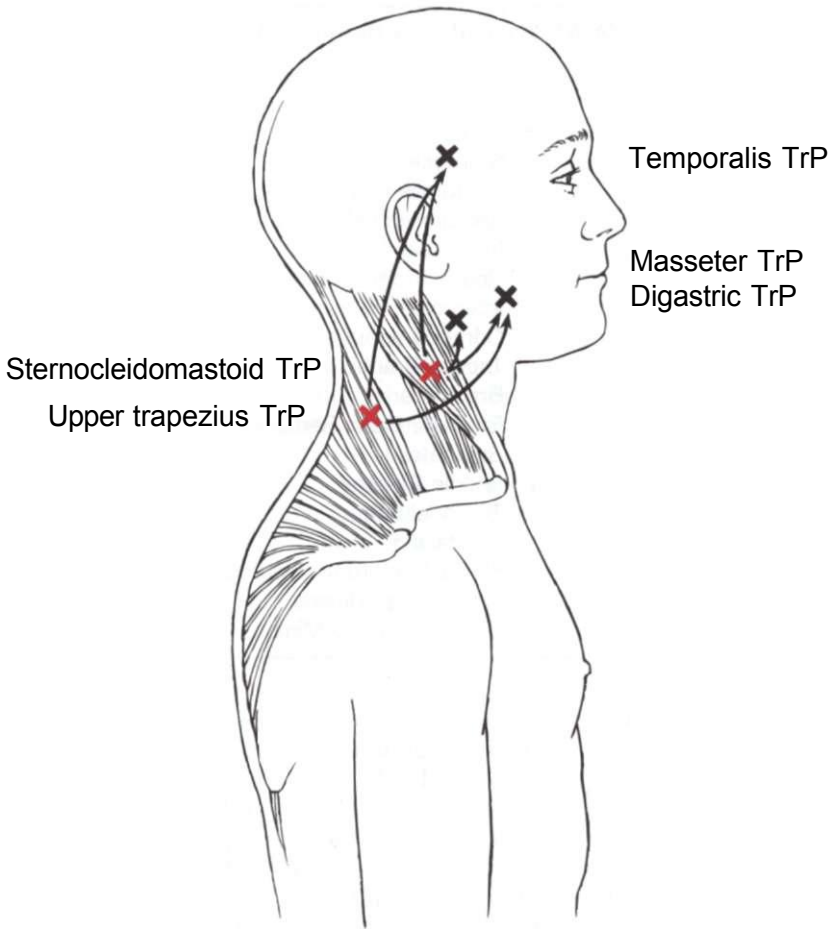


Figure 3.10. Examples of key trigger points (TrPs), shown as *red* **Xs,** and corresponding satellite trigger points *(black* **Xs)** in other muscles. A key TrP in one muscle can induce satellite TrPs in other muscles *(arrows).* This figure illustrates key upper trapezius TrPs initiating satellite TrPs in the temporalis and masseter muscles. It shows a key sternocleidomastoid trigger point initiating satellite trigger points in the temporalis and posterior digastric muscles. (Credit is given to M.J. Tolic, M.D., for suggesting the concept of this figure.)

minutes after inactivation of the responsible myofascial TrPs, which immediately relaxes the taut bands. Effects of more severe compression may require days or weeks for recovery.

11. DIFFERENTIAL DIAGNOSIS

Section 11 was previously named Associated Trigger Points and has been renamed Differential Diagnosis. The associated trigger point information is now covered in a subheading, Related Trigger Points. The material under this subheading identifies the other muscles of the functional unit that are also likely to develop TrPs.

In the first edition, when differential diagnosis was considered as it is here, it was usually included as a subheading under Section 7, Activation of Trigger Points, or distributed throughout the chapter. This section now identifies other diagnoses that are commonly applied to patients when the pain is caused by TrPs. Table 2.5 lists examples of 24 of these conditions. This section also considers the problem of misdiagnosing one of those other conditions as TrPs and not treating it appropriately.

Table 3.4 ***Nerve Entrapments by Myofascial Taut Bands in Muscles That Are Included in This Volume***

Entrapped Nerve	*Muscle*	*Chapter*
Accessory	Sternocleidomastoid	7
Brachial Plexus, lower trunk	Pectoralis Minor	43
Brachial Plexus	Anterior and Middle Scalenes	20
Digital	Interossei, hand	40
Greater Occipital	Semispinalis Capitis	16
Intercostal	Intercostal	45
Musculocutaneous	Coracobrachialis	29
Radial	Triceps Brachii	32
Radial, sensory	Brachialis	31
Radial, superficial sensory	Extensor Carpi Radialis Brevis	34
Radial, deep	Supinator	36
Serratus Anterior, motor nerve	Middle Scalene	20
Spinal, posterior primary rami	Thoracolumbar Paraspinal	48
Supraorbital	Frontalis	14
Ulnar	Flexor Digitorum, deep, superficial	38
Ulnar	Flexor Carpi Ulnaris	38
Ulnar, deep (motor) branch	Opponens Digiti Minimi	40

12. TRIGGER POINT RELEASE

With Contributions by
Mary L. Maloney, P.T.

The title of this section has changed from "Stretch and Spray" to "Trigger Point Release." The change reflects the shift in emphasis from only one manual therapeutic technique to an appreciation of the wide variety of effective techniques available and an indication of the clinical usefulness of each. This section presents a rationale for each technique that is based on the new understanding of the nature of TrPs.[147,148] It calls attention to how various treatment techniques that are commonly used today relate to myofascial TrPs.

There are many techniques published and practiced to relieve the tissue tensions associated with musculoskeletal pain. Each professional group dealing with this problem has its own terminology and favorite methods. Rarely do these practitioners identify what is causing the pain or present a convincing explanation of why their procedure relieves it. Some practitioners do explain why they think the tissues are tense, but these explanations are rarely applicable to the muscle itself. Examined in terms of muscle tension caused by TrPs, many of these procedures are appropriate for treating TrPs, although the authors rarely recognize the possibility (likelihood) that TrPs are a significant part of the picture.

This section is organized in terms of the kinds of procedures used to relieve the TrP tension causing the pain and considers why each approach is likely to contribute to relief. It considers passive muscle stretch techniques, techniques that involve voluntary contraction, manipulation of the TrP, and modalities. In addition, this section identifies helpful accessory techniques, notes methods used simply to relieve the pain rather than eliminate its cause, and lists a number of caveats.

Intensive examination for perpetuating factors is called for when the patient has chronic myofascial pain, or if the severity of involvement is progressive.

When deciding which of the many available treatment methods to use, it is important to consider whether the TrPs being treated are *central* TrPs in the endplate zone of the muscle or are *attachmentTrPs* located where the muscle attaches to its aponeurosis, tendon, or a bone. Stretching (lengthen-

ing) the muscle inactivates central TrPs, but may tend to aggravate the overloaded muscle attachments. The attachment TrPs are more likely to respond to manual therapy that is directed to the regions where central TrPs are located and therapy that concentrates on relieving the strain on the attachments caused by the TrP-induced shortening of the taut band fibers.

Generally, central TrPs become less irritable in response to warmth. However, sometimes patients find relief by application of cold. Attachment TrPs may be more responsive to cold than to heat, especially when they are very irritable. Since the attachment TrPs are the result of the tension from the taut bands of the central TrPs, inactivation of the central TrP is essential; on the other hand, reducing the sensitivity of the attachment TrPs may greatly facilitate inactivation of their central TrPs. The optimal therapeutic intervention for central TrPs compared to that for attachment TrPs is an issue that needs competent experimental investigation.

Recovery of full function may involve more than just TrP inactivation and relief of pain, especially in patients who have chronic pain. If the muscle has learned dysfunction that restricts both its strength and coordination during functional activities, it must be retrained to normal function. This requires adequate monitoring of the rate of muscle fatigue and loss of strength during exercise and functional activity. Surface EMG can be a valuable quantitative tool for measuring fatigue and in addition can provide biofeedback for retraining.

Spray and Stretch

In 1952, Hans Kraus[123] described how he discovered that spraying ethyl chloride on the skin relieves musculoskeletal pain. Kraus was looking for a substitute for alcohol-soaked towels exposed to live steam that were then used in Germany by wrestlers as a treatment for painful sprains.[123] Kraus[93] recommended ethyl chloride spray for initial treatment, and then depended heavily on active range of motion and exercise for eventual recovery. Dr. Travell became aware of his freezing spray technique through his 1941 paper on "surface anesthesia."[92] Her first use of it was on a young girl who had sprained her middle finger knuckle. As the spray was momentarily applied over the joint, the girl was startled and jerked her hand away. Then, mystified, the girl said "That feels better; put some right here." A second brief pass of vapocoolant over the other side of the joint completely relieved her pain and restored the full range of motion.[169] Refrigeration *anesthesia* with frosting of the skin was *not* an essential mechanism.

Rinzler and Travell,[138] and then Travell,[162, 169] succeeded in relieving pain due to acute coronary thrombosis by applying the spray to the skin over the area of pain referred from the heart. This effectiveness of vapocoolant spray in relieving the pain of myocardial ischemia was demonstrated experimentally.[137] The spray did nothing to relieve the ischemia; it relieved only the pain. It inhibited the perception of referred visceral pain.

In our experience, spray and stretch is the single most effective noninvasive method to inactivate acute TrPs. However, many other noninvasive techniques require no supplies and are better suited to use by the patient at home. When the simpler approaches fail to give satisfactory results, many times the addition of spray and stretch (often in combination with other techniques) brings success.

A single-muscle syndrome of recent onset frequently responds with full return of pain-free function when two or three sweeps of spray are applied while the muscle is being extended gently to its full stretch length.[160] In addition, when many muscles in one region of the body, such as the shoulder, are involved and the TrPs are interacting strongly with one another, spray and stretch is a practical means of releasing an entire functional group of muscles together to make more rapid progress toward pain relief. The spray-and-stretch technique does not require the precise localization of the TrP that is needed for injection; it requires only identification of where the taut bands are located in the muscle to ensure that those fibers are released.

The essential therapeutic component is the stretch. "Stretch is the action, spray is distraction." However, the expression "spray and stretch" is preferred to "stretch and spray" because it is important that the spray be applied before or concurrently with, but not after, the muscle is stretched. Stretch

without some additional technique to release muscle tension and suppress pain is likely to aggravate TrPs, especially attachment TrPs.

Myofascial TrPs in the muscles of young children and babies are especially responsive to spray-and-stretch therapy.[10] In this age group, many of the other techniques which require more patient participation are not applicable. Most children have been well trained to be needle-shy.

Spray and stretch is especially useful immediately after TrP injection during the period that the local anesthesia remains. This combination procedure helps to inactivate any residual TrP activity and to attain full stretch range of motion.

Much of the shoulder pain in patients with hemiplegia arises in TrPs caused by the overload of spasticity and strain on the remaining functional musculature. During the first few weeks following a stroke, much temporary relief can be obtained by spray and stretch of both agonists and antagonists in the shoulder region, applied twice daily. Liberson[104] described wheeling a drum of vapocoolant equipped with a hose and spray nozzle through the Physical Medicine and Rehabilitation Ward twice daily to spray and stretch the patients with hemiplegia in order to reduce their pain and increase their function more rapidly during the early weeks of recovery. After 4-8 weeks, as the degree of paralysis and spasticity stabilizes, the relief of TrP pain becomes more lasting. Such relief of pain encourages the patient to strive for function, and influences the results of rehabilitation by improving the patient's efforts to use marginally functional muscles.[35]

Immediately following major trauma such as fracture, dislocation, or whiplash injury, cold packs should be applied to the muscles to reduce tissue swelling. Spray and stretch, with heat, should be deferred until 3-5 days later as the local reaction to trauma subsides. However, the anti-inflammatory effect of the vapocoolant spray alone, when applied at once, is remarkably helpful for relieving the pain of sprains and burns.

Patients who have myofascial pain and hyperuricemia may not respond well to spray and stretch because pain recurs quickly. The response is better to injection of TrPs. This may be explained by the deposition of uric acid crystals in an acid environment at the TrP.

The effectiveness of the spray for helping the stretch to release central TrPs (when attachment TrPs are also present) may depend on the vapocoolant spray's suppression of pain from the attachment TrPs, which would otherwise be intolerant of any additional tension. This effect of the spray would be comparable to its potent analgesic effect on burns, sprains, cardiac ischemia, and referred visceral pain. See Chapter 2, Part B for more on this mechanism.

Vapocoolants. To be effective for releasing TrP tension in order to stretch the muscle, the vapocoolant must be dispensed as a *fine stream,* not as the dispersed spray which is used for spraying paint or hair. Two sprays are currently commercially available: Fluori-Methane® and ethyl chloride. Both are sterile as dispensed and can be sprayed on a sterile field without contaminating it.[1] However, neither is considered to be an antiseptic nor will they kill germs.

Both volatile liquids exert pressure in a closed container at room temperature. The pressure forces a stream of the room-temperature liquid out of the inverted bottle upon opening of the control valve. The warmer is the container, the higher is the pressure. Upon leaving the nozzle, the stream of liquid immediately begins to evaporate, which cools the stream as it passes through the air to the skin. For a distance of approximately half a meter (about 18 inches) the stream continues to get colder until it impacts the skin, where it continues to evaporate and further cool the skin. At short distances from the skin, the stream has less time to cool and so impacts the skin at a temperature nearer to room temperature. When held far enough from the skin and directed at one spot, the stream of either spray can produce subfreezing temperatures; this is to be avoided.

Because ethyl chloride is a potentially serious health hazard and is colder than desirable for TrP applications, Travell[169] assisted in the development of a safe alternative, Fluori-Methane, which is a mixture of two fluorocarbons: 85% trichloromonofluoromethane and 15% dichlorodifluoromethane. Fluori-Methane is nonflammable, chemically stable, non-toxic, non-explosive and does not irritate the skin.

Unfortunately these fluorocarbons cause serious degradation of the upper atmosphere ozone layer and are no longer manufactured or approved for commercial purposes. A temporary medical exception has been granted for Fluori-Methane while a suitable substitute is being developed. A promising substitute is undergoing testing and when approved will be marketed as "Gebauer Spray and Stretch" by the same company that sells Fluori-Methane. The new product will be dispensed from a can with a different valve mechanism rather than from the familiar glass bottle. Most of the illustrations in this edition show the operator using the new product.

Ethyl chloride is too cold for optimum release of TrP tension as usually applied. It is a rapidly acting general anesthetic that has a dangerously low margin of safety, is flammable and is explosive when 4-15% of the vapor is mixed with air.[123] It has been responsible for accidental anesthetic deaths of patients and a physician.[169] If ethyl chloride spray is used, rigorous precautions must be observed. Fire hazards must be eliminated, and neither the patient nor the clinician should inhale the heavy vapor.[160,170] Ethyl chloride should never be given to a patient for home use.

Urticaria owing to cold allergy has not been observed in response to spraying with Fluori- Methane for myofascial therapy, and was observed only once with ethyl chloride.[170] There is no evidence that inhalation of Fluori-Methane in doses and concentrations to which patients are exposed during treatment for TrPs is toxic. It has an odor that is unpleasant to some patients, and unnecessary exposure should be avoided.

Most of the articles by Travell describing the use of spray refer to ethyl chloride because they were written before Fluori-Methane became available. She cautioned readers to substitute Fluori-Methane for ethyl chloride when they refer to those articles.

Spray Technique. Detailed descriptions of the vapocoolant spray technique have been published.[51,119,160,164,170,193] Reproduction of the referred pain by pressure on a TrP helps the patient to more fully understand why treatment is directed primarily to the tender region in the muscle and not primarily to the region of pain complaint.

PATIENT PREPARATION. Adequate body warmth is critical for a favorable muscular response to treatment. If, on arrival, the patient feels chilly or the hands and the feet are cold, a dry heating pad can be applied to the abdomen to raise the core temperature and cause reflex vasodilatation in the limbs. This is important in cold climates, chilly rooms, and whenever a patient feels cool. A blanket should cover the portion of the patient not exposed for treatment.

A simple and often effective alternative to the application of heat is neutral warmth that is obtained by covering the patients with a wool scarf, sweater, or small blanket to keep them warm by conserving their own body heat. However, the muscle relaxation gained by the warmth can be lost due to chilling when the source of warmth is suddenly removed.

Hypoglycemia aggravates TrPs. Before application of a specific myofascial therapy like spray and stretch, the patient should be asked if he or she has eaten recently to avoid potential hypoglycemia. For patients with a suspicious history, a banana, glass of milk, cheese, flavored "drinking" gelatin in orange juice, or a cup of instant soup may prevent a painfully adverse reaction to therapy soon afterward.

The portions of the skin to be sprayed should be bare. Spray penetrates the hair unless it is heavily greased or thickly matted, but wigs and toupees must be removed.

Patients should be given an initial reference with which to judge improvement in their range of motion following treatment, so that they can be aware of progress following treatment. To the patient, a movement feels as if it "goes as far as it can go" both at a restricted range and at the full range of motion. During initial testing, the patients learn the extent of their movement by answering specific questions. "How wide does your mouth open; two or three knuckles?" "How far can you see around behind you?" "Can your fingertips reach around the back of your head and cover your mouth?" or "Can you reach your back pants pocket?" A mirror helps patients to see and remember what they were able to do. The measurement should be retested following treatment so that the patient can fully appreciate the difference. Since we are as concerned with function as with

pain, it is important that patients fully appreciate the improvement in their function.

The involved muscle cannot be effectively stretched if it is not fully relaxed, and full relaxation needs a comfortable, warm, well-supported patient. All the limbs must be positioned comfortably when the patient is in the recumbent position. In the seated position, the patient's pelvis and shoulder-girdle axis must be leveled by adding an ischial lift to compensate for any discrepancy in the size of the two halves of the pelvis. The patient is given a lumbar pad to correct a stooped posture *(see* Chapter 41, Section C). If the operator is extending the patient's head, the patient is asked to lean the head back against the operator, who supports it so that the neck muscles can fully relax.

If the patient is tensely holding the breath, the practitioner can make a remark like, "Don't forget to breathe," to remind the patient to release the tension. As demonstrated by Basmajian,[9] relaxation is not a passive process, but an active one that requires learning how to consciously turn off motor unit activity.

For many patients, the trick is to divert attention from themselves and to concentrate on the *support.* They must feel the armrests of the chair supporting their forearms, or think about the support of the mattress on which they are lying. For those who find this difficult, deep breathing with the *diaphragm* is encouraged; then spray and stretch is applied to the muscle as the patient slowly exhales. For most patients, normal coordinated diaphragmatic (abdominal) breathing is much more relaxing than paradoxical chest breathing.

To effectively stretch a muscle, one end of it must be anchored so that the operator can exert tension on it toward the other end. Frequently, the patient's body weight or gravity can be used as the anchor. Sometimes, the patient can fix one end of the muscle by sitting on the hand when a scalene or upper trapezius muscle is being stretched.

SPRAY PROCEDURE. Figure 3.11 summarizes the sequence of steps in the spray-and-stretch technique, as applied to the trapezius muscle. First, the patient must be positioned *comfortably* and well supported to permit voluntary relaxation. One end of the muscle should be *anchored* so that movement of the head will take up slack in the muscle as it develops. Initial sweeps of the jet stream of spray are applied over the *trapezius muscle* and continued over the *complete pain pattern* to begin releasing muscle tension before taking up the slack to lengthen the muscle toward its stretch position. The spray (or ice) is applied in parallel

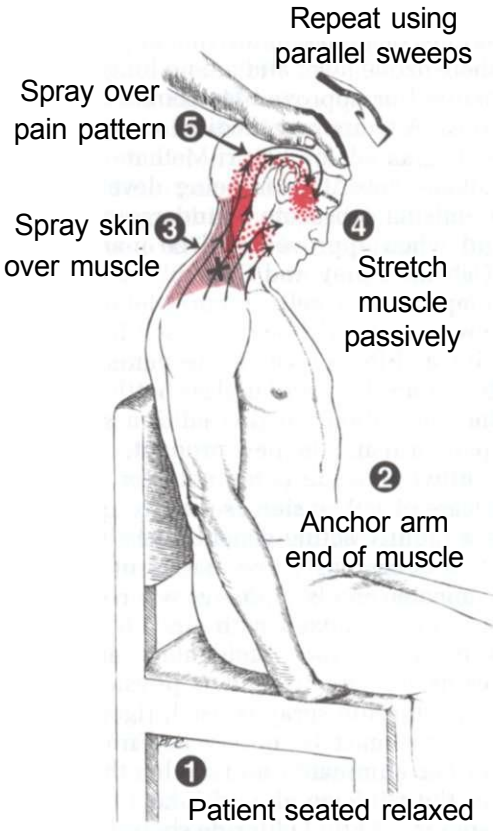


Figure 3.11. Sequence of steps to use when stretching and spraying any muscle for myofascial trigger points, as applied to this partial stretch of the upper trapezius muscle. 7, patient supported in a comfortable *relaxed* position. 2, one end of the muscle *(light red)* anchored. *3,* skin sprayed with repeated parallel sweeps of the vapocoolant over the length of the muscle in the direction of pain pattern *(dark red dots).* All of the muscle belly and its attachments are included. *4,* after the first sweep of spray, pressure is applied to take up the slack in the muscle and is continued as additional sweeps of spray are applied. 5, sweeps of the spray are extended to cover the referred pain pattern of that muscle. 6, steps 3, 4 and 5 may be repeated 2 or 3 times until the skin becomes cold to the touch or when the range of motion reaches maximum. Application of heat and then several cycles of full *active* range of motion follow. See Figure 3.12 for details of the spray technique.

sweeps only in the direction of the referred pain. This spray procedure can be *repeated* until full muscle length is achieved, or no further progress occurs. However, any given area of skin should be covered only two or three times before rewarming. After the skin has rewarmed, several cycles *oifull active range of motion* complete one spray-and-stretch treatment of that muscle.

The valve that seals the nozzle of the Fluori-Methane bottle permits only an on-off application with no intermediate control. Partial opening of the valve results in dripping of vapocoolant from the nozzle and deflection of the stream. The Fluori-Methane bottle must be held inverted so that the liquid will flow from the nozzle. When it is held upright, only vapor emerges. The replacement product that is being developed will come in cans that are held upright and operated much like a hair spray or paint can, and its valve probably will not provide proportional control either.

The closer the bottle is held to the skin, the warmer is the stream of vapocoolant on impact. One can demonstrate this easily on oneself by how cold the stream feels when the bottle is held at various distances from the skin. Notice the sharp pain produced at the site of frosting when one spot is sprayed too long (about 6 seconds) and causes freezing of the skin. *This should be avoided.*[186] An instant of frosting is painful but innocuous. Prolonged frosting can cause a blister and ulceration.

When the spray is initially applied over very irritable TrPs, the skin may be unbearably hypersensitive to the cold. This initial distress can be mitigated by using a bottle saved for its fine-bore nozzle, by holding a bottle (can) close to the skin, and by wafting the jet stream across the skin rapidly.

Many patients who are receiving spray therapy for the first time are severely startled by the cold spray if they are not warned what to expect. The effect of the spray should be demonstrated to them first on the operator's hand, and then on the patient's hand before starting treatment. The jet stream of vapocoolant is most effective when directed at an acute angle to the skin (approximately 30%), not perpendicularly, and when applied in parallel sweeps along the direction of the muscle fibers. The spray sweeps are applied in one direction only, covering first the full length of the muscle and then covering the complete pain reference zone. It is important to include coverage of the attachments at both ends of the muscle as well as the muscle belly.

The bottle is held about 30 cm (12 in) from the skin (Fig. 3.12). Slow, even sweeps that progress over the skin at about 10 cm (4 in)/sec are spaced to provide a slight overlap of the tracks of wet spray. Two or three superimposed sweeps are usually maximum; the skin must then be rewarmed. Six sweeps over the same skin area, without rewarming, are too many because that many sweeps can cool the underlying muscle (Fig. 3.13). It is best to spray a slightly larger area than just the referred pain pattern. No therapeutic harm is done with some additional coverage and it may help to release TrP tension in adjacent muscles.

If the spray is too cold for the patient, moving the line of spray more quickly across the skin will help. If the spray is still too cold, the bottle or can should be held closer to the skin than the usual 12 inches. If a colder spray than usual is desired, the spray distance can be increased to 18 inches.

The patient should tell the operator if it feels as if the spray should be directed over a line of muscle fibers that are being missed. Many times the patient can clearly feel the line of muscle tension that needs to be released and can describe or point to just where the spray needs to be directed to relieve the tension. Vapocooling such an overlooked region usually further releases muscle tension and provides increased range of motion. It is remarkable how precisely the skin (that the patient wants to be sprayed) overlies the abnormally tense muscle fibers. It also is remarkable how the muscle tension sometimes melts away as the stream of spray reaches the most distant portion of the referred pain pattern.

When vapocoolant is applied to the face, the eye on that side should be covered. If Fluori-Methane spray accidentally hits the conjunctiva or the eardrum it is startling and painful, but not damaging. Patients with *asthma* and other respiratory conditions may not tolerate vapocoolant spray near the face unless the practitioner covers the patient's nose with a small cloth or a hand. Ice stroking *(see* below) may *replace* the spray for these patients.

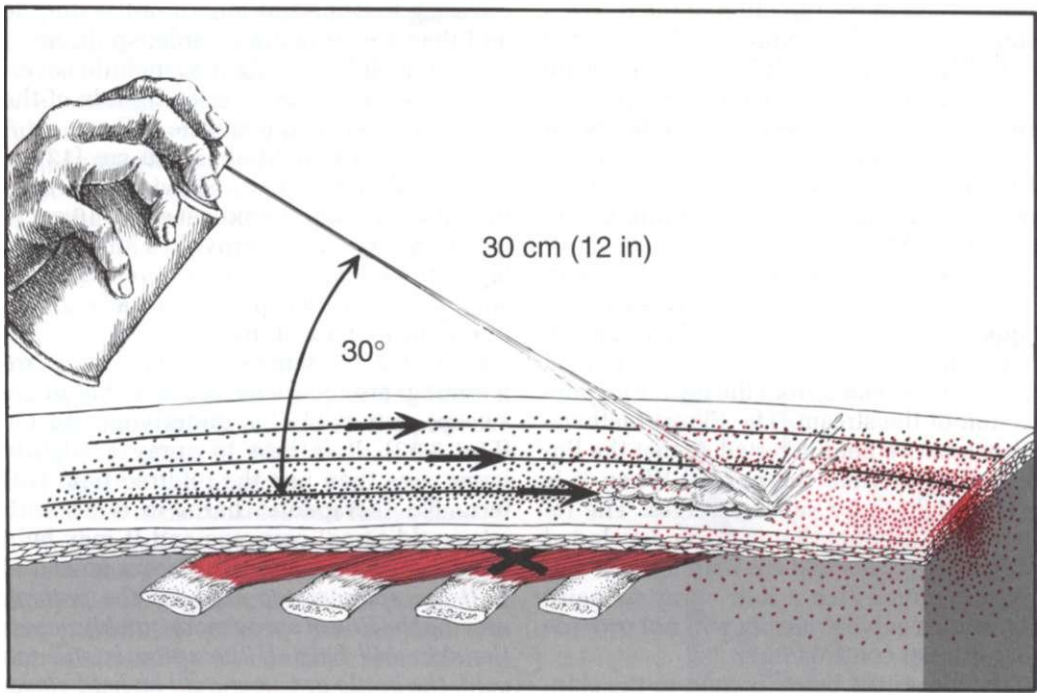


Figure 3.12. Schematic drawing showing how the jet stream of vapocoolant is applied. Unidirectional sweeps cover, first, parallel lines of skin over those muscle fibers that are stretched the tightest, then over the rest of the muscle and its pain pattern. Sequential sweeps of spray *(thick black arrows)* should follow the direction of the muscle fibers, and progress toward the referred pain zone *(red stippling)*. The spray container is held at an acute angle usually 30 cm (12 in) from the skin, as the spray sweeps over the skin at a rate of about 10 cm (4 in)/sec. Held closer, spray is warmer. Farther away, it is colder.

Vapocoolant spray also can be applied advantageously as a **pre-spray** preliminary to other manual techniques such as manual release, myofascial release, a muscle energy technique, or whenever both hands of the practitioner are required for a manual release technique.

Self-spray by the patient with Fluori-Methane can be useful during the transition period, while the perpetuating factors are still being identified and resolved, and by patients who seem unavoidably prone to reactivation of TrPs and therefore need to be able to quickly inactivate TrPs for themselves. Patients generally learn quickly to self-spray their masticatory and calf muscles. However, it requires unusually skillful selective relaxation to effectively spray and stretch by oneself the shoulder-girdle, arm and neck muscles. Fortunately, there are effective alternate techniques for self treatment if patient self-spray is not practical or convenient.

STROKING WITH ICE. The sensory and reflex effects of a jet stream of vapocoolant spray (such as Fluori-Methane) can be obtained also to a considerable degree by stroking with ice. Water frozen in a plastic or paper cup is a convenient form for applying the ice. A stirring stick inserted in the cup before freezing the water provides a convenient handle to hold the ice, or a well-insulated plastic foam cup can be used. The ice is exposed by tearing back part of the cup, and an *edge* of the ice is applied to the skin in unidirectional parallel strokes, following the spray patterns presented in each muscle chapter. The stroking movements progress slowly, at the same rate as the spray (10 cm (4 in)/sec). This application of the sharp edge of ice simulates the jet stream of vapocoolant spray. The practitioner should hold a small cloth ready to blot the skin as needed to prevent melting ice from wetting the skin.

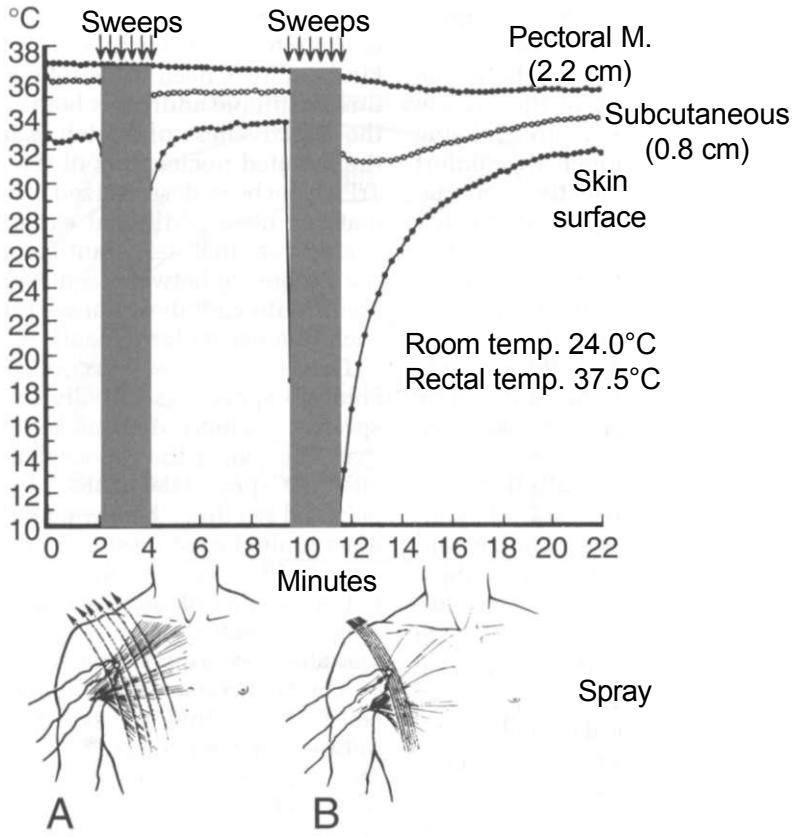


Figure 3.13. Skin surface, subcutaneous and muscle temperature effects produced by the correct **(A)** and incorrect **(B)** use of vapocoolant spray. Temperatures were recorded by a tier of three thermal sensors in needles from: (1) pectoral muscle *(upper line)* at a depth of 2.2 cm, (2) subcutaneous tissue *(middle curve)* at a depth of 0.8 cm, and (3) skin surface (bottom *curve).* The jet of vapocoolant was applied in one direction in six even sweeps at 10 cm/sec for a total of 2 min *(shading).* **A,** *(bottom left)* sweeps covered adjacent parallel skin areas, and only one sweep traversed the tier of sensors. **B,** *(bottom right)* all six sweeps traversed the same path directly over the sensors. When a given skin area was covered only once **(A),** the fall in muscle temperature was negligible, 0.2°C; when the skin and tier of sensors were covered by six superimposed sweeps **(B),** the muscle temperature dropped more, 1.5°C, starting during the application and continuing to drop as the cold penetrated to deeper tissues. This shows the importance of spacing sweeps of spray, of not superimposing them, and of not covering the same area of skin too many times too quickly.

The skin must remain dry, because dampness reduces the rate of the change in skin temperature produced by the ice-stroking. Wetness also prolongs and diffuses the cooling effect, which delays rewarming of the skin. The ice block can be covered with thin plastic as long as the stroking edge of the ice is thin and cold. The clinician should avoid cooling the underlying muscle when stroking with ice, just as when applying vapocoolant spray.

RATIONALE FOR VAPOCOOLING AND ICING. Properly applied, the vapocoolant or ice stroking causes a sudden drop in skin temperature and has a physical impact that produces additional tactile stimulation. The continuous motion of the stream of spray causes a continuing barrage of alarming impulses to the spinal cord. This input has an inhibitory effect on locally generated pain as demonstrated by its effective analgesic effect in sprained ankles, burns,

and ischemic contraction of forearm muscles and as indicated schematically in Figure 3.14. This neural mechanism helps the patient maintain relaxation of the muscle when otherwise the degree of stretch being achieved might cause enough discomfort to initiate involuntary protective contraction of the muscle to prevent further lengthening. As described in Chapter 2 Part C, the autonomic nervous system can significantly influence the intensity of activity of the TrP mechanism at the motor endplate. The spray effect on the skin apparently also can inhibit this autonomic activation at the spinal cord level as illustrated in Figure 3.14.

There are substitute stimuli that also can produce these effects of spray. They include the application of sweeps of ice, and the serial prickling produced by running a neurologist's pin wheel along lines that follow the spray pattern.

> **The close reflex relationship between skin sensation and function of the underlying muscle was demonstrated by studies of human withdrawal reflexes to noxious skin stimuli on both the trunk and lower extremity. Electromyographic recordings of a gentle, sustained voluntary contraction of multiple muscles throughout the region measured increases and decreases in the EMG activity induced by shock stimuli to the skin. Activity of muscles beneath the site of skin stimulation was facilitated, and that of other muscles was generally inhibited.[63 95] Early and late responses were identified. Early responses were likely to cause movement that terminated weight bearing of that limb, and to remain constant, regardless of changes in the location of the stimulus. Late responses were modifiable, and after several trials, they were adapted to move the limb away from the stimulus.[63] Skin reflexes of the back and abdomen induced movement away from the stimulus.[95]**

The direction of spraying, across the muscle and then over the pain pattern, was initially determined by subjective testing on patients by Dr. Travell. She observed the direction of spraying that the patients preferred and that gave the maximum relief of tension and pain. The spray may be especially effective in quieting attachment TrPs and the stretch may be specific for the release of central TrPs. The value of spray and stretch may lie in the fact that both kinds of TrPs need to be relieved and that this technique addresses both. Apparently, the effectiveness of stretch is improved if the irritated nociceptors of the attachment TrPs have been desensitized. These considerations need additional experimental investigation that takes into consideration the difference between central and attachment TrPs and the relative sensitivity of each in a particular patient.

OTHER USES FOR VAPOCOOLANTS. Ethyl chloride spray was initially used for joint sprains;[123] Fluori-Methane is equally effective. The sooner the vapocoolant is applied after the sprain, the more fully it relieves pain and swelling, thereby permitting immediate limited use to restore normal function quickly. Vigorous stretching must be avoided in the presence of torn tissues, but the joint's range of motion should be progressively reestablished as quickly as recovery permits.

The vapocoolant spray is remarkably effective for chilling the skin to numb it for painless **TrP** injections,[186] and for relieving the pain and preventing the blistering of thermal burns. It reduces secondary hyperalgesia, erythema, and swelling as demonstrated in experimental studies.[163 173] Burns of second-degree severity that were sprayed repeatedly (as necessary to keep them pain-free) did not blister, compared with untreated control burns that did blister. The spray is applied to the painful area as soon as possible after the burn (preferably within 5 or 10 seconds) until it stops hurting. It is helpful to keep a bottle in the kitchen by the stove. The spray is reapplied immediately as soon as pain recurs. The number of repetitions required depends on the severity of the burn. On minor first-degree burns, one application may be sufficient to immediately and completely eliminate pain.

Vapocoolant spray applied to the painful regions in acute myocardial infarction can be remarkably effective in relieving the pain without changing the course of the cardiac pathology.[137 162] A few applications can sometimes fully replace morphine or comparable analgesics.

Vapocoolant spray relieved or delayed pain during experimental ischemic contraction of forearm muscles.[174] The same mechanism may apply to relief of attach-

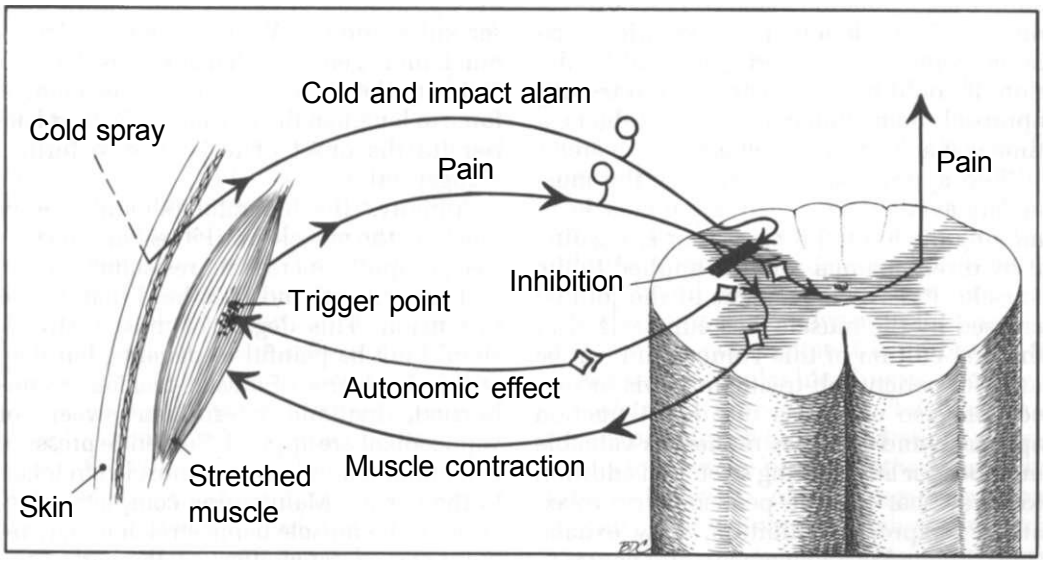


Figure 3.14. Schematic representation of likely neural pathways that could account for the effectiveness of vapocoolant applied to skin overlying an active central myofascial trigger point *(dark red).* The trigger point limits range of motion of the muscle, and an attempt to lengthen the muscle beyond its limited range of comfort causes pain. This pain of stretching can induce involuntary muscle contraction to return the muscle to a comfortable length and can increase sympathetic activity that stimulates the trigger point mechanism. Thus the pain prevents further elongation of the muscle. The sudden cold and the tactile stimulus of the stream of vapocoolant spray inhibit the pain and the reflex motor and autonomic responses in the central nervous system *(black bar).* This pain-suppressing effect now permits more effective relaxation and gentle lengthening of the muscle. In addition, spray over muscle attachment trigger points appears to reduce their sensory irritability.

ment TrP tenderness. The spray also relieves the pain of **bee stings,**[123] and is reported as helpful in controlling the pain of **postherpetic neuralgia.**[157]

Some **veterinarians** and animal trainers use vapocoolant spray to relieve myofascial TrPs, including spot tenderness, in the muscles of horses[123] and dogs.[83,84] Because some animals can react so vigorously to the cold spray, some veterinarians simply reassure the animal in order to use manual TrP pressure release and stretch techniques to inactivate the TrPs. Drs. Travell and Simons have found spray and stretch of TrPs in dogs and cats very effective if intolerable coldness of the spray is controlled by application technique, and if the animal is properly reassured. A veterinarian, Dr. Frank[48] is completing a doctoral thesis that demonstrates the effectiveness of TrP pressure release techniques for inactivating myofascial TrPs and restoring full function in seriously afflicted dogs.

Stretch (Lengthening) Technique. Almost any method that gently stretches (lengthens) a muscle with TrPs and increases its pain-free range of motion is beneficial. In a controlled experiment,[81] application of the spray-and- stretch technique reduced the intensity of referred pain and reduced the sensitivity of the TrPs being treated. However, a rapid, forceful stretch by itself causes pain, protective contraction, and reflex spasm of the muscle, all of which hurt the patient and obstruct further elongation of the muscle. Some method of suppressing these reactions must be added in order to release TrP tension. Rapid stretch and a "bouncing" stretch are to be avoided; they tend to irritate TrPs, not release them. It is often possible, with a newly activated or a moderately irritable TrP, to inactivate it immediately by simply passively, slowly stretching the muscle without spray. However, the release without spray can be expedited and made less uncomfortable when stretch is combined with

simple augmentation maneuvers such as coordinated exhalation, postisometric relaxation, contract-relax, and reciprocal inhibition. It could be very instructive to try this approach **immediately** on yourself the next time you activate a TrP, wherever you are.

Two approaches to stretching the muscle are available: elongating the muscle by moving the joint(s) it crosses or elongating it by direct manual traction applied to the muscle. Passive movement of the joint(s) crossed by the muscle was emphasized in the first edition of this volume and can be used for patient self-treatment. This second edition also embraces the direct traction approach and includes numerous valuable methods for augmenting stretch in addition to spray that include postisometric relaxation, reciprocal inhibition, slow exhalation, directed eye movement, and contract-relax. These various techniques can be used in many different combinations and integrated with augmentation techniques.

The remarkable effectiveness of almost any technique that elongates the muscle and restores it to full stretch length can be explained by the integrated hypothesis (see Chapter 2 Part D). The contracture of the sarcomeres in the contraction knots of a TrP must be released in some way. Lengthening the contractured sarcomeres of the contraction knots by gentle sustained stretch with augmentation techniques apparently induces gradual reduction in the overlap between actin and myosin molecules and reduces the energy being consumed. When the sarcomeres reach full stretch length, there is minimal overlap and greatly reduced energy consumption. This breaks an essential link in the energy crisis vicious cycle. The *sustained* increased tension on contractured sarcomeres may cause tearing of the actin attachments to the Z lines as observed ultramicroscopically by Fassbender.[44] This tearing, when complete, could produce the segments of empty sarcolemma observed by light microscopy among muscle fibers in muscle biopsies selected at TrP sites in dogs[152] and at TrP sites in patients with myogelosis.[136]

STRETCH OF SPRAY AND STRETCH. The **key** to treating TrPs is to **lengthen** the muscle fibers that are shortened by the TrP mechanism. Following publication of the first edition of this volume, a rather forceful passive stretch was sometimes associated with the application of vapocoolant spray. This second edition of Volume 1 emphasizes a much more gentle stretch and uses the term "take up the slack" to identify just enough force to lengthen the muscle to the next barrier (to the onset of resistance to further elongation).

Initially, the operator should gently lengthen the muscle until it reaches the barrier (a rapidly increasing resistance to further movement) and then hold that degree of tension. This degree of muscle stretch should not be painful to patients, but they will feel a degree of muscle tension. As unhurried, rhythmic, intermittent sweeps of vapocoolant are applied, the gentle pressure is maintained to keep the muscle stretched to the barrier. Maintaining complete relaxation of the muscle being stretched may require concentrated effort on the part of the patient, careful monitoring by the operator, and verbal reinforcement as necessary. As the muscle "gives up" and releases its tension, the operator *smoothly* takes up the slack to reestablish a new stretch position that again engages the barrier.

The effectiveness of this spray-and-release technique is often increased by alternating the application of spray with postisometric relaxation that is augmented by coordinated cycles of respiration. Following each period of contraction and relaxation, the muscle is repositioned to engage the new restrictive barrier.

Jerking the muscle or sudden loading by the patient must be avoided during and after stretch. Reaching the full normal length of the muscle is essential for complete inactivation of its TrPs and relief of their referred pain and achieving the final few degrees of stretch may be critical.

A skill that for some operators is difficult to learn is "tuning in" to the tenseness of the patient's muscles and accurately identifying the barrier, which occurs at a very specific position. Some muscles, especially large strong ones, may need a little "coaxing" at the barrier to get them to release, but excessive force hurts the patient, results in post-treatment soreness, and increases the irritability of attachment TrPs. The operator must ask patients to speak up *immediately* if the procedure becomes painful and warn them not to be stoical. The concept of "no pain-no gain" does NOT apply here.

After completing full stretch, the return to resting length must be smooth and gradual, and the patient must not overload a muscle by suddenly lifting the weight of that part of the body with it.

If the muscle seems "stuck" short of full range of motion, instead of repeating exactly the same spray-and-stretch procedure, a number of modifications and alternate techniques may be tried by (1) sweeping the spray over functionally parallel or neighboring muscles that also may be shortened by latent TrPs and could be "hanging up" the muscle being stretched; (2) by asking the patient to help *gently* to stretch the muscle by contracting its antagonists thereby reaping the additional rewards of reciprocal inhibition (however, if the patient tries too hard and cocontracts the involved muscle, it can defeat this purpose); (3) by trying several cycles of postisometric relaxation with directed eye movements and coordinated exhalation, emphasizing coordinated "abdominal" (diaphragmatic) breathing; (4) by applying TrP pressure release; (5) or by having the patient perform several cycles of the full active range of motion for that muscle and then resume spray and stretch.

DIRECT STRETCH RELEASE. Muscle stretch by direct application of manual effort is identified in this volume as Stretch Release. Two hands are used to stretch the muscle by placing the hands near the attachments of the muscle and gently separating them until a tissue barrier is encountered. This tension elongates the *muscle and the associated connective tissues.* This stretch release is preceded by pre-spraying with vapocoolant or stroking with ice to help release the muscle tension.

Authors who write about **Myofascial Release** describe a similar technique but rarely associate its effectiveness with the inactivation of TrPs, and they do not apply intermittent cold. They use more generalized terminology and emphasize release of the fascial tissues rather than release of muscle tightness. Release of both tissues is important.

PERCUSSION AND STRETCH. This technique begins by passively lengthening the muscle just to the onset of resistance. The clinician or patient uses a hard rubber mallet or reflex hammer to hit the TrP at precisely the same place about 10 times. This must be done at a slow rate of no more than one impact per second and at least one impact every 5 seconds; the slower rates within this range are likely to be more effective. This procedure may enhance or substitute for intermittent cold with stretch. Dr. Travell considered it particularly applicable to the quadratus lumbomm (self-applied), brachioradialis, long extensors of the fingers, and to the peroneus longus and brevis muscles. It should *not* be applied to anterior or posterior compartment leg muscles because of a possible compartment syndrome if it caused intramuscular bleeding or swelling.

Poststretch Procedures. The most important poststretch (or postinjection) procedure is to have the patient *actively* perform three FULL cycles of the range of motion that fully lengthens and fully shortens every muscle that was treated. This movement helps to normalize muscle function at the sarcomere level and to reestablish normal coordination with other muscles of its functional unit. It encourages the patient to use that muscle throughout its full normal range in ordinary daily activities. At this time, the practitioner should teach the patient how to perform the same movement(s) at home.

A program of **home stretch exercises** is important, particularly when postural trunk and lower limb muscles are involved. The patient benefits by soaking in a warm bath at home as soon as possible after the spray-and-stretch treatment. The patient should avoid strenuous activity and therefore should NOT go traveling, go sightseeing, or go shopping immediately after treatment, but should allow the muscles to rest and recover normal function. The patient should be instructed in advance to plan appropriately limited activity following treatment. Strenuous swimming should be avoided, but it is desirable for the patient to perform, in a warm pool, unstrained stretching and range-of-motion activities that cause no pain. Thus, "lazy" stretching with the body supported by the water is excellent. Specific stretching exercises for the patient to do at home are essential. They maintain and help to extend the range of motion achieved by treatment.

Although no controlled experiments on the effect of **heat** following TrP therapy are known to have been reported, it was Dr. Travell's conviction that dry heat applied to myofascial TrPs was not as ef-

fective as moist heat and that post-treatment muscle soreness is markedly reduced by applying a hot pack for a few minutes immediately after spray and stretch (or injection). Certainly, use of heat rewarms the skin for reapplication of spray or icing of the same area, if needed. Used at the end of treatment, applied heat leaves the patient feeling warm and reassured, which promotes further reduction of muscle tension by encouraging mental relaxation. The art of medicine can, at times, be as important as the science.

In this manual, when **moist heat** is mentioned, it is assumed to be an Hydrocollator Steam Pack or comparable hot pack. A convenient alternative for home use is a *waterproof* electric heating pad covered with dampened flannel. A piece of plastic that covers the exposed side of the pad can be tucked in around its edges to protect sheets, clothing and hair from getting wet.

Patients who are instructed to use electric heating pads at home must be warned to use the low setting; if they fall asleep with the switch on high, they may burn themselves seriously. A hand-pumped spray bottle of water is a convenient device with which to dampen the cover of the waterproof heating pad. A thin wet towel wrapped around an old-fashioned hot water bag has been used effectively in place of a hot pad or pack.

Voluntary Contraction and Release Methods

These methods all employ some degree of voluntary (active) contraction followed by relaxation. A reduction in muscle stiffness (tension) following the contraction provides an increase in range of motion during the period of relaxation. This approach is the basis for some of the simplest, most available, most popular, and most effective techniques for inactivating myofascial TrPs. Included among them are contract-relax, postisometric relaxation, a combination of postisometric relaxation and reciprocal inhibition, hold-relax, and muscle energy techniques.

The new understanding of the nature of TrPs provides a rationale as to why this approach is so effective. Since the primary TrP etiology appears to be a contraction knot at a dysfunctional motor endplate, gentle intermittent muscular contractions may be very effective at normalizing sarcomere lengths of involved muscle fibers. The action potentials resulting from the voluntary effort cause contraction of the elongated sarcomeres on both sides of the contraction knot. This added tension tends to pull open the contractured sarcomeres in the contraction knot. As soon as this process begins to separate the actin and myosin molecules in the contractured sarcomeres, those sarcomeres consume less energy because fewer myosin heads interact with actin. This tends to relieve the energy crisis which could lead to the reduction of the amount of excessive acetylcholine being released. If this analysis is correct, it *should* be beneficial during each relaxation period to keep taking up slack as it develops in the muscle. Chapter 2, Section D explains this etiological mechanism in detail.

Postisometric Relaxation (PIR). The postisometric relaxation (PIR) technique introduced by Karel Lewit" is a modified contract-relax method that for most muscles incorporates augmentation by coordinated respiration and eye movements. This technique has been specifically identified by Lewit[101] as useful in the treatment of myofascial TrPs with detailed instructions for their treatment in many individual muscles.

The basic concept of PIR is to contract the tense muscle isometrically against resistance and then to encourage it to lengthen during a period of complete voluntary relaxation. Whenever possible, gravity is used to "encourage" release of the muscle tension and take up the slack. For PIR to be effective, the patient must be relaxed and the body well supported. The muscle is passively and gently lengthened to the point of taking up the slack (reaching the barrier or the point of initial resistance). If this initial positioning causes pain, either the extent of the movement has been excessive or the patient has actively resisted the movement.

Postisometric relaxation begins by having the patient perform an isometric contraction of the tense muscle at its maximum pain-free length, while the clinician stabilizes that part of the body to prevent muscle shortening. Contraction should be slight (10-25% of maximum voluntary contraction[101]). After holding this contrac-

tion for 3-10 sec, the patient is instructed to "let go" and to relax the body completely. During this relaxation phase, the clinician gently takes up any slack that develops in the muscle, noting the increase in range of motion. Care is taken to maintain the stretched length of the muscle and not let it return to a more neutral position during subsequent cycles of isometric contraction and relaxation.[101]

Combining PIR with *reflex augmentation* of relaxation[100,101] greatly enhances its effectiveness. Augmentations include the use of coordinated respiration and eye movements as described below. *Reciprocal inhibition* can also be incorporated to enhance release of tight muscles. The effectiveness of the contract-relax technique used in *postisometric relaxation* was demonstrated experimentally as *preisometric contraction.*[110] These are two different names for essentially the same technique. In this controlled study,[110] the authors demonstrated that hamstring muscle stretch which was limited by pain increased significantly ($p < 0.01$) following a 6 second voluntary contraction of the muscle. The authors were perplexed by the fact that measured EMC was essentially unchanged before and after stretch under all conditions, because they had accepted the common assumption that increased resistance to stretch arose from motor unit activity. Their results substantiate our understanding that much increased stiffness of painful muscles is due to the viscoelastic properties of the muscle which can be strongly influenced by the tension of the taut bands that are induced by TrPs.[151a]

A good way to become skilled in the use of PIR is to practice it on oneself to relieve muscle stiffness associated with prolonged immobilization of muscles. This stiffness becomes increasingly apparent with advancing age, when it becomes critically important to maintain full range of motion of muscles, especially of postural muscles, in order to maintain normal mobility throughout the body.

Reciprocal Inhibition. Reciprocal inhibition is not only an involuntary spinal-level reflex but is effective when a contraction is initiated at the cortical level. When one muscle is activated, its antagonist is reflexly inhibited. The use of reciprocal inhibition is valuable for augmenting relaxation and release of muscle tension when stretching a muscle to inactivate its TrPs. To invoke reciprocal inhibition, the muscles that *oppose* the muscle being stretched are voluntarily contracted to actively assist the stretching movement. Thus, the muscle to be stretched is reciprocally inhibited.

This method can be used alone to augment a simple stretch, or it can be combined with other techniques such as spray and stretch. Apparently, this neuromuscular mechanism for releasing TrP tension involves more than inhibition of alpha motorneuron activity. The tension-release mechanisms also may be dependent on autonomic effects that are related to the inhibition of spontaneous electrical activity (SEA) and spike activity of TrPs during exhalation, and their augmentation by inhalation and mental stress.

Contract-relax. The principle of contract-relax appears in many forms with many names throughout the musculoskeletal treatment literature. The "muscle energy technique" is highly regarded by osteopathic physicians and is described under a separate heading below. The term *contract-relax,* as originally taught by Knott and Voss[90,180] was recommended for treatment of marked limitation of the range of passive motion with no active motion available in the muscle opposing the tight muscle. As they described it, contract-relax employed *maximum* contraction in a *pattern movement* followed by relaxation of the tight muscle to permit active shortening of the opposing weak muscle. Release of tightness in that muscle permitted improvement in the range of motion. Through the years, the exact meaning of the term has become somewhat diffuse. There are now numerous variations (and applications) of the basic principle that muscle tension is reduced immediately following voluntary contraction.

In this volume, contract-relax used for treating TrPs is a *gentle,* voluntary, minimally resisted contraction of the tight muscle. The contraction is followed by relaxation to permit passive elongation of the muscle to a new stretch length. Contract-relax is the basic procedure in the PIR method of Lewit.[101]

Hold-relax. Hold-relax is a variant of the contract-relax technique that is not commonly used for treating TrPs, but may

be employed when there is no joint movement desired during or after the procedure. It consists of isometric contraction of the tight muscle followed by relaxation, but *not* by elongation of the tight muscle. When used in the treatment of muscles with TrPs, hold-relax is commonly combined with manual techniques applied directly to the muscle, such as deep stroking massage and TrP pressure release.

Muscle Energy Technique. **Muscle energy** techniques are closely related to techniques used for release of TrPs and therefore are of considerable interest. They are osteopathic procedures that by definition[57] "are used to mobilize joints in which movement is restricted, stretch tight muscles and fascia, improve local circulation, and balance neuromuscular relationships to alter muscle tone."

Kuchera and Kuchera[94] clearly identify three muscle energy techniques. The first is *isometric contraction,* which is the one most commonly used, generally for improving restricted motion at an articulation. This technique corresponds to the contract-relax method described in this manual for restoring restricted motion at an articulation (caused by muscle tightness due to TrPs). The second technique, *isotonic contraction,* is commonly identified as a concentric (shortening) contraction, that in this case is resisted by the clinician. The third muscle energy technique, *isolytic contraction,* is the same as what is commonly identified as an eccentric (lengthening) contraction, but again is resisted by the clinician. These techniques involve voluntary muscle contractions by the patient against a specific counterforce provided by a clinician, whereby the patient, not the clinician, provides the corrective force.

Since two of the four stated objectives of muscle energy recognize the importance of correcting muscular abnormalities in order to effectively mobilize restricted joints, many of the procedures effectively stretch the trouble-making tight muscles with their associated fasciae. In many cases, the resisted patient contractions effectively produce the contract-relax maneuver. Since many of these maneuvers would therefore be effective for the treatment of TrPs, it is not surprising when Greenman[59] observes that frequently adequate treatment of somatic dysfunction relieves the TrPs. The concept of TrPs and many examples in specific muscles are well described by Kuchera and Kuchera[94] in a separate chapter that emphasizes their importance.

There is much to be gained if practitioners identify the active TrPs *and* any associated joint dysfunction so that each can be treated specifically with an appropriate technique. Often, the appropriate technique simultaneously (and serendipitously) corrects both conditions.

Trigger Point Pressure Release

The new term *trigger point pressure release* replaces the previous term and concept of *ischemic compression.* TrP pressure release is known to be effective at central TrPs where there is a rationale for its use. However, its value when applied to attachment TrPs needs to be evaluated experimentally. Clinical evidence and the nature of TrPs indicate that, when applying digital pressure to a TrP to inactivate it, there is no need to exert sufficient pressure to produce ischemia . Since the core of the TrP already is suffering severe hypoxia surrounded by increased tissue oxygen tension, there is no reason to expect that additional ischemia as such would be helpful. Treatment needs to release the contractured sarcomeres of the contraction knots in the TrP.

The technique that was previously described as ischemic compression is essentially what Prudden called myotherapy,[133] and was adopted by a group of practitioners of this technique who identified themselves as myotherapists.

Instead of ischemic compression, we recommend the application of TrP pressure release. This technique is less vigorous than ischemic compression and employs the barrier release concept.[101] The pressure release approach seems to be equally or more effective clinically and is NOT likely to produce appreciable additional ischemia. This approach is tailored to the needs of the individual's muscles, is more "patient friendly", and therefore is more likely to be used by the patient. The patient learns what optimal pressure feels like for subsequent self-treatment. The barrier release approach, however, does require a higher order of manual skill.

To apply TrP pressure release, the clinician lengthens the muscle to the point of increasing resistance within the comfort zone and then applies gentle, gradually increasing pressure on the TrP until the finger encounters a definite increase in tissue resistance (engages the barrier). At that point the patient may feel a degree of discomfort but should not experience pain. This pressure is maintained (but not increased) until the clinician senses relief of tension under the palpating finger. The palpating finger increases pressure enough to take up the tissue slack and to encounter (engage) a new barrier (the finger "follows" the releasing tissue). The clinician again maintains only light pressure until more of the muscle tension releases ("lets go") under the finger. During this period the clinician may change the direction of pressure to achieve better results. This process of TrP pressure release can be repeated for each band of taut muscle fibers in that muscle. The virtue of this technique is that it is painless and imposes no additional strain on any attachment TrPs, and thereby avoids aggravating them. This digital technique is particularly well suited to muscles like the infraspinatus and serratus muscles that are relatively thin and overlie bone.

The effectiveness of this approach can often be enhanced by including supplemental techniques. These additional techniques should not cause pain either. In addition to simply taking up the slack in the muscle before beginning the procedure, the entire muscle can be maintained at a slack-free length throughout the process. Release of the TrP may be further enhanced by occasionally performing a contract-relax maneuver alternated with reciprocal inhibition. The goal is to release the contraction knots in the TrP and release the tension they cause in the muscle fibers comprising the taut band.

This barrier release approach may fail to afford relief because (1) the TrP is too irritable to tolerate any additional mechanical stimulation; (2) the operator misjudged the pressure required to reach the barrier; (3) the operator pressed too hard, causing pain and autonomic responses with involuntary tensing by the patient; and (4) the patient has perpetuating factors that make the TrPs hyperirritable and resistant to treatment.

Shiatzu. Shiatzu[77] and acupressure[28] are terms used to describe a technique comparable to the old concept of ischemic compression but are not conceptually related to TrPs. However, the descriptions of the treatment strongly suggest that TrPs are often the painful condition that is being treated with shiatzu. These treatments are promoted for other conditions in addition to the relief of pain. Shiatzu and acupressure are philosophically quite different from the concept of myofascial TrPs, but in practice many of the treatments appear quite similar.

Deep Stroking (and Other) Massage

The technique of *deep-stroking massage* (which is also called *stripping massage)* was historically the first widely accepted technique for treating fibrositis (many descriptions of which fit myofascial TrPs[142]) and was widely practiced at the beginning of the 20th century. This method is probably the most effective way to inactivate central TrPs when using a direct manual approach, and it can be used to treat TrPs without producing excessive joint movement. The rationale is clear.

Deep-stroking massage is effective in the hands of clinicians who are skilled in its use. Massage should be applied with close attention paid to restrictive barriers and their release. The patient must be positioned comfortably so that the muscle to be treated is completely relaxed and lengthened without pain to the point that there is no residual slack in the muscle as a whole. The skin should be lubricated if the subcutaneous tissues are tense and immobile. The thumbs or a finger of both hands are placed so they trap a taut band between them just beyond the band's TrP. As the digits encounter the nodularity of the TrP that is caused by its contraction knots *[see* Fig. 2.25), pressure is exerted to engage the restrictive barrier. The digits progress no faster than tissue release occurs as the nodularity "gives" to some extent. The purpose of the pressure directed along the length of the taut band is to elongate the maximally shortened (contractured) sarcomeres of the contraction knots to release their tension. The stroking massage should be continued along the length of the remaining taut band beyond the TrP to

the attachment of the band, helping to restore the stretched sarcomeres to normal length by continuing to exert traction on the shortened contraction knots. This also helps to relieve the taut band tension and any enthesopathy in the attachment region.

The next massage stroke should go in the reverse direction starting on the same taut band but on the other side of the nodule to further release the contractured sarcomeres. This stroke now helps to release the abnormal tension on the other half of the taut band and at the other muscle attachment.

Excessive pressure or speed of movement is likely to rupture contraction knots, destroying the corresponding motor endplates as functional structures and increasing the painfulness of the procedure. If the procedure were to rupture the sarcolemmal membrane at the contraction knots, the muscle fiber would spill myoglobin. In addition to the effect of stretch, this rupture process may be one reason for the effectiveness of vigorous application of deep massage, as suggested by the following experiments.

Danneskiold-Samsoe and co-workers[40,41] found that application of deep massage to the "tender nodules" of "fibrositis" or of "myofascial pain" (which were consistent with the clinical characteristics of TrPs) relieved the signs and symptoms of most patients after 10 massage sessions. Those experiencing pain relief had a transient elevation of serum myoglobin levels following the initial therapy sessions, but not after the final sessions when symptoms had been relieved and the tenderness and tension of the nodule being massaged had subsided. The results of this research effort are illustrated in Figure 2.29. Control massage of normal muscle did not appreciably increase serum myoglobin. This finding suggests that the muscle fibers of TrPs and their contraction knots are more susceptible to mechanical trauma than uninvolved fibers and that local tissue manipulation can inactivate the symptom-producing TrPs.

This technique is *not* the deep friction massage of Cyriax,[37] which he applied *across* the long axis of the muscle fibers. The Cyriax technique is more closely related to strumming that is described below.

Strumming. Strumming is similar to deep-stroking massage except that the strumming finger runs across the taut bands at the level of the TrPs over the nodules from one side of the muscle to the other. The operator's finger pulls perpendicularly *across* the muscle fibers rather than along the length of the fibers. This method applies specifically to central TrPs, which are near the middle of the muscle belly.

Strumming consists of pulling the finger across the middle of the muscle fibers slowly until the nodule at the TrP is encountered. Light contact is maintained at that point until the operator senses tissue release under the finger. The finger continues pulling across the nodule in steps as tension releases. Relaxed deep coordinated abdominal (diaphragmatic) breathing by the patient facilitates general relaxation during the exhalation phase.

This technique is particularly applicable to a few muscles like the masseter and medial pterygoid, which permit direct palpation of the muscle through only a thin layer of mucosa rather than through thick cutaneous and subcutaneous tissues.

Friction Massage. The purpose of friction massage is to mobilize the superficial tissues over the underlying structures in order to improve their mobility.[11] This corresponds to the technique of skin rolling to relieve the subcutaneous tightness of panniculosis (Section 8, above) and is also an accessory technique. It is not considered to be a specific TrP therapy.

Ice Massage. Ice massage can be applied in two different ways with different objectives. One method is the intermittent use of ice in lieu of the vapocoolant spray as a variation of spray and stretch, as described earlier in this chapter. The other method is the local application of cold for pain relief, which is considered later in this section.

Periosteal Therapy. Periostbehandlung (periosteal therapy) is essentially an unrelated rhythmic massage technique that is applied to bony prominences of the body[179] and should not to be confused with myofascial TrP therapy. Waves of pressure are applied for 2-4 min; each half-wave of increasing or decreasing pressure lasts 4-10 sec. The finger, thumb, or knuckle pressure is applied to the periosteum near painful areas. We agree with the authors[179] that the mechanism of pain relief in this case is distinctly different from that of *Druckpunkte*

(pressure points), which usually have been described in terms that are compatible with myofascial TrPs.

Indirect Techniques

The osteopathic technique for releasing tender points described by Jones in 1981[85] in terms of strain and counterstrain (the term still used by osteopathic physicians[53]) has now evolved into a 1997 book on positional release therapy by a chiropractor and a physical therapist[39] that is predicated on the osteopathic paradigm of somatic dysfunction. This indirect technique uses body positioning for *releasing* tender points which are conceived of as foci of constriction in the myofascial tissues. The tender points they describe appear to have little relation to fibromyalgia tender points, but may fit into the concept of myofascial attachment TrPs. The authors[39] make no clear distinction between fibromyalgia tender points and myofascial TrPs.

Kuchera and Kuchera[94] characterize Jones's strain-and-counterstrain technique clinically as follows: About 146 tender points can be identified. The muscle pain, weakness, and subjective hurt is on one aspect of the body and the Jones tender point is characteristically on the other aspect of that part of the body, usually in a muscle antagonist. At the position of comfort, the point becomes flaccid on repeated testing and the position is one of mild strain. The position of comfort generally is held for up to 90 seconds, until release is detected by gentle intermittent test palpations, then the body parts are returned SLOWLY to their neutral position. Poor posture may cause recurrence of these "myofascial points" that are presumed to be caused by some form of functional strain.[94]

In the original book on strain and counterstrain by Jones,[85] he illustrated and named the location of 65 tender points. Most of the names identified bony landmarks that did not identify what muscle(s) attached at that location. Of the 65 points, 9 were identified at the attachment region of a named muscle. Forty-four points were located either at the region of a muscular attachment where one might find an attachment TrP, or, occasionally, at the belly of a muscle where a central TrP might be located. Twelve points appeared to be purely bony locations where a muscular attachment would be unlikely.

Lewit[101] considers Jones points as tender spots in soft tissue. These tender spots are often found at muscle attachments where enthesopathy could occur and cause the tenderness.

If these tender points of Jones are often at the same location as myofascial attachment TrPs, it should be simple to verify. First, one could note if a central TrP is present in a taut band of muscle fibers that attach where the Jones tender point has been located. If so, it would be of interest to measure the irritability of both the central and attachment TrP sites before and after treatment. The central TrP could be treated by trigger-point therapy (e.g., by injection or local pressure release techniques), *or* the Jones point could be treated by positional release therapy, orboth treatments could be applied to a third group of subjects. Each therapeutic approach may have advantages that are complementary.

Myofascial Release

Myofascial release is a system of therapy that combines principles and practice from soft tissue technique, muscle energy technique, and inherent force craniosacral technique.[59] It includes a highly subjective transfer of energy from the therapist to the patient.[141] It is strongly promoted by John Barnes[8] and practiced by numerous physical therapists. It is another example of a clinical practice that is sometimes effective for myofascial TrPs, but the patient is not examined for them, so the extent to which the patient is benefitting from relief of TrPs goes unrecognized. Unfortunately, any additional benefit of specifically addressing the patients' remaining TrPs is forfeited.

Accessory Techniques

A number of techniques can assist and supplement the specific TrP therapies described above. Controlled respiration and directed eye movement are two techniques of great value and are applicable to many of the TrP release techniques. The others have more limited application.

Phased Respiration. As one **slowly exhales,** muscles throughout the body generally tend to relax. With inhalation, muscular activity is facilitated.[101] One noteworthy exception is the relaxing effect

that a deep inhalation (yawn) has on the jaw-closing mandibular elevator muscles. Since exhalation encourages relaxation of most muscles, it can be very helpful to coordinate an exhalation phase of respiration with the release phase of most muscle-stretching techniques as described. To be effective, respiration must be sufficiently slow and deep.[101] Effectiveness also improves if the patient uses coordinated diaphragmatic (abdominal) breathing. Abdominal breathing is particularly important when attempting to relax neck muscles. Paradoxical breathing should be avoided; it is inefficient and may send mixed messages to the autonomic nervous system as to whether the individual is inhaling or exhaling.

When phased respiration is used in conjunction with a technique such as contract-relax that involves muscle contraction and relaxation phases, the muscle contraction phase should be synchronized with inhalation and the relaxation phase with exhalation. Patients who have difficulty adopting a slow, deep respiratory pattern may be helped by pausing, breathing naturally several times, and relaxing between each cycle of contraction-relaxation.

For the torso, inhalation facilitates moving toward the neutral erect position and exhalation facilitates relaxation as one leans away from an erect posture. Leaning forward is naturally associated with exhalation and relaxation.

The following experimental evidence suggests a significant relation between respiration and TrP activity. While conducting a study of active loci in human TrPs,[151] the authors confirmed a previous observation of another investigator.[76] In many subjects, spike activity associated with SEA of the TrPs in the upper trapezius muscle was turned on by normal resting inhalation and was turned off by exhalation. The authors also noted a corresponding waxing and waning in the amplitude of SEA. The effect was augmented by exaggerated respiration. Neither set of potentials represented nearby motor unit potentials or distant ones.

Directed Eye Movement, Clinical observations have shown that the direction of gaze facilitates the movement of the head and trunk in the direction of the patient's gaze and inhibits movement in the opposite direction. This applies to lifting the head and torso as well as to stooping and trunk rotation. Direction of gaze does not facilitate movement toward side bending.[100,101] However, looking up does facilitate straightening up from the side-bent position. These eye movements should not be exaggerated, because a maximum-effort movement may have an inhibitory effect.[100,101]

This phenomenon is directly applicable for enhancing stretch-type release techniques used to release TrP tightness in muscles. By looking in the direction of movement needed to release specific muscle tightness, the release process is augmented.

The mechanism for this effect may be similar to the mechanism for the influence that respiration has on the electrical activity in active loci of TrPs, which was noted above. However, the direction of gaze was not specifically tested in those experiments. It should be tested since eye motion and respiration are related. Lewit *et al.*[103] clearly demonstrated a significant synkinetic effect between the rate of alternating the direction of upward-downward gaze and the respiratory rate. The rate of respiration followed the rate of eye motion over a 2:1 range.

Skin Rolling. The manual technique of skin rolling as described in Chapter 2 of this volume with regard to panniculosis has been found to be useful clinically both for diagnosis and treatment of this condition. Therapeutically, skin rolling seems to work best over the shoulders and upper back and least well over the buttocks.[108] The nature of panniculosis and the reason that relieving the panniculosis apparently helps to relieve TrP activity remain speculative. They deserve to be investigated further.

Biofeedback. Biofeedback alone is not specific myofascial TrP therapy, but it can be helpful in two ways. First, it can be used to help the patient avoid unnecessary increased resting muscle activity which contributes to TrP irritability and activation. Many patients express their anxiety and frustration through general muscular tension, which abuses their muscles. Biofeedback training applied to the problem muscles can help these patients to become aware of unnecessarily sustained activation of their muscles. It can be an effective tool for teaching them how to recognize and control the excess tension. Meditation prac-

ticed for this same purpose can enhance the patient's ability to reestablish muscular relaxation and emotional tranquility.

Another, potentially much more important, application of biofeedback is the use of surface EMG for identifying muscular incoordination, referred inhibition, and referred spasm caused by TrP activity. Used as feedback, surface EMG can be an important tool in retraining the affected muscles to normal muscle balance and function following inactivation of the responsible TrPs (*See* Chapter, 2 Part B, Surface EMG).

Heat and Cold. Heat applied to the surface of the skin penetrates poorly. It causes reflex dilatation of skin blood vessels, increasing circulation that quickly removes the heat and distributes it throughout the rest of the body. Thus, the only effect it would be likely to have with regard to underlying TrPs would be a general increase in circulation in that part of the body. It would also tend to make the patient feel cozy and warm, helping in relaxation.

On the other hand, application of surface cold tends to penetrate progressively more deeply into the underlying tissues the longer it is applied. As the cold penetrates, it causes vasoconstriction which reduces the heat that would have been supplied by the local circulation. The cold numbs the tissues, which is why application of cold can be effective for relief of neurogenic pain.

Immediately following major trauma—such as fracture, dislocation, or whiplash injury—cold packs should be applied to the traumatized muscles to reduce pain and tissue swelling without regard to TrPs. When this acute phase has passed in a few days, TrP therapy should be considered.

In the past, clinicians have found that for relief of TrP distress many patients preferred the application of heat rather than cold. However, some patients preferred cold applications to TrPs for relief of their myofascial pain. This seemed contradictory and enigmatic. It may be that central TrPs are more responsive to warmth and that attachment TrPs are more responsive to cold.

No controlled study is known that has explored the effectiveness of heat versus cold when applied to TrPs as therapy. There is a possibility that cold applied to attachment TrPs would reduce the sensory hyperirritability of the enthesopathy. The differences in the response of some patients to the application of heat versus cold to the TrP region deserves critical investigation.

Iontophoresis and Phonophoresis. Iontophoresis is the process of using an electric potential of low voltage direct current to move a solute of ions across a membrane. In the case of TrP therapy, the movement is through the epidermis and dermis into underlying tissues. The degree of penetration depends partly on the barrier properties of the tissues to the penetrating substances. The maximum depth of penetration is probably about 1 cm and the direct current has a caustic and sclerotic effect that must be considered.[140] Iontophoresis has been used to deliver ionic drugs, including hydrocortisone, lidocaine, and salicylate. The use of recombinant DNA technology promises the production of protein and peptide drugs amenable to this therapy.[153]

The effectiveness and any advantages of this modality for medicating attachment TrPs would need to be established by adequately controlled experiments. Usually, injecting the medication directly into the desired location is more direct, reliable, provides better control of the dose, and exposes only the tissue to be treated to the medication. However, injection does involve instrumental invasiveness.

Phonophoresis employs therapeutic ultrasound to drive the substance through the dermis. This medium is commonly used to treat musculoskeletal conditions using hydrocortisone, lidocaine, or aspirin.[140] A controlled study[26] demonstrated effective penetration of dexamethasone and hydrocortisone acetate by ultrasound into the subcutaneous tissue, but not into submuscular tissue. The details of technique can be critical to success.[87]

Although no scientific papers are known to have been published on the usefulness of either of these techniques for the treatment of TrPs, some clinicians have found them useful for the administration of steroids into an active TrP area. Since the injection of steroid into central TrPs rarely appears to prove more beneficial clinically than nonsteroid needle techniques, it is unlikely that attempts to medicate the central TrP region using these modalities would be beneficial. However, steroid application to

attachment TrPs may be a different story, and the beneficial effects observed by clinicians may have been the result of steroid treatment of attachment TrPs. The hazards of steroids also must be fully considered.

Since painfully active attachment TrPs limit the usefulness of stretch techniques, a noninvasive method for markedly reducing their irritability could be quite useful. The advantages and disadvantages of phoretic penetration of steroids as compared to injection need to be investigated with regard to this modality.

Microamperage. Although the use of microamperage therapy for myofascial TrPs has been enthusiastically promoted by manufacturers, we know of no well controlled experimental studies that demonstrate efficacy, nor is there a convincing rationale at this time for its use in the treatment of TrPs. The whole field of cutaneous procedures to treat underlying TrPs needs critical investigation to resolve whether there is an unidentified mechanism operating, or other factors are responsible for whatever favorable clinical results are observed.[150]

Modalities

Therapeutic Ultrasound. Clinically, many therapists find the application of ultrasound an effective means of inactivating TrPs. Unfortunately, no *controlled* study specific to its effectiveness on TrPs is known. Ultrasound transmits vibrational energy at the molecular level, approximately 50% of which reaches a depth of 5 cm. These vibrations not only generate heat within the tissue, but can have additional, but less clearly understood, chemical effects due to intense molecular excitation that may play a role in TrP applications. The clinical use of therapeutic ultrasound is well summarized by Santiesteban.[140]

One clinically successful technique starts with a setting of 0.5 watt/cm^2 and uses a slow dwell technique with a circular motion that completes one circle in 1 or 2 sec.[193] The circle is tight enough to provide a small overlap over the TrP in the center of the circle. In another technique that employs essentially the same movement of the applicator, the power is first increased to the threshold pain level (approximately 1.5 watt/cm^2] and then reduced to one-half of that intensity. Over the next 2 to 3 minutes, the intensity is gradually increased with frequent queries as to patient sensations, until the intensity has been increased to, but not beyond the original pain threshold level. Usually, the patient no longer feels pain at this level of stimulation and the TrP is less tender and irritable.[128]

The Medco-sonolator combines ultrasound with electrical stimulation of sufficient intensity so that the increased current flow through the point of low skin resistance (that frequently, but unreliably occurs over the TrP) generates a prickly sensation. This technique may be helpful in finding a possible location of a TrP for those who have not yet mastered the necessary palpation skills. This combination therapy has been reported to be helpful clinically.[20,129]

The mechanism by which ultrasound could effectively inactivate TrPs is unknown. The ultrasound undoubtedly causes tissue heating, which could aggravate the local energy crisis by increasing metabolic rate at the TrP and thereby stress key TrP tissues to the point of no return. The heat may have more specific effects to inhibit the release of acetylcholine and reduce endplate dysfunction. The mechanical excitation of tissues at the molecular level by ultrasound may play a role in these processes.

Well-designed, well-controlled experimental studies on the effect of ultrasound on competently diagnosed active TrPs are needed to fill this challenging void in our knowledge.

High Voltage Galvanic Stimulation. The waveforms characteristic of this kind of electrical stimulation are relatively high-frequency brief spikes of at least 150 volts with very rapid rise times and no duration of peak-voltage. This form of stimulation is selectively more effective on large diameter motor nerves than on smaller diameter sensory nerves, which makes it better tolerated than square-wave potentials as a way of stimulating muscle nerves electrically.[140] A description of the parameters of various types of electrical stimulation is presented by Kahn.[86]

The use of high voltage (and high frequency) galvanic stimulation is common practice among some therapists as a primary modality for the treatment of TrPs. It sometimes is used as preliminary treatment and more commonly is applied fol-

lowing stretch and/or injections.[134] Clinical experience suggests that one effective technique is to increase the intensity of cyclic (not continuous) electrical stimulation to the point of gentle muscular contractions.

Rachlin[134] recommends electrical stimulation routinely following TrP injection and needling. He describes using an intermittent current (sinusoidal, surged, or ramped) for 15 minutes. If spasm were present, he recommends preceding the intermittent current with 10 minutes of tetanizing current to fatigue the muscle in order to achieve more complete relaxation following stimulation. If the patient rejects the use of electrical stimulation (because of discomfort), he suggests moist heat as a substitute.

Muscle spasms can be released by application of high frequency galvanic stimulation,[140] if the muscle is continuously stimulated to exhaustion.[112]

The application of intermittent or cyclic stimulation sufficient to cause gentle muscle contraction and then relaxation may be considered an involuntary (effortless) form of contract-relax, which, when done voluntarily by the patient, is very effective. The electrical stimulation allows the patient to feel contraction of the appropriate muscle, and this assists the patient to learn an appropriate voluntary contraction for a home exercise.

Pain Relief

Transcutaneous Electrical Nerve Stimulation. Transcutaneous electrical nerve stimulation is well established as one means of obtaining temporary, sometimes prolonged, pain relief. Although it is not a treatment modality for myofascial TrPs, it is an accessory technique. The electrical stimulus consists of relatively low-voltage square waves of variable polarity, duration and frequency. This stimulus is not suitable for muscle stimulation because it tends to stimulate small sensory nerves more readily than the larger motor nerves, and therefore is relatively more painful than high voltage galvanic stimulation. Santiesteban reviewed[140] the treatment parameters and clinical applications of this kind of electrical stimulation.

The nonspecific relief of pain afforded by this modality can, in addition to improving the quality of life, help the patient to achieve increased mobility and a degree of muscle stretching that otherwise might not occur.

Stimulation is sometimes applied along the spine, or over acupuncture points, or over reference zones where the pain is felt, or over the TrPs where the pain originates. Generally, electrode placement is an empiric matter that depends on what positioning provides the patient with the most relief. Stimulation over the central TrPs may give different results than stimulation over the corresponding attachment TrPs. This could be explored systematically.

Drug Therapy. In the management of patients with myofascial pain syndromes, one can consider the role of drugs with respect to pain relief, muscle relaxants, sleep, and trouble-making drugs.

PAIN RELIEF. No nonnarcotic medication is known that is specific for the pain generated by central myofascial TrPs. The new understanding of the pathophysiology of TrPs opens the door to identifying drugs that specifically inactivate the TrP mechanism. The specific effectiveness of drugs on attachment TrPs is unexplored. Since the enthesopathy causing the pain and tenderness at that location is a reaction to the stress of the taut band on attachment structures, it is likely that some anti-inflammatory drugs, including steroids, may provide effective relief if administered locally on site in effective dosage.

Available nonsteroidal anti-inflammatory drugs given orally afford little relief from pain originating in central TrPs. However, they can be very helpful for alleviating the postinjection soreness that is likely to peak a day or two after injection, especially when dry needling without a local anesthetic has been used. This alleviation reflects the fact that the tissue injury of needling induces an inflammatory reaction that is fundamentally different from the pathophysiology of the TrP itself.

When a nonsteroidal anti-inflammatory drug was injected in high concentrations at the TrP, its prostaglandin-suppressing action seemed to help relieve pain originating from TrPs.[49] Prostaglandins are likely one of the more important agents involved in the sensitization of nociceptors in a TrP. This drug would not be expected to have any effect on the primary endplate dysfunction.

Successful management of a *chronic* myofascial pain syndrome relieves the patient's pain so that analgesic medication is no longer needed. However, at the start of specific treatment, before the patient obtains sustained relief, medication may be necessary. To help wean the patient off of drugs, a time-contingent schedule, rather than the usual pain-contingent schedule, should be established. A pain cocktail can be very helpful.[47]

Chapter 5, Section D in this manual outlines a general treatment approach for chronic headaches, facial, neck, or shoulder pain that have a significant myofascial TrP component. It can readily be adapted to the other muscles.

Successful management of musculoskeletal pain depends on an accurate diagnosis. Is the pain muscular, articular, and/or neurologic in origin? In current medical practice most medical practitioners are poorly trained in the diagnosis and manual treatment of the common muscular and articular sources of pain. Common initial reactions of practitioners to these enigmatic musculoskeletal complaints are to (1) procrastinate, hoping it will go away spontaneously, (2) prescribe a drug, (3) consider surgery, or (4) rationalize it as psychogenic or behavioral, which can be considered a form of denial. The patients deserve better.

MUSCLE RELAXANTS. The rationale for the use of muscle relaxants has been based largely on the erroneous concept that muscle pain causes spasm of the same muscle, which in turn causes more muscle pain. Since this pain-spasm-pain concept has failed the test of experimental investigation[121] *[see* Chapter 2, Part C), we see no rationale for muscle relaxants in the treatment of myofascial pain caused by TrPs.

Often the increased muscle tension that is identified as "spasm" related to musculoskeletal pain is actually caused by taut bands of TrPs. Muscle relaxants have no effect on muscle fibers that are in contracture because of dysfunctional endplates. On the other hand, true spasm (identified as EMG motor unit activity) can be reflexly induced by TrPs or by other sources such as joint dysfunctions or ruptured surface fibers of an intervertebral disc *[see* Chapter 41, Part B). This spasm can be responsive to muscle relaxants, but it is important also to accurately identify and treat the source of the spasm.

SLEEP. Most patients with persistent myofascial TrP pain have difficulty sleeping and show abnormal sleep patterns when monitored in a sleep laboratory.[4] In many patients, it is the referred pain generated by active TrPs that disturbs sleep. In patients with pain and in normal subjects, disturbed sleep tends to aggravate pain the next day.[125] This subject has been well reviewed by Moldofsky.[124]

In treating patients with myofascial pain that disrupts sleep, top priority needs to be given to inactivating the TrPs that are chiefly responsible for insomnia. The patient should be shown what sleeping position will minimize myofascial pain and be encouraged to take medication as necessary to obtain restful sleep.

Three antihistamines, which are nonhabit-forming, are recommended for better sleep. Dimenhydrinate (Dramamine) and diphenhydramine hydrochloride (Benedryl) have a common antihistamine that has a soporific effect on most people. The 50-mg tablet of dimenhydrinate is available without prescription and packs a stronger wallop than a 25-mg capsule of diphenhydramine, which is also available in a 50-mg capsule. The 25-mg dose can usually be repeated during the night, if necessary, without excessive morning hang-over. Generally, sleep medication should be taken one-half hour before retiring.

Promethazine (Phenergan) has a longer duration of action than dimenhydrinate, and may be helpful to individuals who fall asleep easily, but have trouble remaining asleep. This antihistamine also has a potent calming effect that is valuable for patients who are anxious. Usually, one 12.5 mg tablet at bedtime suffices.

A natural sleep-inducing hormone, melatonin, is now available without prescription in a bewildering array of amounts available per tablet (from a few hundred micrograms to more than a milligram) with no guidance as to how to use it. It is specifically useful to reestablish a normal sleep-waking cycle. A dose of 200-500 /xg taken one-half hour before bedtime will expedite going to sleep and help to prevent early awakening. Best of all, there is no need for a hang-over effect the next morning because its influence is ef-

fectively negated by continued exposure to bright light. We recommend use of no more than the minimum dose required to obtain the desired effect, and recommend that melatonin be taken only intermittently, because the long-term effect of regular large doses (1 mg or more) has not been reported.

TROUBLE-MAKING DRUGS. Small to moderate amounts of caffeine may help to minimize TrPs by increasing vasodilatation in the skeletal musculature. However, excessive intake of coffee and/or cola drinks that contain caffeine (more than two or three cups, bottles, or cans daily) is likely to aggravate TrP activity. A cup of coffee may contain 50-150 mg of caffeine. As a rule, drip coffee contains more than percolated, which contains more caffeine than instant coffee.[34] Most of the canned soft drinks contain 30-50 mg of caffeine. However, caffeine-free soft drinks are now widely available. Many combination analgesic drugs contain caffeine that may add significantly to the total caffeine load without the patient's realizing it unless someone analyzes in detail the patient's caffeine intake.

Regular excessive alcohol consumption may indirectly perpetuate TrPs through chemically reduced serum and tissue folate levels and because of poor eating habits. Ingestion of alcohol reduces the absorption of folic acid, while increasing the body's need for it.

The habit of tobacco smoking markedly increases the need for vitamin C, which is poorly stored in the body. The marked capillary fragility associated with low ascorbic acid levels greatly increases the tendency for tissue bleeding at injection sites. Injection of TrPs in smokers should be postponed until adequate tissue levels of vitamin C are assured (see Vitamins, in Chapter 4). Clinical experience indicates that tobacco smoking aggravates TrPs directly.

Caveats

Hypermobility. Treatment with a stretching technique that fully lengthens a muscle is *contraindicated* across joints that are truly hypermobile. When there are TrPs in muscles that cross hypermobile joints, these TrPs should be inactivated using techniques applied directly to the central TrPs without stretching the muscle as a whole. These alternative therapies include TrP pressure release, deep stripping massage, hold-relax with mild (not vigorous) contraction, indirect techniques, TrP injection, high voltage galvanic stimulation, and ultrasound. The muscles of these patients may require strengthening with stabilizing exercises.

Hypermobility syndrome is covered in more detail in Volume 2, Chapter 2, Section 7 of this manual.

Shortening Activation (Reactive Cramping). When a tight muscle (e.g., right middle scalene) is suddenly released, shortening activation (a reactive cramp) may develop in an antagonist muscle (e.g., the left levator scapulae). As the tight muscle (right middle scalene) is lengthened well beyond its accustomed limit in the process of inactivating its TrPs, the antagonist (left levator scapulae) is simultaneously shortened to less than its accustomed minimum length. If the antagonist harbors latent (or mildly active) TrPs, they suddenly and strongly may be activated by being placed (and held briefly) in this unaccustomed shortened position. The patient can then experience severe cramplike referred pain from the TrPs in this muscle that is an antagonist to the previously tight muscle. This reaction may be immediate, or it can develop half an hour or so following treatment. The delayed reaction may be caused by the patient making use of the new stretch range of the treated muscle, thus placing the untreated antagonist muscle in the shortened position after leaving the treatment session.

Shortening activation can be avoided by systematically treating both the agonist and antagonist groups of muscles partially, one after the other. The reaction occurs more often in flexors, like the biceps brachii, finger flexor, and hamstring muscles, than in the corresponding extensors. Lengthening treatment of the sternocleidomastoid or quadratus lumborum muscle on one side is likely to activate latent TrPs in its contralateral mate. Treatment of the subscapularis may activate TrPs in the supraspinatus and infraspinatus muscles. Occasionally, stretch of the abdominal musculature initiates paraspinal cramping.

Reasons for Failure. When the patient fails to show lasting improvement following spray and stretch (assuming that myofascial pain due to TrPs is the correct diagnosis, and assuming that the muscle causing the

dysfunction is the one that was treated) the following should be considered:

PERPETUATING FACTORS. When active myofascial TrPs do not subside after correctly applied spray and stretch, one or more perpetuating factors are usually responsible.

INADEQUATE COVERAGE. If the spray is applied only to the reference zone where the patient complains of pain, it usually misses the skin overlying the TrP that is causing the pain. When TrPs in several widely separated muscles refer pain to the same area, stretching and spraying some, but not all, of these muscles will provide only partial relief.

PATIENT TENSION. For effective passive stretch, the patient must fully relax the muscles being treated. Frequently tension in postural muscles spills over, and the patient must assume a relaxed body position and feel relaxed all over to fully relax the muscles being treated.

POOR SPRAY TECHNIQUE. The vapocoolant is less effective if the stream of spray is passed too quickly over the skin, or if the spray container is held too close to the skin. On the other hand, the same skin area should not be sprayed so often, or so slowly, that the underlying muscle becomes chilled. The line of spray must be directed over the line of muscle fibers that are under maximum tension so that the topographically related skin reflex effects of vapocooling can release them.

INCOMPLETE SPRAY AND STRETCH. Additional cycles of spray and stretch, with rewarming after each cycle, need to be repeated as long as the range of motion increases significantly with each cycle, or until full range is reached.

INADEQUATE STRETCH TECHNIQUE. The TrPs will persist if too much or jerky force is used to passively stretch the muscle. Firm stretch *before* spraying can cause painful spasm and seriously impair relaxation.

INCOMPLETE STRETCH. Residual tautness remains when the muscle is stretched to less than its FULL range of motion. Adjacent muscles often need releasing before this full range can be reached. If stretch is limited by structural impediments, such as an old fracture, osteoarthritis or idiopathic scoliosis,[164] local manual release techniques will be required.

POOR POSTTREATMENT. Muscle soreness is likely to be greater if the skin is not rewarmed immediately with a hot pack or pad. Recurrence is more likely if the patient fails to actively move the treated part through its fully shortened and lengthened range of motion several times to reestablish normal function.

CHRONICITY. Pain recurs when aggravating or perpetuating factors are present and reactivate the TrPs. However, chronicity *alone* need *not* prevent an immediate but temporary response to specific myofascial therapy.

13. TRIGGER POINT INJECTION

There are three different approaches to the needle-inactivation of the active loci in a central TrP. Generally, we recommend injection of a local anesthetic without corticosteroid and no adrenalin. Dry needling can be effective but results in more postinjection soreness. Only under special circumstances would one inject Botulinum toxin A. Effective treatment using either the injection of a local anesthetic or dry needling depends on mechanical disruption and inactivation of the active loci in that TrP. Inactivation of TrPs by injecting Botulinum toxin A depends on its specific pharmacological destructive effect on motor endplates.

It is essential to clearly define just what is meant by one injection. The number of injections should be counted in terms of the number of TrP sites injected, not the number of times some solution has been deposited within one TrP site. One TrP site has a highly variable number of active loci that must be inactivated and all of the loci in one TrP can be needled or injected with one skin penetration. Using a nonmyotoxic local anesthetic (which is the kind of anesthetic recommended) or dry needling, many needle movements within the TrP are normally required. When a local anesthetic is used, one should inject only a small amount (< 1 ml) at any one location within the TrP. The clinician must obtain twitch responses from all of the remaining active loci in that TrP in order to ensure effective treatment.

Some clinicians depend on the injection of large amounts of seriously myotoxic drugs like Botulinum toxin A or concentrated long acting local anesthetics in the general vicinity of a point of tenderness, hoping to inject a TrP. When myotoxic drugs are considered unavoidable for injec-

tion of TrPs, it is much better to inject *small* amounts *precisely* where the contraction knots of the TrP are located. Selective injection of small amounts of these substances *wherever the needle elicits an LTR in a TrP* is much less damaging to the muscle as a whole, and is just as effective, as a large amount. EMG monitoring for the spontaneous electrical activity of an active locus is an even more specific indicator of a site for injection.

When reporting TrP injections, for each injection the clinician should specify the muscle injected, and whether it was a central TrP or an attachment TrP that was injected.

Why Inject?

The decision whether to treat TrPs by manual methods (described in the preceding Section 12) or by injection depends strongly on the training and skill of the practitioner. Ideally, both approaches should be equally available to the patient and used when indicated. Manual methods are noninvasive, available for the patient to learn to use for self-treatment, and can be used to release multiple TrPs at the same time in one muscle or a group of TrPs in several muscles that serve the same function. However, manual methods are more likely to require several treatments and the benefit of treatment may not be as fully apparent for a day or two, as compared to injection. It requires considerable time and effort to acquire the skill needed to use manual techniques or to inject TrPs effectively.

Manual methods are specifically indicated when the TrP is acute, when the goal is to train the patient in effective methods of self-management of the pain and dysfunction, when the patient is severely needle-shy, or when the central TrPs in the middle of the muscle belly are not accessible to injection (iliacus and psoas muscles) for most clinicians.

One well-performed injection can fully inactivate a TrP immediately, which is reassuring to the clinician and the patient. Identification and injection of key TrPs can produce impressive results. Success depends strongly on the accuracy of the clinician's aim. This accuracy depends strongly on the precision with which the TrP was localized and on the skill of the clinician.

Injection is indicated when a few TrPs remain that are unresponsive to manual methods, when skilled manual TrP therapy is not available, when there are only a few relatively acute TrPs and the treatment time is severely limited, and when the patient has hyperuricemia and symptoms of gout. Injections can be helpful when the muscle cannot be stretched for mechanical reasons, or when stretch should be limited because of hypermobility.

Patients with both fibromyalgia and myofascial TrPs are much more sensitive to painful therapeutic techniques (which, for them, are likely to be counterproductive) than are patients with TrPs but without fibromyalgia. Patients with both conditions respond to TrP injections, but they do not respond as well as patients with only myofascial TrPs.[72]

It is a serious mistake to judge the efficacy of TrP treatment by manual methods or by injection if the practitioner was not both well trained AND experienced in the techniques used for the muscles reportedly treated. Knowledgeable, skillful practitioners of TrP therapy can be difficult to locate. So often, when patients give a history of being treated for TrPs without benefit, careful questioning makes it clear that treatment was given without adequate examination for TrPs or was not performed in a manner that one would expect to be effective.

What to Inject?

Dry needling is as effective as injection of an anesthetic for relief of TrP symptoms, IF the needle elicits LTRs,[67] which occur when the needle encounters active loci of the TrP. Conversely, if no LTR occurs, dry needling and injection of nontoxic anesthetics are equally ineffective.[67] Postinjection soreness is more likely to occur, is more severe, and is of longer duration following dry needling.[67]

Various injection techniques have included the use of procaine, lidocaine, longer acting local anesthetics, isotonic saline, epinephrin, a corticosteroid, Botulinum A toxin, and several forms of dry needling, each of which will be considered.

Dry Needling versus Injection. In comparative studies[67, 82] dry needling was found to be as effective as injecting an anesthetic solution such as procaine or lidocaine in terms of immediate inactivation

of the TrP. In the Hong study[67] of the response of trapezius muscle TrPs to 0.5% lidocaine or to dry needling, both groups experienced essentially the same amount of improvement immediately and 2 weeks later. However, within 2-8 hours, 42% of the lidocaine-injected patients and 100% of the dry-needled patients developed local soreness. The soreness of the patients treated by dry needling had significantly greater intensity and duration than the soreness of lidocaine-injected patients.

These results indicate that the critical therapeutic factor in both cases is mechanical disruption by the needle. This is consistent with the understanding that disruption of the TrP contraction knots terminates the basis for a local energy crisis and its sensitization of nearby nerves.

Procaine Injection. Dr. Travell recommended a procaine concentration of 0.5% in physiological saline because greater concentrations showed no additional increase in anesthetic effect.[167] No serious harm is done by using a 1% solution. However, the higher concentration has no known advantage. An accidental nerve block will last longer and the higher concentration has a correspondingly greater toxicity to the muscle and systemically. The maximum amount of procaine that should be injected at one time is 1 g **ire** That would permit the injection of 100 ml of 1 % procaine. Since only a few tenths of a milliliter of local anesthetic are deposited at a time within a TrP, it rarely is necessary to inject a total of more than 20 ml at one visit.

With 0.5% procaine, accidental injection of 2 ml into an artery or vein creates no problem, if adequate hemostasis is applied to the vessel. Injection with the same strength solution near a nerve causes only mild sensory loss for a maximum of about 20 minutes, which is well tolerated if the patient was previously warned that this might happen. These statements are ***not*** true if the injected solution contains epinephrine, which is **never** recommended for the injection of TrPs.

Procaine is the least myotoxic among the local anesthetics that are commonly injected. Pain sensation following nerve block reappeared in 19 minutes after 1% procaine, and in 40 minutes after 1% lidocaine.[36] Procaine and chloroprocaine have the lowest systemic toxicity of the commonly used local anesthetics.[29]

Procaine is the ester of p-aminobenzoic acid and ethanol with a tertiary diethylamino group attached at the other end of the alcohol. It is hydrolyzed rapidly in the blood serum by procaine esterase to *p*-aminobenzoic acid and diethylamino ethanol.[56] The diethylamino ethanol is an antiarrhythmic agent, but more weakly so than procaine, and is fortunately an effective anti-convulsant, because convulsions are one of the toxic effects of the whole procaine molecule.[56] The other product of hydrolysis, p-aminobenzoic acid, is sometimes considered a member of the vitamin B complex because it is needed for the synthesis of folic acid by those bacteria that can produce the vitamin.[56] The potassium salt of p-aminobenzoic acid is sold under the name Potaba as an antifibrotic agent.

Most local anesthetics, including procaine, block nerve conduction by competitively replacing calcium at its membrane binding site.[36] Depolarization of the nerve membrane is essential for the propagation of an action potential and depends on the flow of sodium ions through sodium channels from the inside to the outside of the membrane. Normally, the displacement of calcium from its binding site facilitates the flow of sodium ions across the membrane through the channels. Blockage of this calcium binding site impedes the flow of sodium ions, which prevents depolarization and the propagation of an action potential.[29,56]

Local anesthetics based on this mechanism selectively affect small, usually unmyelinated, fibers as compared to large myelinated nerve fibers and thus block pain perception more than voluntary motor control.[56] Unlike most local anesthetics, procaine is *not* rapidly absorbed from mucous membranes.[29]

A bacteriostatic agent commonly added to procaine is sodium bisulfite, which can be irritating and contribute to postinjection soreness. This effect can be reduced by diluting 2% procaine solution to 0.5% with isotonic saline solution, which is not so irritating to the muscles as sodium bisulfite and has local anesthetic properties of its own.[56,193] The 0.5% strength of procaine is not commercially available.

Lidocaine Injection. A 1% solution of lidocaine is commonly used successfully instead of procaine to inject TrPs. The ef-

fectiveness of these two anesthetic agents for reducing postinjection soreness has not been compared experimentally.

Lidocaine is not merely a longer-acting form of procaine. These two agents have different intermediate chains and different aromatic residues.[56] Unlike other amide local anesthetics, lidocaine is an aminoacyl amide. Lidocaine is more effective in a neutral solution; procaine is more potent in an alkaline solution.[36, 139] Procaine is hydrolyzed in the blood stream; lidocaine is removed from tissues through solubility in fat and is metabolized primarily in the liver.[29]

Isotonic Saline Injection. Sola and Kuitert[154] treated a series of 100 patients with myofascial TrPs by injecting isotonic saline (which also contained a bacteriostatic agent) with multiple needle penetrations in a fanwise pattern. These patients experienced therapeutic results equal to those previously reported with the injection of a local anesthetic. Frost *et* a/.[50] did a controlled, double-blind comparison between isotonic saline and a long-acting anesthetic, mepivacaine. They injected tender areas of the muscle that showed localized changes in the consistency of the muscle, and from which the patient's pain could be evoked (TrPs). Using these TrP criteria for the precise localization of the injection, they found that the saline afforded equal, or more pain relief than injection of the same volume of 0.5% mepivacaine, which is myotoxic. Most bacteriostatic saline for injection contains at least 0.9% benzyl alcohol as the bacteriostatic agent, which has local anesthetic properties of its own.[56193]

Corticosteroid Injection. Corticosteroids are potent anti-inflammatory agents and therefore appropriate for the treatment of conditions characterized by an inflammatory reaction. The pathophysiology of a central TrP in the muscle endplate zone involves sensitization of nociceptors secondary to a local energy crisis. The clinical experience to date indicates that nonsteroidal anti-inflammatory drugs are not effective in reducing the nerve sensitization in central TrPs. Addition of injected steroids here apparently offers no advantage. On the other hand, the nerve sensitization at attachment TrPs is the result of chronic mechanical stress which may produce aspects of an inflammatory reaction that would be responsive to corticosteroids. The common practice of relieving the pain of enthesopathy with injection of corticosteroids supports this possibility.

The definitive treatment for attachment TrPs is inactivation of the central TrPs responsible for them. However, prompt reduction in the tenderness and irritability of the region of enthesopathy at the attachment TrP is therapeutically beneficial to the patient's comfort and most likely helps to reduce the irritability of the corresponding central TrP.

Although we know of no controlled study that critically evaluated the effectiveness of steroid therapy specifically for attachment TrPs, the report by Day *et al.*[12] on the treatment of tennis elbow is strongly suggestive that steroid therapy is quite effective in this situation. This study is summarized and its relation to attachment TrPs is presented in Chapter 36, Section 13.

The use of long-acting (deposit) steroids is not recommended for the injection of TrPs. Such a preparation may, by itself, be destructive to muscle fibers.[132] It can increase the danger of muscle and connective tissue tears. Long-acting steroids are generally irritating to nerves and can produce complications.[58] Use of deposit steroids enhances the danger of a systemic Cushingoid reaction with repeated injections.

Repeated use of corticosteroid injections followed by ultrasound has been reported to produce depression and atrophy of skin and subcutaneous tissue that resulted in the need for surgical repair.[88]

Myotoxicity. Procaine and lidocaine are the least myotoxic of the local anesthetics that are commonly injected intramuscularly, and lidocaine is clearly more myotoxic than procaine. Myotoxicity, particularly of the longer-acting anesthetics, is strongly related to the concentration injected. It is unlikely that solutions stronger than 0.5% are any more effective when injecting TrPs. Solutions stronger than 1% become increasingly and significantly myotoxic. Longer acting anesthetics tend to be more myotoxic than shorter acting ones. Epinephrine severely increases myotoxicity without conferring any appreciable clinical advantage when injecting TrPs.

Intramuscular injection of a 1% or 2% solution of procaine and of a 1% solution of lidocaine in rats produced a mild infiltration of neutrophils, lymphocytes, and

macrophages within 24-72 hours.[132] There were no, or at most only occasional, damaged muscle fibers; such fibers were eventually phagocytized. No changes could be detected beyond 7 days, except for a few remaining leukocytes. Perineural injection of the same solutions produced no histological changes within the nerves that had been anesthetized, but a temporary inflammatory reaction developed in 24-72 hours, with nearly complete recovery in 2 weeks. Repeated intramuscular injections of isotonic sodium chloride also caused a similar response.[132] Single intramuscular injections of 2% procaine or isotonic saline caused no muscle necrosis.[25] These findings indicate that the responses were essentially innocuous temporary foreign body reactions.

The intramuscular injection of longer-acting local anesthetics, like 0.5% dibucaine and 1% tetracaine, produced in the ensuing 24-48 hours moderate infiltration of the muscle with lymphocytes and macrophages as the predominant cells and occasional coagulation (severe) necrosis of the central muscle mass. In addition, adjacent muscle showed an intensification of eosinophilic infiltration with vacuolization, loss of cross striations, and some phagocytosis of muscle fibers (minimal necrosis). Regeneration of the muscle was complete in about 7 days.

Intramuscular injection of 2% lidocaine,[13,16,25] cocaine,[16] bupivacaine,[13,16] and mepivacaine[16] caused muscle necrosis, chiefly of the white muscle fibers.[16] Intramuscular injection of 0.5% bupivacaine destroyed chiefly red muscle fibers.[16]

By 4 days after intramuscular injection of 1.5% and 2% lidocaine in rabbits and mice,[24] any atrophy of the muscle was difficult to measure because of the pronounced inflammatory and degenerative changes in many fibers, with abundant endomysial cellular proliferation. By 16 days after injection, the reaction had subsided leaving centrally located muscle fiber nuclei and small round fibers with significant atrophy, but no fibrosis.[24] A subsequent study showed that damage to associated tissues and vascular supply was minimal or absent, so that muscle regeneration followed rapidly.[13]

No such muscle fiber destruction resulted from procaine in 2% solution[16,24] or from 0.5% solutions of lidocaine,[13,16] cocaine,[16] mepivacaine,[13,16] or prilocaine[13] in contrast to the stronger (2%) concentrations. Forty-eight hours after intramuscular injection of 3% mepivacaine, the muscle showed extensive necrosis that was specifically related to the increased intracellular concentration of free calcium that it produced.

Addition of epinephrine in strengths of 1:100,000 or greater potentiated the muscle damage caused by local anesthetics.[14] The muscle necrosis caused by supramuscular 2% lidocaine with 1:50,000 epinephrine regenerated completely in 16 days;[14] however, five successive daily injections left evidence of retarded regeneration and microscarring in some areas.[15]

Twenty-four hours after the intramuscular injection of 0.5 ml of 2% lidocaine with 1:100,000 epinephrine into rat gastrocnemius, the muscle was almost totally necrotic.[191] Eighteen hours after the same injection into human sternocleidomastoid muscle, the region of necrosis, which extended along fascial planes, was much more extensive, but the damage was less intense than in the smaller rat muscle. Muscle-enzyme levels in the blood serum increased in both groups and were characteristic of muscle destruction.[191]

Botulinum Toxin A Injection. Botulinum toxin type A (BTA) binds irreversibly to presynaptic cholinergic nerve terminals, which includes the terminals of motor nerves supplying skeletal muscle-fiber endplates. Once internalized, the BTA blocks exocytosis of the neurotransmitter acetylcholine (ACh), which permanently terminates any neurogenic muscle fiber contractions meditated by the affected endplates. The skeletal muscle that has been thus chemically denervated remains paralyzed until a motor nerve sprouts new axons and forms new synaptic contact to reestablish a functional neuromuscular junction for each of the affected muscle fibers.

Toxin potency is expressed in mouse units. One unit represents the estimated LD_{50} (median lethal dose) for 18-20 g female Swiss-Webster mice, which is approximately 0.4 ng of BTA.[30] There is typically a 24-72 hour delay between administration of toxin and onset of clinical effects, al-

though patients may experience results immediately. Axon sprouting and muscle fiber reinnervation terminate the clinical toxic effect of BTA, usually in 2-6 months.[23]

Botulinum toxin A has become well recognized as an effective therapy for spasticity caused by upper motor neuron lesions such as spinal cord injury because it terminates motor activity of the affected motor endplates. The increasingly extensive use of BTA for treatment of spasticity has recently been reviewed in depth.[23]

Since the primary dysfunction of motor endplates associated with the TrP phenomenon appears to be excessive release of ACh, injection into the TrP of a substance like BTA which only blocks ACh release should be specific TrP therapy. BTA injection for the treatment of myofascial TrPs has been reported by several authors to be clinically effective.[2,30,192]

> One randomized, double-blind, placebo-controlled study[30] in 6 subjects compared the effect of TrP injections into cervical paraspinal and shoulder girdle muscles. Four patients experienced at least 30% reduction in TrP symptoms and signs following BTA but not saline injection as measured by visual analog scale, verbal descriptors for pain intensity and unpleasantness, palpable muscle firmness, and pressure pain thresholds. Significant reduction in symptoms was not seen at 30 minutes following injection, but was found 1,2,3,4, and 8 weeks later. This corresponds to the usual 1- to 3-day delay in the onset of clinical effects. One subject had no response to either type of injection, and the other had an equal response to both types. This study employed valid criteria for diagnosing a TrP, but noted no confirmatory evidence that the injection was in the TrP. Since dry needling and saline injection of TrPs have also been reported effective treatment when properly placed, the placebo control may have had some therapeutic effect. This study[30] strengthens the expectation that BTA would be an effective therapeutic agent for injecting TrPs.

It is important when using BTA to inject the minimum amount necessary and only in the TrP, since BTA destroys normal and dysfunctional TrP endplates alike. Ottaviani and Childers[130] emphasized the importance of injecting BTA only where endplates were located and recommended that it be injected where a systematic search revealed endplate potentials. Since these spontaneous endplate potentials are highly correlated with TrPs,[151] this is an ideal way to determine exactly where to inject the BTA for maximum TrP effectiveness and would greatly reduce unnecessary destruction of innocent endplates.

It is unknown whether the newly formed endplates following BTA denervation are more or less vulnerable to the development of TrP dysfunction than those endplates that they replaced.

Dry Needling. Injection is effective using a dry needling technique.[67,82,98,167] However, Kraus[93] stated that although dry needling is effective, postinjection pain follows immediately. Supporting this, Berges[17] found that local anesthetics reduce the painfulness of TrP injection, as compared with isotonic saline and dry needling. Hong[67] reported that injecting lidocaine reduced postinjection soreness.

Lewit[98] reported that *accurately localized* dry needling is effective, without quantitatively comparing it to procaine injection. He preferred dry needling to the use of a local anesthetic, because dry needling permitted location of all of the TrPs in a region by fully preserving their telltale pain reaction.

Many practitioners of acupuncture use several TrP criteria to locate pain acupuncture points and, in fact, are successfully performing dry needling of TrPs that they speak of as acupuncture therapy (See Chapter 2, Section B)

How to Inject?

Preinjection. Before injecting or needling a patient's TrPs, the practitioner should consider patient positioning, vitamin C and aspirin intake with regard to possible increased bleeding tendency, needle selection, proper cleansing, painless skin penetration, and the value of preinjection blocks.

PATIENT POSITIONING. The patient should be recumbent for *any* injection, to avoid psychogenic syncope and falling to the floor. When the patient sits in a chair (or worse is standing), injections can be hazardous in susceptible individuals.[155,159] Recumbency also greatly facilitates locating

the TrP, since the patient is more comfortable and relaxed. It is then easier to adjust muscle tension so that the bands containing TrPs stand out in a background of relaxed muscle fibers.

Syncope is more likely to occur in apprehensive patients. The circulatory arrest observed in one recumbent patient receiving a venipuncture to draw blood was attributed to cardiac arrest by one author,[159] but was interpreted as extreme sinus bradycardia by a cardiologist who reported similar reaction while an electrocardiographic recording was being made.[155]

VITAMIN C AND ASPIRIN. The increased capillary fragility characteristic of a low serum vitamin C level can cause excessive bleeding in muscles injected for TrPs. Capillary hemorrhage augments postinjection soreness and leads to unsightly ecchymoses. A frequent source of increased bleeding due to low vitamin C is tobacco. Mega-dose vitamin C therapy daily for 1 week should correct this deficiency. At least 500 mg of timed-release vitamin C three times daily is recommended for a minimum of 3 days prior to injection of TrPs. The importance of vitamin C for smokers was reviewed in Section 12 of this chapter.

A daily dose of aspirin increases the susceptibility to bleeding. The patient should take no aspirin for 3 days before TrP injection or needling.

NEEDLE SELECTION. For the techniques recommended here, the needle length must be sufficient to reach the contraction knots in the TrP to disrupt them. The needle diameter is more a matter of personal preference and skillfulness developed through practice, except in locations where pneumothorax is a serious consideration. A precision technique with maximum control is then needed.

Table 3.5 relates the diameter of the needle in millimeters (metric system) to the gauge size (English system). The larger-diameter 22-gauge needles are less prone to bend within the tissues, thus providing a more accurate feel for the texture of the tissues being penetrated by the needle tip. The larger needles also provide more tactile feedback as to the density and texture of the tissue being penetrated by the needle. On the other hand, the thinner 27-gauge needles (nearly acupuncture-needle size) cause less tissue damage with each penetration and are well suited to a fast-in, fast-out technique.[68]

Table 3.5 ***Corresponding Needle Sizes in the Metric (Millimeter) and English (Gauge) Systems***

Metric (millimeters)	*Gauge Size*
0.30	30
0.33	29
0.36	28
0.40	27
0.45	26
0.50	25
0.55	24
0.60	23
0.70	22
0.80	21
0.90	20
1.1	19
1.2	18
1.3	17
1.6	16
1.8	15
2.1	14
2.4	13
2.7	12
3.0	11
3.4	10

A 22-gauge, 3.8-cm (1.5-in) needle is usually suitable for most superficial muscles. In hyperalgesic patients a 25-gauge, 3.8-cm (1.5-in) needle may cause less discomfort, but will not provide the clear "feel" of the structures being penetrated by the needle and is more likely to be deflected by the dense contraction knots that are the target. When capillary fragility with bleeding is a major concern, or subsequent ecchymosis is especially undesirable, the thinner 25-gauge needle is advantageous. A 27-gauge, 3.8-cm (1.5-in) needle is even more flexible; the tip is more likely to be deflected by the contraction knots and it provides less tactile feedback for precision injection.

In thick subcutaneous muscles, such as the gluteus maximus or paraspinal muscles, in non-obese persons, a 21-gauge, 5-cm (2-in) needle is usually necessary. For injecting a TrP, the needle should be long enough to reach the TrP *without inserting the needle to its hub.*

A 21-gauge, 6.4-cm (2.5-in) needle is generally long enough to reach TrPs in the deepest muscles, such as the gluteus minimus and quadratus lumborum, and is available as a disposable *hypodermic* needle. However, TrPs in such deep muscles in obese patients may occasionally require as much as a 8.9-cm (3.5-in) needle. The longer lengths of disposable needles are available only as 22-gauge *spinal* (not hypodermic) needles. The spinal needle is not as effective for TrP injection as the hypodermic type because of the spinal needle's flexibility and diamond-shaped tip, which pushes the TrP aside, rather than penetrating it. This problem may require obtaining nondisposable *hypodermic* needles 8.9-cm (3.5-inch) in length and ensuring that they are properly sterilized. The skin-indentation technique described later may solve the problem with a 2.5-inch needle. If indentation of the skin provides inadequate safe penetration, and sterilizing hypodermic needles is not feasible, an alternate manual treatment approach will be required *[see* Section 12).

CLEANSING. An aseptic technique is ensured by careful cleansing of the skin with a suitable antiseptic, avoiding areas suggestive of local infection, and by using uncontaminated sterile solutions and properly sterilized or disposable needles and syringes.

PAINLESS SKIN PENETRATION. Some patients are terribly afraid of the skin pain caused by needle penetration. This fear of the needle is usually acquired in childhood and creates obstacles to a good doctor-patient relationship.[1'5"'169] Most patients find the sharp skin pain more threatening than the deep, aching (sometimes more severe) pain of needle contact with the TrP. The skin pain is avoidable with the use of cold anesthesia (described below), but their fear is not avoidable.

First, the patient must be reassured that the needle penetration of the skin will be minimally painful. This can be done by demonstrating the spray procedure on the patient's brachialis muscle where the patient can watch, but only *after* explaining to the patient why it will not hurt this time. Vapocoolant spray is recommended in this situation because it is reliably effective when properly administered and convinces the patient you are doing something to eliminate the pain.

In more ordinary situations, a time-honored approach is to mask the needle pain with a strong distracting stimulus such as stretching, pinching, or slapping nearby skin, precisely when the needle is inserted; this requires a high degree of coordination and skill to be effective. Timing is critical.

In adults, vapocoolant spray provides the simple answer of cold anesthesia,[92166186] which effectively blocks nerve conduction when the skin temperature falls to 10°C (50°F). After carefully disinfecting the skin with alcohol, one applies the vapocoolant spray from a distance of about 45 cm (18 in) for 5 or 6 sec (just short of frosting), and then introduces the needle quickly after the stream of spray stops and the spray has evaporated leaving the skin nearly dry.[166186]

For young children who dislike the sudden cold impact of the vapocoolant jet stream, a sterile, fluffy, small cotton ball is saturated with vapocoolant until it is dripping wet. The wet cotton is held *lightly* against the skin for about 10 sec, and then removed. *At the instant* that the skin dries, the needle is inserted painlessly.[186]

Three less reliable, but more convenient, techniques that can be combined are to (1) insert the needle *very quickly* through the skin with a flick of the wrist, (2) place the skin under marked tension so that the additional tension of the needle penetration is hardly noticeable (this can be done by the operator strongly spreading his or her fingers apart against the skin and inserting a needle between them), and (3) increase skin tension by pinching a fold of the skin between the thumb and fingers and inserting the needle through the tightly folded skin. The latter two techniques are recommended **ONLY** for the initial penetration with a fresh disposable needle that has not yet penetrated the skin of the patient, in case the needle accidentally penetrates a finger of the clinician.

When the skin has been cleansed with an alcohol wipe, a film of liquid alcohol remains for a while. If the needle is inserted through the wet alcohol it produces a stinging sensation as the needle carries some of it into the skin. This can be avoided by simply waiting until the alcohol dries, or by washing the alcohol away with vapocoolant spray which is sterile as dispensed. The vapocoolant evaporates more rapidly than the alcohol. The particular technique used

is less important than the communication to the patient that the practitioner *cares* and *knows how* to insert the needle painlessly.

Before injection, the patient should be warned that successful needle contact with a TrP may produce a flash of distant pain and likely will cause the muscle to twitch. The patient should be asked to note exactly where that pain is felt, permitting an accurate description afterward of the precise pattern of pain referred by that TrP. In this way, the operator can confirm the referred pain pattern of that TrP, and the patient can realize the connection between his or her pain and the TrP in that muscle. This reassures both the operator and the patient as to the importance of inactivating it. Patients learn to welcome this painful harbinger of a successful injection and future relief.

PREINJECTION BLOCKS. It is now well established that even brief exposure to considerable pain can cause long-lasting neuroplastic changes in the spinal cord that tend to enhance pain. For patients who are particularly pain-sensitive, or who have found the pain produced by needle encounter with TrPs seriously distressing, a preinduction block can be helpful. This is a newly introduced procedure and must be adopted with due caution. It is described in detail by Fischer,[46] who presents two methods. One involves diffuse infiltration of local anesthetic proximal to the area to be injected, and the other involves infiltration of the entire TrP area with local anesthetic before needling individual active loci. It is important, if one does these infiltrations, to use 0.5% procaine because of its lower myotoxicity, its relative innocuousness if a vessel were accidently injected, and the more rapid recovery of normal nerve function.

Precision Technique. Although there are a number of alternate TrP injection techniques now in use, the following precision technique is the one that was presented in the first edition of this volume. It is a basic technique that is applicable to central TrPs in any muscle location that can be reached with a needle.

LOCALIZING THE TRP. Localization of a TrP is done mainly by the practitioner's sense of feel, assisted by patient expressions of pain and by visual observation of LTRs. The TrP is identified by gentle palpation for the taut band in the muscle, next for a firmer nodule in the taut band, and then for exquisite spot tenderness of the nodule. The tender spot in the nodule (the TrP) is also the most responsive spot for eliciting LTRs by snapping palpation or by needle insertion.

The three methods of palpation (flat palpation, pincer palpation, and deep palpation) are fully described in Section 9 of this chapter, under Palpable Tender Nodules and Taut Band. The more precisely the TrP is localized the more satisfactory will be the injection.

When flat palpation is used to locate the TrP for injection, its position can be confirmed precisely by pushing the nodular TrP back and forth between two fingers (Fig. 3.15A and B). The TrP can then be fixed for injection by pinning it down midway between the finger tips (Fig. 3.15C). This identifies for the practitioner the plane that passes through the TrP perpendicular to the skin. The needle can then be aimed half way between the fingers precisely in that plane and angled to whatever depth is necessary to reach the TrP.

When pincer palpation has been used to locate the nodule and its TrP, the degree of tension placed on the muscle fibers can be fine tuned by varying the distance that the muscle is pulled away from underlying tissues. The nodule is located by rolling sequential portions of the taut band between the digits (Fig. 3.8). The nodule lies in the endplate zone which is near the center of the muscle fibers. For injection, the TrP is held tightly between the thumb and fingertips. An additional description of this method as applied to the teres minor muscle is found in Chapter 23, Section 13.

When deep palpation is necessary to locate the TrP, the position of the finger on the skin and the precise direction of maximum tenderness are carefully noted. The needle is then inserted exactly where the finger was and directed in precisely the same direction as the maximum tenderness.

For injecting central TrPs when employing any of the three methods of palpation, the muscle fibers of the taut band are placed on sufficient stretch to take up any slack but not enough stretch to cause additional pain. This tautness is necessary to help hold the TrP in position. If the muscle is slack there is a tendency for the dense contraction knots

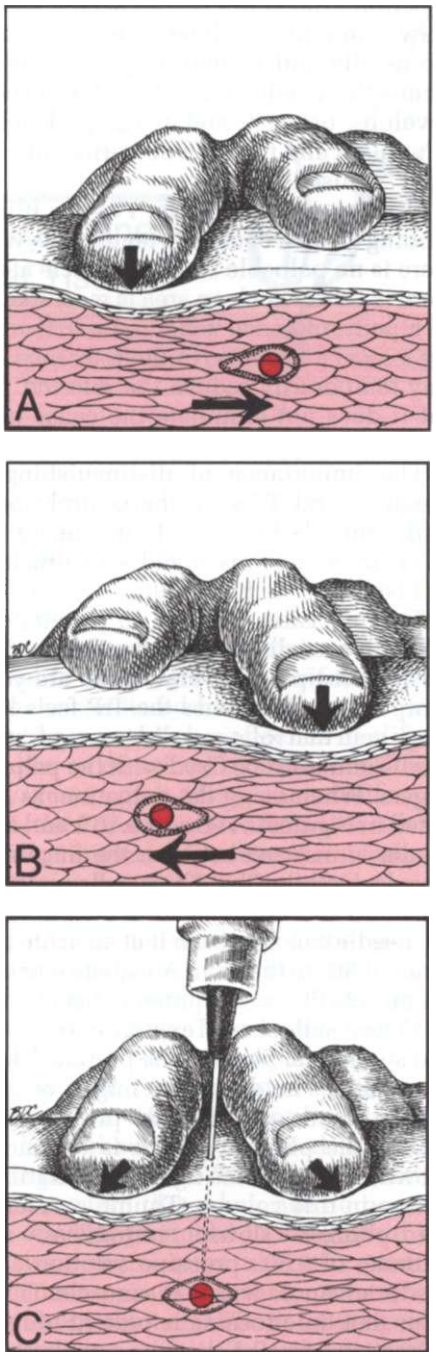


Figure 3.15. Cross-sectional schematic drawing of flat palpation to localize and hold the trigger point *(dark red spot)* for injection. **A** and **B,** use of alternating pressure between two fingers to confirm the location of the palpable nodule of the trigger point. **C,** positioning of the trigger point half way between the fingers to keep it from sliding to one side during the injection.

of the TrP to slide to one side, like a tough vein, as the needle tip encounters them.

To inject central TrPs in superficial layers of muscle close to the skin, the needle tip can be brought precisely to the TrP by first carefully locating the tender nodule with the finger and then, after inserting the needle subcutaneously, pressing it against the finger through the skin to accurately localize the TrP. Finally, the needle tip is directed into the TrP by means of this "tactile vision" provided by palpating both the needle and the TrP at the same time.

The same technique is useful to inject TrPs in the area of the muscle opposite the puncture site when using pincer palpation. The location of the needle and the TrP can be identified by palpation as the needle approaches the skin after penetrating most of the muscle.

Attachment TrPs are identified as spots of marked tenderness and usually some palpable induration in the region of the muscle attachment. The end of the muscular contractile tissue and the structure(s) to which it attaches are identified by palpation and the muscle tissue is examined to determine whether a taut band runs to the region of tenderness. The region of tenderness is then injected with anesthetic.

There is a need for controlled experimental studies to resolve the relative advantages of dry needling or injecting fluids like saline, local anesthetics, and corticosteroids into the region of enthesopathy. There is *no basis* for injecting botulinum toxin A into attachment TrPs.

HEMOSTASIS. Injecting TrPs is a full-time job for both hands of the practitioner. The injecting hand is busy placing the needle and controlling the plunger of the syringe for injection. The palpating hand constantly maintains hemostasis and often must fix the TrP to help the needle penetrate it. It also must be ready to detect any palpable LTRs. Hemostasis is important.[193] Local bleeding is irritating to the muscle, causes postinjection soreness, and can produce an unsightly ecchymosis. Ecchymosis is usually preventable; when it occurs, only time (which may be assisted by ultrasound *if steroid was not injected]* eradicates it.

To prevent bleeding, the fingers of the palpating hand should be spread apart, maintaining tension on the skin (Fig.

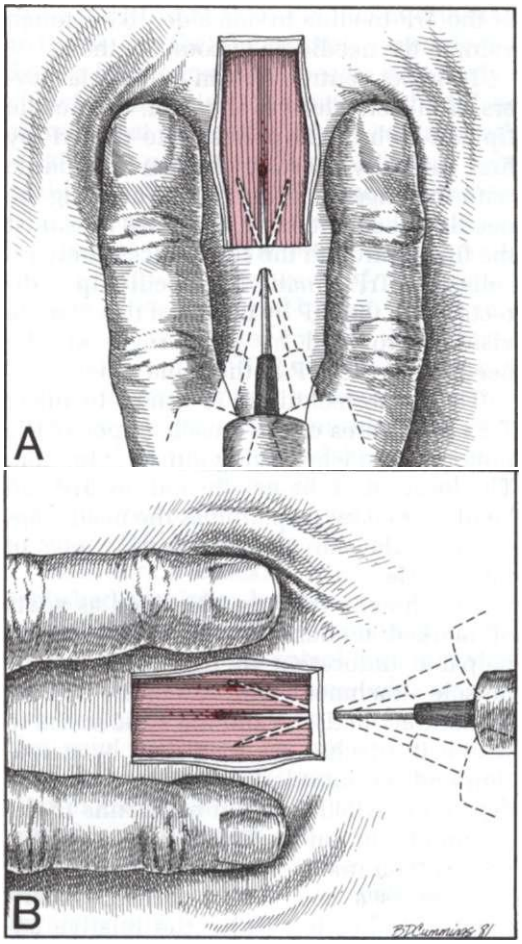


Figure 3.16. Schematic top view of two approaches to the flat injection of a trigger point area *(dark red spot)* in a palpable taut band *(closely spaced black lines)*. **A,** injection away from fingers, which have pinned down the trigger point so it can not slide away from the needle. *Dotted outline* indicates additional probing to explore for additional adjacent trigger points. The fingers are pressing downward and apart to maintain pressure for hemostasis. **B,** injection toward the fingers, with similar finger pressure. Additional trigger points are often found in the immediate vicinity by probing with the needle.

3.16A) to reduce the likelihood of subcutaneous bleeding where the needle has penetrated. Also, during the injection, the fingers exert pressure around the needle tip to provide hemostasis in deeper tissues. When the angle of the needle is changed, the direction of pressure changes. The pressure should be applied *throughout* the injection procedure. As the needle is withdrawn, one finger slides over the track of the needle and instantly applies pressure where the needle was. If visible bleeding develops, pressure and a cold pack should be applied and the patient warned of a possible "bruised" spot.

NEEDLING THE TRIGGER POINT. Blindly probing an area of diffuse tenderness where there is no palpable band or muscle attachment is futile. Such an area is most likely to be a pain reference zone, not a TrP. Injecting a local anesthetic in the reference zone may temporarily reduce the referred pain, but it does not eliminate the cause of the pain.

The importance of distinguishing between central TrPs (in the central portion of the muscle belly) and attachment TrPs when injecting was noted and illustrated by Fischer[46] (see Fig. 3.19).

The precision required to penetrate the TrP with a needle is a skill that for most people requires practice. How good are you at venipuncture? At times the TrP feels like a tough vein that rolls and slides away from the needle and must be fixed with the palpating fingers. When using flat palpation, as illustrated in Figures 3.15C and 3.16A and B, the needle is inserted between the fingers that have located the TrP. The needle penetrates the skin 1 to 2 cm away from the TrP so that the needle can approach it at an acute angle of about 30° to the skin. Adequate tension of the muscle fibers is required to penetrate the TrP. The needle should explore both the deep and superficial fibers of the muscle. The syringe may be held between fingers of the injecting hand, and thumb pressure used against the plunger, which is the method shown in most of the figures illustrating injection in this volume. Thumb pressure on the plunger slowly introduces small amounts of 0.5% procaine solution as the needle advances within the muscle. This ensures that the procaine is present to relieve pain at the instant that the needle tip encounters an active locus of the TrP.

The clinician should avoid inserting the needle to the hub where the needle is most likely to break off. Some additional depth of penetration can be obtained safely by indenting the skin and subcutaneous tissues with a finger beside the needle as illustrated in Figure 3.17.

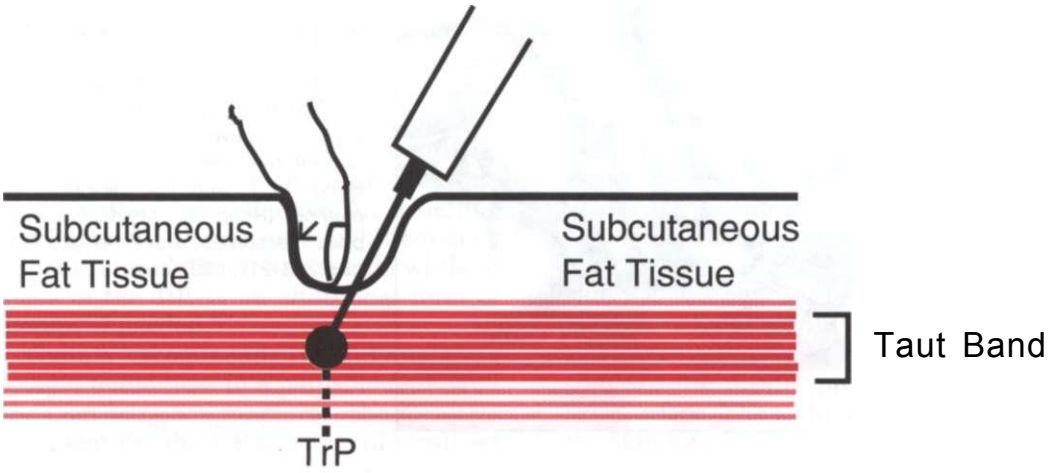


Figure 3.17. Finger pressure beside the needle is used to indent the skin, subcutaneous, and fat tissues so that the needle can reach the trigger point in a muscle that would be inaccessible otherwise. (Credit is given to CZ Hong, M.D., for suggesting this technique.)

The dense contraction knots in a TrP often feel to the practitioner as if the needle tip has encountered hard rubber that is resistant to penetration and tends to slide to one side, as described by Gold and Travell many years ago.[54] Using the needle as a probe, the TrP sometimes feels like a dense globule, 2-3 mm in diameter;[54] resistance to penetration helps to identify it.[93] Occasionally, TrP contact with the needle feels gritty. Adequate tension of the muscle helps to stabilize the position of the TrP to permit precise penetration by the needle, especially for deep TrPs which cannot be easily fixed in position by palpation.

If an LTR and referred pain were elicited from the TrP prior to injection, then both should be observed when the needle penetrates the TrP during injection. Hong showed[67] that when needle penetrations of a TrP produced LTRs, those injections were much more likely to result in subsequent pain relief than penetrations that did not elicit an LTR. Following effective needling, most TrP characteristics should have disappeared; no LTR, no evoked referred pain, and no spot tenderness should remain.[1793] The tense band is more relaxed following effective needling and may no longer be distinguishable by palpation.

Whenever an LTR or pain response occurs, an additional 0.1-0.2 ml of procaine solution is injected to minimize postinjection soreness. The patient can usually describe the exact distribution of the referred pain elicited by needle contact with an active locus of the TrP, but only if he or she had been alerted beforehand to pay attention to it.

Sometimes a cluster of TrPs, each with a discrete taut band, are present in one part of the muscle. This fact is often recognized when the muscle is initially palpated for TrPs. When one of these TrPs has been inactivated, the area is peppered[54] in a fan-like manner,[17] or in a full circle,[93] in an effort to ensure that all remaining TrPs in the group are inactivated, as illustrated in Figure 3.16B. After each probing movement, the needle tip must be withdrawn to subcutaneous tissue and redirected before the next movement. When this probing search of the spherical region is completed, the site is palpated for any remaining spots of tenderness. If one is found, it is accurately localized with the fingers and injected. ***All*** tender spots in that region should be eliminated before withdrawing the needle through the skin.[185]

Hong Techniques. Hong[68] introduced two new injection techniques: one was a safer way to hold the syringe and the other was a different way to perform the injection itself.

HOLDING THE SYRINGE. When one injects TrPs in locations that pose a hazard should

Figure 3.18. Injection of trigger points using a technique for holding the syringe that minimizes the danger of accidentally inserting the needle farther than intended if the patient makes a sudden unexpected movement. Drawn from an original photograph, courtesy of John Hong, M.D. who first described this method. (Hong CZ. Myofascial trigger point injection. *Crit Rev Phys Med Rehabil* 5:203-217, 1993.)

the patient make a sudden unexpected movement—such as a startle reaction, sneeze, or cough—Hong[66,68] recommends a way to hold the syringe that is safer than the usual way. His technique ensures that the syringe will move with the patient and not enter unintended tissue and that the finger on the plunger of the syringe will move with the syringe and not cause an accidental injection. The hand that is holding the syringe must be firmly supported by the patient's body; this is readily accomplished with his technique, as illustrated in Figure 3.18. The syringe is held between the thumb and lesser fingers, and the plunger is depressed with the index finger. This technique is particularly valuable when injecting over the lung or when the needle is directed toward major arteries or nerves.

RAPID TECHNIQUE. Hong[68] also described his "fast in, fast out" method of injecting a TrP that has been precisely located by palpation. The palpating finger should stay over or straddle the taut band in order to guide the needle insertion directly to the TrP. The syringe is held by the other hand. With the thin (27-gauge) needle remaining deep to the subcutaneous tissue, the muscle fibers of the TrP are carefully explored with multiple needle insertions.

The needle movement is rapid, "fast in" and "fast out." Hong has modified the technique as originally described. He now takes 2 or 3 seconds between insertions.[70] The pause following each insertion permits time to consider the tissue textures traversed by the needle and where to redirect the needle, time for identification of an LTR, and time to immediately inject anesthetic solution into the same needle track when a twitch occurs.

The needle is inserted deep enough to fully penetrate the taut band (TrP) region and then is pulled back to the subcutaneous tissue layer, but not out of the skin. A drop of 0.5% procaine (or lidocaine) is injected into the taut band following every LTR, which is detected by the feeling of needle tip movement (from the hand holding the syringe), by palpating the twitch contraction (with the hand doing taut-band palpation), or by seeing the movement of a visible twitch. The local analgesic agent should be injected only if an LTR accompanies needle insertion.

This rapid technique avoids muscle fiber damage from LTRs. Experience during research studies showed that LTRs are elicited more frequently when the needle is moved quickly rather than slowly. The track of needle insertion is usually very straight and the needle is less likely to be deflected by the dense contraction knots when the needle is inserted at high speed. For this reason, this "fast in, fast out" technique is well suited to the use of acupuncture needles. It may require a considerable period of practice before one becomes skillful in this rapid needle movement/injection technique.[68]

Dry Needling. As noted above, the experimental evidence available indicates that dry needling is as effective for inactivating TrPs and relieving TrP pain as injection with a local anesthetic (lidocaine).

However, the patients receiving dry needling are significantly more likely to experience postinjection soreness that is more severe for a longer period of time.[67]

Many practitioners familiar with both acupuncture techniques and the identification of myofascial TrPs find the acupuncture needles very effective for needling myofascial TrPs. Some add high frequency galvanic stimulation to the needle while in place in the TrP as an additional form of therapy if needed.[60]

Gunn[61] recommends identifying TrPs by spot tenderness in a palpable taut band and then using acupuncture techniques. He first identifies the TrP as a spot of localized tenderness in a taut band and then identifies the precise skin location through which to insert the acupuncture needle using a dermometer (point finder or skin resistance detector). He then inserts the needle through this location to the TrP where he feels a "grabbing" sensation at the needle tip, which is often associated with aching pain, as the needle enters the TrP. An LTR is often observed. Gunn identifies this TrP injection technique as Intramuscular Stimulation.[61]

Special Precautions.

CONTRAINDICATIONS TO TRP INJECTIONS.

1. Patients on anticoagulation therapy.
2. If the patient has taken aspirin within 3 days of injection.
3. Tobacco smokers unless they have stopped smoking and have taken at least 500 mg of timed release Vitamin C for 3 days prior to injection.
4. Patients who have an inordinate fear of needles.

CAVEATS.

1. By NEVER aiming the needle at an intercostal space the clinician avoids the distressing complication of a *pneumothorax*. The only exception is when there is need to inject intercostal muscles, and this is done only with GREAT care. The patient may sneeze or jump; the operator may startle unexpectedly. As a resident, Dr. Travell found in her early experience of doing many pleural taps for pleural effusions, that patients consistently reported a salty taste in the mouth whenever the pleura was punctured. The patient might say, "Oh, I can taste the solution." When the lung is punctured and collapses, dyspnea, cough, and chest pain characteristic of a pneumothorax follow.
2. A needle is prone to *break* where it attaches to the hub. The needle should never be inserted solidly to its hub because of the difficult situation that would ensue should it break off at the hub and disappear under the skin. Recovering the needle can be a time-consuming, frustrating process. A long-enough needle should be used, or the skin indented around it, to ensure that some of the needle projects above the skin surface. The technique of indenting the skin by displacing subcutaneous tissue with finger pressure on either side of the needle is illustrated in Figure 3.17.
3. The *location of the needle tip* can readily be misjudged when using a long slender needle. It is especially important to insert the needle straight and avoid any side pressure that might bend the needle, deflecting the tip an unknown distance to one side.
4. A needle with a *burr at the tip* must not be used. When the tip of a disposable needle contacts bone, the impact frequently curls the tip to produce a "fishhook" burr that feels "scratchy" and drags as the needle is drawn through tissues; it causes unnecessary bleeding, and should be replaced immediately. It is especially important to avoid using such a barbed needle when injecting TrPs in muscles like the scaleni, which lie near nerve trunks.

How Many Injections?

Note the definition of one injection at the beginning of this Section 13. The number of TrP sites that need to be injected per visit and the number of visits required are strongly dependent on the patient's condition and the practitioner's skill and judgement. To date, no medical specialty has adopted the diagnosis and treatment of myofascial TrPs as an official part of the training program, nor have *specialty* standards of training and practice been established for this diagnosis. The *International Association for the Study of Pain* has published recommended standards of TrP training.[45]

Since some practitioners request reimbursement for unreasonable numbers of TrP injections, and there is no assurance as

to the competence of individual practitioners, third party payers are beginning to establish arbitrary limits on the number of injections. Unfortunately, at this time, procedures are not available to readily determine if large numbers of injections were sometimes performed because of lack of training and skill on the part of the practitioner, because of unusual needs of the patient, or because of no clear definition of what constitutes one TrP injection.

Recently activated (acute) myofascial TrPs that have no perpetuating factors or additional tissue damage because of mechanical injury to other tissues (i.e., TrPs that are uncomplicated) should resolve with one or two injections. This is especially true if, after injection, the patient is trained and then performs exercises to maintain full range of motion of the involved muscle(s). When both central TrPs and attachment TrPs are present (Fig. 3.19), both sites must be injected, which would count as separate injections.

When initial TrP therapy is delayed and symptoms have not subsided with time, the longer the period of delay before starting TrP therapy, the larger the number of injections that will be required over a longer period of time.[73] Some chronic TrP problems could involve dozens of injections over months of treatment. In this situation, the primary guideline is that the period of relief from TrP pain and dysfunction should become progressively longer with successive injections.

When there are multiple active TrPs in functionally related muscles, there is a distinct advantage to inactivating them as a group. Thus, 5 or even 10 injections at one visit can be appropriate. Since a properly performed and effective injection produces

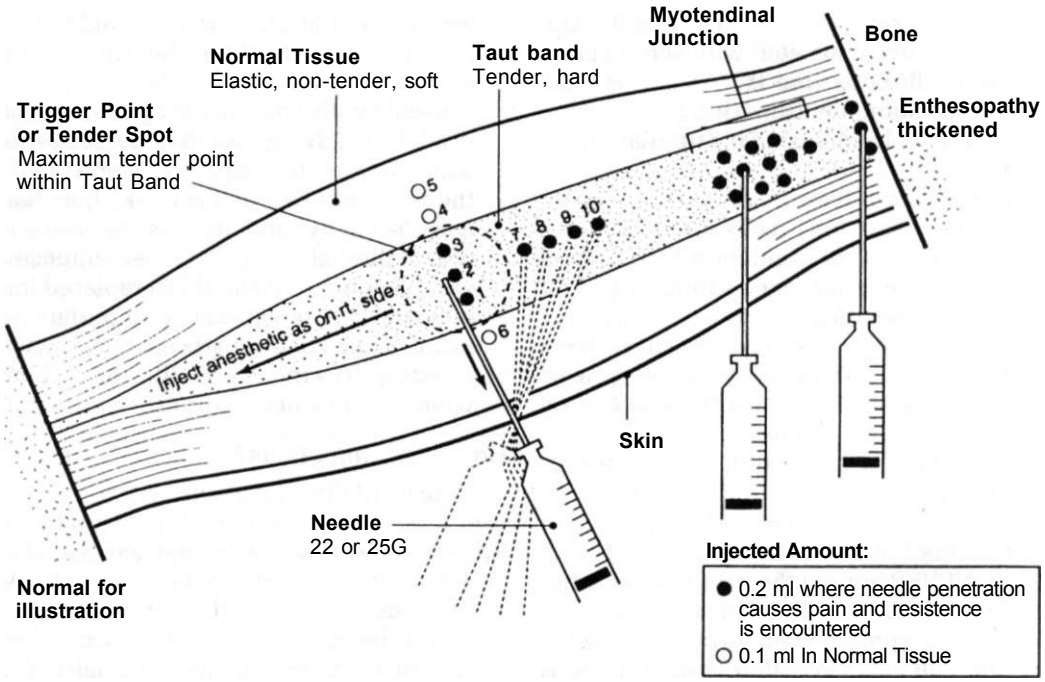


Figure 3.19. Diagrammatic representation of preinjection sites *(open circles),* and injection sites *(solid circles)* of local anesthetic in relation to the trigger point *(large broken circle).* The taut band is represented by the *enclosed stippled area.* This diagram distinguishes the central trigger point within the broken circle from the attachment TrPs located at the myotendinous junction and at the attachment of the tendon to the bone. Each of these three TrP regions can be identified by their individual spot tenderness and anatomical locations. No rationale is apparent for injecting the part of the taut band that lies between the central trigger point and the attachment TrP (solid circles numbers 7-10). (Reproduced with permission from Fischer AA. New approaches in treatment of myofascial pain: myofascial pain-update in diagnosis and treatment. *Phys Med Rehabil Clin North Am* 8(7J.i53-169, 1997.)

an LTR which is often associated with considerable pain, there is a limit as to how many painful injections should be performed at one visit out of respect to the patient's emotional and autonomic distress level.

The presence of unrecognized perpetuating factors *[see* Chapter 4) will lead to unnecessary injections. The presence of associated joint dysfunctions that need manipulation can cause poor response to injection and prompt recurrence of the TrP activity. After appropriate treatment of the joint(s) one or two more injections should resolve the problem. The presence of concurrent fibromyalgia will increase the number of injections required and can justify recurrent injections every 6-8 weeks since the fibromyalgia acts as a perpetuating factor that has no cure. Inactivating their TrPs can provide significant pain relief for many of these patients.

Ligamentous Sprains

The pain of ankle and wrist sprains has been reported to be relieved in most cases by injection with procaine, either with[127] or without[115, 161, 163] epinephrine. Either 0.5%[161] or 1%[115] procaine is effective. Best results are obtained if all of the tender spots in the sprained joint are injected as soon as possible (less than 12 hr) following injury. The joint should be pain free following injection, which should permit use of the joint at once, including some slow walking. It should be used gently throughout its normal range to remain free of pain, aided by an elastic support to remind the patient to protect the joint.

Postinjection Procedures

Stretch following TrP injection is an integral part of that treatment. Zohn and Mennell[193] emphasized that failure to stretch following injection can mean failure of treatment. Kraus[93] devoted the bulk of his therapeutic instructions to stretching and strengthening exercises that are to be done by the patient following injection of myofascial TrPs.

Immediately following injection (before the effect of injected anesthetic has worn off) the patient should actively move each muscle injected through its FULL range of motion 3 times, reaching its fully shortened and its fully lengthened position during each cycle. The muscle usually feels stiff toward the end of full stretch range of motion on the first cycle, less on the second, and begins to feel comfortable through its full range by the third cycle. It is important that the patient move the muscle slowly to explore the end range of motion for additional release.

The process is facilitated if the clinician applies a few sweeps of vapocoolant spray in parallel lines over each muscle and its referred pain pattern during its first stretch. Vapocoolant application should be followed by a hot pack or pad placed over the areas sprayed with vapocoolant. The moist heat also seems to help reduce postinjection soreness.

The postinjection stretch is important because it helps to again equalize sarcomere lengths throughout the length of affected muscle fibers, which relieves their abnormal tension and can eliminate the palpable taut bands. Voluntary movement also relieves residual stiffness at full range of motion, helps the patient to appreciate fully the improved range of motion, and provides the patient stretches that will be incorporated in the home program. In addition, this range of motion activity establishes the patient's conscious awareness of normal function in that muscle while reprogramming the cerebellum to incorporate the newly restored full-range capability of the muscle into the patient's daily activities.

Lewit[98] noted muscle soreness after dry needling and after a local anesthetic injection, but made no mention of applying heat as part of the treatment. The postinjection soreness, *per se,* is not unfavorable if the patient's related pattern of referred pain has been relieved. However, it is wise to let the muscle recover completely from postinjection soreness, which ordinarily lasts at most 3 or 4 days, before injecting its TrPs again. Soreness also can be caused by ineffectually needling close to, but not into, TrPs. For patients who are troubled by postinjection soreness, acetaminophen is usually as effective as aspirin and less irritating to the stomach. The practitioner should teach the patient a home exercise program that includes the postinjection stretches which the patient just performed.

If two or three treatments by injection fail to produce improvement of the TrPs in a muscle, repeated injections are rarely the answer. The perpetuating factors that are making the TrPs so irritable must be identified and managed.

Reasons for Failure of Injection of Trigger Points

1. Aside from a complete misdiagnosis, disregarding *perpetuating factors* is probably the most important reason for failure.
2. Injecting a latent TrP, not the responsible active TrP.
3. Injecting the area of referred pain and referred tenderness, not the TrP.[193] This error provides only incomplete, temporary relief.
4. Needling the vicinity of the TrP, including needling of the taut band, but missing the TrP itself.
5. Using a needle for precision injection that was finer than 25-gauge may allow the tip of the needle to be shoved aside by the dense contraction knots which are at the heart of the TrP and which must be disrupted for maximum effectiveness.
6. Injecting a solution with an irritating or allergenic bacteriostatic preservative, such as sodium bisulfite; sodium hyposulfite is less irritating.
7. Inadequate hemostasis followed by irritation of the TrP due to local bleeding.[193]
8. Overlooking other active TrPs that are contributing to the patient's pain.
9. Forgetting to have the patient perform active range of motion following injection with the result that the muscle's full range was not incorporated into daily activities.
10. Omitting regular passive stretch exercises at home, which would have maintained the full length of the muscle and suppressed further TrP activity.

14. CORRECTIVE ACTIONS

This section reviews what *the patient* must do or avoid, to achieve lasting recovery; Chapter 4 reviews the perpetuating factors that *the doctor and therapist* must identify and resolve. Also the practitioner is referred to Chapter 5, Section D for a general treatment approach for *chronic* headaches, facial, neck, or shoulder pain with a significant myofascial TrP component.

Patients must learn to respect their muscles. Muscles are designed to contract, relax, and be kept mobile through their full range of motion. They are *not* designed to be held for long periods in sustained contraction or in a fixed position, particularly not in the fully shortened position. Most patients need to apply some myofascial therapy at home, such as moist heat, stretch exercises, and TrP pressure release (as illustrated in Figure 3.20). Patients also need to practice good movement postures that prevent excessive muscle tension and stress *[see* Chapter 41, Section C).

Patient Compliance

Patients may fail to perform corrective actions effectively because of over-enthusiasm, misunderstanding, or lack of interest and motivation.

Over-enthusiasm. Some patients are hard-driving over-achievers who live by the philosophy that if one is good, two must be better, and three much better. They

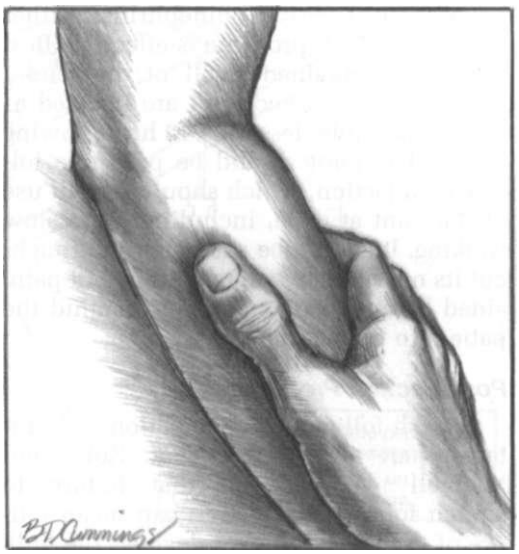


Figure 3.20. Technique for applying trigger point pressure release to trigger points in the right extensor carpi radialis brevis muscle. Pressure is gradually increased until the finger encounters a barrier of increased resistance. That pressure is maintained until some release occurs and the finger gently advances (follows the release of tissue tension) to the next barrier. This procedure should cause (at most) mild discomfort and not pain.

tend to be Spartan and are determined to be "good sports," performing activities regardless of exhaustion or pain, and they refuse to quit. These patients abuse their muscles, rather than use them within their normal limitations.

Misunderstanding. People routinely misunderstand verbal instructions, sometimes even when the instructions are written and handed to them (a valuable routine). By having the patient *demonstrate* the exercises on return to the office, exactly as performed at home, the clinician learns (1) what exercise the patient has actually been doing, (2) how the patient has been doing it, and (3) how much improvement in function has occurred, if any. The reason for lack of pain relief often is apparent when one sees how incorrectly the patient has been doing a stretch exercise. This also gives the examiner an opportunity to discuss with the patient the reason for each exercise, specifically what muscle, or muscles are involved, and an opportunity to strongly reinforce skillful, conscientious exercise performance. Many patients benefit by deciding what reward they will give themselves for doing their exercises faithfully each week.

When investigating what medications and nutritional supplements patients are taking, the clinician should draw a distinction between what they were *told* to take and what they *actually* took. Asking "When did you take your folic acid the last time?" or "When do you usually take it?" reveals whether the patient takes it regularly, or whether it is a hit-or-miss operation. Plastic pill boxes with seven separate compartments, each marked for one day of the week, conveniently help patients to take the correct medication each day and remind them when they forgot.

Lack of Interest and Motivation. Patients with chronic myofascial pain will not do well unless they understand that this is **their** muscle problem and that the clinician's essential role is to help them learn how to deal effectively with their muscles. Encouraging conscientious compliance with their home exercise program and teaching them to distinguish between muscle use and muscle abuse are integral parts of their medical management.

Frequently, patients have seen many doctors, have been given many different diagnoses, and have received many different treatments, none of which afforded significant relief. At first, they have no reason to believe that yet another physician will do any better. These patients need prompt tangible evidence that their pain originates in the muscles, not in their bones, nerves, or in the head, and that it responds to myofascial TrP therapy.

Many patients are justifiably frustrated with, and distrustful of, the medical profession's ability to identify a cause of their disabling pain that will respond to treatment. Medical pronouncements, based on X-rays, that the patients have "pinched nerves" or "arthritis of the spine," leads to the belief that this is the cause of their pain, a pain without hope of relief except through pain pills, and that their disabilities are permanent.

Patients with pending disability compensation are likely to be subconsciously ambivalent about losing their pain. One approach to this problem of ambivalence is first to give the patient an opportunity to reorient life toward function, not disability. One must take the time and effort to establish the myofascial basis of the pain and then to educate the patient in its nature and probable response to treatment. Recovery of *function* becomes the primary goal, with *guarded* promises as to prompt pain relief.

It is up to the patient to decide what he or she really wants to do: try for compensation or try for return to full function. In cases involving litigation, it is very helpful to call attention to the fact that a lawyer may be emphasizing the pain and disability, whereas the clinician is trying to relieve or minimize the patient's symptoms and disability. These two objectives are in conflict.

When treatment is successful, as the patient's myofascial TrPs are inactivated and the limitations imposed by learned pain behavior are replaced by normal function, the pain complaints also fade.[47] One must treat both the TrP sources of pain and chronic pain behavior, which would have been avoided if the myofascial causes had been recognized initially and treated promptly and properly.

Appropriate Activities

After a treatment session, the patient must understand what kind and dose of activity are appropriate, and must eliminate

or modify habitual movements that are perpetuating TrPs.

Posttreatment Activity. Strenuous activities should be avoided for at least the 2 or 3-day period of muscle soreness, and preferably for about 1 week. That includes avoidance of tennis playing, serious gardening, moving furniture, and traveling to conventions. On the other hand, patients are encouraged to use their muscles in a gentle, normal way through their *full* range of motion. They must avoid placing their muscles in a fixed, shortened position for a prolonged length of time.

The patient should learn ways of moving that will avoid loading recently injected muscles. For example, in getting out of bed, the patient who has received an injection in one sternocleidomastoid muscle can turn so that the uninjected opposite sternocleidomastoid and other muscles hold the weight of the head.

Perpetuating Movements. When the patient's TrPs are extremely hyperirritable, the muscles can be *overwhelmed* by the TrP activity and generate pain nearly all of the time, even at rest; almost any activity makes them worse. However, as the muscles improve, some activity is well tolerated. Then, when the patient does the *wrong* thing and pain recurs, an awareness develops as to which activities are now tolerated and which cause pain.[172] This is the *discriminating* phase when the patient can recognize overstress of the muscles and how to avoid it. Any activity that produces pain for more than a few seconds after the effort should be avoided.[122] As all remaining TrPs are inactivated, full *recovery* occurs and the patient can do the *normal* things that were done before the pain developed, but not more; he or she never could lift a piano!

In this discriminating stage, the clinician helps the patient decide which aggravating activities are unnecessary and must be eliminated (for example, lifting a paperweight 50 times a day to test whether it still hurts), *versus* those which are essential; the latter must be modified so that they are done without damaging stress. The patient learns how to become fully functional within the limits of the muscles.

Patients should learn a few basic rules. NEVER bend over and lift, or pull something, with the back twisted. ALWAYS lift by extending the knees and hips, holding the back in an erect-forward-facing position. Similarly, NEVER get up from, or sit down in, a chair while leaning forward in the stooped position with the trunk rotated; that is "asking" for low back strain.

To recognize the pain-perpetuating activities, the patient should first be alerted as to what kinds of movements are likely to abuse the affected muscles and reactivate their TrPs. The patient is asked to report at the next visit any activity that caused referred pain from the stressed muscles, and to note any habitual repetitive movement that would overload those muscles.

When the offending movements are unnecessary, it is a matter of the patient's unlearning bad habits. When the activity is a necessary one, such as turning the doorknob to open a door, then at least one satisfactory alternate method of performing the activity must be developed (e.g., use the other hand, or rotate the shoulder rather than the forearm, or eliminate the cause of the problem and lubricate the door latch mechanism).

Some people characteristically make rapid, jerky, movements. These movements are poorly coordinated and are likely to initiate additional reflex responses of muscles and unnecessary stress. Slower, smoother, better coordinated movements can be learned (using appropriate exercises and equipment) and should become habitual. Surface EMG biofeedback can be helpful in reaching this goal.

For patients with acute scalene, serratus anterior or quadratus lumborum TrPs (muscles with rib attachments), sneezing or coughing can be exquisitely painful and aggravating to the TrPs. The sneeze may be inhibited by promptly biting *high* on the upper lip or by firmly squeezing the upper lip or nostrils to induce distracting pain in the nose area. These painful anti-sneeze stimuli are effective only if started early enough in the sneeze. Otherwise, the patient can learn to keep the glottis open during the sneeze to minimize increased intrathoracic pressure and the overload which the closed glottis imposes on the accessory respiratory muscles.

Students, or other readers, ordinarily place a book on a flat surface or on the lap, bending the head and neck forward to read. This requires that the posterior neck mus-

cles maintain *sustained contraction* in order to checkrein the heavy weight of the head against gravity. The ensuing neck strain can be avoided by placing the book on a book rack, or by propping it up at eye level. Thus tilted at a convenient angle, the book can be read easily when the head is held erect and balanced, without neck strain.

Activity Goals

Not only is WHAT to do important, but also HOW to do it. On performing a task, the patient MUST learn to keep the muscles mobilized, and not held fixed in a contracted position. Muscle fibers need to alternately contract and relax to provide blood flow and replenish their energy supply. Using the motor unit training technique of Basmajian,[9] normally, even a type I motor unit will not sustain a minimal contraction indefinitely, but will drop out and be replaced by another motor unit. However, during strong muscular contraction when most of the motor units have been recruited and are sustaining moderately high firing rates, the brief periods of rest provided by the alternation of motor units become inadequate for energy replenishment.

Strenuousness of Effort. The patient must avoid using the muscles at maximum effort, when they are most likely to be strained. Lifting, pulling or pushing something should use less than maximum strength, always leaving some reserve, especially in the case of muscles susceptible to TrPs.

Chronic overload of anterior and lateral neck muscles due to paradoxical breathing must be corrected by learning to synchronize contraction of the diaphragm with contraction of the intercostal muscles (coordinated abdominal and chest breathing) *(see* Section 14 in Chapters 20 and 45).

Mobility. Lying still in bed with muscles in a shortened position aggravates TrP activity; being up and doing nonstrenuous activities help to mobilize the muscles and reduce TrP activity. A mobilizing and relaxing activity is rocking in a physiologically well-designed rocking chair. To avoid becoming stiff and restricted in range of motion, muscles need to be extended to their full stretch range of motion every day. The muscle performs better if activities stretch it while *lightly* loaded. The patient should learn to move the part frequently throughout the day in ways that provide gentle, complete stretching of all the muscles and particularly TrP-prone muscles. The forcefulness of *stretching should always be within the limits of pain, and should never produce a lasting ache after the stretch.*

Travell[168,169] described the application of these principles to housework.

1. Vary your task each day so as not to overuse any one group of muscles in repetitive work, like ironing for hours at a stretch. Especially, don't combine too many jobs that involve standing and stooping as they place a heavy load on the low back muscles. To achieve variety of movement, you will have to scramble your housework.
2. Slow your working pace to a speed no faster than your muscles will tolerate. Fatigue of any one of your muscles is a warning signal—learn to pay attention.
3. Cultivate a rhythm of movement. It discourages the prolonged fixed contraction that tires a muscle, and it encourages pauses during which the working muscles fill up with new blood and fresh fuel. Do your housework as if you were dancing—music helps.
4. Take short rests frequently. After every hour or less of housework, lie down to rest for a couple of minutes, if only on the floor. The anti-gravity muscles of the neck and back that hold you erect do not relax fully unless your body is supported in a nearly horizontal position.
5. Don't sit too long in one position. When watching TV, or at a movie, or in the theater, move around in your seat. At intervals, turn your head from side-to-side and rotate your shoulder blades. When you drive far, pull off the road every hour and walk around your car two or three times. That doesn't take long. At home, you can sit in a rocker. This constantly changing position prevents resting muscles (electrically silent) from building up the tension (electrical activity) that inevitably occurs when you stay motionless for several minutes or up to one-half an hour, as electromyographic studies have shown.[107]

6. Don't try to lift a heavy piece of furniture by yourself, or to carry large awkward things that extend the leverage length of your arm. The extra leverage multiplies the weight transmitted to your low back muscles. Keep the load close to your body, and just before you lift it, raise your head smartly and look up. That tightens the long spinal muscle and prepares your back for the load.

Relaxation. For relaxation in the sitting position, a well designed chair with adequate lumbar support and the correct height armrests is necessary *(see* Chapter 41, Part C).

When standing or walking, the patient should focus on the floor under the feet, trying to feel the texture and hardness of the rug, linoleum, or concrete with each step. This helps to relieve unnecessary muscle tension. When resting, sensation should be concentrated on the bed that supports the body, on the texture of the sheets, and on the shape of the supporting surface. This concentration on the underlying *support* beneath the body encourages relaxation.

Muscles relax more fully immediately following a gentle contraction. When lying down and trying to relax, one can feel the difference in muscle tension before and after the contraction of individual muscle groups. Relaxation is an active process that requires intense concentration. This concentration on relaxation helps to clear the mind for sleep.

Training in biofeedback and mind management, such as meditation, can help people learn how to relax their muscles.

Between cycles of an exercise, a pause for several deep breaths greatly aids muscular relaxation, permits time for return of circulation, and trains the patient how to reduce chronic tension of the muscles.

Application of Heat

Allowing the body to become chilled, as by a cold draft across the shoulders, invites activation of TrPs. A sweater in the home by day and an electric blanket at night can make the differences between comfort and pain.

Cold applied to the skin penetrates quickly due to progressive vasoconstriction. Surface heat does not penetrate; the excess heat is quickly carried away by the increased blood flow due to vasodilatation. Prolonged cold over a TrP tends to activate it, but prolonged cold applied over the pain reference zone may relieve discomfort by partially anesthetizing sensory nerve function locally.

Whenever the muscles become chilled, especially after exercise, a warm shower or bath warms and relaxes them. Professional athletes do not wait long after playing competitive sports, but run immediately for the hot shower.

Heat that the patient applies to sore muscles is usually more soothing if it is moist rather than dry. Either a wet pack soaked in hot water, or a wet-proof heating pad covered with a damp cloth, serves the purpose. The moist heat is applied over the TrP, if its location is known. If a patient with pain asks where to place the moist heat, it is advisable to try several different regions, for approximately 5 min each, to see which location affords the greatest relief. Daily application of moist heat to active TrPs can progressively quiet them.

Posture and Positioning

Activity Posture. Good posture avoids sustained contraction or prolonged shortening of muscles. Strain of the upper trapezius is lessened by providing armrests that properly support the elbows. These are needed when sitting, reading, telephoning, and driving or riding in a car. Placing the work level low enough so that the shoulders need not be raised to reach it (e.g., a keyboard) also is important.

Correct Standing and Sitting Posture. The criteria of good posture, and the techniques for achieving it, are presented in Chapter 41, Part C.

Reading Position. Tilting the plane of reading glasses, so that the lower rim is against the cheek, allows the patient to read by turning the eyes downward, rather than by bending the neck forward, as is described in Section 7 of Chapter 16 *(see* Fig. 16.4).

When reading, the light should be placed so that the book is well illuminated when it is held straight in front of the reader without the reader having to turn the head. For reading in bed, an overhead light that clips onto the bed, or is mounted on the wall or ceiling, is recommended *(see* Fig. 7.3A).

Sleeping Position. Muscles should rest in a neutral or slightly stretched position at night and NEVER be kept in the *fully* shortened position. Sleeping with the calf mus-

cles in the shortened position encourages night cramps.

The shoulders should not be allowed to creep up toward the ears when one is lying in bed. Correct positioning is helped by tucking the corners of the pillow between the chin and shoulder on each side. When lying on the side, the patient should pull the corner of the pillow around between the lowermost shoulder and chin, to avoid shortening the front-of-the-neck muscles and to support the mandible. The patient should use only one comfortable pillow under the head (not under the shoulders) to keep the head and neck in a neutral position, when lying on the back. Tilting the entire bed frame by elevating the head end of the bed with 3.5- to 4-inch blocks under its legs creates helpful gentle traction on the neck, elongating the scalene and sternocleidomastoid muscles.

As a rule, lying on the side is most comfortable; lying on the abdomen with the head and neck extended and twisted to the side is the position most aggravating to neck TrPs. Many people with the scalene TrP syndrome prefer to lie on the affected side, but the shoulder-girdle muscles must be tolerant of the sustained pressure of body weight.

The pillow should be filled with a nonspringy material, such as feathers or shredded Dacron; foam rubber should be discarded. Special pillows designed to maintain the head in a normal alignment with the body, retaining a moderate cervical lordosis, are the Cervipillo designed by Ruth Jackson, MD,[78] the Wal-Pil-O designed by Lionel Walpin, MD,[182] and others.

The elbows and wrists should not be held sharply flexed at night. A pillow in the axilla, between the arm and chest wall, prevents painful shortening of the muscles in TrP syndromes that can occur in the subscapularis, pectoralis major, latissimus dorsi, triceps brachii, infraspinatus, and teres major and minor muscles. A pillow at the feet lengthens the gastrocnemius/soleus calf muscles and prevents sustained plantar flexion.

Exercises

An exercise should be designed primarily for lengthening, strengthening, or conditioning specific muscles. Exercise to lengthen the involved muscles is the key to sustained relief of myofascial pain. Improved conditioning (exercise tolerance or stamina) and increased strength of a group of muscles, achieved through exercise, reduces the likelihood of their developing TrPs. However, in most patients with *active* TrPs, conditioning and strengthening exercises can further activate the TrPs, encourage substitution by other muscles, and aggravate symptoms. On the other hand, these exercises render latent TrPs less prone to reactivation if properly paced at a gradual rate of progression.

The kind of exercise prescribed depends largely on the irritability of the TrPs responsible for the pain. When the patient is experiencing **rest pain** for a considerable part of the time, the TrPs are very active and rarely respond favorably to anything more than gentle release and moist heat. At this stage, movement in warm water with rhythmic and gentle, active or passive stretching is very helpful. The object is to unload and restore normal range of motion to the overworked sore muscles; at that stage, active exercise that loads a contracting muscle is not indicated.

Exercise should be regarded as a prescription, much as one prescribes medication. Like a drug, there is a right kind, dose, and timing of exercise. The exact exercise to be performed should be demonstrated and explained to the patient, who then *does* a return demonstration to confirm understanding of the instructions. The rate, number of repetitions, frequency in one day, and the conditions under which it should be done (e.g., not when the muscles are tired or cold) should be specified. For any repetitive exercise, whether stretch or strengthening, a pause to relax and breathe should be interposed between each cycle of the exercise. The number of counts (time) during the pause should equal the number required to perform the movement.

As the TrPs are inactivated, and rest pain fades, a *carefully graded* exercise program is needed to improve conditioning and endurance. The program should start with ***lengthening,*** not shortening exercises.

Patients should avoid activities that produce repetitive muscular loads, such as shoveling snow, raking leaves, vacuum cleaning, painting a wall, or unloading a dishwasher. If such tasks must be performed, then the movements should be varied and sides of the body alternated so that contralateral muscles are used in turn.

The number of repetitions of the movement should not exceed 6 or 7 times, with pauses to allow the muscle to rest.

Stretch Exercise. In this manual, muscle lengthening exercises are described and illustrated in detail, because they are so important to the recovery from dysfunction and pain due to active TrPs, and because they are often the only kind of exercise tolerated by hyperirritable TrPs. A daily home program of passive stretch exercise that achieves FULL range of motion of the affected muscles helps to insure continued relief. It is important that the patient use an objective measure of the full range of motion, so that any gradual loss is recognized.

Stretching exercises should be started on a daily schedule, and variety of movement should be emphasized. These stretch exercises can include postisometric relaxation and other augmentation techniques to enhance their effectiveness. An exercise that increases referred pain during or after its performance should be reduced or stopped.

Postisometric Relaxation. Postisometric relaxation (PIR)[101] and combinations of it with reciprocal inhibition are the preferred approach to home exercises. Gravity is preferred to take up slack as it develops, or contraction of opposing muscles can help release the TrPs and take up slack. These exercises may be slightly uncomfortable as the patient feels the muscle being released, but should NOT be painful. Some Spartan individuals assume that "the more pain, the better" and thus aggravate their TrPs, rather than inactivate them.

People who are prone to develop TrPs do well to emulate the cat, which rarely tries to walk after sleeping without first stretching its limb muscles. Such active stretching should be accomplished slowly, with a smooth, sustained cat-like effort that avoids any jerking movements.

An exercise that involves rolling the head around in all directions at full range of motion is NOT recommended. Sudden overload of a tight shortened muscle can activate TrPs.

Strengthening Exercise. To strengthen a muscle, one needs to hold a *maximal* contraction for only 5 or 10 seconds, once a day. Strengthening exercises may be isotonic or isometric. During isotonic exercise, the muscle moves against a uniform force. During isometric exercise, the muscle exerts a variable force in a fixed position. When dealing with muscles that contain myofascial TrPs, the movement associated with an isotonic exercise is preferable to the fixed position of the isometric exercise.

A muscle has significantly greater strength and efficiency during a lengthening contraction than during a shortening contraction. A muscle usually shortens as it works; it contracts and becomes shorter. Lengthening contraction occurs when the muscle is overpowered by a greater force than it is producing; its force of contraction controls (resists) the lengthening (for example, the quadriceps muscle when walking down a mountain). A "sit up" (Fig. 49.13C) requires a shortening contraction of the abdominal muscles; a "sit back" (Fig. 49.13A) involves a lengthening contraction of the same abdominal muscles. A lengthening contraction exerts more force with less energy than does a shortening contraction. It is safer for the patient, initially, to do unloaded exercises that lengthen, rather than shorten the muscle. The muscle is required to do less work, and lengthening contractions may help to equalize the length of sarcomeres in muscle fibers.

Hill[65] constructed a special bicycle ergometer on which two subjects did exactly the same amount of work in opposite directions. At high pedaling speeds the oxygen consumption of the subject who performed shortening contractions was 6 *times* that of the subject doing lengthening contractions, which agreed with their subjective impressions of the relative effort required.

An example of a lengthening contraction exercise for the biceps brachii and brachialis muscles would be a "chin down," (which is a "chin up" in reverse). Instead of pulling the body up to bring the chin to the bar, as in a chin up, the patient steps up on a box and lets the arms control the rate at which the body and chin drop down away from the bar. A quadriceps lengthening exercise would be a "step down" when going downstairs, as compared to the shortening contraction of a "step up" when going upstairs.

When the patient can do 10 lengthening contractions easily, it is time to replace this exercise with one shortening contraction, which is gradually increased in number on subsequent days. With this approach, the patient is less likely to overload and over-

stress a weak or tired muscle that harbors TrPs. It restores normal muscle function more quickly than a program of shortening contractions.

If an exercise causes pain that lasts after the exercise, it should be reduced or postponed. When mild muscular soreness disappears after the first day, the exercise can be repeated on the second day. If soreness lasts into the second day after exercise, the next exercise session should be postponed until the third day and the amount of exercise reduced. If the muscles are still sore on the third day, the exercise should be changed. Also, if the patient on a home program calls to complain of annoying (but not incapacitating) muscle soreness due to exercise or overenthusiastic activity, he or she should be told that with limited activity and moist heat, the postexercise soreness and stiffness should not last longer than 72 hr.

Conditioning Exercise. To condition both the cardiovascular system and a particular set of muscles, the exercise is continued at submaximal strength to the point of fatigue. Swimming, bicycling, tennis, treadmill, jogging, and jumping rope are examples of conditioning exercises. Although not essential for recovery from myofascial TrPs, a *regular* conditioning exercise program at least twice a week, or preferably every other day, is strongly recommended for optimal health and to minimize the chance of reactivating TrPs.

When a warm pool is available, swimming provides excellent exercise for many muscles and poses a minimal hazard of strain. Bicycling is less traumatic than jogging. The least traumatic bicycle exercise is on a machine that the patient pedals from behind, while lying supported in the semirecumbent position. When riding an upright stationary bicycle, the patient should not hold the handlebars, but should sit up straight and swing the arms from time to time. The trunk-forward, head-up position of bicycling, which is all too common, severely overloads the posterior cervical muscles. Whatever exercise is undertaken, the first efforts should remain *well* within tolerance, by underestimating rather than overestimating how much can be done at the start. On an exercise bicycle or treadmill, an increment is added *gradually,* to only one factor at a time—either duration, rate (speed), or load (angle of belt). Overexercising when one is out of condition can be severely counterproductive; when jogging, one can take a route that allows a shortcut home if needed in order to avoid overexercising.

REFERENCES

1. Abeles M, Garjian P: Do spray coolant anesthetics contaminate an aseptic field? [Letter]. *Arth Rheum* 29:576, 1986.
2. Acquadro MA, Borodic GE: Treatment of myofascial pain with botulinum A toxin [letter]. *Anesthesiology 80(31:705-706,* 1994.
3. Agnew LR, Aviado DM, Brody JI, *et al.: Dorland's Illustrated Medical Dictionary.* Ed. 24. W.B. Saunders, Philadelphia, 1965.
4. Baker B: Personal Communication, 1981.
5. Baker DM: Changes in the corium and subcutaneous tissues as a cause of rheumatic pain. *Ann Rheum Dis* 14:385-391, 1955.
6. Bardeen CR: The musculature, Sect. 5. In: *Morris's Human Anatomy.* Ed 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921.
7. Bargmann W, Batrawi AM, Beau A, *et al: Nomina Anatomica.* Excerpta Medica Foundation, Amsterdam, 1966.
8. Barnes J: *Myofascial Release: the Search for Excellence.* Self-published, 1990.
9. Basmajian JV: *Muscles Alive.* Ed. 4. Williams & Wilkins, Baltimore, 1978 (pp.103-114, 115-129).
10. Bates T, Grunwaldt E: Myofascial pain in childhood. *JPediatr* 53:198-209, 1958.
11. Beard G, Wood EC: *Massage: Principles and Techniques.* W.B. Saunders, Philadelphia, 1964 (pp. 38-45, 51).
12. Bell WH: Nonsurgical management of the pain-dysfunction syndrome. / *Am Dent Assoc* 79:161-170, 1969.
13. Benoit PW: Effects of local anesthetics on skeletal muscle. *AnatRec* 169:276-277, 1971.
14. Benoit PW: Reversible skeletal muscle damage after administration of local anesthetics with and without epinephrine. *J Oral Surg* 36:198-201,1978.
15. Benoit PW: Microscarring in skeletal muscle after repeated exposures to lidocaine with epinephrine. / *Oral Surg* 36:530-533, 1978.
16. Benoit PW, Belt WD: Some effects of local anesthetic agents on skeletal muscle. *Exp Neurol* 34:264-278, 1972.
17. Berges PU: Myofascial pain syndromes. *Postgrad* Med 53:161-168, 1973.
18. Boeve M: Personal communication, 1990.
19. Bogduk N, Simons DC: Neck pain: joint pain or trigger points? Chapter. 20. In: *Progress in Fibromyalgia and Myofascial Pain,* Vol. 6 of *Pain research and Clinical Management.* Edited by Vaerey H, Mersky H. Elsevier, Amsterdam, 1993 (pp. 267-273).
20. Bonica JJ: Management of myofascial pain syndromes in general practice. *JAMA* 164:732- 738, 1957.
21. Boos R. Pannikulose und Pannikulitis. In: *Fortbildungskurse fiir Rheumatologie, Der Weichteilrheumatismus.* Edited by Kaganas G, Miiller W,

Wagenhauser F, Vol. 1. S. Karger, Basel, 1971 (pp. 35-48).
22. Brand PW, Beach RB, Thompson DE: Relative tension and potential excursion of muscles in the forearm and hand. / *Hand Surg* 6:209-219, 1981.
23. Brin MF (Ed.): Spasticity: etiology, evaluation, management, and the role of Botulinum toxin type A. *Muscle & Nerve* Suppl. 6, 1997.
24. Brun A: Effect of procaine, carbocaine and xylocaine on cutaneous muscle in rabbits and mice. *Acta Anaesthesiol Scand* 3:59-73, 1959.
25. Burke GW, Jr., Fedison JR, Jones CR: Muscle degeneration produced by local anesthetics. *Va Dent* J 49:33-37, 1972.
26. Byl NN, McKenzie A, Halliday B, *et al.:* The effects of phonophoresis with corticosteroids: a controlled pilot study. / *Orthop Sports Phys Ther* 38(5j:590-600, 1993.
27. Cardenas DD, Stolov WC, Hardy MS: Muscle fiber number in immobilization atrophy. *Arch Phys Med Rehabil* 58:423-426, 1977.
28. Chan P: *Finger Acupressure.* Ballantine Books, New York, 1975.
29. Chemick WS: Local anesthetics. Chapter 11. In: *Drill's Pharmacology in Medicine.* Ed. 4. Edited by DiPalma JR. McGraw-Hill, New York, 1971 (pp. 190-193, 196-199).
30. Cheshire WP, Abashian SW, Mann JD: Botulinum toxin in the treatment of myofascial pain syndrome. *Pain* 59:65-69, 1994.
31. Chu J: Dry needling (intramuscular stimulation) in myofascial pain related to lumbosacral radiculopathy. *Eur J Phys Med Rehabil* 5(4):106-121, 1995.
32. Chu J: Twitch-obtaining intramuscular stimulation: its effectiveness in the long-term treatment of myofascial pain related to lumbosacral radiculopathy. *Arch Phys Med Rehabil* 78:1024, 1997 (Abstr).
33. Clemente CD: *Gray's Anatomy of the Human Rody,* American Ed. 30. Lea & Febiger, Philadelphia, 1985.
34. Consumer Reports: Caffeine: how to consume less. *Consumer Reports* 597-599, October, 1981.
35. Copeman WS, Ackerman WL: "Fibrositis" of the back. *QJMed* 13:37-51, 1944.
36. Covino BG: Local anesthesia (Parts One and Two). *NEngl J Med* 286:975-983, and 1035- 1042, 1972.
37. Cyriax JH: Clinical applications of massage, Chapter 7. In: *Manipulation, Traction and Massage.* Ed. 2. Edited by Rogoff JB. Williams & Wilkins, Baltimore, 1980 (pp.152-155).
38. Dalessio DJ: *Wolff's Headache and Other Head Pain.* Ed. 3. Oxford University Press, New York, 1972 (p. 553).
39. D'Ambrogio KJ, Roth GB: *Positional Release Therapy.* Mosby, St. Louis, 1997.
40. Danneskiold-Samsoe B, Christiansen E, Andersen RB: Regional muscle tension and pain ("Fibrositis"). *Scand J Rehab Med* 15:17-20, 1983.
41. Danneskiold-Sams0e B, Christiansen E, Andersen RB: Myofascial pain and the role of myoglobin. *Scand J Rheumatol* 15:174-178, 1986.
42. Day BH, Govindasamy N, Patnaik R: Corticosteroid injections in the treatment of tennis elbow. *Practitioner* 220:459-462, 1978.
43. Eisler P: Die Muskeln des Stammes. Gustav Fischer, Jena, 1912.
44. Fassbender HG: Non-articular rheumatism. Chapter 13. In: *Pathology of Rheumatic Diseases.* Springer-Verlag, New York, 1975 (pp. 303-314).
45. Fields HL (Editor). *Core Curriculum for Professional Education of the International Association for the Study of Pain.* IASP Press, Seattle, 1995.
46. Fischer AA: New approaches in treatment of myofascial pain. In: *Myofascial Pain-Update in Diagnosis and Treatment.* Edited by Fischer AA. *Phys Med Rehabil Clin North Am* 8f2j:153-169, 1997.
47. Fordyce WE: *Behavioral Methods for Chronic Pain and Illness.* C.V. Mosby, St. Louis, 1976.
48. Frank, E: Personal Communication, 1997.
49. Frost A: Diclofenac versus lidocaine as injection therapy in myofascial pain. *Scand J Rheumatol* 15:153-156, 1986.
50. Frost FA, Jessen B, Siggaard-Anderson J: A control, double-blind comparison of mepivacaine injection versus saline injection for myofascial pain. *Lancet* 1:499-501, 1980.
51. Gardner DA: The use of ethyl chloride spray to relieve somatic pain. *J AO A* 49:525-528, 1950.
52. Gerwin RD, Shannon S, Hong CZ, *et al.:* Interrater reliability in myofascial trigger point examination. Fain 69:65-73, 1997.
53. Glover JC, Yates HA: Strain and counterstrain, Chap. 58. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams and Wilkins, Baltimore, 1997 (pp.809-818).
54. Gold H, Travell J: Cornell conference on therapy: management of pain due to muscle spasm. *NY State J Med* 45:2085-2097, 1945 (pp. 2095-2096).
55. Good MG: Acroparaesthesia—an idiopathic myalgia of elbow. *Edinburgh Med J* 56:366- 368, 1949.
56. Goodman LS, Gilman A: *The Pharmacological Basis of Therapeutics.* Ed. 4. Macmillan, London, 1970 (pp. 372-376, 382, 1662-1663).
57. Goodridge JP: Muscle energy technique procedures, Chapter 53. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams and Wilkins, Baltimore, 1997 (pp.691-696).
58. Gottlieb NL, Riskin WG: Complications of local corticosteroid injections. *JAMA* 243:1547-1548,1980.
59. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 39-49, 99-103, 539).
60. Gunn C. Chan: Personal Communication, 1995.
61. Gunn CC: *The Gunn Approach to the Treatment of Chronic Pain-Intramuscular Stimulation for Myofascial Pain of Radiculopathic Origin.* Churchill Livingstone, London, 1996.
62. Gunn CC, Milbrandt WE: Early and subtle signs in low-back sprain. Spine 3:267-281, 1978.
63. Hagbarth KE, Finer B: The plasticity of human withdrawal reflexes to noxious skin stimuli in lower limbs. *Prog Brain Res* 1:65-78, 1963.
64. Headley BJ: EMG and myofascial pain. *Clinical Manage* 10:43-46, 1990.
65. Hill AV: The mechanics of voluntary muscle. *Lancet* 2:947-951, 1951.
66. Hong CZ: Myofascial trigger point injection. *Crit Rev Phys Med Rehabil* 5:203-217, 1993.
67. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Rehabil* 73:256-263, 1994.

68. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. / *Musculoske Pain* 2f2j:29-59, 1994.
69. Hong CZ: Persistence of local twitch response with loss of conduction to and from the spinal cord. *Arch Phys Med Rehabil* 75.12-16, 1994.
70. Hong CZ: Personal Communication, 1998.
71. Hong CZ, Chen YN, Twehous D, *et al.:* Pressure threshold for referred pain by compression on the trigger point and adjacent areas. *J Musculoske Pain 4(3)61-79,* 1996.
72. Hong CZ, Hsueh TC: Difference in pain relief after trigger point injections in myofascial pain patients with and without fibromyalgia. *Arch Phys Med Rehabil 77(11'*.1161-1166, 1996.
73. Hong CZ, Simons DC: Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. *J Musculoske Pain 1(1):89-131,* 1993.
74. Hong CZ, Torigoe Y: Electrophysiological characteristics of localized twitch responses in responsive taut bands of rabbit skeletal muscle. / *Musculoske Pain 2(2):17-43,* 1994.
75. Hong CZ, Torigoe Y, Yu J: The localized twitch responses in responsive taut bands of rabbit skeletal muscle fibers are related to the reflexes at spinal cord level. *J Musculoske Pain 3(l):15-34,* 1995.
76. Hubbard D: Personal Communication, 1994.
77. Irwin Y, Wagenvoord J: *Shiatzu.* J.B. Lippincott, Philadelphia, 1976.
78. Jackson R: *The Cervical Syndrome.* Ed. 4. Charles C. Thomas, Springfield, 111., 1977 (pp. 310, 311).
79. Jacobs AW, Falls WM: Anatomy, Chapter 3. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997, pp. 27-43 (see p. 35).
80. Jaeger B: Differential diagnosis and management of craniofacial pain, Chapter 11. In: *Endodontics.* Ed. 4. Edited by Ingle JI, Bakland LK. Williams & Wilkins, Baltimore, 1994 (pp. 550-607).
81. Jaeger B, Reeves JL: Quantification of changes in myofascial trigger point sensitivity with the pressure algometer following passive stretch. *Pain* 27:203-210, 1986.
82. Jaeger B, Skootsky SA: Double blind, controlled study of different myofascial trigger point injection techniques [Abstract]. Pain 4(Suppl):S292, 1987.
83. Janssens LA: Trigger points in 48 dogs with myofascial pain syndromes. *Vet Surg* 20:274-278,1991.
84. Janssens LA: Trigger point therapy. *Probl Vet Med* 4:117-124, 1992.
85. Jones LH: *Strain and Counterstrain.* The American Academy of Osteopathy, Colorado Springs, 1981.
86. Kahn J: Electrical modalities in the treatment of myofascial conditions, Chapter 15. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 473-485.
87. Kahn J: Phonophoresis technique questioned. *Physical Therapy* 76(i2j.l348-1349, 1996. [Letter]
88. Kamen LB, Miller LT: Complications of trigger point steroid injections and therapeutic ultrasound. *Arch Phys Med Rehabil 69:778,*1988. [Abstract]
89. Kelly M: New light on the painful shoulder. *Med J Aust* 1:488-493, 1942 (Case 2, p. 489).
90. Knott M, Voss DE: *Proprioceptive Neuromuscular Facilitation.* Ed. 2. Harper & Row, New York, 1968 (pp. 97-99).
91. Kraft GH, Johnson EW, LeBan MM. The fibrositis syndrome. *Arch Phys Med Rehabil* 49:155-162. 1968.
92. Kraus H: The use of surface anesthesia in the treatment of painful motion. *JAMA* 16:2582- 2583,1941.
93. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970.
94. Kuchera WA, Kuchera ML: *Osteopathic Principles in Practice.* Second Edition Revised, Greyden Press, Columbus, 1994.
95. Kugelberg E, Hagbarth KE: Spinal mechanism of the abdominal and erector spinae skin reflexes. *Brain* 81:290-304, 1958.
96. Laskin DM: Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 79:147-153, 1969.
97. Lewis C, Gevirtz R, Hubbard D, *et al.:* Needle trigger point and surface frontal EMG measurements of psychophysiological responses in tension-type headache patients. *Biofeedback Self Reg* 19(3): 274-275, 1994.
98. Lewit K: The needle effect in the relief of myofascial pain. Fain 6:83-90, 1979.
99. Lewit K: Muskelfazilitations- und Inhibitionstechniken in der Manuellen Medizin. Teil II. Postisometrische Muskelrelaxation. *Manuelle Med* 19:12-22, 1981.
100. Lewit K: Postisometric relaxation in combination with other methods of muscular facilitation and inhibition. *Manual Med* 2:101-104, 1986.
101. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 11, 81, 186).
102. Lewit K: Personal Communication, 1996.
103. Lewit K, Berger M, Holzmiiller G, *et al.:* Breathing movements: the synkinesis of respiration with looking up and down. *J Musculoske Pain* 5(4):57-69, 1997.
104. Liberson WT: Personal communication, 1979.
105. Lieber RL: *Skeletal Muscle Structure and Function.* Williams & Wilkins, Baltimore, 1992 (see pp. 42-45).
106. Lloyd DP: Integrative pattern of excitation and inhibition in two-neuron reflex arcs. / *Neurophysiol* 9:421-438, 1946.
107. Lundervold AJ: Electromyographic investigations during sedentary work, especially typewriting. *Br J Phys Med* 14:32-36, 1951.
108. Lynn, Paulette: Personal Communication, 1993.
109. Macdonald AJ: Abnormally tender muscle regions and associated painful movements. *Pain* 8:197-205, 1980.
110. Magnusson SP, Simonsen EB, Aagaard P, *et al.:*Mechanical and physiological responses to stretching with and without preisometric contraction in human skeletal muscle. *Arch Phys Med Rehabil* 77:373-378, 1996.
111. Maigne R: Low back pain of thoracolumbar origin. *Arch Phys Med Rehabil* 61:389-395, 1980.
112. Maloney, M: Personal Communication, 1996.
113. Margoles MS: Letter to the editor. Fain 8:115-117, 1980.
114. McKeag PW: Fibrositis and panniculitis. *Br J Phys Med* 8:107-109, 1933.
115. McLaughlin CW Jr: Procaine infiltration in treatment of acute sprains. *Milit* Surg 97:457- 460,1945.
116. McNulty WH, Gevirtz RN, Hubbard DR, *et al.:* Needle electromyographic evaluation of trigger point response to a psychological stressor. *Psychophysiology* 31(3):313-316, 1994.

117. Melzack R: The McGill pain questionnaire: major properties and scoring methods. ***Pain*** 1:277-299, 1975.
118. Mennell JM: ***Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.*** Little, Brown and Company, Boston, 1964 ***(see*** pp. 3-5).
119. Mennell J: Spray-stretch for relief of pain from muscle spasm and myofascial trigger points. ***J Am Podiatry Assoc*** 66:873-876, 1976.
120. Mense S: Referral of muscle pain: new aspects. ***Am Pain Soc J*** 3:1-9, 1994.
121. Mense S, Simons DC: ***Muscle Pain.*** Williams & Wilkins, Baltimore. In Press.
122. Modell W, Travell J, ef ***al.:*** Treatment of painful disorders of skeletal muscle. ***NY State J Med*** 48:2050-2059, 1948.
123. Modell W, Travell J, Kraus H, ***et al:*** Relief of pain by ethyl chloride spray. ***NY State J Med*** 52:1550-1558, 1952.
124. Moldofsky H: The contribution of sleep-wake physiology to fibromyalgia, Chapter 13. In: ***Myofascial Pain and Fibromyalgia, Advances in Pain Research and Therapy, Vol. 17.*** Edited by Fricton JR, Awad EA. Raven Press, New York, 1990 (pp. 227-240).
125. Moldofsky H, Scarisback P, England R, ef ***al.:*** Musculoskeletal symptoms and non-REM sleep disturbance in patients with "fibrositis syndrome" and healthy subjects. ***Psychosom Med*** 37:341-351,1975.
126. Morgan GJ Jr: Panniculitis and erythema nodosum, Chapter 75. In: ***Textbook of Rheumatology.*** Edited by Kelley WN, Harris ED, Ruddy S, ***et al,*** Vol. 2. W.B. Saunders, Philadelphia, 1981 (pp.1203-1207).
127. Nagler JH: Injection treatment of sprains. ***Milit Surg*** 96:528-529, 1945.
128. Nielsen AJ: Personal communication, 1981.
129. Novich MM: Physical therapy in treatment of athletic injuries. ***Tex State J Med*** 61:672- 674, 1965.
130. Ottaviani LB, Childers MK: Localization of neuromuscular junctions through needle electromyography. ***Arch Phys Med Rehabil*** 76:1045,1995. (Abstr)
131. Patton IJ, Williamson JA: Fibrositis as a factor in the differential diagnosis of visceral pain. ***Can Med Assoc*** /58:162-166, 1948.
132. Pizzolato P, Mannheimer W: ***Histopathologic Effects of Local Anesthetic Drugs and Related Substances.*** Charles C Thomas, Springfield, 111., 1961 (pp. 40, 41, 60, 71).
133. Prudden B: ***Pain Erasure: The Bonnie Prudden Way.*** M. Evans & Co., New York, 1980 (pp.18, 19).
134. Rachlin ES: Trigger point management. Chapter 9. In: ***Myofascial Pain and Fibromyalgia.*** Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 173-195).
135. Rasch PJ, Burke RK: ***Kinesiology and Applied Anatomy.*** Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp.46, 47).
136. Reitinger A, Radner H, Tilscher H, ***et al.:*** Morphologische Untersuchung an Triggerpunkten [Morphologic study of trigger points]. ***Manuelle Medizin*** 34:256-262, 1996.
137. Rinzler SH, Stein I, Bakst H, ***et al:*** Blocking effect of ethyl chloride on cardiac pain induced by ergonovine. ***Proc Soc Exp Biol Med 85:329-333,***1954.
138. Rinzler SH, Travell J: Therapy directed at the somatic component of cardiac pain. ***Am Heart J*** 35:248-268, 1948 (p.250).
139. Ritchie JM, Ritchie BR: Local anesthetics: effect of pH on activity. ***Science*** 162:1394- 1395, 1968.
140. **Santiesteban AJ: Physical agents and musculoskeletal pain, Chapter 9. In: *Orthopaedic and Sports Physical Therapy,* Vol 2. Edited by Gould JA III, Davies GJ. CV Mosby, St. Louis, 1985:199-211.**
141. **Shea MJ, Keyworth D: Myofascial release: blending the somatic and orthopedic models. *Clin Bull Myofascial Ther 2(1)85-75,* 1997.**
142. **Simons DG: Muscle pain syndromes—parts I and II. *Am J Phys Med* 54:289-311, 1975, and 55:15-42, 1976.**
143. **Simons DG: Electrogenic nature of palpable bands and "Jump Sign" associated with myofascial trigger points. In: *Advances in Pain Research and Therapy.* Edited by Bonica JJ, Albe-Fessard D. Raven Press, New York, 1976 (pp. 913-918).**
144. **Simons DG: Referred phenomena of myofascial trigger points, Chap. 28. In: *New Trends in Referred Pain and Hyperalgesia, No. 27 in the series Pain Research and Clinical Management.* Edited by Vecchiet L, Albe-Fessard D, Lindblom U, ef *al.* Elsevier Science Publishers, Amsterdam, 1993:341-357.**
145. **Simons DG: Neurophysiological basis of pain caused by trigger points. Am Pain Soc J 3:17-19,1994.**
146. **Simons DG: Book Review of: Muscles, Masses and Motion. The Physiology of Normality, Hypotonicity, Spasticity and Rigidity, by E. G. Walsh. *J Musculoske Pain 2(4):153-154,* 1994.**
147. **Simons DG: Triggerpunkte und Myogelose [Trigger points and myogelosis]. *Manuelle Medizin* 35(6):190-294, 1997.**
148. **Simons DG: Myofascial trigger points: the critical experiment. *J Musculoske Pain* 5(4J.-113-118,1997.**
149. **Simons DG, Dexter JR: Comparison of local twitch responses elicited by palpation and needling of myofascial trigger points. / *Musculoske Pain 3(1)A9-81,* 1995.**
150. **Simons DG, Hong CZ: Comment to Dr. Baldry's dry needling technique. *J Musculoske Pain* 3(4):81-85, 1995.**
151. **Simons DG, Hong CZ, Simons LS: Nature of myofascial trigger points, active loci [Abstract]. *J Musculoske Pain 3(Suppl* 1):62, 1995.**
151a. **Simons DG, Mense S: Understanding and measurement of muscle tone related to clinical muscle pain. Pain 75.1-17, 1998.**
152. **Simons DG, Stolov WC: Microscopic features and transient contraction of palpable bands in canine muscle. *Am J Phys Med Rehabil* 55:65-88, 1976.**
153. **Singh P, Maibach HI: Iontophoresis in drug delivery: basic principles and applications. Crif *Rev TherDrug Carrier Sys 11(2-3):181-213,* 1994.**
154. **Sola AE, Kuitert JH: Myofascial trigger point pain in the neck and shoulder girdle. *Northwest Med* 54:980-984, 1955.**
155. **Stern S, Keren A: Extreme sinus bradycardia following routine venipuncture. *JAMA* 239:403-404,1978.**
156. **Sternbach RA: *Pain Patients.* Academic Press, N.Y., 1974 (pp. 5-11).**
157. **Tavernor D: Alleviation of postherpetic neuralgia. *Lancet* 2:671-673, 1960.**
158. **Telling WH: The clinical importance of fibrositis in general practice. *Br Med J* 1:689-692, 1935.**
159. **Tizes R: Cardiac arrest following routine venipuncture. *JAMA* 236:1846-1847, 1976.**
160. **Travell J: Rapid relief of acute "stiff neck" by ethyl chloride spray. *J Am Med Worn Assoc* 4:89-95,1949.**

161. Travell J: Basis for the multiple uses of local block of somatic trigger areas (procaine infiltration and ethyl chloride spray). Miss *Valley Med J*71:13-22, 1949.
162. Travell J: Early relief of chest pain by ethyl chloride spray in acute coronary thrombosis, Case Report. *Circulation* 3:120-124, 1951.
163. Travell J: Pain mechanisms in connective tissue. In: *Connective Tissues, Transactions of the Second Conference, 1951.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1952 (pp. 90, 92-94, 105, 119, 121).
164. Travell J: Ethyl chlorides spray for painful muscle spasm. *Arch Phys Med Rehabil* 33:291- 298, 1952.
165. Travell J: Referred pain from skeletal muscle: the pectoralis major syndrome of breast pain and soreness and the sternomastoid syndrome of headache and dizziness. *NY State J Med* 55:331-339, 1955 (pp. 332, 333).
166. Travell J: Factors affecting pain of injection. *JAMA* 158:368-371, 1955.
167. Travell J: Temporomandibular joint pain referred from muscles of the head and neck. / *Prosthet Dent* 10:745-763, 1960.
168. Travell J: Use and abuse of the muscles in housework. *J Am Worn Med Assoc* 18:159-162, 1963.
169. Travell J: *Office Hours: Day and Night.* The World Publishing Company, New York, 1968 (pp. 260, 262, 269, 270, 272, 273, 276, 283).
170. Travell J: Myofascial trigger points: clinical view. In: *Advances in Pain Research and Therapy* Vol 1. Edited by Bonica JJ, Albe-Fessard D, Raven Press, New York, 1976 (pp. 919-926).
171. Travell J: Identification of myofascial trigger point syndromes: a case of atypical facial neuralgia. *Arch Phys Med Rehabil* 62:100-106, 1981.
172. Travell JG: Chronic myofascial pain syndromes: mysteries of the history, Chapter 6. In: Advances in *Pain Research and Therapy, Vol 17.* Edited by Fricton JR, Awad E. Raven Press, Ltd., New York, 1990 (pp. 129-137).
173. Travell J, Koprowska I, Hirsch BB, *et al.:* Effect of ethyl chloride spray on thermal burns. / *Pharmacol Exp Ther* 101:36, 1951.
174. Travell J, Rinzler SH: Influence of ethyl chloride spray on deep pain and ischemic contraction of muscle. *Fed Proc* 8:339, 1949.
175. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 11:425-434, 1952.
176. Vandam LD: Local anesthetics, I. *N Engl J Med* 263:748-750, 1960.
177. Vecchiet L, Giamberardino MA, Dragani L, *et al:* Latent myofascial trigger points: changes in muscular and subcutaneous pain thresholds at trigger point and target level. *J Man Med* 5:151-154, 1990.
178. Vecchiet L, Giamberardino MA, de Bigontina P, ef *al.:* Comparative sensory evaluation of parietal tissues in painful and nonpainful areas in fibromyalgia and myofascial pain syndrome, Chapter 13. In: *Proceedings of the 7th World Congress on Pain, Progress in Pain Research and Management,* Vol. 2. Edited by Gebhart GF, Hammond DL, Jensen TS. IASP Press, Seattle, 1994, pp. 177-249.
179. Vogler P, Kraus H: *Periostbehandlung, Kolonbehandlung: Zwei reflextherapeutische Methoden.* George Thieme, Leipzig, 1975 (pp. 52-69).
180. Voss DE, Ionta MK, Myers BJ: *Proprioceptive Neuromuscular Facilitation.* Ed. 3. Harper & Row, Philadelphia, 1985.
181. Voss H: Tabelle der Muskelwichte des Mannes, berechnet und zusammengestellt nach der Untersuchungen von W. Thiele (1884). *Anat Anz* 103:356-360, 1956.
182. Walpin LA: Bedroom posture: the critical role of a unique pillow in relieving upper spine and shoulder girdle pain. *Arch Phys Med Rehabil* 58:507, 1977.
183. Walsh EG: *Muscles, Masses & Motion. The Physiology of Normality, Hypotonicity, Spasticity &• Rigidity.* MacKeith Press, Distributed by Cambridge University Press, 1992.
184. Weber EF: Ueber die Langenverhaltnisse der Fleischfasern der Muskeln in Allgemeinen. Berichte uber die Verhandlungen der Koniglich Sachsischen Gesellschaft der Wissenschaften Zu Leipzig 3:63-86, 1851.
185. Weeks VD, Travell J: Postural vertigo due to trigger areas in the sternocleidomastoid muscle. J Pediatr 47:315-327, 1955.
186. Weeks VD, Travell J: How to give painless injections. In *A.M.A. Scientific Exhibits 1957.* Grune & Stratton, New York, 1957 (pp. 318-322).
187. Whiteside J: Personal communication, 1995.
188. Willis WD Jr, Grossman RG: *Medical Neurobiology.* C.V. Mosby, St Louis, 1973 (p. 103).
189. Wilson TS: Manipulative treatment of subacute and chronic fibrositis. *Br Med J* 1:298-302, 1936.
190. Wu CM, Chen HH, Hong CZ: Inactivation of myofascial trigger points associated with lumbar radiculopathy: surgery versus physical therapy [Abstract], *Arch Phys Med Rehabil* 78:1040-1041, 1997.
191. Yagiela JA, Benoit PW, Buoncristiani RD, et *al:* Comparison of myotoxic effects of lidocaine with epinephrine in rats and humans. *Anesth Analg* 60:471-480, 1981.
192. Yue SK: Initial experience in the use of botulinum toxin A for the treatment of myofascial related muscle dysfunctions [Abstract]. *J Musculoske Pain 3(Suppl 1):22,* 1995.
193. Zohn DA, Mennell JM: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Little, Brown and Company, Boston, 1976 (pp. 126-129, 190-193).

Suppliers

Cervipillo, TRU-EZE Mfg. Co., 27635 Diaz, Temecula, CA 92390

Dramamine, Searle Pharmaceuticals, Inc., Box 5110, Chicago, IL 60680

Fluori-Methane and ethyl chloride spray, Gebauer Chemical Co., 94100 St. Catherine Ave., Cleveland, OH 44104

Hydrocollator Steam Pack, Chattanooga Corporation, 101 Memorial Drive, Chattanooga, TN 37405

Medco-sonolator, Medco Products Co., Inc., P.O. Box 50070, Tulsa, OK 74150

Phenergan, Wyeth Laboratories, P.O. Box 8299, Philadelphia, PA 19101

Potaba, Glenwood, Inc., 83 N. Summit St. Tenafly, NJ 07670

Wal-Pil-O, RoLoke Co., Box 24DD3, West Los Angeles, CA 90024

CHAPTER 4
Perpetuating Factors

With Contributions by Robert D. Gerwin, MD

HIGHLIGHTS: The **CLINICAL IMPORTANCE** of factors that perpetuate myofascial trigger points (TrPs) is generally underestimated. Perpetuating factors are numerous and often require special knowledge to recognize their importance to TrPs. They are commonly overlooked and neglected. In patients with chronic myofascial pain, attention to perpetuating factors often spells the difference between successful and failed therapy. **MECHANICAL STRESSES** frequently perpetuate the TrPs in patients with persistent (chronic) myofascial pain syndromes. The most common sources of such physical stress are skeletal asymmetry and disproportion. Asymmetries include a lower limb-length inequality-a 0.5 cm (3/16 in) difference can be critical-and a small hemipelvis. Skeletal disproportions are a long second metatarsal bone (Morton foot configuration) and short upper arms. Other sources of muscular stress, such as misfitting furniture, poor posture, abuse of muscles, constricting pressure on muscles, and prolonged immobility, are frequently significant and nearly always correctable. **NUTRITIONAL INADEQUACIES** are often crucial perpetuating factors and commonly occur along with sources of mechanical stress. Low "normal" levels of vitamins B_1, B_6, B_{12}, and/or folic acid, are suboptimal, and frequently are responsible when only transitory relief is obtained by specific myofascial treatment of involved muscles. Abnormally low values consistently aggravate TrPs. Vitamin C deficiency increases bleeding at sites of injection; low levels of this vitamin are very common in smokers. Vitamin inadequacies are confirmed by measuring blood serum levels; symptoms usually respond to oral supplements. Iron deficiency and anemia aggravate TrPs. Adequate calcium, potassium, and several trace minerals also are essential for normal muscle function. Borderline anemia is an important factor. **METABOLIC AND ENDOCRINE INADEQUACIES** that commonly perpetuate TrPs are hypometabolism due to suboptimal thyroid function, hyperuricemia and hypoglycemia. Apparently, whatever impairs muscle metabolism, including anemia and low thyroid function perpetuates TrPs. Adequate laboratory tests are now available to identify marginal thyroid function affecting TrPs. **PSYCHOLOGICAL FACTORS** that can delay recovery include depression, tension caused by anxiety, the "good sport" syndrome, secondary gain, and learned sick behavior. **CHRONIC INFECTION** due to either viral or bacterial disease, and some parasitic infestations, can prevent recovery from myofascial pain syndromes. **OTHER FACTORS,** such as allergy, impaired sleep, radiculopathy and chronic visceral disease, prolong treatment. The routine **SCREENING LABORATORY TESTS** that are most useful to identify perpetuating factors are serum vitamin levels, a blood chemistry profile, complete blood count with indices, the erythrocyte sedimentation rate, and thyroid hormone levels.

A. CLINICAL IMPORTANCE

The importance of correcting perpetuating factors is illustrated by the apocryphal story of the man who stepped in a hole in the sidewalk and broke his leg. He was treated and the bones of his leg healed, but 2 months later he stepped in the same hole and again broke the leg. *No one had patched the hole.* If we treat myofascial pain syndromes without "patching the holes" by not correcting the multiple perpetuating factors that promptly reactivate TrPs, the patient is doomed to repeated cycles of treatment and relapse. For patients who have suffered myofascial pain for many months or years, we find it necessary to spend most of our time patching holes. For patients with chronic myofascial TrPs, this is fiie *most important single chapter* in this manual; it concerns the most neglected part of the management of myofascial pain syndromes.

The answer to the question, "How long will the beneficial results of specific myofascial therapy last?" depends largely on how long the active TrPs have persisted untreated, and on the perpetuating factors that remain unresolved. In the absence of such factors, the muscle with fully inactivated trigger points (TrPs) should be no more susceptible to TrP activation than that muscle had been previously.

One also can view perpetuating factors as predisposing factors, since their presence can make the muscles more susceptible to the activation of TrPs.

The present chapter concerns the group of mechanical and biochemical factors that perpetuate existing TrPs. The previous chapter noted many common mechanical stresses that initiate TrPs by overloading muscles (Section 7). Frequently with acute TrPs, one stress activates a TrP, then other factors perpetuate it. Occasionally, these perpetuating factors are so important that their elimination permits spontaneous inactivation of the TrPs.

B. MECHANICAL STRESS

Three types of mechanical stresses are considered below: structural inadequacies, postural stresses, and constriction of muscle.

Structural Inadequacies

Common structural inadequacies can be potent perpetuators of TrPs. A lower limb-length inequality (one leg shorter) can cause a tilted pelvis in standing. This usually results in a compensatory scoliosis that is maintained by sustained muscular effort, which is a potent perpetuating factor for TrPs in those muscles. A small hemipelvis (when the subject is standing or seated) can tilt the sacral base also producing a compensatory scoliosis that produces the same results. Short upper arms (in relation to torso height) leave the shoulders without adequate support in most seated positions. This leads to overloaded shoulder elevator muscles. It also produces compensatory distorted postures that can overload torso muscles and perpetuate their TrPs. The short first, long second metatarsal variation (Morton's foot configuration) causes muscle imbalance that can extend from the leg to the head and perpetuate TrPs in those muscles.

Lower Limb-Length Inequality

IMPORTANCE. The clinical experience of practitioners is strongly convincing that lower limb-length inequality (LLLI) is fre-

quently a critically important perpetuating factor. Correcting the inequality is often essential for lasting inactivation of TrPs in muscles that are overloaded by the length discrepancy. The diagnosis of LLLI is covered in detail in Volume 2, Chapter 4, Section 8. The treatment is covered in the same chapter of Volume 2, Section 14 and also in this volume, Chapter 48, Section 14.

Although no controlled studies are known that have specifically examined the relation between LLLI and the perpetuation of TrPs, the available literature strongly suggests such a relation. Trigger points in hip and torso muscles (Chapter 41, Parts A and B) commonly cause back pain. Many studies agree that there is a strong correlation between the presence of LLLI and back pain which is frequently relieved by correcting the inequality with a lift.[90,135,195,215,246] Myofascial TrPs are the most likely muscular cause of the pain relieved by restoring functional body symmetry.

Hudson, *et al.*[129] reported an experiment in which one normal subject who had been pain free added a 1.9 cm (3/4 in) elevation to the heel of the left shoe. On the third day, the subject experienced aching in the buttocks and after 1 week, tightness and pulling in the dorsolumbar area. After 3 weeks, regular night pain was experienced in these regions. With removal of the elevation, symptoms disappeared in 2 weeks. Maigne[171] reported relief of intractable headaches by equalizing leg length with a heel lift.

Noteworthy was Redler's observation[215] that 1.3-1.9 cm (V2-3/4 in) leg-length discrepancies in children between 1.5 and 15 years of age were outgrown (disappeared) in 7 of 11 children when leg length was equalized with a heel correction for 3-7 months. This evidence for need of a structural correction in growing children by the temporary addition of a compensatory heel lift was supported in a later 3-year study of elementary, junior and high school boys.[149] Research is needed to explain why leg-length inequalities in children disappear with correction.

IDENTIFYING LOWER LIMB-LENGTH INEQUALITY. Frequently, one side of the patient is slightly smaller than the other. One lower limb is shorter than the other, the same side of the pelvis is smaller and that side of the face is smaller. When asked, many patients remember having been told that one leg was shorter than the other at a previous examination. Patients may know that they need a longer length of pant-leg on one side, or need a larger size of shoe on one foot.

On first observing these patients, body asymmetry may be revealed by facial asymmetry. The distance from the corner of the eye to the corner of the mouth on one side is less than on the other. They may walk with a tilt or lurch to one side.[21,215] When standing they are likely to assume a short-limb stance. That is, they stand with body weight on the shorter limb and the foot of the longer limb either forward with the knee slightly flexed,[215] or with the longer limb placed diagonally to the side.

When a lower limb-length discrepancy is suspected, the patient should first be examined for quadratus lumborum TrPs and, if present, they should be inactivated.[247] Any TrP shortening of the quadratus lumborum is likely to produce a misleading result, as described and illustrated in Volume 2, Chapter 4, Figure 4.9.

For examination, the undressed patient stands with the back to the examiner and with both knees straight, preferably facing a full-length mirror. The feet are brought together and an estimate of length difference is made quickly by palpating the iliac crests and the posterior superior iliac spines. An approximate correction is placed promptly beneath the shorter limb, making sure that the patient finds it comfortable. Pages of a pad or small magazine are convenient. The patient is engaged in conversation for a minute or two, and is encouraged to relax and let the weight settle on both feet. As the muscles are relieved of their attempt to compensate for the difference in limb length, they release their protective control and relax. It is then possible to accurately compensate any remaining limb-length inequality by adding correction until the pelvis and shoulders are level and, most importantly, the spine is straight.

To confirm the accuracy of the correction, a millimeter or two of lift may be added to see if the pelvis, and perhaps the shoulders, tip the other way due to overcorrection. Many patients are immediately aware of this unfamiliar strain.

The necessity for the correction is convincingly demonstrated to the patient by removing the correcting heel lift and then calling attention to the body distortion as seen in a full length mirror. When the correction is then briefly transferred to the longer limb (doubling the discrepancy) most patients are acutely distressed by the increased distortion of symmetry of the body. The correction is quickly returned to the short side to relieve the sense of muscle strain.

Some additional points in the examination are helpful. The arm that is on the same side of the body as the shorter leg tends to hang away from the body, while the arm on the other side rests against the body. Narrowing at the waist and the bulge of the hip appear greater on the side of the longer limb. The border of the gluteal fold appears lower on the shorter side.[135,246] Skin folds are present or more numerous in the flank of the concave side of the lumbar spine.

The flank skin may be pushed up bilaterally to bring the index fingers of the examiner as close as possible to the uppermost portion of the iliac crests, in order to compare the level on each side.[21,39,135,215,246] The most prominent bony portion of each ilium posteriorly (posterior superior iliac spines) may be palpated and accurately located with the thumbs, and then compared visually for levelness.[39,148,246] Comparison of the level of the dimples that correspond approximately to the posterior superior iliac spines is helpful when they are clearly visible. Variation in the levels of these iliac spines is sometimes more clearly revealed by having the patient lean forward 90% at the hips, while the examiner sights across the sacrum to determine any difference in elevation between the two sides.[21,39]

Similarly, the height of the greater trochanters can be compared.[246] In an obese patient, the trochanter is located by palpating for it while the patient bends forward, flexing the thigh at the hip.[21]

The patient also may be asked to swing first one foot back and forth, then the other; the foot of the shorter limb is easily moved with little disturbance in body positioning, whereas swinging the longer limb requires upward displacement of the pelvis on that side for the foot to clear the floor.[171]

The spine should be examined for scoliosis. If the positions of the spinous processes are difficult to determine, they may be emphasized by asking the patient to lean forward, flexing the spine slightly. If scoliosis is present, the examiner usually will see a prominence of the rib cage posteriorly on one side as the patient flexes forward.

Tilting of the shoulder-girdle axis is often readily apparent in the standing patient. Accurate evaluation of shoulder tilt may be hampered when increased tension of the upper trapezius muscle on one side distorts the silhouette of the shoulders. The positions of the scapulae are most accurately determined by palpating the relative levels of their lower poles. A tilt of the shoulder-girdle axis is especially important in patients with head, neck, shoulder-arm, and upper back pain.

When various indicators of disparity in lower limb-length disagree, especially when spinal scoliosis remains after the hips are leveled, the problem may be that the sacrum is tilted in the pelvis between the ilia, or that the lumbar spine is angulated. These conditions are discussed in detail in Volume 2, Chapter 4.

CORRECTION. The procedure for correcting an LLLI is presented in Chapter 48, Section 14 of this volume and in Volume 2, Chapter 4, Section 14. Sometimes corrections of as little as 3 mm (1/8 inch) can make an impressive difference in TrP irritability.

Discriminating patients are able to feel the reduction in muscle strain when standing, and at heel strike when walking, after the short leg is corrected. Some patients may require several days to adjust to the correction. The patient should never walk in bare feet and should have the bedroom slippers corrected. Walking on slanted surfaces, like a beach, should take into consideration that when walking in one direction, the effects of the limb-length discrepancy are aggravated, and in the other direction they are compensated.

All permanent shoe corrections should be checked for their accuracy.

Small Hemipelvis. When the pelvis is smaller on one side than the other, it can tilt the sacral base producing a compensatory scoliosis whether the subject is sitting or standing. The limb length discrepancy produces this effect only when the

subject is standing. The examination for this condition is presented in Chapter 48, Section 14 and in more detail in Volume 2, Chapter 4, Section 8. The correction is summarized in Section 14 of these two chapters.

Patients with a pelvis that is small in its vertical dimension on one side tend to sit crookedly, leaning toward the small side. They often cross one knee over the other to cantilever up the low side (see Fig. 48.10A). A seesaw effect tilts the pelvis when sitting, if one side of the pelvis is smaller than the other. This tilt is magnified by the normal closeness of the weight-bearing ischial tuberosities. The effects of this tilt *(see* Fig. 48.10B) on the spine and muscles above the pelvis are comparable to the effects of the pelvic tilt caused by a lower-limb length inequality (see Fig. 48.9B). When the subject is sitting, a small hemipelvis affects posture. When standing, both pelvic asymmetry and LLLI affect it. When both of these parts of the body are asymmetrical, they are usually both shorter on the same side.

The quadratus lumborum is the muscle primarily affected by axial deviations in the lumbar and pelvic regions;[277] the scalene and sternocleidomastoid muscles of the neck are heavily overloaded by tilt of the upper thorax. The small hemipelvis is more commonly overlooked than LLLI as a structural difference that is likely to produce chronic muscle strain. Lowman[169] reported that 20-30% of those examined in an orthopedic practice were found to have a small hemipelvis, which can occur separately or with an LLLI, usually shorter on the same side.

The results of the initial examination for a small hemipelvis can be confusing if the pelvis is twisted around the horizontal axis through the sacroiliac joints. Such an obliquity is detected by placing the thumbs on the posterior superior iliac spines and resting the hands over the crests of the ilia, pointing each index finger to an anterior superior iliac spine, fingertips at equal distances from the spines bilaterally. When the seated patient rocks the pelvis backward, the relative heights of the anterior and posterior spines are noted on each side of the pelvis. Then the patient rocks the pelvis forward for comparison. When all points on one side are lower than the corresponding points on the other side, regardless of the position of the pelvis, that half of the pelvis is smaller. If, however, one anterior spine dips much lower than the other when the pelvis rocks forward, the pelvis is twisted. This obliquity can, of itself, be a source of pain, and distorts the evaluation of a small hemipelvis; before making a final determination, the obliquity should be corrected as described by Bourdillon[40] and by Maigne.[172]

The amount of seated correction for a small hemipelvis is determined by adding increments of lift beneath the ischial tuberosity on the small side until the spine is straightened and the pelvis is leveled with the patient seated on a hard surface. The correction determined on a hard surface must be approximately doubled for a moderately soft chair seat, and tripled for a very soft sofa. Since the torso leans toward the short side *(see* Fig. 48.10B), the weight borne on that side is increased, depressing the buttock further into a soft seat, requiring a thicker lift, as illustrated in Figure 48.10D. By paying attention to this strain on the muscles, many patients develop a high degree of sensitivity to balance and learn to avoid this unnecessary seated stress.

For permanent correction, the patient uses a "sit-pad,"[169] or an "ischial lift." This may be a pad of felt of desired thickness sewn into the underwear or placed in a long back pants pocket, or it may be a small magazine slipped beneath one ischial tuberosity during sitting. The same effect can be obtained when sitting on either a domed or scooped chair seat by sliding the hips to the side that levels the pelvis. A chair that is used regularly may be fitted with a divided pneumatic seat cushion that permits separate inflation of either half (e.g., the TWIN-REST cushion").

Soft automobile seats are a common source of poor support, which can be remedied by use of a SACRO-EASE[b] seat insert; usually the wide model BR is used, which

"TWIN-REST Cushion, Fashion Able, Rocky Hill, NJ 08553.
''SACRO-EASE, McCarty's Sacro-Ease Division, 3320 Piedmont Ave, Oakland, CA 94611

provides a stable base on which to sit and also a firm support for the upper back. The SACRO-EASE may be tilted by placing a book or other material under one side to compensate for pelvic asymmetry. The patient should beware of unwittingly tilting the pelvis by sitting on a wallet in the back pocket,[103] by sitting on a tilted seat in an office chair that lacks coasters under its feet on one side, or by sitting on a sideways tilted piano bench.

Short Upper Arms. Shortness of the upper arms in relation to torso height is a rarely recognized, but not uncommon, source of muscle strain and perpetuation of TrPs in the shoulder-girdle musculature. This disparity places undue stress on the shoulder-girdle elevators, thus perpetuating TrPs in the upper trapezius and the levator scapulae muscles. Short upper arms are characteristic of the body structure of the Native Americans, but are not limited to this race. If the shoulder-elbow segment of the upper limb is short in proportion to the rest of the body, when the subject is standing, the elbows do not reach the iliac crests; when the person is sitting, the elbows fail to reach the armrests of the usual chair *(see* Fig. 6.13C). For most adults, the average armrest height from the compressed seat bottom is 22 cm (8.5 in), and ranges from 18-25 cm (7-10 in).[70]

The diagnosis of short arms is presented in Volume 2, Chapter 4, Section 8 and well illustrated in Figure 4.13 of that volume. The corrective actions to compensate for this body structure are found in Volume 2, Chapter 4, Section 14. Treatment is also described in this volume, Chapter 6, Section 14, and illustrated in Figure 6.13.

Short First, Long Second Metatarsal. The patient with a relatively short first, and long second, metatarsal bone has what is known as a Dudley J. Morton or "classic Greek" foot configuration. It is of special interest because it is likely to perpetuate myofascial pain in the low back, thigh, knee, leg, and dorsum of the foot, with or without numbness and tingling.[274] Patients with this foot configuration consistently give a history of weak ankles and report having frequently turned and sprained these joints, and had difficulty learning to ice-skate.

This condition is discussed here because problems in the foot can produce asymmetries (in the lower limb and upward) that affect the posture of the upper part of the body. Postural stresses can then activate and perpetuate TrPs in muscles of the trunk, neck, and shoulders, as well as in the lower limb.

The clinical diagnosis of this condition is covered in detail in Volume 2, Chapter 20, Section 8, and the corrective actions needed to relieve the muscular stress and overload that it causes are found in the same chapter, Section 14.

According to Morton,[182-183] during normal weight bearing the first metatarsal head should carry half of the body weight; others disagree.[109] When the first metatarsal is relatively short, the second metatarsal bears more weight. The foot, balanced on the second metatarsal, rocks as if on a knife edge.[183] To compensate for this, most people modify the gait in a way that the lateral side of the heel and the medial side of the sole of the shoe show excessive wear. Usually in such cases, the foot is slightly toed-outward at heel strike, and during stance phase. The ankle rocks inward (excessively pronates) during the stance phase. Also, during stance phase, the knee swings in toward the other knee as the thigh undergoes excessive medial rotation.

This gait usually activates myofascial TrPs in the posterior part of the gluteus medius muscle. These TrPs refer pain to the low back. The rocking foot also strains the peroneus longus muscle, which activates TrPs in it that refer pain to the ankle.[276] The taut bands of these TrPs may entrap the peroneal nerve against the fibula immediately below its head, producing numbness and tingling across the dorsum of the foot and sometimes motor weakness with foot drop. Extension of TrP activity to the posterior part of the gluteus minimus, which laterally rotates the thigh at the hip, causes posterior thigh and calf pain. Extension of TrPs to the vastus medialis causes medial knee pain[276] and may progress to the buckling knee syndrome.[274] These symptoms mimic radiculopathy, a diagnosis sometimes mistakenly made in these patients to explain their symptoms.

A study[109] of 7,167 feet in a group of 3,619 Canadian enlisted men who were unselected for symptoms found that 1596 (22%) of their feet had first and second metatarsals of equal length, 2,878 feet (40%) had a first metatarsal shorter than the second by 0.1-1.2 cm, and 2,693 feet (38%) had a first metatarsal longer than the second by 0.1-1 cm. The relative length of the metatarsals was measured from the posterior end of the calcaneus to the head of each metatarsal bone. This result suggests that the Morton foot configuration is an expression of normal variation that can cause clinical symptoms.

This syndrome is greatly aggravated by pressure from a shoe that is tight because it is too small or has a tight cap over the toes, and by high heels. If the subject has LLLI, symptoms appear primarily in the shorter leg (which experiences heavier impact) even though both feet may have the same disproportion of first and second metatarsal bones.

Although Morton[182,183] never specifically claimed that calluses under the second' metatarsal head were caused by a long bone, many authors have assumed, because of his detailed description of the weight-bearing changes, that this disproportion was responsible. In the study of 3,619 Canadian enlisted men by Harris and Beath,[109] the authors displayed graphically the concentration of weight borne throughout the plantar surface of the foot and related this to the relative lengths of the first and second metatarsals, as determined by foot **X**-rays, and to callus formation. Concentration of weight under the central metatarsal heads correlated well with callus formation, but showed no convincing relation to the relative lengths of the first and second metatarsals. Of the 35 feet showing focal concentration of weight bearing under the second to fourth metatarsal heads, 14 (40%) had short first metatarsals and 21 (60%) did not; this was also the percentage of short first metatarsals in the whole study. Apparently, some other factor was chiefly responsible for the formation of callosities under the metatarsal heads of these soldiers. Clinical experience suggests that a concurrent fungus infection of the foot may make the difference.

An Army[109] study found that 332 of 10,000 soldiers developed *painful feet* during 6 months of military training. Thirty-four, or 10%, developed symptoms attributed to the long second metatarsal syndrome of Dudley J. Morton. Of this group, 76% could be returned to duty by using the shoe insert recommended by Morton, suggesting that the inserts corrected a painful muscle imbalance. Morton's[182] identification of posterior displacement of the sesamoid bone proximal to the head of the first metatarsal as a cause of foot imbalance was not verified by the study.[109]

Clinically, it appears that the added support under the short first metatarsal is usually sufficient to relieve the calluses along the sides of the feet, but may not relieve a pressure-callus problem beneath the long metatarsal heads. The callus can be removed by a podiatrist and any dermal fungus infection should be treated daily.

Even toddlers can benefit by compensating for this foot structure with shoe correction. A 2-year-old child with Dudley J. Morton type of foot imbalance was toeing-in and frequently falling over his feet. After adding first metatarsal toe pads and medial-side heel fillers, the child at once walked without toeing-in and without tripping.

Surprisingly, TrPs in the lower limb muscles can interact with TrPs in muscles of the head and neck to restrict movement of the latter. Release of tension in the lower limb muscles by inactivation of their TrPs (such as those perpetuated by a short first, long second, metatarsal relationship) has been observed to increase a TrP-restricted interincisal opening of the jaws by 20 or 30%.

The Dudley J. Morton foot configuration is unrelated to the metatarsalgia of Morton's neuroma, described by Thomas G. Morton[2] as due to pressure on an interdigital neuroma of the plantar nerve, usually between the third and fourth metatarsal heads.

Postural Stresses

This heading concerns postural stresses due to misfitting furniture, poor posture, abuse of muscles, immobility, and repeti-

tive movement overload. Important additional postural issues are considered in detail in Chapter 41, Part C of this volume.

Misfitting Furniture. Prolonged sitting in a chair not designed for comfort, or in a well-designed chair used for the wrong purpose, quickly tires and strains muscles. Seating should be such that, as the muscles relax and the body tends to sag, correct posture is maintained by the chair and not by sustained effort of the muscles. The chair should do the work.

Travell[273] has listed nine common faults of most household chairs: "No support for your low back, armrests too low or too high, too scooped a backrest in its upper-portion, backrest nearly vertical, backrest short, failing to support your upper back; jackknifing effect at hips and knees, high front edge of the seat, shutting down the circulation in your legs; seat bottom soft in the center, creating a bucket effect which places the load on the outer side of your thighs, rather than on bony points in the buttocks; an excellent chair may be the wrong size for you."[273] Body proportions that are the basis for the design of comfortable chairs have been meticulously detailed.[70] The value of an adequate lumbar support is illustrated in Figure 41.4E; auto seats are among the worst offenders in this respect.

Poor Posture. This is another frequent source of chronic muscular strain that perpetuates myofascial TrPs. Common examples of poor posture that contribute to continued TrP activity are unphysiologic positioning at a desk or work surface *(see* Fig. 16.4C) and head tilt resulting from poorly adjusted reading glasses *(see* Fig. 16.4A), as described in Section 14 of Chapter 16.

Reading and copy material should be placed at eye level to avoid sustained forward tilting of the head and to relieve the posterior neck and upper back muscles of prolonged checkreining overload.[268] Correction of the kyphotic, round-shouldered posture when standing *(see* Fig. 41.4 A, B and C, and Figs. 41.6 and 41.8) and when sitting *(see* Fig. 41.4 D and E, and Fig. 41.5) relieves the upper back and more caudal back muscles, as well as easing chronic shortening of the pectoral muscles that results from a round-shouldered posture. Standing posture with the weight on the heels tends to shift the head forward as a counterweight, resulting in a loss of the normal cervical and lumbar lordotic curves.

Disability that continuously influences posture, such as unilateral deafness or an old injury that restricts range of motion, are potent sources of habitual muscle strain.

Other common sources of postural strain include malpositioning of materials that a person uses, such as placing documents flat at one side without a stand, writing on the lap, or using the neck and shoulder muscles to hold the receiver of the telephone against the ear.

Various ways of correcting poor posture are described and illustrated in Chapter 41, Section C.

Abuse of Muscles. People abuse muscles and thus perpetuate TrPs by poor body mechanics that render movements needlessly stressful, by sustained isometric contraction or immobility of the muscles, with too many repetitions of the same movement, and by excessively quick and jerky movements.

A common example of *poor body mechanics* is leaning over while twisting sideways to lift an item from a shelf or the floor.[268] The same effect is often produced when a person leans over the sink to brush the teeth, or stoops forward to get in and out of a chair *(see* Fig. 48.12A) instead of using the sit-to-stand or stand-to-sit technique *(see* Fig. 48.12B), as discussed in Chapter 48, Section 14 or the method described in Chapter 41, Section C.

Standing on one leg to put on a skirt or trousers is likely to strain gluteal and low back muscles; the person should sit to do this, or at least lean the weight against a support. When writing, pressing hard on the paper with a vertically-held, small-barrel ball-point pen overloads intrinsic hand muscles; using a felt-tip pen, held flatter, is less likely to perpetuate TrPs.

Troublemaking sources of *sustained contraction* include reaching up to a keyboard that is positioned too high, painting a ceiling, hanging drapes, holding a chain saw or other power tool in a fixed position,

holding a rope tight on a sailboat, or merely standing still in one place-stiffly at military attention, or tensely impatient.

Some individuals perpetuate myofascial TrPs by *jerkiness of movement.* Rapid movements that start and stop suddenly usually overstress the muscles. Optimal efficiency is obtained by smoothly coordinated movements, much as optimal gasoline mileage is obtained with smooth steady driving of an automobile at a moderate pace, without sudden changes in speed.

A sustained shortened position of the calf muscles is caused by wearing high heeled shoes or cowboy boots.

Immobility. Lack of movement, especially when a muscle is in the shortened position, tends to aggravate and perpetuate myofascial TrPs. This commonly occurs when people sleep in a position that places a muscle in its shortest length, when the muscle cannot be moved through its full range of motion due to a fracture, deformity, or articular disease; in individuals who concentrate on an activity, such as writing or reading, so intently that they forget to change position regularly; when patients have acquired habits of guarding against movement due to pain; or because they have been advised to restrict movement of a part of the body.

Repetitive Movement. A frequent repetitive movement can overload muscles and initiate TrPs. Trigger points are frequently activated by tasks that require repetitive use of the same muscles in the same way for long periods of time, such as sorting tasks at the post office or on an assembly-line job or movements by a performing artist.[257] The TrP cause of the pain reported in repetitive strain injuries is commonly overlooked, which often leads to inadequate treatment.

When patients say, "I can't do it without it hurting," they may be testing to see whether a certain painful movement can be made without pain. Repeated dozens of times daily, this unconscious testing can serve as the activity stress that is perpetuating TrPs.

Bruxism and emotional tension can interact to overload the masticatory and neck muscles, perpetuating their TrPs which cause head and face pain *[see* Chapter 5).

Constriction of Muscles

Myofascial TrPs are perpetuated by prolonged constricting pressure on a muscle, for example, by the pressure from the strap of a ponderous purse hung over the shoulder,[83] or by narrow brassiere straps that support heavy breasts and groove the upper trapezius. Constriction by a tight hosiery elastic compromises the gastrocnemius muscle, a brassiere tight around the chest compresses the latissimus dorsi, a tight shirt collar or necktie compresses the sternocleidomastoid, and a tight belt around the waist compresses the paraspinal, abdominal oblique and rectus abdominis muscles. The front edge of a chair seat that is so high that the feet do not rest firmly on the floor compresses the hamstring muscles. The hand should slip easily under the thigh, which assures ample clearance between the thigh and the seat.[5]

C. NUTRITIONAL INADEQUACIES

Nutrients of special concern in patients with myofascial pain syndromes are the water-soluble vitamins B_1, B_6, B_{12}, folic acid, vitamin C, and certain elements: calcium, iron, and potassium. These will be considered individually after some general comments.

Extensive material on vitamins is presented here because they are so important to the management of myofascial pain syndromes. Nutritional deficiencies, especially those involving water-soluble vitamins, are especially common when persons have poor dietary habits, drink excessive alcohol, or have chronic co-morbid diseases.[69,110,128,231] Nearly half of the patients whom we see with chronic myofascial pain require resolution of vitamin inadequacies for lasting relief. The complexity of this subject matches its importance. This complexity is increased by the interdependence of certain vitamins on one another, by the individual variations of human enzyme systems, and by the variable responses of individuals to metabolic distress. Although nutritional factors are not mentioned in many chapters of this manual, they ***must*** be considered in most patients with chronic myofascial TrPs if continuing relief of pain is to be achieved.

A vitamin is a nutrient that plays an essential role in normal body metabolism as a coenzyme to an apoenzyme (that requires the coenzyme to perform its metabolic function), but is not synthesized by the body. A need for better vitamin nutrition appears at *three levels:* vitamin insufficiency, vitamin deficiency, and vitamin dependence.

An apoenzyme that requires a lacking vitamin as a coenzyme will be least affected if the apoenzyme has a high affinity for the vitamin. A relatively small amount of the vitamin (coenzyme) is sufficient. Enzyme systems in which this affinity is low may be almost completely inactivated by moderate lack of the vitamin. As deficiency progresses, vitamin-dependent enzyme reactions with higher affinities also stop functioning. In general, the reactions most essential to life tend to be the last to fail.

A vitamin insufficiency requires the body to make some degree of metabolic adjustment because the amount of the coenzyme (vitamin) is limited. Myofascial pain syndromes are aggravated by insufficient levels of at least four B-complex vitamins, as listed above.

Insufficiency states may not be associated with overt disease as deficiency states are. Deficiency of vitamin C can lead to the disease scurvy, which can be prevented by 10 mg of ascorbic acid (vitamin C) per day. However, the minimum recommended daily requirement is more than 10-fold greater. The signs of vitamin inadequacy can be subtle, but can usually be found by careful history-taking and careful physical examination. Levine and Hartzell [158] discuss this concept in depth in relation to ascorbic acid. They point out that vitamin C is an essential vitamin cofactor in eight different enzymatic reactions, including the synthesis of norepinephrine and serotonin, both important in central modulation of pain transmission. Optimal levels of ascorbic acid would permit any one of the enzymatic reactions dependent on it to run at the maximum rate required at any time. Ascorbic acid concentration could therefore be rate limiting for any of the reactions for which it is an essential cofactor. As yet we do not know the actual serum level of ascorbic acid needed to become the limiting factor.

Herbert's[113] additional nonroutine laboratory test results became abnormal for one individual before they deviated beyond the accepted range of normal for routine laboratory testing. This is because the normal range for an individual is often narrow and can be within the broader population-based normal ranges. For these water-soluble vitamins, it is the low end of the normal range that is of most concern.

Vitamin **insufficiency** is used in this text to denote levels of vitamins in the lower range of normal, generally in the lower quartile of normal, that are associated with biochemical or metabolic abnormalities that indicate suboptimal function, but that are not necessarily associated with the recognized clinical symptoms or signs. The insufficiency is associated with increased irritability of TrPs. The term is also applied to those situations where the low serum level of vitamins is associated with subtle clinical symptoms or signs, but not overt disease. Thus, the terms inadequacy and insufficiency can be used interchangeably. Vitamin **deficiency,** on the other hand, is a level of serum vitamin, generally below the accepted lower normal range, that is associated with overt clinical disease such as pellagra or pernicious anemia.

Vitamin inadequacy apparently increases the irritability of myofascial TrPs by several mechanisms. Since an energy crisis is a key link in the chain of histochemical changes that are characteristic of TrPs, it is to be expected that anything that interferes with the energy supply of the muscle will aggravate TrPs. In addition, the muscles behave as though neural feedback mechanisms that perpetuate TrPs are augmented and as if TrP-referred phenomena are intensified.

The vitamin inadequacy becomes a **deficiency** when effects due to impaired function of essential enzymes are grossly apparent, and it has already seriously involved many of the less critical enzyme functions.[19] A vitamin deficiency may be established by laboratory evidence of abnormally low serum and tissue values for the vitamin,[282] by excretion of abnormal metabolic products, by the therapeutic effect of vitamin supplementation, or by some combination of the three.

There is good reason to expect that serum vitamin levels within the normal range do not ensure *optimal* levels of nutrition. Persons selected as normal controls are seldom screened for the subtle symptoms of vitamin inadequacy, such as chronic pain syndromes, leg cramps, depression or loss of energy. Individuals who typically serve as normal controls were found[13] to have deficient activity of glutamic oxaloacetic transaminase and a deficiency of pyridoxal phosphate in their erythrocytes. In this "normal" group, the tissue stores of this vitamin were depleted to the point of significantly reducing at least one pyridoxal-dependent enzyme function.

This issue of average *versus* optimal vitamin nutrition takes on added significance when the availability of a vitamin coenzyme is related to the production of one of its apoenzymes. A 55-68% increase in the specific activity of erythrocyte glutamic oxaloacetic transaminase after pyridoxine therapy in 10 pyridoxine-deficient patients indicated the biosynthesis of more apoenzyme in response to an adequate supply of coenzyme.[80] Vitamin supplementation may increase the body's production of the enzymes that the vitamin activates, relieving a double deficiency.

In a group of 12 elderly subjects who had taken 50-300 mg of pyridoxine daily for at least 1 year,[88] the specific activity of erythrocyte glutamic oxaloacetic transaminase was remarkably constant. However, 5-11 weeks of pyridoxine supplementation were required to reach this same level in pyridoxine-deficient individuals.[88]

The measurement of circulating vitamins *per se* detects the vitamin inadequacy before biochemical and classical clinical signs appear. For example, the plasma ascorbate level fell to an undetectable level after 41 days of ascorbate depletion, whereas clinical signs of scurvy did not appear for 134 days.[19] Similarly, upon elimination of folate from the diet, it required only 3 weeks for depressed serum folate levels to appear, but 14-18 weeks for a biochemical defect to become apparent, and 20 weeks for the clinical symptoms to develop.[115]

Vitamin **dependence** is observed in only a few individuals who have a congenital *deficiency* of an enzyme that requires that vitamin as a coenzyme. This defect may require the ingestion of pharmacological (megadosage) amounts of the vitamin to compensate for the congenital lack of the enzyme that requires that vitamin.[124] The larger group of people who have a congenital *insufficiency* of the enzyme will also have a congenital increased need for the corresponding vitamin.

The five *vitamins of special importance* to myofascial pain syndromes are vitamins B_1, B_6, B_{12}, folic acid, and vitamin C. This does not imply that the others are unimportant for optimal health, only that, based on current knowledge, they are less critical for the relief of myofascial TrP symptoms. Each vitamin fills multiple metabolic roles by serving as an essential coenzyme to several enzyme systems.

Vitamin B_1 (thiamine) is most critical as an energy vitamin and for the synthesis of neurotransmitters; the need for it increases with increased caloric expenditure by the body. Vitamin B_6 (pyridoxine) is essential to the metabolism of many proteins including several neurotransmitters. Cobalamins (forms of vitamin B_{12}) are critical for energy and protein metabolism. Both cobalamins and folates are required for the synthesis of deoxyribonucleic acid (DNA), which is necessary for cell replication. Either a deficiency or an excess of folate increases central nervous system irritability; adequate amounts of folate are critical for normal development of the central nervous system.

From the viewpoint of muscle, vitamin C reduces postexercise stiffness and corrects capillary fragility caused by lack of the vitamin. Adequate tissue levels may be very important to the successful management of myofascial pain syndromes in some patients, and are of importance to optimal health in all patients.

Several factors may ***cause vitamin insufficiency:*** (1) inadequate ingestion of the vitamin, (2) impaired absorption, (3) inadequate utilization, (4) increased metabolic requirement, (5) increased excretion, or (6) increased destruction within the body.[119, 121] Several groups of people are especially vulnerable to vitamin deficiencies: the elderly,[217] pregnant and lactating

women,[17] adherents to some cultural dietary customs,[18] substance abusers (most often ethyl alcohol),[119,292] "crash" dieters and food faddists, the economically disadvantaged, the emotionally depressed,[53] and the seriously ill—a list that adds up to a significant portion of the population.

Several of these factors are likely to appear in combination (e.g., among the elderly who are poor), which increases the vulnerability to deficiency. Vitamin nutrition of the elderly is often compromised in three ways: decreased nutritional intake for a number of reasons, decreased absorption that is at least partly due to folate deficiency, and increased need that is caused by the decreasing efficiency of some enzyme systems with age.

The *prevalence of unrecognized hypovitaminosis* is distressingly high. In a randomly selected municipal hospital population,[17] 105 of 120 patients (88%) had abnormally low levels of 1 or more of 11 vitamins; over half the patients were low in 2 or more vitamins. Serum folate was low in 45%; this was the commonest vitamin deficiency. Despite the low blood levels, there was a history of inadequate dietary intake in only 39% of the patients with hypovitaminosis. Moreover, hypovitaminosis was clinically apparent in only 38% of the entire group.[17]

Since the levels necessary for optimal health are unknown and the health cost of vitamin inadequacy (low normal range) is relatively unexplored, the prevalence of such vitamin inadequacy and the toll it exacts appear to be greater than is generally realized.

The ***toxicity*** of oil-soluble vitamins A, D and E is much greater than that of the water-soluble B-complex group. An excess of these oil-soluble vitamins is stored in the body fat and can readily accumulate to toxic levels, whereas an excess of water-soluble vitamins is largely discharged in the urine. Confusingly, hypervitaminosis A may cause bone or joint pain and severe throbbing headache, which can readily be confused with myofascial symptoms related to hypovitaminosis.[170]

No toxic effects of thiamine (vitamin B_1) administered by mouth have been reported in man. Rats ingested 100 times their daily requirement for three generations without harmful effects.[190] Pyridoxine, (vitamin B_6) at doses of 500 mg per day, and occasionally at doses as low as 200 mg per day, can cause peripheral neuropathy. Cyanocobalamin (vitamin B_{12}) in doses 10,000 times the daily requirement was without ill effects,[121] including one patient who had received 1 mg daily by injection for more than 1 year. Folic acid is potentially toxic; if substantiated, this would contraindicate megadosage unless specifically indicated. Thirteen of 14 normal volunteers on 15 mg daily developed gastrointestinal symptoms or mental changes and sometimes sleep disturbance.[131] However, other investigators reported 15 mg daily as innocuous.[121]

Although megadoses of vitamin C have been identified as theoretically causing cystine and oxalate stones in the urinary tract, it is becoming apparent that patients with normal renal function can tolerate exceptionally high dosage of vitamin C. One patient took 15 g of vitamin C daily for 4 months without ill effects.[289]

When dealing with vitamin requirements, one must recognize the enormous variation in nutritional needs among individuals. For instance, 64 weaning rats of four strains were fed an exclusive diet of white bread. Individual life spans ranged from 6-144 days with weight gains of from 2-212 g. This inborn individuality has a sound biological basis; the evolutionary process could not have taken place without it.[290] This means that normal laboratory values for any one patient are at best only a statistical estimate of a likelihood that the normal laboratory values apply to the needs of this individual.

If a vitamin insufficiency or deficiency aggravates TrPs, then it follows that these individuals are more likely to develop active TrPs. This would explain why an extraordinarily high percentage of patients with TrPs have one or more of these vitamin insufficiencies or deficiencies.

Thiamine (Vitamin B_1)

Discovery

In 1884, Takaki of Japan decreased the disastrous incidence of beriberi in the Japanese navy by adding meat, vegetables and condensed milk to the rice diet of the

sailors.[190] By 1912, the therapeutic effectiveness of rice polishings had been demonstrated, and in 1936, Williams and his coworkers announced the chemical structure and synthesis of the active principle, thiamine.[283]

Functions. The active form of vitamin B_1 in the body is thiamine pyrophosphate. Thiamine has been relatively unexplored in relation to myofascial pain syndromes. It is considered potentially important because it is essential for the oxidative metabolism of glucose that leads to the production of pyruvate. Pyruvate in turn is converted to acetyl CoA, which enters the Krebs citric acid cycle, by another thiamine pyrophosphate (TPP)-dependent reaction. TPP is also required for another Krebs cycle reaction, and also for the anaerobic glycolytic pathway as a coenzyme for transketolase. It is therefore essential for normal energy production within the cell, and may be a factor in the energy crisis that is part of the pathophysiology of a TrP *(see* Chapter 2, Part D). Furthermore, thiamine is essential for normal nerve function. Neuropathy can be a significant factor in the development of myofascial trigger points.[59,60] These issues urgently need well-designed research.

Thiamine Insufficiency. We see many patients with thiamine inadequacy as indicated by a low normal, or marginally abnormal serum thiamine level. The muscles of these patients have increased susceptibility to myofascial TrPs that are resistant to local therapy until the serum thiamine level is raised to the mean normal level, or above. On clinical examination, thiamine insufficiency can be detected by the presence of peripheral neuropathy characterized by diminished distal pain and temperature perception in the legs and feet, and by a loss of vibration sense. Ankle tendon reflexes may also be lost, but not necessarily so in mild sensory neuropathy.

Some thiamine-inadequate and many thiamine-deficient patients have nocturnal calf cramps, mild dependent edema, constipation, fatigue, and decreased vibratory perception in relation to nerve fiber length. When given thiamine parenterally, they may promptly lose several pounds by diuresis with resolution of the edema, have softer stools (the body is no longer removing the moisture from the bowel contents to supply the edema), and are relieved of nocturnal calf cramps.

In contrast to the *painful* calf cramps sometimes associated with thiamine deficiency, *painless* contractions of the hand or other muscles may be due to a lack of pantothenic acid, and relieved by its oral supplementation. Tinnitus may be relieved by a combination of thiamine *and* niacin therapy, but not by one vitamin alone if both are low.

Thiamine Deficiency. The **abuse of alcohol** can lead to signs and symptoms that are a variable composite of three diseases; alcoholism, thiamine deficiency, and liver dysfunction. Not only is the diet of the alcoholic likely to be deficient in thiamine, but the intake of ethyl alcohol seriously reduces thiamine absorption in either the presence[20] or absence[265] of liver disease. The liver disease itself can seriously impair the conversion of ingested thiamine to its active form, aggravating the thiamine deficiency.[239] The 74% of 43 alcoholic patients who showed enzyme evidence of thiamine deficiency also had gait and oculomotor disturbances; the others did not.[153]

Laboratory Tests. Tests for thiamine include chemical identification, microbiologic assay, erythrocyte transketolase activity, and blood levels of pyruvate and a-ketoglutarate. The photometric thiochrome procedure is the most widely used of the chemical tests for thiamine, but its results are easily distorted by interfering substances. *Lactobacillus viridescens* is the most widely employed organism for microbiologic assay, but the phytoflagellata *Ochromonas danica* appears to be the most sensitive indicator of thiamine deficiency, especially in the presence of severe liver disease.[19]

Erythrocyte transketolase (ETK) activity decreases in thiamine deficiency and correlates well with clinical symptoms;[34,165] it should be more than 800 |xg hexose/ml/hr.[283] Supplemental information is obtained by the in vitro addition of thiamine pyrophosphate to measure its stimulatory effect on transketolase activity. Increased ETK activity with thiamine supplementa-

tion indicates either a thiamine deficiency or an increased proportion of young erythrocytes.[267]

The fasting blood pyruvate is elevated above 1.0 mg/dl[283] in patients with thiamine deficiency. Following ingestion of glucose, serum pyruvate peaks in nearly 1 hour due to the disturbed glycogenesis; this is a more specific indicator of thiamine deficiency than increased serum a-ketoglutarate.[43]

Requirement and Sources. The need for thiamine is directly related to caloric intake when this corresponds to energy expenditure. The recommended daily allowance (RDA) established for adults by the National Academy of Sciences[189] is 0.5 mg/1000 kcal of energy expended, with a minimum of 1 mg/day of thiamine for older persons regardless of their activity level. Most adults expend between 1500 and 2500 kcal/day, which would require 0.75 to 1.25 mg/day. The RDA is increased for pregnant and lactating women.[189] Normal thiamine reserves usually provide at least 5 weeks protection from severe thiamine deprivation.[292]

Thiamine is widely distributed in both animal and vegetable foods, but few are rich in it. Lean pork, beans, nuts and certain whole grain cereals are the best sources available; kidney, liver, beef, eggs and fish contain helpful amounts.[101] In cereal grains, the vitamin is present almost exclusively in the germ and hull. Since these are lost in milling and refining, processed grains need to have the thiamine replaced.[7]

Causes of Insufficiency. Thiamine can be destroyed by heating above 100°C (212°F). It is quickly leached out of foods during washing or boiling.[7] It resists destruction in acid solutions at temperatures up to boiling, but is rapidly degraded in foods fried in a hot pan, foods cooked under pressure (increased temperature), and in an alkaline medium.

Canned vegetables generally contain only about 30% of the thiamine initially available. Retention in preprocessed meats ranges from 40-85%. Increasing the roasting temperature of beef or pork reduced thiamine content from 62-51% of the original. Pasteurization of cow's milk destroys from 3-10% of its thiamine, whereas the additional heat in processing evaporated milk reduces its thiamine by 30%.[7]

In addition to inadequate ingestion of thiamine, a number of factors can increase the need for the vitamin. Thiamine absorption is impaired by alcohol ingestion,[265] liver injury,[20] magnesium deficiency,[264] tannin in tea,[285] and also antacids. Thus, tea and gastric alkalinizers taken with food, and also alcohol, should be avoided. Vitamin B_1 is destroyed by thiaminase, which is found in a wide variety of fish, and in bracken fern, which grows in upland pastures where it can pose a hazard to foraging animals.[190] Excretion (loss) of thiamine is potentiated by diuretics[100,286] and probably by regularly drinking large amounts of water, which also causes a diuresis.

Conversion of dietary and synthetic thiamine to thiamine pyrophosphate, the physiologically active form, is seriously compromised in liver disease, which reduces thiamine availability and further aggravates the liver damage. Overloading the tissues with glucose may precipitate deficiency if the thiamine level is borderline low.[100]

Therapy. Thiamine is available over-the-counter in 10-, 50- and 100-mg tablets. It is also available for injection as Betalin S[c] in 1 ml ampules, and in 10 ml and 30 ml vials, at a concentration of 100 mg/ml of thiamine. The therapeutic oral dose usually recommended is 10 mg daily for several weeks, or until all evidence of deficiency has disappeared. Increasing this to 50 mg daily will cause no harm and will insure providing for patients with an exceptional need for the vitamin. A B-50 vitamin supplement contains 50 mg of thiamine and is an ample daily dose to protect nearly all individuals from thiamine insufficiency and can be taken indefinitely as a safe, inexpensive form of health insurance.

When taken in much larger amounts, excess thiamine is excreted in the urine and has no reported human toxicity. Intolerance to oral thiamine is extremely rare; daily doses of 500 mg have been administered for as long as a month without ill effects.[7] However, in rare instances, intravenous thiamine has produced fatal ana-

[c]Eli Lilly and Company Medical Department, 307 East McCarty St., Indianapolis, IN 46285.

phylactic shock. Most of these reactions occurred in patients who had previously received large doses of thiamine by injection[190]. They apparently developed sensitivity to additives in the injected solution.

In the experience of the authors, the absorption of orally administered thiamine may be inadequate even with a supplement of 100 mg three times daily. Since toxicity is not of concern, there is no known contraindication to this dosage level. In one study,[265] increasing an oral intake of thiamine above 10 mg increased neither its blood level nor the amounts excreted in the urine, supporting the belief that intestinal absorption of thiamine was likely the limiting step.

Injection of thiamine bypasses a malabsorption problem, but only a part of each injection is retained. Biweekly intramuscular injections of 100 mg are given for 3 or 4 weeks to bring the serum concentration of this vitamin up to an optimal level; however, smaller doses may be effective. This intramuscular therapy also can be used as a therapeutic trial to ensure that oral doses, which ordinarily should be adequate, actually are sufficient for patients who have poor intestinal absorption or an exceptional need.

Thiamine seems to potentiate the effectiveness of thyroid hormone. Both are essential to energy metabolism. In our experience, when patients with low thiamine levels and evidence of low thyroid function are given supplemental thiamine, their symptoms of low thyroid function may disappear, and laboratory tests of thyroid function improve without thyroid therapy. Patients already taking a thyroid supplement who receive sufficient thiamine to correct a deficiency of that vitamin may then develop symptoms of excess thyroid hormone, and the dose of thyroid supplement must be reduced.

Conversely, in the presence of thiamine insufficiency, even a small dose of thyroid hormone may precipitate symptoms of acute thiamine deficiency, which, in some respects, mimics thyrotoxicosis and may be misinterpreted as intolerance to the thyroid medication. After the thiamine deficiency has been corrected, the same small dose, and often larger doses, of thyroid hormone are well tolerated.

Pyridoxine (Vitamin B_6)

Pyridoxine (vitamin B_6) is considered important in myofascial pain syndrome (MPS) because of its role in energy metabolism and in nerve function. It is also critical for the synthesis and/or metabolism of nearly all of the neurotransmitters, including norepinephrine and serotonin, which strongly influences pain perception. There are no clinical research studies at this time that have critically assessed the contribution of low levels of pyridoxine to the perpetuation of myofascial TrPs.

Discovery

In 1934, Szent Gyorgyi identified a dietary factor that prevents rat acrodynia, a dermatitis of the tail, ears, mouth and paws characterized by edema and scaliness of the skin; later he named this substance vitamin B_6.[206] Vitamin B_6 is a complex formed from three distinct, chemically different compounds, pyridoxal (an alcohol), pyridoxal (an aldehyde), and pyridoxamine (an amine). These are the dietary precursors of the active coenzyme forms. The precursors are phosphorylated in the body, chiefly in the liver, by pyridoxal kinase to become the active coenzymes, pyridoxal phosphate and pyridoxamine phosphate.[155, 230] The activity of pyridoxal kinase increases as the concentration of pyridoxal phosphate drops, under the control of an unspecified feedback mechanism.[288]

This vitamin proved essential to man when, in the early 1950s, its absence in an infant formula caused an epidemic of convulsions that were curable by pyridoxine injection.[62,230] In 1968, the National Academy of Sciences recognized its essential nature in human nutrition by assigning it a required daily allowance (RDA).[189]

Functions. Pyridoxal phosphate has been implicated as critical in lipid metabolism because its deficiency causes myelin degeneration in man.[62,230] Vitamin B_6 deficiency also is characterized by anemia and hormonal imbalance expressed as growth retardation.[79] In pyridoxine deficiency, glutamic oxaloacetic transaminase (GOT) and glutamic pyruvate transaminase (GPT) activity in the blood and its components are reduced.[230]

Deficiency of pyridoxine involves other vitamins. Its deficiency results in reduced absorption and storage of cobalamin, increased excretion of vitamin C, and blocked synthesis of nicotinic acid (niacin). Vitamin B_6 acts synergistically with vitamin E to control the metabolism of unsaturated fats, and with vitamin C in tyrosine metabolism.[79]

More than 100 pyridoxal phosphate-dependent enzymes are known to man. Many of the most important functions of this vitamin concern **amino acid metabolism.** For these functions, pyridoxine provides essential coenzyme reactions that include transamination (the reversible transfer of an a-amino group between an amino acid and an a-keto acid), oxidative deamination of an amino acid to an aldehyde, the interconversion of the L and D isomers of an amino acid, decarboxylation, the interconversion of glycine and serine, and the conversion of homocysteine and cystathione to cysteine. Failure of the methionine-to-cysteine pathway leads to homocystinuria. The failure of cystathione conversion leads to cystathioninuria. Pyridoxal phosphate is essential to the cleavage step in the pathway of tryptophan to niacin. Hence, in the absence of an adequate exogenous source of niacin, pyridoxine deficiency enhances a niacin deficiency.[62]

Although it has no primary effect on metabolism, vitamin B_6 deficiency indirectly influences both anaerobic and aerobic metabolism. Pyridoxal phosphate plays an important conformational or structural role in the enzyme phosphorylase, which is essential to the release of glucose from glycogen for *anaerobic* metabolism, pyruvate, is normally the chief substrate for *oxidative* metabolism in muscle.[155]

The vitamin contributes to aerobic metabolism through the degradation of at least 11 amino acids, making the corresponding a-keto acid analogue of the amino acid available to enter the energy-releasing tricarboxylic acid cycle. Deficiency of pyridoxal phosphate interferes seriously with the disposal of used amino acids, and their reconfiguration for synthesis to new amino acids.[155]

Practically all of the compounds identified as **neurotransmitters** in the brain are synthesized and/or metabolized with the aid of pyridoxal phosphate. These include dopamine, norepinephrine, serotonin, tyramine, tryptamine, taurine, histamine, 7-aminobutyric acid (GABA), and indirectly acetylcholine.[79] Serotonin is derived, with the help of pyridoxal phosphate, from 5-hydroxytryptophan. Glutamic acid decarboxylase with pyridoxal phosphate catalyzes the formation of GABA, which is a central nervous system inhibitor derived from glutamic acid.[79]

In hemoglobin synthesis, pyridoxal phosphate plays an essential role as a cofactor in the synthesis of porphyrin, which is a part of the hemoglobin molecule.[230] Adults with proven pyridoxine deficiency may show a microcytic hypochromic anemia that fails to respond to iron, but the anemia improves dramatically following treatment with small doses of pyridoxine.[62]

Pyridoxine Insufficiency and Deficiency. The specific enzymatic functions of vitamin B_6 that must be lacking to cause increased neuromuscular irritability and perpetuation of TrPs has not been established. Clear-cut symptoms of pyridoxine deficiency are unusual. Pyridoxine deficiency rarely occurs alone, but usually is seen with deficiency of the other vitamins of the B-complex. Milder, equivocal symptoms appear with inadequate amounts of the vitamin. At risk for pyridoxine insufficiency are the elderly,[76] and women taking an oral contraceptive.[221]

Initially, patients on poor diets were observed to have ill-defined central nervous system syndromes of weakness, irritability and nervousness, insomnia, difficulty in walking, loss of "sense of responsibility," and abnormal electroencephalograms. These changes did not respond to treatment with other members of the vitamin B-complex, but were relieved within 24 hours by ingesting pyridoxine.

The role of insufficient pyridoxine as a significant factor in carpal tunnel syndrome (CTS) and its use as a treatment for that condition are controversial. One study found that pyridoxine supplementation for 12 weeks was effective in the

treatment of CTS compared to placebo.[82] However, a subsequent study failed to support their findings.[94] In some cases, pyridoxine insufficiency may increase the vulnerability of peripheral nerves to entrapment enough to cause the symptoms of CTS.

In a group of 154 patients admitted to the psychiatric unit of a general hospital,[53] the pyridoxine-deficient patients showed a disproportionately high incidence of depression when compared to psychiatric patients without such a deficiency.[163] A degree of depression and pyridoxine inadequacy are common findings in patients with chronic myofascial pain. In depressed patients with chronic myofascial TrPs the blood level of pyridoxine is worth investigating.

Diabetic patients who complained of leg cramps, swelling of the hands, and impaired tactile sensation were relieved of their symptoms while taking 50 mg/day of pyridoxine orally.[81]

Since vitamin B_6 is required for the conversion of tryptophan to niacin, dermatological lesions of pellagra (niacin deficiency) may result secondarily from vitamin B_6 deficiency, producing mixed symptoms of pyridoxine and niacin deficiencies.[62]

Pyridoxine Dependence. The need for very large amounts of pyridoxine occurs when one of the specific enzyme systems that require this vitamin is congenitally incomplete. Megadoses (10 times the RDA, or more) of pyridoxine at least partially compensate for the metabolic abnormality. Metabolic dependence on the vitamin is established clinically when both the symptoms and the characteristic abnormal metabolic intermediates recur promptly after resumption of an unsupplemented normal diet.

One should expect considerable variability among patients in their need for pyridoxine.[290] Patients with chronic myofascial pain are a select group who show a high prevalence of vitamin inadequacies. Many of these patients do well on large vitamin supplements. One likely explanation for this apparent partial dependence on pyridoxine by a number of patients would be the partial expression of one or more of the genetic enzyme deficiencies described here. This condition would be analogous to the differing degrees of penetrance of symptoms often seen among various members of families with inherited myopathies and neuropathies.[42]

Laboratory Tests. In experimental, fulminating deficiency, measurement of circulating serum vitamin B_6 permits detection of the deficit before biochemical and clinical signs appear. Decrease in this blood vitamin level is the earliest warning signal of an acute clinical deficiency. In mild-to-moderate chronic deficiency, the symptoms may depend as much on concomitant secondary deficiencies as on the blood level of pyridoxal phosphate.

Valid biological assay for the presence of vitamin B_6 requires time and/or special care.[111] A yeast, *Saccharomyces carlsbergensis,* is the test organism commonly used because it is responsive to pyridoxal, pyridoxal, and pyridoxamine. Unlike most other test microorganisms, it is unable to use D-alanine to satisfy its vitamin B_6 requirement. *S. carlsbergensis* is, therefore, suitable for tests on human blood. Plasma pyridoxal phosphate (PLP) concentration, determined by a radioactive tyrosine and apodecarboxylase assay, reliably reflects vitamin B_6 levels in humans.

Requirement and Sources. Vitamin B_6 is highly conserved in the body. Excretion of vitamin B_6 and its metabolites is rapidly adjusted to changes in the intake of the vitamin. The vitamin B_6 requirement rises roughly in proportion to the increase in protein intake,[48,162] and with age.[189] The 1989 National Research Council (Great Britain) RDA for vitamin B_6 is 1.6 mg for adult females, and 1.4 mg for adult males,[93] whereas the 1989 National Academy of Sciences (USA) RDA remains at the previous level of 1.4 mg for adult females and 2.0 mg for males. The current RDA of 2 mg/day may be more than is necessary to maintain the *minimum* health of a *normal* adult (with no exceptional needs).[77]

Vitamin B_6 is widely distributed in nature, but not in large amounts. The most available sources of this vitamin include liver, kidney, white meat of chicken, halibut, tuna, English walnuts, soybean flour, navy beans, bananas, and avocados. Helpful sources are yeast, lean beef, egg yolk, whole wheat, and milk.[61,230]

Fresh milk contains 0.6 mg of vitamin B_6/L (0.14 mg/8 oz serving). Very little is destroyed in milk during processing, but much is lost when milk is exposed to sunlight for more than a few minutes.

The usual synthetic form of vitamin B_6 is pyridoxine hydrochloride, which is stable in acid solution, but rapidly destroyed by sunlight when in neutral or alkaline solution.[230] This synthetic form is heat stable through most food processing. Animal sources of vitamin B_6 are less susceptible to loss of the vitamin because of cooking or preserving than are plant sources.[62]

Most of the vitamin B_6 taken orally is well absorbed in the upper intestine by passive transport, where the relatively high pH facilitates absorption. Once absorbed, all three forms of vitamin B_6 are converted to pyridoxal phosphate.

Body stores normally contain about 0.60 mg (0.55-0.66 mg) of pyridoxal phosphate/0.45 kg (1 lb) of body weight. For an 82-kg (180 lb) individual, the total amount would approximate 108 mg of pyridoxine. Most of it is stored in two tissue compartments. The bulk, 90%, resides in a slow turnover compartment with a half-life of nearly 33 days, representing tightly bound tissue stores. The remaining 10% is held in a fast-turnover compartment with a half-life of about 16 hours. During this time, the exogenous vitamin is either excreted or turned over to the slow compartment for storage. The major part is stored in *muscle,* liver and blood.[237]

Causes of Deficiency. In addition to inadequate dietary intake, tropical sprue and alcohol interfere with its absorption. Several things increase the need for vitamin B_6 including oral contraceptives, pregnancy and lactation, excessive alcohol consumption, antitubercular drugs, corticosteroids, hyperthyroidism, and uremia.

The majority of **oral contraceptive** users had abnormal tryptophan metabolism characteristic of pyridoxine deficiency; the estrogenic component of the contraceptive pill was responsible[221] with no evidence of impaired absorbtion.[200] There is no known contraindication to **regularly supplementing** the diet of oral contraceptive users with 5-10 mg of vitamin B_6 daily, except minimal cost; to many individuals, there are important advantages. **Oral supplementation of at least 10 mg per day of vitamin B_6 is strongly recommended for those taking an oral contraceptive.**

During **pregnancy** and **lactation,** the requirement for pyridoxine is markedly increased. Augmenting the basic 2.0 mg RDA of vitamin B_6 by 2.5 mg to a total of 4.5 mg daily was not sufficient to raise the blood level of pyridoxal phosphate in pregnant women to that found in nonpregnant women; the metabolic basis for this increased need was not identified.[68] Obstetricians have used supplemental pyridoxine to combat the nausea and vomiting of early pregnancy for many years.[81,230] Dr. Travell found that one or two intramuscular injections of 100 mg of pyridoxine may promptly terminate these common distressing symptoms of early pregnancy. Vitamin B_6 therapy also has provided effective prophylaxis against motion sickness in nonpregnant individuals, both adults and children.

The strong association of pyridoxine deficiency with **excessive alcohol consumption** is widely recognized.[62,160,230] Pyridoxine deficiency is aggravated in alcoholics by (1) a reduced dietary intake of the vitamin through substitution of alcohol for food, (2) by impaired absorption of the natural dietary forms of vitamin B_6, and (3) by interference with the conversion of vitamin B_6 to the active phosphorylated form by both the alcohol and liver disease. Acetaldehyde, an oxidation product of ethanol, interferes with the metabolism of vitamin B_6 by promoting the degradation of pyridoxal phosphate.[240]

Two **antitubercular drugs,** isonicotinic acid hydrazide (INH or isoniazid) and cycloserine, are potent pyridoxine antagonists.[254] Symptoms of pyridoxine deficiency due to INH interaction can be prevented by 50 mg/day of oral pyridoxine;[254] higher doses are likely to neutralize the effectiveness of the INH.

Supplemental **corticosteroids** increase the need for pyridoxine.

The need for vitamin B_6 is increased in **hyperthyroid** patients.[102,230]

Pyridoxine deficiency often occurs in both dialyzed and undialyzed **uremic** patients.

Therapy. Pyridoxine is available over-the-counter in 10-, 25- and 50-mg tablets,

and in larger amounts by prescription. Parenteral pyridoxine hydrochloride is supplied in vials of 10 and 30 ml in a concentration of 100 mg/ml.[d] A single intramuscular injection of 100 mg of pyridoxine effectively raises the serum level of the vitamin.

An adequate pyridoxine supplement is needed for individuals who eat marginal or poor diets, those who have relatively high protein intake, pregnant and lactating women, and those on an oral contraceptive. Interactions with other drugs also can be important. Pharmacological doses of vitamin B_6, ranging from 10-100 mg or more daily, are indicated for the pyridoxine-dependent conditions described and are nontoxic. A B-50 vitamin supplement contains 50 mg of pyridoxine and is an ample daily dose to protect nearly all individuals from pyridoxine insufficiency. That supplement can be taken indefinitely as an inexpensive form of health insurance.

Doses of 500 mg per day given chronically (6 months or longer) produce a peripheral sensory neuropathy and ataxia.[233] Doses over 100 mg per day are unnecessary. Doses as low as 200 mg per day have produced a sensory neuropathy,[202] and constitute a warning against the use of such high pharmacologic doses of the vitamin.

Cobalamin (Vitamin B_{12}) and Folic Acid

Cobalamin and folic acid are considered together because their metabolism and function are intimately linked. These two independently essential enzyme cofactors (essential because they must be supplied by exogenous sources as they cannot be synthesized by humans) are required for DNA synthesis in erythropoiesis and in rapidly dividing cells such as those in the gastrointestinal tract, and for fatty acid synthesis that is critical for nerve myelin formation.

Discovery of Vitamins B_{12} and Folic Acid

In 1926, Minot and Murphy successfully treated pernicious anemia by feeding patients liver. Previously, the disease had been invariably fatal.[22] In 1948, the responsible agent, a cobalamin, was finally discovered and crystallized. Hodgkin won the 1964 Nobel Prize in Chemistry for delineating the structure of this complex molecule. Its central cobalt atom is linked to a variable anionic group. This group is -CN in cyanocobalamin (the common synthetic form), -OH in hydroxocobalamin (the major form in plasma), and -CH_3 in methylcobalamin. At least three other forms are known.[121] It has been officially recommended[73] that the term vitamin B_{12} be reserved specifically for the cyanocobalamin form; "cobalamin" may apply to any of its forms. Methylcobalamin and 5'-deoxyadenosinecobalamin are the only two forms of the vitamin known to be physiologically active.[124] Cyanocobalamin is physiologically inactive and must be converted to other forms, first to be absorbed, and then to be metabolically useful.

Understanding of the overlapping contributions of folic acid and vitamin B_{12} to the etiology of macrocytic anemia evolved slowly. Pteroylglutamic (folic) acid was purified in 1943 by Stokstad and was crystallized from liver in the same year by Pfiffner and associates. By 1948, Angier and his coworkers synthesized it and identified its structure. It then became clear that folic acid was the Wills factor, the vitamin M previously found in dry brewers' yeast, and the vitamin B_c of yeast identified in chick experiments.[121]

Role in Myofascial Pain Syndromes. Vitamin B_{12} and folate insufficiency and deficiency states can be seen in chronic myofascial pain syndromes. In one study of chronic MPS and fibromyalgia, 16% of 57 myofascial pain subjects tested for vitamin B_{12} had serum levels below 261 pg/ml, while 3 of 7 (43%) fibromyalgia syndrome (FMS) patients (without myofascial trigger points) had vitamin B_{12} levels below 258 pg/ml.[95] Ten percent of the MPS subjects had low serum or erythrocyte folate levels. These figures, which are more convincing for MPS than for FMS because the FMS numbers are so small, are suggestive of a relationship between the effects of low vitamin B_{12} and/or folic acid and the persis-

[d]Hexa-Betalin, Pyridoxine hydrochloride injection, Eli Lilly & Co., Medical Department, 307 East McCarty St., Indianapolis, IN 46206.

tence of chronic MPS. Two of the three FMS subjects with vitamin B_{12} deficiency cleared completely with cobalamin replacement (Gerwin, unpublished data).

An explanation of why inadequacy of either of these two vitamins would aggravate the painfulness of TrPs is not clear and needs research investigation. Lack of these vitamins reduces blood cell production. The blood cells transport oxygen to muscles, oxygen that is essential for their energy metabolism. A severe local energy crisis exists in the region of the dysfunctional endplates of TrPs. The crisis releases substances which sensitize local nociceptors, causing pain and local tenderness. Anything that aggravates the energy crisis by intensifying the hypoxia would be expected to increase the nociceptor sensitivity. The extent to which this increased sensitization feeds back to increase acetylcholine release from the nerve terminal would further aggravate the TrP dysfunction. When and how this second step also occurs should be resolvable with appropriate research experiments.

In addition, the role of both vitamin B_{12} and folic acid on nerve function raises the possibility that these vitamins produce central or peripheral nerve dysfunction that predisposes to altered nerve/muscle junction or motor endplate dysfunction (see Chapter 2, Part D). That vitamin B_{12} inadequacy or deficiency causes a myelopathy has long been known. It is now known that there is also a peripheral neuropathy associated with vitamin B_{12} deficiency. Folic acid deficiency has also been reported to cause a peripheral neuropathy that is less common than that seen with vitamin B_{12} deficiency.[35,36] Neuropathy is associated with increased TrP irritability.[59,60] The mechanism in MPS patients is not clear.

Persons with acute lumbar or cervical radiculopathy can present with an acute MPS before there is any clinical sign of radiculopathy. Likewise, postlumbar-laminectomy scarring with nerve root entrapment can present with MPS in the distribution of the entrapped nerve root. These observations, made by Dr. Gerwin, support the concept that at least some cases of MPS are the result of nerve injury. By analogy, it may be that metabolic nerve dysfunction (injury) can also result in the formation or the persistence of the myofascial trigger point.

Functions. Cobalamins serve numerous essential metabolic functions that include (1) deoxyribonucleic acid (DNA) synthesis, (2) regeneration of intrinsic folate, which is also critical to the synthesis of DNA, (3) the transport of folate to, and its storage in, cells, (4) fat and carbohydrate metabolism, (5) protein metabolism, and (6) the reduction of sulfhydryl groups. Since cobalamin and folic acid are required for the synthesis of DNA, both are necessary for normal growth[121] and tissue repair.

Folate deficiency impairs the **synthesis of deoxyribonucleic acid,** causing megaloblastosis in all duplicating cells of the body, most commonly observed in bone marrow cells. The impaired hematopoiesis produces a pancytopenia.

> The cobalamins are involved in both fat and carbohydrate metabolism since the conversion of methylalanate to succinate is cobalamin-dependent. It has been proposed, but not proved, that the neurological deficits characteristic of cobalamin deficiency are due to compromise of the lipid portion of the lipoprotein myelin sheath surrounding the affected nerve fibers. In both the central and peripheral nervous systems, cobalamin deficiency is associated with inadequate myelin synthesis that leads to, first, demyelination, then axonal degeneration, and finally neuronal death.[14] Comparable neurologic disease is less frequently caused by folate deficiency.[121] Lesions of the myelinated peripheral nerves due to cobalamin deficiency occur more frequently and earlier than the central nervous system lesions of the myelinated posterior and lateral cords of the spinal column. The latter advanced deficiency is known as subacute combined degeneration, combined system disease, posterior lateral sclerosis, or funicular degeneration.[121]
>
> The metabolic pathways of vitamin B_{12} and folic acid are intertwined. Cobalamin is essential for the methylation of homocysteine to methionine through a reaction involving methionine synthase, for which methylcobalamin (Me-Cbl) is the cofactor.

The conversion of homocysteine to methionine is a key reaction in the synthesis of DNA, and requires both Me-Cbl and tetrahydrofolate (THF). The methyl donor is Me-THF (methyltetrahydrofolate). Folic acid is stored intracellularly as a polyglutamate, which is the form that is also necessary for its enzyme cofactor function. When cobalamin is lacking, Me-THF cannot be demethylated, an essential conversion prior to polyglutamation. Hence, the polyglutamated form of THF is decreased in serum and intracellularly when Cobalamin is inadequate. In cobalamin insufficiency, Me-THF cannot proceed through the steps of methyl-group transfers to ultimately convert deoxyuridylate to thymidylate, thus impairing DNA synthesis. However, THF can correct the impairment of thymidylate synthesis in vitamin B_{12} deficiency.[127] There is evidence to suggest that impairment of methionine synthesis may lead to peripheral neuropathy in cobalamin deficiency.[107,281] Methionine is metabolized to S-adenosylmethionine, which is required for myelin synthesis.

Serum vitamin B_{12} has two fractions, one bound to transcobalamin II, the transport protein for Cobalamin, and haptocorrine, its storage protein. Depletion of vitamin B_{12} first produces a fall in holo-transcobalamine II (cobalamin attached to transcobalamin II), before there is a decrease in haptocorrine or serum cobalamin levels.[114] When homocysteine cannot be converted to methionine, or methylmalonyl-CoA to succinyl-CoA, because of a shortage of cobalamin, there is an accumulation of both homocysteine and methylmalonic acid.

Folate is critical to development of the brain and essential for its normal functioning after birth.[178]

Insufficiency. The symptomatology of a marginal amount of **cobalamin** in the body may be highly variable and difficult to interpret. Nonspecific depression, fatigability and increased susceptibility to myofascial TrPs are likely to predominate. An exaggerated startle reaction to unexpected noise or touch is occasionally a helpful guide.

Insufficiency of **folate** is the most common vitamin inadequacy and among those inadequacies likely to perpetuate myofascial TrPs. The symptoms described by patients with myofascial pain who have marginally low serum folate levels are similar in kind to, but less intense than, many of the symptoms reported by patients with obvious neurologic disorders responsive to folic acid therapy. Increased muscular irritability and susceptibility to myofascial TrPs are commonly observed in patients with low normal (lowest quartile) or subnormal serum folic acid levels. They tire easily, sleep poorly, and feel discouraged and depressed. In our experience, these patients also frequently feel cold and have a reduced basal temperature, as do patients with thyroid hypofunction; their symptoms are often relieved by multivitamin therapy including folic acid.

Deficiency. Prompt recognition and resolution of cobalamin and/or folate deficiency is of utmost importance to the patient from the general health point of view. It is also essential for effective management of their myofascial TrPs. It now appears that neurologically cobalamin deficiency has the greatest impact on the cord and peripheral nerves, whereas folate deficiency is more likely to be associated with mental disorders that concern affect and intellect.[121]

The clinical presentations of megaloblastic anemia (pernicious anemia) and the neurologic dysfunction that is caused by **vitamin B_{12}** deficiency occur as two distinct syndromes,[241] although there is a considerable overlap in that 67% of persons with pernicious anemia with pancytopenia will have some neurologic disorder.[245] Neurologic dysfunction can occur in the absence of megaloblastic anemia, and progress independently of it.[112,141] Symptoms are those of combined degeneration of the spinal cord, which include loss of vibratory and position sense (posterior spinal cord column functions) and weakness and spasticity (lateral spinal cord column motor functions), and of peripheral neuropathy. The latter is both an axonal and demyelinating neuropathy [176] that tends to be predominantly, but not exclusively, sensory. Gait ataxia and spasticity with weakness produce neuromuscular stress in addition to that of the nerve dis-

order itself, and may further predispose to myofascial TrP formation. Diarrhea, sore tongue and other gastrointestinal complaints, reflect the disturbance of DNA synthesis in the rapidly dividing cells of the gastrointestinal tract. Constipation occurs when bowel motility is impaired. Fatigue, syncope, personality change and memory loss are less specific symptoms that raise suspicion of vitamin B_{12} deficiency. Additional symptoms of cobalamine deficiency are seen in more severe cases that are not likely to present as muscle pain syndromes, and include dementia, visual loss, and psychosis. The neurologic symptoms were once thought to be related to an abnormality of fatty acid metabolism and abnormal myelin formation, but more recent studies indicate that impairment of methionine synthesis is more likely to be the cause of the neuropathy.[263]

Pernicious anemia due to **cobalamin** deficiency occurs in 1-3% of persons of European ancestry over the age of 60,[55] and is more common in younger persons, especially women, of Hispanic and African ancestry.[49, 251] Deficiency of both vitamin B_{12} and folic acid is much more prevalent in the elderly population, vitamin B_{12} deficiency occurring in as many as 40% of subjects as determined by measuring homocysteine and methylmalonic acid levels.[134, 161, 294] Five percent of healthy elderly and 19% of hospitalized elderly were deficient in folic acid. In the cases of both vitamin B_{12} and folic acid, metabolic deficiency was found in subjects whose serum vitamin levels were within the accepted range of normal. The causes of the deficiency states were more often dietary than lack of gastric intrinsic factor. The Schilling test is more often normal than not in elderly persons with low cobalamine levels, but without megaloblastic anemia.[232]

Abnormalities of absorption, whether due to endogenous disorders (e.g., achlorhydria) or parasitic infections and gastrointestinal diseases that impair absorption like Crohn's disease, can result in cobalamin deficiency.[86]

Folic acid deficiency is associated with fatigue, diffuse muscular pain, and restless legs.[35] In addition, megaloblastic anemia, depression, peripheral sensory loss, and diarrhea can occur. A subnormal serum folate level in time causes megaloblastic hematopoiesis[117] and anemia. The differential diagnosis of anemia is well described by Herbert.[119] Evidence of peripheral neuropathy was found in 21% of one group of folate-deficient patients.[244] Similar findings in another group responded to folic acid therapy.[37] Folate deficiency alone can cause signs and symptoms of subacute combined degeneration of the cord, as in vitamin B_{12} deficiency.[36,106,207,208]

Experimental deprivation of folate for 6 months[115,116] produced the following effects: in 3 weeks, low serum folate; in 7 weeks, hypersegmentation of polymorphonuclear leukocytes; in 14 weeks, increased urinary excretion of formiminoglutamic acid; in 18 weeks, low erythrocyte folate and macroovalocytosis; and in 19 weeks, megaloblastic bond marrow and anemia. During the fourth month, sleeplessness and forgetfulness appeared and gradually increased through the fifth month. The mental symptoms disappeared within 48 hours after starting oral folic acid therapy.[115,116]

A disproportionately high percentage of psychiatric patients are folic acid deficient.[52, 139, 266] Depression is their most probable psychiatric diagnosis.[52] The pain these patients complain of is likely to be due to TrPs.

Dependence. Moderate impairment of one of the metabolic pathways that requires **cobalamin** results in a need for more than the usual amount of cobalamin and, depending on which cobalamin-dependent enzyme is involved, may or may not show low serum levels of the vitamin.[6]

Congenital abnormalities in folate-dependent pathways are generally seen initially in children with severe and often irreversible mental retardation and/or megaloblastic anemia. Some are greatly improved by megadoses of folic acid or folacin. Liver enzyme studies revealed a markedly decreased activity of 5-methyltetrahydrofolate transferase.[117] Patients with methylenetetrahydrofolate reductase deficiency exhibit homocystinuria responsive to folate therapy. In contrast,

cystathionine synthase deficiency, which also causes homocystinuria, requires supplemental vitamin B_6.[84, 124, 196, 224] Deficiency of **glutamic formiminotransferase** is less rare and blocks the formation of glutamate from histidine[84,124] causing increased excretion of formiminoglutamate (FIGLU) in the urine.[196,224] Incomplete expression of such congenital enzyme deficiencies can significantly increase the dietary folate requirements of an individual.

Laboratory Tests and Diagnosis. In an appropriate clinical setting, including any patient who has chronic MPS, especially with lassitude, easy fatigability, constipation, and impaired vibration sense in the toes, vitamin B_{12} and folic acid levels should be obtained, as well as erythrocyte folate. When the vitamin B_{12} level is 350 pg/ml or lower, serum and urine homocysteine and methylmalonic acid levels should be obtained. If these values are normal, and there is strong clinical suspicion of vitamin B_{12} deficiency, cystathionine and holotranscobalamin II (HTC II) levels should be obtained. The Schilling test is useful in planning maintenance treatment, as it addresses the ability of the individual to absorb oral vitamin B_{12}, but one must heed the cautionary statements made below, that it can be normal even when there is impaired absorption.

The diagnosis of **cobalamin** deficiency had been considered relatively easy to make in the past. Megaloblastic anemia or signs of subacute combined degeneration of the spinal cord and neuropathy, a smooth tongue, and lemon-yellow skin suggested the diagnosis that was confirmed by a low serum level of vitamin B_{12}. We now know that the presentation of cobalamin deficiency can be much more subtle, and that the assay of serum levels of vitamin B_{12} alone can be insufficient to make the diagnosis.[212] Indeed, several of Dr. Gerwin's cases presented only with fatigue, disturbed sleep and diffuse muscle pain, all of which improved with cobalamin replacement.

Vitamin B_{12} serum level determinations use competitive inhibition of radiolabeled cobalamin and cobalamin-binding proteins by serum cobalamin. Assay kits containing **R** binding proteins that bind other cobalamin analogues will result in falsely higher values of vitamin B_{12}, even into the normal range, when there is cobalamin deficiency. Even laboratory tests using purified intrinsic factor will result in false-negative tests, indicating normal vitamin B_{12} levels when there is deficiency. Noncobalamin corrinoids that are inactive analogues of vitamin B_{12} can falsely elevate serum B_{12} levels if the assay method does not use pure intrinsic factor. Large amounts of vitamin C or other reducing agents can destroy vitamin B_{12}, giving falsely low values.[120, 243] Acquired immunodeficiency syndrome can also give falsely low serum values of cobalamin.[145] Studies have shown that persons with vitamin B_{12} levels in the normal range can have other laboratory or clinical evidence of vitamin B_{12} deficiency. In one study, 14% of persons with vitamin B_{12} levels in excess of 350 pg/ml had B_{12} deficiency.[197]

The diagnosis of vitamin B_{12} deficiency cannot be made reliably only by measuring serum vitamin B_{12} levels. Measurements of other metabolites involved in cobalamin-related metabolic pathways have been used to refine the diagnosis and make it more reliable. Cobalamin is essential in the conversion of homocysteine to methionine, a reaction that is folate-dependent, and in the conversion of methylmalonyl-CoA to succinyl-CoA, a reaction that is folate independent. Hence, in cobalamin deficiency both homocysteine and methylmalonic acid will accumulate in the serum and urine, while only homocysteine accumulates in folic acid deficiency.[212] Measurement of both serum and urine levels of homocysteine and methylmalonic acid not only identify the metabolic deficiency state, but serve to differentiate between folic acid and vitamin B_{12} deficiency. Measurement of HTC II (a binding protein that functions in the transport of cobalamin) will identify early or mild cases of cobalamin deficiency.[123] Cystathionine, a metabolite of homocysteine, is elevated in both vitamin B_{12} and folic acid deficiency.[134] The deoxyuridine suppression test, measuring suppression of radiolabeled cobalamin into DNA, is a very sensi-

tive indicator of vitamin B_{12} or folic acid deficiency.[50] However, it is an in vitro test performed on bone marrow, and is not readily available.[241]

Additional information about the status of vitamin B_{12} can be obtained by evaluating antibodies to intrinsic factor[55] and to gastric parietal cells.[236] Antibodies to intrinsic factor are present in over half of persons with pernicious anemia, but are lacking in diagnostic sensitivity because they are absent in approximately **40%** of persons with pernicious anemia. Antiparietal cell antibodies are present in about **90%** of persons with pernicious anemia, but lack specificity for the diagnosis. Nevertheless, these tests enhance the ability to diagnose pernicious anemia. They do not address cobalamin deficiency based on inadequate dietary intake.

The Schilling test is used to evaluate the cause of cobalamin deficiency as a lack of absorption of vitamin B_{12}. The test assesses the absorption of an oral dose of radiola**beled** vitamin B_{12} by measuring the fraction of the ingested dose that is excreted in the urine over **24** hours. The stage I test without intrinsic factor should always be abnormal in pernicious anemia, and should be corrected by the concurrent administration of intrinsic factor in the stage II test. However, the test has serious limitations, because the crystalline form of vitamin B_{12} is not the same as food-bound vitamin $B_{12,}$ and is absorbed more readily[113] Hence, the stage I Schilling test can be normal, even in the presence of pernicious anemia, particularly since only about **10%** of the normal level of intrinsic factor is needed to absorb vitamin B_{12}. A more physiologic stage I Schilling test is performed by adding the vitamin to a raw egg and feeding the resultant omelet as the vitamin B_{12} dose.

Routine laboratory testing of **folate** levels in blood serum and in blood cells (tissue level) is now available. Normal human serum contains approximately **7-16** ng/ml of folate in the serum. Contrary to expectation, among hospitalized patients, a high mean corpuscular volume (MCV) of **95** cu mm or more had only an **0.18** correlation with folate deficiency, and therefore, would not have been useful to screen for it.[105] In some of the patients, other conditions caused the macrocytosis; or blocked macrocytosis despite the folate deficiency; in other patients, the tissue folate had not yet been sufficiently depleted to produce the macrocytosis.

Low serum cholesterol levels were correlated with low serum folate values at or below **6.2** ng/ml in **46** patients, r = **0.58.** No such correlation was obtained between cobalamin deficiency and the serum cholesterol level.[30] Low thyroid function of thyroid (but not of pituitary) origin is likely to be associated with an increased serum cholesterol.[133]

Requirement. The daily requirement needed to maintain body stores of **vitamin B_{12}** is between **1-6** u.g. [104,212] The enterohepatic circulation is so frugal in conserving vitamin B_{12}, that little is lost each day. It can take nearly a year to deplete body stores of cobalamine.[22]

The total **folacin** activity recommended as a daily dietary allowance is **400** u.g/day for adults and adolescents. During pregnancy, this allowance is set at **800** u,g/day and during lactation, **500** ug/day.[187] Evidence of depleted body stores of folacin appear in **2** months and symptoms become severe after **4** months of folic acid deprivation.[115, 116]

Sources. Among vitamins, **cobalamins** are unique because the only primary food source is from bacteria. The cobalamins are synthesized by certain microorganisms that are found in soil, sewage, water, intestines, or rumen; herbivorous animals depend entirely on microbial sources for their cobalamin.[22] The vitamin is not found in vegetable food sources, and is available to man only from animal food products or supplements. Brewers yeast, still used by some as a source of B vitamins, does not contain vitamin B_{12} unless the yeast is grown on a special cobalamin- containing media.

The dietary sources of **folate** are leafy vegetables (foliage), as the name indicates. Sources also include yeast, liver and other organ meat, as well as fresh or fresh-frozen uncooked fruit or fruit juice, and lightly cooked fresh green vegetables, such as broccoli and asparagus. Although folates are ubiquitous in nature, being pre-

sent in nearly all natural foods, they are highly susceptible to oxidative destruction; 50-95% of the folate content of foods may be destroyed in processing and preparation. All folate is lost from refined foods, such as hard liquor and hard candies.[119, 121, 217]

Causes of Insufficiency and Deficiency. The complicated chain of events required for the absorption of **cobalamin** presents many links that can fail. Absorption begins with the freeing of ingested cobalamins from their polypeptide linkages in food by gastric acid and by gastric and intestinal enzymes. The freed cobalamins form complexes with the intrinsic factor that is produced by normal gastric parietal cells. On reaching a protein receptor on the microvillar membrane of the terminal ileum, in the presence of ionic calcium and at pH about 6, the cobalamin passes through the mucous membrane into the portal venous blood. There it must join the transport protein, transcobalamin II, which carries it to the liver.

Several drug interactions may reduce serum cobalamin levels. Folate is essential for several cobalamin-dependent metabolic steps. Therefore, in the presence of a folic acid deficiency, large doses of folic acid increase the utilization of cobalamin and, when cobalamin reserves are **already** depleted, can precipitate a serious cobalamin deficiency. Drugs including neomycin, colchicine, p-aminosalicylic acid, slow-release potassium chloride, biguanide therapy (e.g., metformin),[142,218,271] and ethanol have been associated with malabsorption of cobalamin. Persons ingesting large doses of vitamin C for long periods may risk cobalamin deficiency.[119]

Tissue deficiency in **folate** is common even in high-income states, in 15% of the white population and in over 30% of the black and Spanish-American groups.[223] The four commonest causes of folate deficiency are advanced age (an increasing segment of our population), pregnancy or lactation, dietary indiscretion, and drug abuse, most commonly of alcohol.

In a study of 210 elderly patients,[177] folate deficiency was found in 24% of those from homes for the aged, in only 7.8% of similar patients from their own homes, and in 5% of a younger control group. Physical disability is seriously underestimated as a cause of impaired nutrition. This situation is compounded by the social isolation, confusion, and interacting drug effects to which the elderly are especially prone.[227]

One-third of all pregnant women in the world develop a folate deficiency so severe that they have megaloblastic anemia.[119] If the prevalence of folate *deficiency* is so high, especially in vulnerable groups, how many more individuals must have *insufficient* folate nutrition? The following study gives some idea of the proportion. Among 269 pregnant low-income patients in Gainesville, FL, 15% were *deficient* in serum folate (< 3 ng/ml), and 48% were low *(insufficient)* in serum folate (3-6 ng/ml), on their first maternity visit.[15] Be sure to check your patients with chronic myofascial TrPs for low normal or abnormal serum folate levels.

Therapy. Vitamin B_{12} is only derived from animal products, whereas folic acid is available from both animal and vegetable foods. Persons on limited diets of animal foods are at high risk for vitamin B_{12} deficiency. Treatment of the deficiency states means replenishing body stores of the nutrients in question and then maintaining them at optimal levels. In pernicious anemia, treatment is lifelong. However, in dietary deficiency, alteration of the diet may suffice, once body stores have been replenished. It is generally accepted that initial replacement of vitamin B_{12} is by intramuscular administration of 1000 ug of cyanocobalamin weekly, although some protocols specify daily dosing. Weekly injections of vitamin B_{12}, 1000 ug, will generally restore the body pool to normal levels. Monthly injections thereafter will usually maintain adequate blood levels of vitamin B_{12}. For those who can absorb vitamin B_{12}, oral administration of vitamin B_{12} 500-1000 (ug may maintain serum levels, but serum levels of vitamin B_{12} and of homocysteine and methylmalonic acid levels should be obtained at 6 month intervals for a period of 2 years to ensure that there is adequate absorption of vitamin B_{12}, because the Schilling test is not a reliable predictor of absorption. Passive absorption

of ingested vitamin B_{12} 1000 ug in the absence of intrinsic factor is sufficient to provide about 3 ug/day. This has led to oral replacement therapy, rather than using parenteral cyanocobalamin, in persons with pernicious anemia. Intranasal vitamin B_{12} gel is available as well. In such instances, it is advisable to periodically obtain serum B_{12} levels to ensure that adequate cobalamin levels are maintained.

Some persons cannot convert cyanocobalamin to hydroxocobalamin because of a genetic defect in cobalamin metabolism. These individuals do well with hydroxocobalamin given as replacement therapy. However, though used in the United States in the past, it is no longer available except in Europe where its use is widespread.

Folic acid replacement and maintenance dose recommendations are determined both by the daily requirement needed to minimize the occurrence of neural tube defects in newborns, and by the concern that high doses of folic acid will aggravate the neurological deficits of vitamin B_{12}, and obscure the early hematological signs warning of possible combined degeneration of the spinal cord by correcting the megaloblastic anemia alone. This argument should not be valid because every physician should know NOT to administer folic acid without checking the vitamin B_{12} level. Daily intake of 400 ug of folic acid can aggravate the effects of vitamin B_{12} deficiency, and will also reduce elevated homocysteine levels associated with folic acid deficiency. However, reduction of elevated homocysteine levels to the point that there is no increased mortality from cardiac and cerebral thrombosis[199] requires a higher daily dose of about 700 ug. Hence, a daily dose of 1 mg has been considered adequate. Higher doses of folic acid may in fact be required, and may be determined by the level of homocysteine, but should be given only if vitamin B_{12} levels are normal as well. Patients should be cautioned that folic acid absorption is impaired by the simultaneous ingestion of antacids.

In Dr. Gerwin's experience, fatigue and sleep disturbance improve after 2-4 weeks of folate replacement therapy, and reduction in the irritability of myofascial trigger points takes 4-6 weeks.

Practical Considerations. Vitamin B_{12} inadequacy or deficiency should be considered in persons with clinical evidence of peripheral neuropathy, in vegans or persons on a predominantly vegetarian diet who do not supplement their diet with vitamin B_{12}, diabetics and others who may not absorb cobalamin, and in persons over the age of 50, since gastric mucosal atrophy is progressive as age increases and impairs vitamin B_{12} absorption. Persons with a macrocytic anemia are also suspect. Serum levels of vitamin B_{12}, serum folate and red blood cell (RBC) folate [which also requires a complete blood count (CBC)] are obtained. Whenever serum levels of vitamin B_{12} are less than 300 pg/ml, supplementation with cyanocobalamin should be given. When serum levels are between 300-400 pg/ml, serum and urine homocysteine and methylmalonic acid levels are obtained, and if any one of them are elevated, supplementation should be given. If the situation is still unclear, (borderline or normal levels of homocysteine or methylmalonic acid, but a high suspicion) then cystathionine and HTC II levels are obtained. Intramuscular administration of cyanocobalamin is given, 1000 ug weekly, for 10 weeks. Folic acid supplementation is always given along with cyanocobalamin, at 1 mg/day orally. The Schilling test is an unreliable indicator of oral absorption of vitamin B_{12}, and oral supplementation should always be monitored by subsequent serum levels of the vitamin.

It is wise to routinely prescribe adequate amounts of vitamin B_{12} and folic acid together, not just one. They are both water soluble vitamins, inexpensive, available without prescription, and can be taken orally as a 500 mg tablet of B_{12} and a 1 mg tablet of folic acid daily. This dosage is safe and effective. Some practitioners are tempted to prescribe the vitamins without checking the level of insufficiency or deficiency. Knowledge of the laboratory values is important to understand the cause of the patient's symptoms. If the patient knows that their vitamin levels were inadequate with their customary diet (which ordinarily would be considered an adequate diet),

this information can identify an exceptional increased need for the vitamin. In that case, patients needs to know that supplementation is essential for them for the rest of their life. If they depend on their previous diet alone, their TrP perpetuating factor will recur. Most patients require a lot of convincing and reinforcement to change their eating habits substantially.

Ascorbic Acid (Vitamin C)

This vitamin is of clinical importance to the muscles because it can prevent much postexercise muscle soreness or stiffness, it corrects the increase in capillary fragility associated with ascorbic acid deficiency, and it interacts strongly with numerous other vitamins important to muscle function.

Discovery

In 1928, Albert Szent-Gyorgyi isolated a chemical that protects some fruits against discoloration and infection when bruised. The chemical is now known as ascorbic acid, or vitamin C.[46] For its discovery, he won the Nobel Prize in 1937.

Some birds[57] and a few mammals are unable to convert D-glycuronic acid to L-ascorbic acid. Man, monkeys, the guinea pig, and the Indian fruit bat are unable to synthesize ascorbic acid, which makes them dependent on exogenous sources.[154] Three exceptional guinea pigs out of several thousand were apparently able to synthesize it,[97] a capability occasionally observed in this species by other investigators;[159] a few people may possess a similar capability.

Through recorded history, scurvy was the scourge of armies, explorers, and sailors on extended trips without fresh food, until they learned to include an adequate source of vitamin C, such as lime juice, in their diet. On one trip, Vasco da Gama lost 100 of 160 sailors from scurvy.[126]

Functions. Ascorbic acid is involved in a remarkable number of essential body functions, including collagen synthesis, degradation of amino acids, and the synthesis of two neurotransmitters. Also, it is one of the most active reducing agents known to occur naturally in living tissue;[235] it provides a ready source of hydrogen atoms, since it is easily oxidized.[284] This protects many vital tissues from oxidation damage.

The most abundant protein in mammals is **collagen.** It constitutes nearly one-quarter of the protein in body tissues.[235] The strong reducing action of ascorbic acid is needed for the hydroxylation of the amino acids lysine and proline to form the protocollagen molecule. This function may be assisted by ascorbic acid inhibition of hyaluronidase.[45] At least two other important body components have an amino acid sequence similar to collagen; the Clq subcomponent of complement and the basement membrane of cells.[61,130]

Without vitamin C to provide the collagen needed for a firm vessel wall, the patient experiences marked capillary fragility and easy bruising, with diffuse tissue bleeding following only minor trauma.[235] Scorbutic patients are especially liable to develop postinjection hematomas and ecchymoses, a complication of TrP injections that should be avoided.

As an example of the structural importance of vitamin C and of the unreliable clinical significance of *normal* laboratory values, the rate of healing of pressure sores was nearly doubled by increasing serum ascorbic levels *within the normal range,* from low normal to high normal levels.[262] The low normal values were clearly suboptimal. Collagen (and therefore vitamin C) is essential for the deposition of calcium phosphate crystals to form bone.[235] In the authors' clinical experience, vitamin C can be important in the treatment of low back pain, presumably because it improves the quality of the connective tissue.

A 70-kg person on an average diet metabolizes about 400 g of protein/day, of which 100 g of **amino acids** undergo oxidative degradation in a complicated manner that provides the many building blocks for regeneration of protein structures. With no protein ingestion, some 30 g of indigenous protein continues to be oxidatively degraded. Ascorbic acid is essential to the oxidative degradation of two amino acids, phenylalanine and tyrosine.[61,126,154]

This vitamin is required for the synthesis of the essential **neurotransmitters** norepinephrine and serotonin,[61,126] both important in the modulation of pain transmission in the central nervous system. Vitamin C is the only reducing substance that specifically regulates dopamine beta-monooxygenase activity in chromaffin cells (adrenal gland medullary cells) in the synthesis of norepinephrine.[158]

Ascorbic acid is **readily oxidized** to dehydroascorbic acid, which retains 80% of its effectiveness, but further oxidation renders it inactive.[235] The vitamin also protects the tissue thiol (-SH) group, which is needed to convert plasma transferrin to liver ferritin,[61] enhances the absorption of iron in the gastrointestinal tract,[235] and contributes to fatty acid metabolism through the synthesis of carnitine.[192]

In addition, the vitamin contributes to the **stress responses** of the body. Tissue levels in the adrenal gland parallel those of the corticosteroids; both decrease markedly in response to stress.[150] Since ascorbic acid participates in the synthesis of corticosterone and 17-hydroxycorticosterone, adrenal stores of ascorbic acid may be depleted by its release to the circulation, by its utilization for the replacement of corticosteroids, or both.[130,235]

Ascorbic acid is important to enzymes that protect animals from some toxic substances. It has protected experimental animals against the formation of bladder tumors by 3-hydroxyanthranilic acid and against the hepatotoxic combination of sodium nitrite and aminopyrene.[130]

Increased susceptibility to infectious diseases has been observed consistently among people with scurvy.[126] The claim by Linus Pauling[204] that megadoses of vitamin C protect from the common cold generated much controversy. The immune systems of female children and young female adults are apparently more responsive to ascorbic acid than are those of males.[235] The vitamin does influence the immune system, but its role remains unclear.[280] Ascorbic acid combined with acetylsalicylic acid caused a significant stimulation of interleukin-6.[125] It may also stimulate lymphocyte transformation and polymorphonuclear leukocyte motility.[8]

In the authors' clinical experience, ascorbic acid helps to terminate bouts of diarrhea due to food allergy, and to decrease toxicity and TrP irritability caused by chronic infection.

There is evidence of decreasing tissue levels of ascorbic acid with increased age. Damage to membranous cell structures by lipid peroxidation appears to contribute to the deterioration of cells in the absence of ascorbic acid's reductive protection of the tissue thiol groups.[130,203] Vitamin C reverses some of the electrocardiographic findings associated with increasing age.[58] Deficiency in guinea pigs caused dystrophic disorganization of muscle structures, including fragmentation of myofilaments, swelling of mitochondria and excessive glycogen.[146]

The soreness and stiffness experienced the day after unusually strenuous exercise is prevented or markedly reduced by 1 g or more of ascorbic acid taken shortly before, or at the time of, the exercise. Eccentric exercise is primarily responsible for this phenomenon.[193] Supplementation of 3 g per day blunted reported soreness, the greatest effect occurring at the peak of the delayed-onset of muscle soreness.[138] This postexercise soreness is reviewed in the Appendix of Volume 2 of this manual and does not seem to be related to TrPs.

Ascorbic Acid Insufficiency and Deficiency. In the United States, scurvy due to inadequate dietary intake of ascorbic acid is most likely to occur in smokers, alcoholics, older people, infants fed primarily on cow's mild (usually between the ages of 6 and 12 months), food faddists, and psychiatric patients. A series of 35 patients with alcohol-related illness had a 91% prevalence of ascorbic acid deficiency.[16] Antacids destroy the effectiveness of ascorbic acid and should be taken separately so they are not mixed in the stomach.

Scurvy develops after 4-7 months of an insufficient diet.[235] Elderly patients, who were in a chronic disease hospital and on

an institutional diet with little fresh fruit had an average whole blood vitamin C level of only 0.35 mg/dl. Eight ounces of orange juice daily raised the level to 1.52 mg/dl.[54]

Decreased absorption of ascorbic acid is seen in diarrheal diseases, and increased utilization occurs in thyrotoxicosis. A frequent cause of vitamin C deficiency is cigarette smoking.[3244 130 192] Either the smoker utilizes more ascorbic acid, or less of the vitamin is available from the same dietary intake.[205]

The symptoms of frank scurvy are easily diagnosed while borderline or subclinical cases are difficult to recognize.[235] Initially, scorbutic patients present with nonspecific symptoms of weakness, lassitude, irritability, and vague aching pains in the joints and muscles. They may complain of weight loss. As the disease progresses, they are aware of easy bruising and even hematomas in the skin and muscles. The gums become swollen, red, and bleed easily. The teeth become loose and may fall out. Gum symptoms develop only in response to contact with irritants (plaque) on the teeth, and are absent in edentulous patients.[235]

Experimentally, the first sign of scurvy was perifollicular hyperkeratotic papules on the buttocks, thighs and legs, later on the arms and back. As the hairs became buried in the papules, petechiae appeared around the lesions.[235]

> Ascorbic acid blocks the formation of the carcinogen nitrosamine in vitro, and enhances the cytotoxic effectiveness of certain chemotherapeutic agents. Much of the speculation about the role of ascorbic acid in preventing or treating cancer is based on its ability to block nitrosamine synthesis.

Laboratory Tests. Determination of plasma L-ascorbic acid, based on its reducing properties, is available through medical laboratories.[126] A simple, lingual screening test for ascorbic acid deficiency has been developed[164,291] and marketed."

"Lingual Ascorbic Acid Test, Mineralab, Inc. Available through Medical Diagnostic Services, P.O. Box 1441, Brandon FL 33511.

Requirement and Sources. The body pool of ascorbic acid averages about 1500 mg, and the daily rate of metabolism approximates 3% of the existing body pool. At this rate, it would require 45 mg/day to replenish the pool. Without any replacement, a *filled* body pool is depleted to the scorbutic level in about 2 months.[126]

In the United States, the basic recommended daily allowance is 200 mg/day.

Ordinary farm animals, like horses and pigs, that synthesize ascorbic acid show average plasma concentrations of 0.33-0.40 mg/dl. By comparison, in man the value is stated as:[126]

Well nourished > 1.0 mg/dl,
Adequately nourished 0.6-1.0 mg/dl,
Poorly nourished 0.3-0.6 mg/dl,
Deficient < 0.3 mg/dl.

Excellent potential **sources** of ascorbic acid that contain more than 100 mg/100 g of *raw* food are broccoli, Brussels sprouts, collards, kale, turnip greens, guava and sweet peppers. Less rich, but valuable, sources of ascorbic acid are cabbage and potatoes, because commonly large amounts of these vegetables are eaten.[126] Citrus fruits are well known to supply vitamin C. The *fresh* juice of a large orange contains about 50 mg of the vitamin; thus 4 oranges would supply the 200 mg daily need. However, loss of the vitamin in processing or storage may be large.

Canned tomatoes retain a high percentage of their ascorbic acid content (20 mg/100 g) because of the acid environment.[61]

> Vitamin C is readily absorbed from the upper small intestine and excess is quickly excreted by the kidney, very little *via* other portals. There is no extensive storage. The *maximum* body pool ranges between 1.5 and 5 g,[99] but may be as low as 1 g.[1] The half- life in man ranges from 13-30 days; the larger the intake, the shorter the half-life. Following ingestion, the major portion of the vitamin is excreted through the urine, and also *through the expired* air;[1] the latter pathway is often overlooked. The adrenal cortex is one tissue that is normally richly supplied with ascorbic acid.[126] The human digestive tract absorbs ascorbic acid efficiently

at low levels of intake, but becomes less efficient at higher dose levels; approximately 70% of 180 mg, 50% of 1.5 g, and 16% of 12 g is absorbed, respectively. Unabsorbed vitamin C may cause diarrhea due to an osmotic effect.[126]

The concentration of isotope-labeled ascorbic acid in the adrenal gland, liver and kidney closely paralleled the decreasing concentration in the serum during the 24 hours after intravenous injection in rats.[175] Values in the brain and in one muscle continuously increased throughout this period, suggesting that an active transport system was functioning. Another muscle maintained a constant value, indicating that no active transport system was operating in it at that time.[175] Though an active transport system for water soluble vitamins including vitamin C has been demonstrated from serum into the cerebrospinal fluid (CSF) in humans, low CSF concentrations correlated with low serum levels. This suggests that high serum concentrations of vitamin C should be maintained to ensure high CSF concentrations.[261]

Causes of Deficiency. Cigarette smoking is a major cause of ascorbic acid deficiency and was demonstrated as such in guinea pigs placed on a cigarette smoker for 10 min twice daily.[85] After 28 days, both the smoking and control nonsmoking groups had equal concentrations of ascorbic acid in the liver and testes, but its concentration in the adrenal glands of the "smokers" was 29% less than for the controls and the body weight of the smokers was 30% less.[85]

A study of 17 human volunteers who smoked more than 20 cigarettes/day showed that they required 140 mg of vitamin C daily to maintain a steady state plasma ascorbic acid level compared to a daily intake of only 100 mg of ascorbic acid in nonsmoking controls.[137] Another study showed that smokers needed an additional 65 mg/day of ascorbic acid on average to maintain serum levels equivalent to those of nonsmokers.[249]

Ascorbic acid is rapidly oxidized in water to dehydroascorbic acid, which is only 80% as active as ascorbic acid biologically. Further oxidation renders it inactive. Oxidation in solution is accelerated by heat, light, alkalinity, and a metallic iron or copper vessel. This vitamin is highly soluble in water and is often discarded in the pot liquor of cooked foods.

Treatment. Prescription of ascorbic acid can be based on the new recommended dietary allowance of 200 mg/day.[157] At single doses of 500 mg and higher, the percent of vitamin C that was absorbed declined. Absorption is complete at a dose of 200 mg, but less than 50% of a 1250 mg dose is absorbed. Plateau plasma vitamin C is nearly maximal with the ingestion of 200 mg/day with no adverse effects, and is maximal at 400 mg/day.

Vitamin C daily doses above 400 mg have no evident value. Oxalate and urate excretion were elevated at 1000 mg/day of vitamin C, increasing the risk of renal stone formation. Safe doses are therefore less than 1000 mg/day. There is no rationale, therefore, for higher of megadoses of vitamin C in healthy individuals. No comparable data has been developed for ascorbic acid metabolism in persons in poor health. A physiologic dose of 400 mg daily ensures a **normal metabolic pool** of ascorbic acid to meet emergency demands.[98]

The optimal intake required depends on highly variable stress factors. In sickness there is greater tolerance for vitamin C than in good health; this suggests that megadoses may be therapeutic when in poor health.[144] The vitamin C requirement in women taking estrogen, or an oral contraceptive agent, may increase 3- to 10-fold, requiring daily amounts of the vitamin up to 500 mg.[235] Scorbutic symptoms may develop in persons suddenly withdrawn from megadose therapy, just as these symptoms may appear postpartum in babies born to megadose-treated mothers.[289]

Ascorbic acid exhibits a number of interactions with other vitamins. It apparently is important in the absorption of folic acid and in its conversion to coenzyme form, so that ascorbic acid deficiency in infants between 6 months and 1 year of age may present with the hematologic signs of folic acid deficiency. Scorbutic anemia may be microcytic, due to an associated iron deficiency caused by

blood loss, or macrocytic due to associated folic acid deficiency.[235]

The absorption of folic acid is increased by oral supplements of ascorbic acid in the presence of liver disease.[28] It had been thought that ascorbic acid destroyed vitamin B_{12} levels in food, but that is now thought to be unlikely.[174] The increased absorption of some metallic ions produced by supplemental vitamin C is desirable, as in the case of iron, but undesirable in the case of mercury. Ascorbic acid supplementation increases the amount of warfarin required to maintain the same therapeutic effect on blood clotting.[235] Supplemental vitamin C lowers the prothrombin time in patients on warfarin.[72] A daily megadose can cause watery diarrhea[72] that has been misdiagnosed as spastic colon, and can cause a nonspecific urethritis that has unnecessarily led to extensive studies for venereal infection.[89]

Increased urinary excretion of vitamin C in man due to high plasma levels produces a mild uricosuric effect, probably because of competition with uric acid for renal tubular reabsorptive transport.[28]

Patients should be encouraged to stop smoking for many reasons; the depression of their vitamin C level is only one. Smokers who have stopped smoking should be encouraged to keep their hands busy. Helpful activities include needlepoint, knitting, or embroidery. Others may prefer to carry a string of beads to run through the fingers when the urge becomes great to smoke. Chewing gum has helped some to quit smoking. Any of these activities carried to excess can abuse the muscles and activate TrP.

Dietary Minerals and Trace Elements

Several minerals, especially iron, calcium, potassium, and magnesium, are needed for normal muscle function. Clinical observations indicate that deficiency of the first three tends to increase the irritability of myofascial TrPs. Iron is an essential part of the hemoglobin and myoglobin molecules, which transport oxygen to and within the muscle fibers. Calcium is essential to muscle for release of acetylcholine at the nerve terminal and for the excitation-contraction mechanism of the actin and myosin filaments. Potassium is needed for rapid repolarization of the nerve and muscle cell membranes following an action potential. Magnesium is essential to the contractile mechanism of the myofilaments.

Essential to life, but not as critical for muscle contraction and TrP responsiveness, are other elements: zinc, iodine, copper, manganese, chromium, selenium, and molybdenum. In some patients, a close relationship exists among hypomagnesemia, hypocalcemia, and hypokalemia.

Iron. The relation of iron to muscle pain has several facets. One is the essential role of iron in energy production and oxygenation that affects the ability of muscle to meet its energy demands. This energy factor relates strongly to the TrP mechanism *(see* Chapter 2, Section D). Another is the role of iron in the regulation of hormonal functions like thyroid hormone that again plays a critical role in energy metabolism and clinically are important in chronic myofascial pain syndromes. Finally there is the role of iron in body temperature regulation that may affect both body temperature and the perception of coldness that is often seen in persons with chronic myofascial pain.

FUNCTIONS. Iron is essential for oxygen transport as is well known. It is also required for enzymatic reactions that have to do with tissue respiration, oxidative phosphorylation (cytochrome oxidase reactions are iron dependent), porphyrin metabolism, collagen synthesis, and neurotransmitter synthesis and catabolism.[29]

INSUFFICIENCY AND DEFICIENCY. Iron deficiency is estimated to be present in 9-11% of adolescent girls and women of childbearing age in the United States.[166] The prevalence of iron deficiency is slightly higher worldwide, at 15%.[67] Manifestations of iron deficiency other than anemia that are of interest to the physician treating chronic pain include impaired work performance, thermoregulation, and catecholamine metabolism.

Iron deficiency occurs in several stages: (1) depletion of tissue stores of iron that is detected by serum ferritin levels, (2) depletion of essential iron stores associated with metabolic and enzymatic activity, and finally, and (3) deficient erythropoiesis that leads to iron deficiency anemia.[278] Detection of iron insufficiency before anemia develops is most important, because decreased work capacity and impaired en-

ergy metabolism may produce a total body incipient "energy crisis" that predisposes to myofascial TrP formation, yet is easily correctable.

Essential iron stores are heme proteins involved in oxygen transport, and nonheme proteins and iron-dependent enzymes. Nonessential iron, stored primarily as ferritin, is mobilized to replace essential iron stores. Depletion of tissue iron is reflected in the lowering of serum ferritin levels, as nonessential iron stores are depleted first.

Iron deficiency anemia is associated with impaired thermoregulation, or ability to maintain body temperature, with impaired triiodothyronine response to a cold stressor, and impaired catecholamine response to environmental cold.[23-25,71] Increase in catecholamine levels may represent the body's attempt to raise core temperature.[71] Iron deficiency anemia in young women impaired the ability to maintain body temperature when exposed to a moderately cold environment.[25] Plasma triiodothyronine and thyroxine levels were both decreased in women with iron-deficiency anemia.

Impaired thermoregulation has not been demonstrated in chronic pain patients, but the symptom of coldness was present in 57% of patients with myofascial pain syndrome in one study, and of these, depletion of tissue iron was found in 65%.[96] Work capacity is reduced in iron deficient women.[140] This may relate to the clinical experience of increased fatigue and reduced endurance in iron deficient persons. The cause of the reduction in physical work capacity may be found in the impaired oxygen metabolism in skeletal muscle mitochondria associated with a decrease in iron-containing electron transport chain components as shown in iron deficient animals.[173] Lactic acid accumulates in iron deficient animals as a result of impaired glycolysis, and is also postulated to be the cause of reduced physical activity.[87] The effect of iron on energy metabolism is of special interest because of the hypothesis that the myofascial trigger point is a localized region of "energy crisis" that reflects the metabolic distress of the muscle stress.

LABORATORY TESTS. Measurement of serum ferritin is an accurate way of assessing tissue iron stores.[122] Normal serum ferritin levels are as high as 300 ng/ml. Levels of 30-50 ng/ml may signify iron loss without adequate replacement. Depletion of tissue stores of nonessential iron occur when serum ferritin levels reach 20 ng/ml.[113] Serum iron levels have a two-fold diurnal variation and are less sensitive to the state of tissue iron stores than ferritin.

REQUIREMENTS. Iron requirements are determined by daily iron losses, which are about 0.8-1.0 mg daily, except in menstruating women whose losses are 1.4-2.4 mg/day. About 10% of dietary iron is absorbed, with a ceiling of 4-5 mg/day in anemic individuals.[38] Reduced iron stores must be replenished in iron deficient persons, although iron supplements may be difficult for some persons to take because of gastric irritation, constipation, or diarrhea that develops in almost half of those taking them.

SOURCES. Dietary iron is present as easily absorbed heme iron or as poorly absorbed nonheme iron. Nonheme iron absorption is enhanced by absorption promoters, the most potent of these being ascorbic acid or vitamin C.[56] Inhibitors of nonheme iron absorption include phytates and calcium.[38] Calcium in milk, cheese or as a supplement can decrease nonheme iron absorption by 50%, and can also significantly reduce absorption of heme iron.[108] Calcium supplements should NOT be taken together with iron supplements. Phytic acids are components of cereal grains, and constitute 1-2% of many cereals, nuts and legumes. They chelate heavy metals, and are potent inhibitors of iron absorption, but the presence of phytic acids in nuts and soy are offset by the high iron content of these foods. The strong iron absorption promoter ascorbic acid can overcome the effect of dietary inhibitors to a significant degree.

CAUSES OF INSUFFICIENCY AND DEFICIENCY. Insufficient dietary intake of iron to replace menstrual blood loss places menstruating women at risk of iron insufficiency or deficiency. Iron deficiency in men usually indicates a specific illness like carcinoma that must be identified. Gastric irritation with microscopic blood loss can occur in both men and women who take nonsteroidal anti-inflammatory drugs. Iron deficiency is also associated with pernicious

anemia, occurring in 43% of persons diagnosed with this condition.[51] Moderate exercise has also been shown to reduce iron stores as measured by serum iron levels.[186, 198,213,269] On the other hand, moderate exercise increases iron absorption.[234]

TREATMENT: A PRACTICAL GUIDE. **Suspect** iron inadequacy when myofascial trigger points persist despite appropriate therapy, when fatigue or coldness are prominent symptoms, when NSAIDs have been taken regularly for pain relief, and in menstruating women, particularly those whose menstrual flow is heavy. Low erythrocyte volume or low mean cell hemoglobin concentration are indicative of iron depletion.

Measure iron stores by the serum ferritin test. Levels of 20 ng/ml or less signify iron store depletion. Levels of 30-50 ng/ml may indicate need for replacement of iron stores.

Treat iron depletion at ferritin levels of 30 ng/ml or lower, and even levels up to 40 ng/ml to prevent depletion. At ferritin levels of 30 ng/ml or less, iron supplements containing 150 mg of iron (equivalent to 50 mg of elemental iron) are taken twice daily if tolerated, or once daily if necessitated by constipation or gastric irritation. They are not taken with calcium supplements or with meals of dairy foods. However, taking them with vitamin C helps absorption. Folic acid 1 mg taken with iron lessens the symptom of gastric irritation. Supplements are available with stool softeners and in different formulations, so that finding one that is tolerable is usually possible. Once the serum ferritin level reaches 30-40 ng/ml, a small daily supplement of 12-15 mg, commonly found in most multivitamin with mineral preparations, is enough to maintain tissue iron stores.

Warning: iron supplementation should *always* be monitored to avoid excessive iron storage and hemochromatosis. Serum ferritin levels every 3 months are adequate to monitor supplementation at higher doses, and every 6 months until stable for lower dose maintenance. Iron supplements should not be given unless iron insufficiency is established through the measurement of serum ferritin levels, because iron overload can lead to hemochromatosis, ischemic heart disease and poorer outcome after stroke.[65]

Calcium. Optimum calcium intake is estimated to be 1200-1500 mg/day for adolescents and young adults, 1000 mg/day for women between the ages of 25 and 50, and for postmenopausal women taking estrogen replacement therapy, 1500 mg/day for postmenopausal women not taking estrogen replacement therapy, and 1000 mg/day for adult men. The recommended daily intake for all persons over the age of 65 is 1500 mg. Vitamin D is essential for optimal absorption of calcium. Calcium intakes up to 2500 mg/day do not result in hypercalcemia in normal person.[12]

There is no study that has linked an abnormality of calcium metabolism to myofascial pain syndromes. In the experience of one of the authors (RDG), disturbances in serum calcium levels is extremely uncommon in patients with chronic MPS. Nonetheless, calcium is of great interest in MPS (see Chapter 2) because of its role in the contraction of muscle, and also because of its role in modulating pain responses at the nociceptor cell level through voltage-gated calcium channels, at the triad where the sarcoplasmic reticulum communicates with the T tubule, and in the dorsal horn of the spinal cord.

> A normal value of total serum calcium does not ensure adequate calcium nutrition. The physiologic effects of calcium depend on the free ionic calcium; the total calcium, much of which is bound to protein, has no direct correlation with the concentration of serum ionized calcium.[12]
>
> A simple way to meet dietary calcium needs is to eat at least 2 servings daily from the milk group. One can avoid increased intake of saturated fat when eating dairy foods by using low-fat or no-fat dairy products. For those who cannot drink milk because of allergy or lactose intolerance, 30 g (1.5 oz) of brick cheese, a serving of yogurt, or 2 cups of cottage cheese suffice. For the many people who are lactose intolerant, calcium may be obtained from milk that is predigested by the enzyme lactase, sold as Lactase'; this

Lactase, 25 mg tablets, Rugby Laboratories, Inc., Rockville Centre, NY 11570.

hydrolyses some of the lactose that, undigested, tends to cause diarrhea. Nonfat dried milk can be added inconspicuously and acceptably as a dry ingredient in the preparation of foods. A few other foods, such as green leafy vegetables, legumes, canned salmon, clams, oysters, dried fruits and soybean curd (tofu), also supply calcium in the diet.

If the patient cannot tolerate dietary sources, a supplement such as calcium phosphate or calcium carbonate should be prescribed, such as Os-Cal[s] from ground oyster shell, which has vitamin D added. Three 250-mg tablets daily provide 750 mg of elemental calcium and 375 units of vitamin D_2. However, the large 500-mg tablets contain no vitamin D. Adequate absorption of calcium clearly requires sufficient vitamin D, with evidence that fluoride, phosphate, magnesium, and sometimes estrogen are also important for its absorption and utilization. Calcium supplements have the same bioavailability as calcium supplied by drinking milk.[181]

The importance of calcium to normal membrane function is now unfolding. Calcium has long been known to be essential to the transmission of an action potential across the myoneural junction and to normal excitation-contraction of the myofilaments in muscle.[4]

In excitation and contraction of skeletal muscle, depolarization of the T-tubule membrane results in the opening of Ca^{2+} (ionized calcium) release channels in the sarcoplasmic reticulum. Intracellular Ca^{2+} plays a greater role that extracellular Ca^{2+} does in this response to neural stimulation. Removal of Ca^{2+} depresses the twitch tension, and there is a dependence of muscle contraction on extracellular calcium concentration.[167] Extracellular calcium concentration or blockade of Ca^{2+} entry can modulate contractile responses. (See Chapter 2 for a detailed discussion of calcium and muscle contraction.)

Hypocalcemia that develops as the result of magnesium deficiency improves only with the administration of magnesium as well as calcium.[242] Low serum calcium from this cause will usually return to normal levels within a week after initiating magnesium repletion by oral supplements of antacid or laxative preparations containing magnesium.[242]

Potassium. The recommended daily allowance for potassium is at least about 2 g (50 mEq), but more is needed if there are unusual losses.[188] The normal concentration of serum potassium ranges from 3.5-5.0 mEq/L. Total body potassium is low in hypothyroidism and high in hyperthyroidism. In addition to clinical observations that hypokalemia aggravates myofascial TrPs, potassium deficiency disturbs function of smooth muscle and of cardiac muscle, as shown by an abnormal electrocardiogram.[214] Studies are needed that critically examine the clinical effect of hypokalemia on TrP activity.

A healthful diet for normal persons is high in potassium and low in sodium. This is not true of those with adrenal insufficiency. Foods particularly rich in potassium are fruits (especially bananas and citrus fruits), potatoes, green leafy vegetables, wheat germ, beans, lentils, nuts, dates, and prunes. The pot liquor of cooked vegetables should be saved and reused to conserve its potassium.

A diet high in fat, refined sugar and oversalted food is high in sodium, low in potassium, and can lead to potassium deficiency.[206] Diarrhea, laxatives and certain diuretics increase potassium loss.

Patients with the autosomal dominant disorder, hypokalemic periodic paralysis, experience episodes of low serum potassium and abnormal function of the sodium and potassium channels. During an episode of periodic paralysis, the muscle membrane shows marked increase in irritability evidenced by multiple fibrillation potentials and sharp waves. The reduction in the number, amplitude, and duration of motor unit action potentials is consistent with failure of neuromuscular transmission at the endplate. A careful search demonstrates reduced endplate activity that is lower in amplitude and harder to find.[78] This indicates reduced release of excessive acetylcholine characteristic of active loci of TrPs and does not

[s]**Os-Cal 250 tablet with vitamin D, Marion Laboratories, Inc., 10236 Bunker Ridge Rd., Kansas City, MO 64137.**

clarify why TrPs would be aggravated clinically in normal patients by low serum potassium levels. A reduction in ACh release should reduce, not increase TrP irritability. Research that manipulates serum potassium levels on experimental TrPs in animals with normal sodium and potassium channels should help clarify this issue.

Magnesium. There has been some interest in magnesium deficiency in fibromyalgia. Romano and Stiller[220] measured low RBC Mg levels. However, one of the authors (RDG) has not been able to replicate the low levels that are said to occur in FMS (unpublished data). The same author has looked at magnesium (Mg) levels in MPS subjects and failed to find low levels of RBC or serum Mg. Romano, however, found erythrocyte magnesium levels to be significantly lower in patients with myofascial pain.[219]

Measurement of Mg levels as they apply to muscle function is subject to great error,[229] and makes interpretation of studies of Mg concentrations [(MG)] in musculoskeletal disorders such as FMS and MPS difficult. Using phosphorous 31 magnetic resonance spectroscopy to measure ionized Mg levels in skeletal muscle, Ryschon et al.[229] found no correlation between RBC (MG), mononuclear cell (MG), and muscle ionized (MG). A negative correlation was found between serum (Mg) and muscle ionized (Mg). Hence, future studies of magnesium in MPS or FMS may need to use magnetic resonance spectroscopy in order to accurately reflect skeletal muscle (MG).

Magnesium is the second most abundant cation in intracellular fluid, and is a cofactor for over 300 cellular enzymes, predominantly related to energy metabolism.[229] About 50-60% of magnesium is in bone, most of the remainder is intracellular and only 1% is extracellular. Mg homeostasis is primarily maintained through renal excretion and reabsorption. Mg excess is uncommon, but Mg deficiency is related to a number of clinical conditions.[185] It is unlikely to occur for purely dietary reasons in the general public, but is more likely to occur as a result of malabsorption, fluid and electrolyte losses, renal dysfunction, or malnutrition (e.g., alcoholics).[75] Symptoms of Mg deficiency include neuromuscular hyperexcitability with Chvostek and Trosseau signs and seizures, and also weakness and fasciculations. Mg deficiency is often complicated by secondary hypokalemia, which aggravates muscular weakness. Likewise, hypocalcemia is commonly seen in moderate to severe Mg deficiency. Neither the hypokalemia nor the hypocalcemia are correctable until the low Mg is corrected.

Dreosti[75] reviewed the role of Mg in exercise. Mg loss is reported to occur after strenuous physical activity, and may persist for months thereafter. Mg deficient animals have reduced capacity for exercise. Studies of Mg supplementation and exercise indicate that they improve efficiency of aerobic metabolic pathways and improve cardiorespiratory performance.

The recommended dietary intake (RDI) of Mg is 4.5 mg/kg body weight, or about 250-350 mg/day for adults. Many older individuals do not achieve this level of Mg intake, and yet take calcium supplements. In these individuals, the optimal Ca/Mg ratio of 2:1 is not reached, and may reduce the efficiency of Mg absorption, accentuate the effects of low estrogen, and result in lowered Mg entry into bone, with consequent increased risk of osteoporosis.

Therapeutic Approach to Nutritional Deficiencies

Patients with chronic myofascial pain are a select group which, in our experience, has a remarkably high prevalence of vitamin inadequacies and deficiencies. When the patient fails to respond to specific myofascial therapy or obtains only temporary relief, vitamin deficiencies must be ruled out as a major contributing cause and, if present, corrected.

Treatment for either folate deficiency or cobalamin (vitamin B_{12}) deficiency should not be pursued without establishing the level of, or supplementing, the other vitamin; their symptoms overlap so widely and they interact so strongly that treatment of one may mask or precipitate a deficiency of the other.[119]

A full evaluation of the total vitamin status of the patient is prohibitively difficult because of the many overlapping and nonspecific signs and symptoms of vitamin deficiency, multiple inadequacies, marked individual variations in the daily requirement, multiple causes of inadequacy, and the expense of those laboratory tests. Some laboratories helpfully provide vitamin panels. However, high standards of performance are required at every step to ensure meaningful results that reliably tell the state of the patient's vitamin nutrition.

When a full battery of vitamin tests is not available, we find that a complete balanced supplement is a safe and usually effective alternative. Williams[290] recommends ingesting several times the recommended daily allowance of the water-soluble vitamins, but well below any possible toxic levels. One must be careful not to overload the body with the fat-soluble vitamins, particularly vitamin A. The supplement should include close to a recommended daily allowance of the essential minerals. This provides one form of inexpensive health insurance. This amount is harmless if it is the only supplemental source, and it ensures a margin of safety against inadequate levels of essential nutrients.

When the clinical picture indicates a vitamin deficiency or inadequacy, and after blood has been drawn for vitamin assays, if the most rapid relief possible is indicated, intramuscular injections may be given in addition to oral supplements. A mixed injection of 100 mg each of vitamin B_1 and B_6, 5 mg of folic acid, 1 mg of vitamin B_{12}, and 2 mg of procaine is given intramuscularly. Folic acid is sometimes deleted since it is usually well absorbed by mouth in mild to moderate deficiencies. Four or five injections may be required to quickly bring a severely depleted reservoir of these vitamins to a functionally adequate level.

Balanced mixtures of B-complex vitamins are preferred to supplementation with only one or two vitamins; multiple B-complex deficiencies are very common. In addition, the reciprocal interaction among several B vitamins due to the intertwining of their metabolic functions may precipitate deficiency of an unsupplemented vitamin.[119] For this reason, a mixed B complex such as Plebex[h] may be added to the regimen for intramuscular injection.

An adequate blood level of vitamin C is important to optimal health. This vitamin is poorly stored, and its dietary intake is commonly inadequate. We consider it wise to supplement the diet routinely with 500 mg of a timed release preparation daily. This supplementation program is another cost-effective form of health insurance. Vitamin C supplementation becomes increasingly critical with advancing age.

D. METABOLIC, AND ENDOCRINE INADEQUACIES

Clinically, any compromise of the energy metabolism of muscle appears to aggravate and perpetuate myofascial TrPs. Anemia has been reviewed under Vitamin B_{12} and under Iron in this chapter. Hypometabolism is covered in depth here because, when present, the results of specific therapy for MPS can be utterly frustrating until the hypometabolism is corrected; this perpetuating factor is not uncommon. Hypoglycemia is another perpetuating factor related to impaired energy metabolism. The last of this group, gouty diathesis, is a metabolic disturbance not directly related to energy metabolism.

Hypometabolism

Hypometabolism, or thyroid inadequacy, describes the condition of someone whose serum levels of thyroid hormones are in the low euthyroid, or just below the "normal" two standard deviation limit. The level of thyroid-stimulating hormone (TSH) may or may not be increased. Clearly *hypothyroid* patients have thyroid hormone levels below normal and an elevated TSH.[133] Patients referred to us with MPS often arrive untreated for their slightly low thyroid function because they have only mild symptoms of hypothyroidism and borderline low, or low normal, thyroid tests. Experience has shown that these patients are more susceptible to myofascial TrPs;[275] they obtain only temporary pain relief with specific myofascial therapy. This increased irritability of their muscles and

[h]**Plebex Injection, Wyeth Laboratories, P. O. Box 8299, Philadelphia, PA 19101.**

their poor response to therapy are greatly improved by supplemental thyroid, if they have no other major perpetuating factor.[275] In *hyperthyroidism,* active TrPs are uncommon, but respond well to therapy. Dr. Travell could not remember seeing a hyperthyroid patient with TrPs unresponsive to specific myofascial therapy.

Muscle pain, stiffness, weakness, muscle cramps, and pain on exertion are commonly cited manifestations of hypothyroidism.[136, 168,226,260] studies showing thyroid dysfunction in fibromyalgia have emphasized the subtle nature of the disorder, the laboratory demonstration of abnormal thyroid function was an abnormal response to the administration of thyrotropin releasing hormone (TRH). In some instances this was shown to be the consequence of primary hypothyroidism, such as in thyroiditis.

In other instances this represented a failure of the hypothalamic-pituitary-thyroid axis, or a disorder in the regulation of thyroid hormone, as shown by a blunting of the usual response to TRH. Neeck and Riedel[191] showed that FMS patients tend to have lower thyroid hormone levels with the exception of free levothyroxine (T_4), do not show the normal increase in free 3,5,3'-triiodothyronine (T_3) or free T_4 in response to TRH stimulation, and do not have an increase in TSH levels.

Finally, a third defect has been identified as peripheral resistance to thyroid hormone.

Role of Hypometabolism in Myofascial Pain. As many FMS patients have persistent or recurrent TrPs,[95] and as none of the studies excluded myofascial TrPs as a cause of tender points, FMS findings are likely to be relevant to chronic myofascial pain as well. Despite these reports, the relationship of hypothyroidism to widespread muscle pain, whether fibromyalgia or myofascial pain, remains a controversial issue, and is not widely accepted by endocrinologists. This may be true largely because, until very recently, the causes of those two pain diagnoses were not convincingly identified.

Gerwin[95] identified hypothyroidism in 10% of a cohort of chronic myofascial pain patients, using clinical symptomatology and determinations of T_3, T_4, FT_4, TSH, or TRH stimulation test. A striking feature of these patients was the widespread distribution of myofascial TrPs (Gerwin, unpublished data).

Rosen has reported the occurrence of myoedema in response to TrP injections[222] which he attributes to histamine sensitivity. However, myoedema is a well-described phenomenon in hypothyroidism, though seen in other disorders as well such as malnutrition, and suggests that such patients should be evaluated for hypothyroidism.

Sonkin[252] reviewing his experience with myofascial pain and hypothyroidism, emphasized the value of the basal metabolic rate in the assessment of oxidative metabolism, though this test is no longer available. The test measures the overall efficiency of oxidative metabolism, and will identify hypometabolism that results from thyroid gland disease, pituitary failure, or failure of peripheral utilization of thyroid hormone, which is difficult to measure otherwise. In his review, Sonkin relates his study of 174 therapeutic trials in symptomatic, but chemically euthyroid, subjects. The second most common symptom was myofascial pain (the most common symptom was fatigue). Seventy-three percent of the patients treated with thyroid supplementation had symptomatic improvement. Responsiveness was correlated with the degree of change in the basal metabolic rate and in cholesterol levels.[252]

Sonkin points out that diffuse muscle tenderness may be the major physical finding in mild hypothyroidism.[252] Serum thyroxine (T_4), free thyroxine index, and TSH may be within the normal range in mild cases. Measurement of serum creatine kinase (CK) and cholesterol, both of which become elevated in hypothyroidism, may be useful. The TRH stimulation test produces an abnormal elevation of TSH in hypothyroidism, and he found it useful in the diagnosis of mild hypothyroidism. Mild hypothyroidism, as discussed later in this section, may be the result of too little thyroid hormone in a particular individual, but with thyroid function tests still within the broad range of normal. Mild hypothyroidism can also be the result of impaired peripheral utilization despite adequate circulating thyroid hormone.

In one author's experience (RDG), treatment of hypothyroidism (whether mild or

more severe) makes TrPs more responsive to therapy that includes both physical therapy and TrP injections. However, thyroid hormone therapy *alone* may not clear the TrPs any more in these hypothyroid patients than they might recover spontaneously if they were euthyroid patients. On the other hand, one author (RDG) has repeatedly seen considerable reduction in TrPs and even full recovery from MPS within 4-6 weeks of achieving a TSH of 0.5-2.0 mlU/L in hypothyroid myofascial pain patients. This corresponds to spontaneous recovery from acute TrPs in patients without any perpetuating factors. Other authors (JGT and DGS) have had many similar clinical experiences with thyroxine supplementation.

Forms of Hypothyroidism.

MILD HYPOTHYROIDISM. The issues relating to hypothyroidism in patients who have chronic myofascial pain more often concern mild hypothyroidism rather than overt, clinically advanced disease. Mild hypothyroid failure is often called subclinical hypothyroidism. Danese et al.[64] defined this condition as an elevated serum TSH in the presence of a normal serum free T_4, and noted that it may or may not be symptomatic. The condition is more common in women than men, and increases in frequency with age. Some studies report the prevalence to be as high as 17% in women and 7% in men.[201] Identification and treatment of individuals with subclinical hypothyroidism can reverse subtle clinical symptoms of thyroid hormone deficiency,[253] including multiple muscles with myofascial TrPs that may not be thought of as a manifestation of thyroid disease.

THYROIDITIS. Chronic autoimmune (Hashimoto's) thyroiditis is a common disorder, causing the majority of cases of hypothyroidism. Autopsy prevalence rates of significant thyroiditis are as high as 15% in women and 5% in men. When iodine deficiency is not an issue, 50% of individuals with serum TSH levels > 5 mU/L, and 80% of those with TSH levels > 10 mU/L had thyroid antibodies[66] characteristic of thyroiditis. The presence of antithyroid microsomal antibodies indicates autoimmune thyroiditis.

EFFECTS OF THYROID HORMONES. The thyroid hormones influence growth, energy production, and energy consumption. Thyroxine (T_4) affects growth by increasing the rate of microsomal protein synthesis through a direct effect on translation that does not require synthesis of RNA. On the other hand, T_3 increases both ribosomal RNA and protein synthesis through an increase in RNA polymerase activity. Thyroxine selectively increases the activity of some enzymes 5-10 times.[216] This helps to explain why adequate thyroid hormone is critical for the replication of many kinds of cells.

The chief product of oxidative phosphorylation is adenosine triphosphate (ATP), the primary source of energy for muscular contraction.[28] The production of ATP by mitochondria is significantly increased when the concentration of T_3 increases. The hormone acts at the inner membrane of the mitochondrion, which is the site of oxidative phosphorylation.[255]

A major mechanism by which T_3 causes increased energy expenditure is the increase of adenosine triphosphatase (ATPase) activity in cell membrane. ATP supplies the energy for muscle contraction and drives the sodium-potassium pump that maintains gradients of these ions across a cell membrane.[216] These gradients are essential to the excitability of muscle and nerve fibers and apparently have a "vent" system so that, although overactivity of the pump expends additional energy, it does not produce serious hyperpolarization of the membrane.

Muscle changes occur in hypothyroidism that may be reflected in the clinical signs of weakness and fatigue. Myosin develops the characteristics of slow fibers.[132] Certain mitochondrial enzymes show reduced activity.[194] Argov et al.[10] studied the bioenergetics of muscle using phosphorus-31 nuclear magnetic spectroscopy. The ratio of phosphocreatine to inorganic phosphate (PCr/Pi) was low at rest in two patients with hypothyroidism, PCr depletion during exercise was increased, and postexercise recovery of PCr/Pi was delayed. Similar findings

after exercise, but not a rest, were found in thyroidectomized rats. These changes may be the result of impaired mitochondrial function resulting in abnormal oxidative metabolism of chiefly type I fibers and impaired glycolytic metabolism affecting type 2 fast-twitch muscle fibers in hypothyroidism.

COLD INTOLERANCE. Hypometabolism patients nearly always experience **cold intolerance;** occasionally they are intolerant of both heat and cold. They tend to wear additional clothing (a sweater, jacket, or pullover) when others do not, rarely sweat, and frequently complain of cold hands and, especially, of cold feet. These patients are "weather conscious," and muscular pain increases with the onset of cold, rainy weather.

CONFUSING SYMPTOMS. Inadequate metabolism may cause **additional symptoms** that are suggestive of myxedema or, in some patients, just the opposite. The latter group of patients are thin, nervous, and hyperactive, as if to keep warm. Constipation is much more likely than diarrhea. Disturbed menses may be evidenced by menorrhagia,[133] amenorrhea, or irregular menses. When due to hypometabolism, these irregularities are correctable with supplemental thyroid. Hypometabolic patients are likely to suffer from dry, rough skin, which they often mask with an emollient skin cream. Some individuals of this group have difficulty losing weight, which, according to rat experiments,[9] would be aggravated by a thiamine deficiency.

MOLECULAR BASIS. Brent[41] has reviewed the molecular basis of thyroid function. Inactive thyroxine (T_4) is the primary product of the thyroid gland and the dominant form of circulating thyroid hormone. It is converted to the active form triiodothyronine (T_3) by thyroxine 5'-deiodinase. The functions of thyroid hormone are primarily mediated through the action of T_3 receptors of the cell *nucleus.* The receptors are hormone-responsive nuclear transcription factors determining which genes are stimulated or suppressed by T_3. Interaction of the T_3-receptor complex with DNA regulatory regions modifies gene expression. Transport of T, from outside the cell to the cell nucleus is a complex chain of events beyond the reach of current clinical laboratory testing. TRH is regulated through such interaction of T_3 and its receptors in the brain.

The clinical syndrome of hypothyroidism is thus the expression of the combined effects of many gene products that are regulated by T_3, that cause such varied manifestations as hypercholesterolemia and hypertension. Muscle relaxation is controlled by the balance between fast and slow forms of calcium ATPase in the sarcoplasmic membrane of skeletal muscle. The genes for transcription of these two forms of ATPase are controlled by T_3. Likewise, lipogenesis, lipolysis, and levels of total serum cholesterol and low-density lipoprotein cholesterol are controlled by T_3 receptor-regulated genes.

Thermogenesis is regulated in part by T_3 and adrenergic receptors on brown-fat-specific genes found in rodents and recently found in humans.[152] Growth hormone synthesis in the pituitary gland is T_3 regulated, and is decreased in hypothyroidism, including nocturnal secretion of growth hormone and secretion of insulin-like growth factor 1. Of interest is the finding that growth hormone and insulin-like growth factor 1 are decreased in patients with FMS,[27–228] raising the possibility of a T_3 gene receptor regulatory effect in this facet of the syndrome. T3 regulates the transcription of the genes for thyrotropin in an inverse relationship. Finally, Brent points out that resistance to thyroid hormone is associated with abnormalities in the T_3-receptor-beta gene where many different mutations have been identified.[41]

Measurement of Thyroid Function. The measurement of thyroid function has undergone great changes in the past two or three decades. The basal metabolic rate test gave way to thyroxine-based testing that in turn has been replaced by the newer sensitive thyrotropin (sTSH) assays, as reviewed by Klee and Hay[147] sTSH is a reliable assay for stable ambulatory patients with normal pituitary function, because the pituitary gland is a sensitive monitor of the body's requirement for thyroid hormone. Linear

changes in free thyroxine (FTJ concentrations away from an individuals "set-point" for thyroxine results in logarithmic changes in thyrotropin secretion.

Alterations in the binding of thyroxine to serum thyroid transport proteins makes thyroxine concentrations less reliable than sTSH in sick or hospitalized patients. Almost all T_4 and T_3 is bound to one of the three major transport proteins, primarily thyroxine-binding-globulin (TBG). However, only the 0.1% free hormone concentration is active. Drugs that alter the binding of T_4 and T_3 to these proteins will alter total serum levels of T_4 and T_3, but do not affect the serum concentrations of free T_4 and T_3. Elevated sTSH indicates primary hypothyroidism or inadequate thyroid hormone replacement therapy. A very low sTSH level of less than 0.1 mlU/L indicates hyperthyroidism, either exogenous or primary.

Free thyroxine (FTJ measurement gives an indication of the severity of the thyroid dysfunction. FT_4 is elevated in hyperthyroidism and is low in hypothyroidism. Free triiodothyronine (FTJ is useful in the assessment of hyperthyroidism, and is appropriately assessed when sTSH is low and FT_4 is normal.

The most recent third generation TSH assay technique is 100-fold more sensitive than the first generation assay, and can measure O.OlmlU/L. This degree of sensitivity is most useful in evaluating primary hyperthyroidism, to ensure that thyrotropin is truly suppressed, or to monitor the effectiveness of suppression therapy with thyroid hormone. sTSH determinations are not affected by renal or hepatic disease, or by estrogen therapy. Pituitary tumors can sometimes produce TSH, and can cause hyperthyroidism. Pituitary failure causes secondary hypothyroidism, the low sTSH then is accompanied by low FT_4.

The one caveat worth remembering is that persons with acute neuropsychiatric disorders can have altered thyroid function tests, including TSH, and may need multiple studies in order to clarify their thyroid status. This is rarely a problem in myofascial pain patients, however.

RECOMMENDATION. Klee and Hay[147] recommend as a scheme for evaluating thyroid function employing a second generation TSH test that can measure to 0.1 mlU/L. If that is normal, no further testing need be done. If it is elevated, both FT_4 and microsomal antibody tests are done. If it is low (less than 0.3 mlU/L), FT_4 is obtained. If it is normal, FT_3 is obtained. If the second generation sTSH is below 0.1 mIU/1, a third generation sTSH is performed. A laboratory can do this "thyroid cascade" on the initial sample of blood, thereby providing a rapid turnaround time, and minimizing patient discomfort and inconvenience.

Drug Effects on Thyroid Function. Drugs that effect thyroid hormones alter the serum levels of protein bound T_3 and T_4. They may or may not alter free thyroid hormone levels and therefore thyroid function. Drugs like Lithium can also alter secretion of thyroid hormone, resulting in abnormal thyroid function.

Anticonvulsant drugs (phenytoin and carbamazepine) displace thyroid hormone from their binding to serum proteins, resulting in lower serum T_4 and T_3 levels. However, this results in increased free hormone fractions, resulting in normal free T_3 and T_4 concentration.[258] These drugs, as well as phenobarbital, also increase the rate of metabolism of T_4 and T_3, and can cause hypothyroidism in patients treated with thyroxine. However, serum free T_3 and T_4 are normal when measured in undiluted serum.[259] TSH measurements will adequately assess the thyroid function of these patients.

Lithium inhibits the secretion of thyroid hormone. Subclinical hypothyroidism (abnormalities of thyroid function tests) and clinically overt hypothyroidism each occur in 20% of patients taking lithium on a long term basis.[33,259]

Hypothyroidism can be produced by inorganic iodine in excess of that normally present in the diet, and by organic iodine in pharmacologic preparations such as the antiarrhythmic agent amiodarone, the asthma drug combination elixophyllin-KI, and intravenous contrast agents. This is especially true in patients with autoimmune thyroiditis or otherwise impaired damaged thyroid.

TBG concentrations are decreased in patients taking androgens, and glucocorticoid steroids, although free T_3 and T_4 con-

centrations remain unchanged. Salicylates in chronic high doses (>2.0 g/day, or >2.0 g salsalate per day) inhibit the binding of T_3 and T_4 to TBG, but do not affect the serum free T_4 concentration.[31]

Estrogen raises serum TBG concentrations, resulting in elevations of serum T_4 concentrations of 20-35% at usual doses of estradiol (20-35 ug per day). The authors' clinical experience is that TrPs are more common in women with a chronic deficiency of estrogen, and that estrogen supplement decreases TrP activity. Sonkin[252] noted an increase in TrPs with the onset of menopause corrected by estrogen replacement. Thyroid supplementation in hypothyroid patients must be increased during pregnancy, the additional dose determined by the serum TSH level.

Chronic opiate use is becoming more prevalent in treating persons with nonmalignant pain. Hence, physicians treating patients with chronic myofascial pain are now more apt to see the use of drugs like methadone and slow release morphine and oxycodone than previously. Methadone increases serum TBG concentrations, thus raising the serum T_4 concentration, but not necessarily increasing the active, free fraction of the hormone. As with other drug effects, assessment of thyroid function is best made by measuring serum TSH levels in these individuals.

Glucocorticoids in large doses decrease the activity of T_4 5'-deiodinase, inhibiting the conversion of T_4 to T_3, resulting in significant decreases of serum T_3. There is no data available indicating if this change has an effect on presence of TrPs. Serum free T_3 levels usually diminish to low-normal levels, and serum TSH remains normal.

Treatment of Hypothyroidism. Levothyroxine (T_4) is the treatment of choice for hypothyroidism.[248, 270, 279] Adults require about 1.7 ug/kg of body weight for complete replacement of thyroid hormone. In younger individuals, treatment can be initiated at the full dose. In persons over the age of 50, the replacement dose needed may be less, and the starting dose should be 0.025-0.05 mg of levothyroxine daily. In persons with peripheral resistance to thyroid hormone, the eventual dose of T_4 needed to normalize function can be quite high. The maintenance dose is monitored by measuring serum TSH, which should be in the lower normal range. Thyroxine has a half life of about one week. Therefore, the steady state of serum T_4 is not reached for about 4 weeks after initiation of therapy. Tests of serum TSH levels to monitor the dose of thyroxine should be done no sooner than every 4-5 weeks. T_4 is physiologically converted to T_3 at rates that are determined by the state of the individual. Over 80% of circulating T_3 is derived by deiodination of extrathyroidal T_4. The most physiological means of providing T_3, therefore, is to give thyroxine and to let the body needs regulate the rate of conversion of T_4 to T_3.

"Intolerance" to low-dose thyroid therapy repeatedly has been due to this dose aggravating symptoms of vitamin B_1 deficiency. After supplementation with thiamine, administration of the same or larger dose of thyroid medication is well tolerated. Once given for hypothyroidism, thyroid hormone is generally continued for the lifetime of the individual. Several generic and brand name levothyroxine products have been compared and found to be bioequivalent,[74] an important factor in a drug that is being used for long periods of time and in many persons.

Before starting treatment with thyroid hormone it is important that the patient have an adequate vitamin B, level. Since thyroid increases metabolism, and thiamine requirements are metabolism-dependent, thyroid therapy can convert a vitamin B_1 inadequacy to a severe vitamin B_1 deficiency. If there is doubt, the patient can first be given a sufficient supplement of vitamin B_1 to establish a safe level (25-100 mg, three times daily, for at least 2 weeks before starting thyroid medication). Thiamine in a reduced dosage should be continued during thyroid therapy.

Smoking impairs the action of thyroid hormone and will accentuate the clinical features of hypothyroidism, including raising thyrotropin levels, total and LDL cholesterol levels, and CK levels, and prolonging the ankle reflex duration.[184] Every effort should be made to help the patient stop smoking and to prevent others from becoming addicted.

Practical Considerations. Hypothyroidism should be considered in any individual with widespread myofascial pain or widely distributed TrPs. Symptoms of chronic fatigue, coldness or cold intolerance, constipation, and signs of dry skin, dry hair, husky voice, or mild pretibial edema, slowed ankle reflex return, are all tip-offs that there may be hypothyroidism. TSH should be obtained. If it is clearly elevated, then treatment with levothyroxine (T_4) should be started. If the TSH is between 4.0 and 6.0 mIU/L, the sTSH and FT_4 should be evaluated. If these levels are borderline, the CK and serum cholesterol levels can help reach a determination of thyroid status. If either are elevated, then thyroid supplementation can be started. Once supplementation is started, sTSH is used to monitor the result, the target range being 0.5-2.5 mIU/L.

Hypoglycemia

Myofascial TrP activity is aggravated and the response to specific myofascial therapy is reduced or shortened by hypoglycemia. Recurrent hypoglycemic attacks perpetuate myofascial TrPs. The prevalence of hypoglycemia is controversial, largely because the symptoms of hypoglycemia are caused chiefly by increased circulating epinephrine. Other conditions, such as anxiety, also increase epinephrine levels, but without hypoglycemia. Clinically, the responses are often indistinguishable. Two kinds of hypoglycemia are generally recognized, fasting and postprandial; they occur for different reasons, but present the same symptoms.

Symptoms. The initial symptoms of hypoglycemia or of increased epinephrine are usually sweating, trembling and shakiness, a fast heart rate, and a feeling of anxiety. Activation of sternocleidomastoid TrPs may cause headache and dizziness. With progressively severe hypoglycemia due to unusual circumstances, symptoms similar to those of hypoxia develop and are caused by inadequate energy to sustain brain function: visual disturbances, restlessness, impaired speech and thinking, and sometimes syncope.[92]

Fasting Hypoglycemia. Fasting does not cause hypoglycemia in a normal person because the liver releases glucose as the blood glucose starts to fall. Fasting hypoglycemia may result from failure of the liver to release the glucose, failure of the adrenal medulla to produce epinephrine that stimulates the liver to release the glucose, or failure of the anterior pituitary to stimulate the adrenal gland. Liver disease can impair this function of the liver. Alcohol ingestion when glycogen stores in the liver are depleted can precipitate severe hypoglycemia. Rarely, fasting hypoglycemia may be due to the deficiency of an enzyme, such as glucagon.[92]

Postprandial (Reactive) Hypoglycemia. Symptoms of postprandial hypoglycemia typically occur 2 or 3 hours after ingestion of a meal rich in carbohydrates, overstimulating the release of insulin. The insulin triggers a compensatory epinephrine response. The hypoglycemia caused by the insulinemia appears transiently for 15-30 min until it is terminated by the liver's response to an increased epinephrine level. Generally, the epinephrine causes most of the symptoms usually attributed to hypoglycemia. This form of hypoglycemia is associated with high anxiety levels and is most likely to occur during periods of emotional stress.

An individual who has had part of the stomach removed or other gastric surgery may empty the stomach too rapidly. This, too, causes an abrupt rise in blood glucose level, initiating the same sequence of events and causing the same symptoms. The cause of the patient's symptoms is seen more clearly if the symptoms during a glucose tolerance test are correlated with periodic measurement of both blood glucose and serum insulin levels. In the experience of Drs. Travell and Simons, when a glucose tolerance test is done to detect fasting hypoglycemia, a positive result (very low glucose value) is more likely to be obtained if the patient is active rather than resting in the intervals between blood samples.

Fasting hypoglycemia appears many hours after eating and tends to persist while postprandial hypoglycemia is self-limited. A reactive hypoglycemia secondary to mild diabetes is most likely to occur between the third and fifth hours of a glucose tolerance test.[92]

An identifiable organic disease process is usually responsible for fasting hypoglycemia, but not for postprandial hypoglycemia. Diagnosis of postprandial or fasting hypoglycemia requires demonstration of the hypoglycemia while the symptoms are present.

Treatment. In either fasting or postprandial hypoglycemia, the fundamental cause should be identified, if possible. For both, symptoms are relieved by eating smaller meals more frequently and by selecting a diet that is low in carbohydrates (75-100 g), high in protein, and includes sufficient fat to maintain caloric requirements. Exercise tends to aggravate hypoglycemia. However, exercise may help to reduce anxiety and, therefore, symptoms that depend on adrenaline release due to anxiety. In addition, patients must remember that coffee, tea, and colas that contain caffeine or theophylline should not be used because they stimulate the release of adrenaline. Alcoholic beverages should be avoided, particularly on an empty stomach. The nicotine in tobacco stimulates the release of adrenaline, so smoking and exposure to cigarette smoke should be eliminated.

Gouty Diathesis

Clinically, myofascial TrPs are aggravated in patients who have hyperuricemia or gout. The reason is unknown. These patients are susceptible to TrPs and when hyperuricemic respond poorly to myofascial therapy, particularly spray and stretch. Gout is a disorder of purine metabolism; the first indication usually is an elevated serum uric acid (>7.0 mg/dl in men, >6.0 mg/dl in women).[143]

Diagnosis. About 5% of asymptomatic hyperuricemic people (by the above criteria) develop acute gouty arthritis, with deposits of crystals of monosodium urate monohydrate in and around the joints, and sometimes in other tissues.[143]

The saturation value of monosodium urate at the pH of serum is about 7.0 mg/dl;[143] it is less soluble in the more acid medium of injured tissue. A more advanced stage of gout with tophi is now rarely seen since the advent of effective drugs for control of hyperuricemia.[143] Symptoms are more likely to occur in patients on a diet with meats high in purines.

A definite diagnosis of gout is made by identifying uric acid crystals in fluid aspirated from inflamed tissue. The crystals also may be obtained from asymptomatic metatarsophalangeal joints in patients who have had symptoms of gouty arthritis with hyperuricemia.[3]

The deposition of calcium pyrophosphate crystals produces symptoms similar to gout, but no metabolite is known to be present in excess in calcium pyrophosphate disease.

Treatment If hyperuricemia is a probable factor in perpetuating the patient's myofascial TrPs, it should be managed according to well-established principles.[143] Many diuretics increase serum uric acid levels. Vitamin C in relatively large amounts (several grams per day) is an effective uricosuric agent.

The TrPs of patients with a gouty diathesis respond better to treatment when the hyperuricemia is under control, and better to injection than to spray and stretch.

E. PSYCHOLOGICAL FACTORS

A number of psychological factors can contribute to perpetuation of myofascial TrPs. Most important, the physician must be careful *not to assume* that the psychological factors are primary. It is all too easy for the physician to blame the patient's psyche for the inability of the physician to recognize the musculoskeletal sources of the patient's pain. This wrong assumption can be-and often is-devastating to the patient. We have so much to learn about pain, especially pain from muscles!

Patients who misunderstand the nature of their condition may be depressed, may exhibit anxiety tension, or may be victims of the "good sport" syndrome; some may be exhibiting secondary gain and/or sick behavior; a very few will evidence conversion hysteria. Each must be diagnosed on its own merits.

Hopelessness

Patients who have been erroneously convinced that their pain is due to unbeatable physical factors, such as degenerative joint disease, a "pinched nerve" that is inoperable, or "rheumatism" which they must learn to live with, often live in dread

of aggravating their condition by any movement or activity that begins to elicit the pain. The result is that they avoid all painful movements, including those that would stretch the muscles and help them recover function. When their pain is primarily due to myofascial TrPs, this excessive restriction of movement and activity aggravates and perpetuates their TrPs.

An essential first step with these patients is to convince them that their pain is of *muscular origin* and *treatable,* and that they must understand and respect their muscles. Acceptance of this revises the patients' concept of the prognosis. As they learn what activities to avoid and what they themselves can do to inactivate the TrPs, they realize they are gaining control of the source of their pain. This new confidence in the future of their neuromuscular function lifts a great load from their shoulders.

Depression

Depression and chronic pain are closely associated,[256] especially when patients have no satisfactory explanation for the cause of their pain, fear how much worse it may become, are convinced that nothing can be done to correct the source of pain, and believe they must accept it on these terms. The depression is partly a product of chronic pain and dysfunction, so that the longer the duration and the greater the intensity of the pain, the greater the depression is likely to be.[91] *Vice versa,* depressed patients are more aware of pain,[238'256] which contributes to their dysfunction.

The recovery of many patients with myofascial TrPs who are also depressed is expedited by combining antidepressant medication with specific myofascial therapy. Tricyclic drugs are most commonly used, but must be prescribed in sufficient dosage to be effective. Relief of depression permits the patient to take more responsibility for the care of their muscles and to engage in the exercises and activities that will help them to recover. These activities, especially under the direction of a therapist, are an effective antidepressant themselves. In less than antidepressant dosage, tricyclics can reduce pain and improve sleep.

Anxiety and Tension

In some individuals, high levels of anxiety are expressed in the form of muscle tension. Many muscles are held in sustained contraction that overloads- them and perpetuates myofascial TrPs. These patients are easily identified as they sit up stiff and straight, leaning away from the backrest of a chair, maintaining their shoulders in an elevated position, and displaying a tense facial expression. Generally, they are unaware of these muscular expressions of tension. Biofeedback and relaxation therapy can help many of them to discriminate between unnecessarily tense muscles and relaxed ones. They then need to learn conscious techniques of relaxation and how to turn excess tension off. Identifying the major sources of anxiety and emotional tension and adopting the changes in lifestyle necessary to abate them, may be required to reduce this perpetuating factor enough for lasting relief.

"Good Sport" Syndrome

The "good sport" syndrome is the opposite of hypochondriasis. The "good sport" has a stoical attitude and is determined to ignore pain. He or she charges forth engaging in activities with total disregard, if not outright defiance, of the pain, thereby overloading the muscles and aggravating TrPs.

Good sports often believe that their pain is a sign of "weakness" and that they must push on to demonstrate their mastery of it. They must learn how this abuse of their muscles contributes to their pain, and how new ways of doing things can let them perform the activities important to them safely and comfortably.

Psychological and Behavioral Aspects

A psychologically healthy person finds the functional restrictions imposed by a myofascial pain syndrome frustrating and unrewarding. However, among some persons secondary gain can perpetuate pain behavior. Determining whether the loss of function and the pain behavior is primarily psychological or chiefly neurophysiological can be difficult and may be necessary only when the patient fails to respond to

myofascial therapy. Three questions are helpful.

1. How effective were the patient's skills in coping with the problems of life prior to the onset of pain? Ineffective coping skills foster disability and respond best to counseling that is function oriented.
2. Does the patient concentrate on finding ways to do things that circumvent the pain, or focus on reasons why not? The latter suggest that the patient may have a psychological need of the disability.
3. Is function something the patient tries to do, or only talks about? The latter can represent an emotional need of dysfunction, but not necessarily.

In psychological terms, **primary gain** occurs when neurotic patients *unconsciously* develop psychosomatic symptoms (physically expressed) that tend to relieve their high level of anxiety and tension.[47] In the process secondary gains accrue when *some* patients discover that the privileges of a sick person offer exemption from the normal responsibilities of work and/or mature social interactions; they become accustomed to the rewards of having pain. These patients also may simultaneously realize gratification of other unconscious needs, such as a dependency relationship upon a parent-figure, who may be the physician, a spouse, or other care giver. Psychiatrists see **secondary gains** as resulting primarily from psychogenic dysfunction.[47] It is not always that complicated.

Some patients who experience long-standing disabling myofascial pain, not promptly diagnosed and treated, discover advantages that fit this same pattern of secondary gain. The prospect of the beneficial settlement of a law suit or disability claim may loom as a very important secondary benefit to some, but not all, patients. In the presence of neurological or other damage that precludes complete recovery, the financial need is very real. When this issue is discussed openly and the patient's perception of the situation is clearly understood, it usually becomes clear whether the patient considers it in his or her best interest to be as disabled as possible, or to be as functional as possible between now and when the suit is settled.

Sick behavior is behavior that is appropriate to one who is suffering from pain and includes verbalizations, posturing, taking of medication, restriction of activity, increased rest, etc. In time, these reactions to illness can become a self-perpetuating way of life.[91] Elimination of a TrP source of pain can help greatly, but does not automatically reverse this process. The patient, and those with whom he or she lives and interacts closely (including the physician), must replace the reinforcements of the sick behavior with inducements that reinforce normal productive function. The principles of operant conditioning offer a method of treatment in these instances.[91]

Identifying sick behavior that is out of proportion to the pain and suffering experienced by the patient is difficult and hazardous. Only the patient can feel the pain. It is all too easy for the health care professional who is treating the patient to blame treatment failure on psychogenic factors, especially if the professional has found no organic source of the pain such as TrPs. Identification of objective and semi-objective characteristics of myofascial TrPs including motor and autonomic dysfunctions are most helpful.

The answers to two questions also are useful:

1. What was the level of the patient's function before the event that initiated the pain? A higher level of function is not a realistic goal.
2. As TrPs are inactivated, is the patient resuming activities and responsibilities that he or she had been accustomed to, or looking for reasons why it is not possible to take a step forward in function? The latter reaction requires treatment of more than just the TrPs.

Myofascial pain patients with pending law suits or disability claims are faced with the serious dilemma that any relief of their pain and disability would reduce their chances of receiving remuneration. Since a group of patients intuitively senses that the symptoms are critical to the success of the suits, their minds unconsciously concentrate on an awareness of symptoms rather than on function, whether they intend it or not. How much

the patient expects the settlement to mean financially is very important. If it appears to the patient as a major sum, he or she literally cannot *afford* to get better. It is often very helpful for them to clarify in their own minds just how much of the settlement they will receive and how much will go to the lawyers. In the management of these patients, it is essential that they realistically understand the nature of their dilemma. They are strongly encouraged to resolve the dilemma before proceeding with therapy.

F. CHRONIC INFECTION AND INFESTATIONS

Several persistent disease conditions are likely to aggravate myofascial TrPs: viral disease (especially herpes simplex), any chronic focus of bacterial infection, and infestations by certain parasites. The mechanism by which these diseases perpetuate myofascial TrPs is not clear, but the importance of controlling them to obtain lasting relief from myofascial pain has been demonstrated.[287]

Viral Disease

The activity of myofascial TrPs and muscle soreness in general tends to increase markedly during any systemic viral illness; the increased muscle soreness and stiffness may last for several weeks following an acute viral infection, such as the "flu." A common source of increased susceptibility and perpetuation of myofascial TrPs is an outbreak of herpes simplex virus type 1. Neither genital herpes (herpes simplex virus type 2) nor herpes zoster seem to aggravate TrPs as much as herpes simplex virus type 1.

Diagnosis. Because of its recurrent nature, it is important to identify and control outbreaks of the type 1 herpes virus, which causes the common cold sore, canker sores, and often aphthous mouth ulcers; it also may appear on the skin of the body or extremities as crops of isolated vesicles filled with clear fluid. The small vesicles develop a reddened areola and form an eczematous patch on the skin,[156] which may remain for several weeks, if untreated. After the small blisters that are filled with watery fluid (never with pus) break, they become crusted red spots.

Lesions have been reported in the esophagus, and symptoms of vomiting and diarrhea strongly implicate gastrointestinal involvement comparable to that of the mouth.

Treatment. No drug is known to cure herpes simplex. However, by using a multipronged attack, one can greatly reduce the frequency and severity of recurrences of herpes simplex virus type 1. This includes medicinal application to the lesions, oral ingestion of niacinamide and Lactinex[1] and, if necessary, intramuscular injections of human immune serum globulin. Because of the increased irritability of the muscles during an outbreak of herpes simplex virus type 1, it is unwise to inject the muscles for TrPs until a few weeks after the herpes attack has subsided. Treated sooner, the muscles respond poorly to local therapy and are prone to excessive posttreatment soreness.

For local treatment of the herpetic dermal and mouth lesions, idoxuridine (Stoxil[2]) is rubbed into the lesion several times a day. Experience to date suggests, but does not prove, that adenine arabinoside (ara-A, Vira-A[4]) is useful in cutaneous herpes simplex virus type 1.[156] It is sold as a 3% ophthalmic ointment, which also is rubbed into the lesion two or three times a day. It appears to us that ara-A is as effective as idoxuridine. The package insert notes that ingesting as much as a tube of Vira-A should produce no adverse effects. The newly released Zovirax[5] (acyclovir), 5% ointment, is promoted for treatment of initial attacks of herpes simplex virus type 2; it also may prove to be effective for type 1 herpes simplex.

Administration of niacinamide, 300-500 mg/day, helps the mucous membrane combat the gingivostomatitis of oral herpes simplex (type 1). At the same time, it is important also to correct any folic acid deficiency.

[1]Lactinex Tablets and Granules, Hynson, Westcott & Dunning, Division of Becton Dickinson & Co., Charles & Chase Sts, Baltimore, MD 21201.
[2]Stoxil, ophthalmic ointment, 0.5%, and ophthalmic solution 0.1%, Smith Kline & French Laboratories, Division of SmithKline Corporation, 1500 Spring Garden St., P. O. Box 7929, Philadelphia, PA 19101.
[4]Vira-A, ophthalmic ointment, Parke-Davis Division of Warner-Lambert Company, 201 Tabor Road, Morris Planes, NJ 07950.
[5]Zovirax Ointment 5%, Burroughs Wellcome Co., 3030 Cornwallis Road, Research Triangle Park, NC 27709.

Empirically, the symptoms due to extension of herpetic lesions into the small intestine are relieved by taking 1 packet of granules or 3 tablets of Lactinex* 2 or 3 times daily for at least a month, with subsequent reduction in dosage, unless the oral lesions reappear. A similar course of Lactinex (or yogurt with active live cultures) is usually valuable after antibiotic therapy that suppresses normal intestinal bacteria. Lactinex is a preparation of living *Lactobacillus acidophilus* and L. *bulgaricus.* The intestinal component of herpes is an unseen and generally unappreciated site of infection. *Lactobacillus* therapy is an important part of the total treatment plan.

Patients who have recurrent episodes of diarrhea associated with outbreaks of oral herpes, also tend not to drink milk. When asked, they are not sure why; they "just don't like it." In fact, they may have a lactose intolerance, and as a result, milk causes diarrhea. It is, therefore, important in these cases to measure their serum *ionized* calcium, which is often low even though the serum *total* calcium is normal. An adequate calcium intake must be provided.

When the patient has a series of herpetic recurrences, or a crop of herpes reactivates TrPs, human immune serum globulin can be injected intramuscularly, 0.04 ml/kg (0.02 ml/lb). This usually amounts to a total dose of 2-3 ml/injection. The effectiveness of the viral antibodies from the pooled serum is temporary.

Bacterial Infection

Absorption of bacterial (and viral) toxic products favors the development of active TrPs when minor mechanical stress is added.[272] Common locations of chronic bacterial infection are an abscessed tooth, a blocked sinus, and the urinary tract. Such a chronic infection may increase the erythrocyte sedimentation rate, which is a useful screening test. Specific myofascial therapy is unlikely to produce lasting benefits while a focus of chronic infection persists.

Abscessed or Impacted Tooth. The chronic infection of a tooth is suspected from a careful dental history and confirmed by a dental evaluation with an X-ray examination. Impaction of a wisdom tooth can perpetuate TrPs in the masticatory muscles, even when local infection is not present.

Sinusitis. Sinusitis is characterized by a sense of fullness in the sinus area, postnasal discharge that may be purulent, and failure of the occluded sinus to transilluminate clearly. If there is an allergic component, the patient is likely to have an eosinophilia. Control of inhalant allergies is generally a prerequisite to a lasting resolution of sinusitis. If there is additional mechanical blockage to sinus drainage as by a deviated nasal septum, this also may require correction in order to resolve recurrent sinus infection.

Chronic Urinary Tract Infection. The symptoms of nocturia, dysuria and urgency should arouse the suspicion of a urinary tract infection, especially in female patients. The infection is confirmed by urinalysis and urine culture; it is best managed by the urologist. This specialist can determine the extent of the infection and whether there is incomplete emptying of the bladder, or another cause of the infection.

Infestations

Three infestations are likely to perpetuate myofascial pain symptoms. The fish tapeworm is the worst offender; next is giardiasis. Occasionally amebiasis perpetuates myofascial TrPs. The first two tend to impair absorption of nutrients or consume vitamin B_{12}; the third may produce myotoxins that are absorbed.

Fish Tapeworm. The adult worm of *Diphyllobothrium latum* resides in the intestinal lumen. The infestation develops after ingestion of raw infected fish. Infestation is relatively common in a number of foreign countries in temperate climates where it is common practice to eat raw fish, also in Florida, in the northern central United States, and in south-central Canada.

A worm located high in the jejunum may consume 80-100% of ingested labeled vitamin B_{12}, and thus deprive its host of that vitamin.[118] Since the eggs are discharged in large numbers into the stool, they are easily diagnosed by stool examination for ova and parasites.[211]

Giardiasis. The single-celled protozoan, *Giardia lamblia,* is a significant

cause of traveler's diarrhea, particularly in the Caribbean countries, Latin America, India, Russia, and the Far East.[210] It is a pear-shaped, flagellated parasite that lives in the human duodenum and jejunum, where it multiplies. It was isolated in 3.8% of stools examined in the United States.

The infestation is often asymptomatic, but may cause nausea, flatulence, epigastric pain, and watery diarrhea with bulky malodorous stools. The acute symptoms are usually limited to a few weeks, but chronic giardiasis can cause malabsorption of carbohydrate, fat and vitamin B_{12}. The lack of vitamin B_{12} perpetuates myofascial TrPs.

Diagnosis is made by identifying the cysts in formed feces, or by finding the trophozoites in diarrheal stools, in duodenal secretions, or in jejunal biopsies. In chronic cases, excretion of the organism is often intermittent, and stool specimens must be collected at weekly intervals for 4-5 weeks to exclude this diagnosis.[210]

Amebiasis. Only *Entamoeba histolytica* is pathological among the amebas that parasitize the human intestinal tract. The mature *E. histolytica* lives in the lumen of the large intestine, feeding on bacteria and debris; occasionally it invades the mucosa, causing ulcerations.[209]

Stool surveys reveal the prevalence of this parasite in the United States to be between 1 and 5%, but rates are much higher in tropical areas where the levels of sanitation are low and among groups who spread it by direct fecal-oral contact between sexual partners.[209]

The diagnosis depends on the identification of the organism in the stool or tissue from the large intestine. The microscopic demonstration of this infestation may be difficult. Serological tests using purified antigens are positive in most patients with acute amebic dysentery, but are generally negative in asymptomatic passers of cysts.

These tests should be useful in myofascial pain patients because aggravation of myofascial TrPs by *E. histolytica* probably requires tissue invasion. Antibody titers may be elevated for months to years after complete cure.[209] Treatment is difficult and a cure generally requires a combination of drugs.[209]

G. OTHER FACTORS

Three additional factors, allergic rhinitis, impaired sleep, and nerve impingement, should be considered in the management of myofascial pain syndromes.

Allergic Rhinitis

Many patients with active myofascial TrPs, who also have active symptoms of allergic rhinitis, have been found to respond only temporarily to specific myofascial therapy. When the allergic symptoms are controlled, the muscle response to local TrP therapy usually improves significantly. Hypersensitivity to allergens, with histamine release, seems to act as a perpetuating factor for myofascial TrPs.

Koenig, *et al.*[151] examined 20 "fibrositis" patients with histories that were compatible with a diagnosis of myofascial TrPs and tender areas that responded to palpation with a "jump sign." Of the 20 patients, 9 (45%) had convincing histories of either prior or current allergic rhinitis, and 11 of the 20 had positive family histories of allergy. However, none of the 20 patients showed elevated immunoglobulin E levels or an increased total eosinophile count. From this, the authors concluded that it was unlikely that type 1 hypersensitivity played a role in the pathogenesis of fibrositis. It appears that myofascial pain syndromes are not likely to be activated by an allergy. However, we do find that among a certain number of patients with an active allergic state, the allergy significantly perpetuates the activity of their myofascial TrPs. This uncontrolled study[151] did not address the question, "Does the presence of allergy impede the response to treatment of TrPs?" This question needs to be critically evaluated in a research study.

Diagnosis. Allergic rhinitis is characterized by episodic sneezing, rhinorrhea, obstruction of the nasal passages, conjunctival and pharyngeal itching, and lacrimation. Allergic rhinitis predisposes to upper respiratory infection.[11] The initial diagnosis depends largely on the correlation between exposure to the allergen and appearance of symptoms, both as related to time and place. The peripheral blood and nasal secretions of patients with active allergic rhinitis are rich in eosinophiles. Total

serum immunoglobulin E is frequently elevated, and the demonstration of antibodies to a specific antigen confirms an etiologic diagnosis. A number of radioimmune tests are now used.[11]

Skin testing is useful for detecting sensitivity to inhalant allergens, but questionable for food allergens. Food allergies are common and potent,[63] and should be considered as a possible perpetuator of myofascial TrPs. Some patients exhibit an idiosyncratic muscle reaction to alcoholic beverages, experiencing an attack of myofascial pain soon after, or the day, following indulgence.

In most patients, the upper respiratory tract and eyes, the bronchi, the skin, or the joints are the shock organs for allergic reactions. However, in other patients, the skeletal muscles appear to serve as the shock organ for allergies.

Treatment. Most important is avoidance of exposure to the allergen. For inhalant allergies, a room model electrostatic air cleaner is effective, if the air in that room is independent of the air circulating throughout the house. Some portable room models are suitable for use on trips.

Antihistamines effectively control one mediator of allergy, the mast cell-derived reaction, and can be valuable for controlling symptoms of allergic rhinitis. Either Dramamine, 50 mg, or Phenergan, 12.5 or 50 mg, taken shortly before bedtime help to induce sleep. Dramamine is relatively short acting and can be repeated during the night, if needed. These antihistamines are discussed under Drugs in Chapter 3.

If antihistaminics provided inadequate control, treatment by hyposensitization can be helpful.[11]

Impaired Sleep

Impaired or interrupted sleep, in our experience, occurs with greater frequency in patients with more severe myofascial pain syndromes. Smythe,[250] when redefining "fibrositis," considered disturbed sleep so important that he made it one of four essential diagnostic criteria. Sleep studies[180] in 10 patients with "fibrositis" revealed a decrease in the amount of slow wave activity and intrusion of a rapid alpha rhythm during stages 3 and 4 of sleep. All patients showed an overnight increase in the tenderness of the tender points in their muscles. This redefinition of fibrositis is now know as fibromyalgia.

In many patients with myofascial TrPs, the sleep disturbance can be specifically related to referred pain caused by lying on a TrP, or sleeping with an involved muscle in the fully shortened position. Inactivation of the TrP permits return to a clinically normal sleep pattern. Other patients are disturbed by noise, which can be corrected with cotton in the ears or suitable ear plugs. Some patients are disturbed by depression, which should be managed by antidepressant medication as indicated.

However, Moldofsky and Scarisbrick[179] found muscle tenderness and a sense of physical tiredness in the morning in healthy university students when the slow wave non-REM (rapid eye movement) sleep had been disrupted throughout the night. This finding demonstrates the basis for a vicious cycle. The painful muscles interrupt sleep, and disrupted sleep can make the muscles more painful.

History. A careful inquiry as to the precise nature of the sleep disturbance helps to determine what is causing it. Is the difficulty primarily falling asleep, or staying asleep? Anxious and tense patients have trouble falling asleep, depressed patients are likely to awaken during the night. When, during the night, does the patient awaken? This information helps to identify the cause. Was the patient chilly, or in pain? What was the sleeping position? The position helps to identify what TrPs may be responsible for pain. Some patients with a severe myofascial pain syndrome can sleep in the sitting position only. How does the patient get back to sleep again? Is the lack of sleep at night compensated by sleep during the day?

Treatment. Inactivation of the TrPs that are disrupting sleep holds top priority. If going to sleep is a problem, a warm bath and/or a glass of milk before retiring may help induce sleep (provided the patient likes and digests milk).

An electric blanket is most helpful to prevent chilling of the body and eliminate compensatory muscular contractions to generate heat. The thermostat should be

adjusted to slightly above room temperature before retiring by turning the blanket on and the temperature control up, just beyond the "on" click.

Pillow positioning can be the key to restful sleep. When neck and shoulder muscles are involved, the corners of the pillow can be tucked between the ear or chin and the shoulder to prevent tilting of the head and neck to keep the shoulder from riding up against the neck. Blocks under the feet of the head end of the bed are very helpful in this case, as noted in Chapter 3, Section 14. Excessive neck flexion should be avoided and the pillow should be flat enough to maintain the normal lordotic curve of the cervical spine. An additional small pillow can be positioned to prevent shortening of involved shoulder-girdle and arm muscles during the night. Specific details are described in the individual muscle chapters.

The use of drugs has been mentioned in the previous section, and is discussed in Chapter 3. The value of melatonin to reset a disturbed sleep cycle is reviewed under **Pain Relief** in Section 12 of Chapter 3.

Nerve Impingement

Both myofascial TrP syndromes and peripheral nerve entrapments including radiculopathies are very common. An EMG study of TrPs in lumbar muscles that also examined for early EMG evidence of nerve compromise found a significant correlation,[59] which was reinforced by a subsequent study.[60] The authors[293] of another study examined patients with radiculopathy due to disc lesions before and 4 weeks after surgery. There was a tendency for active TrPs to be present in muscles of the involved extremity corresponding to the level of root involvement, especially for L_5 innervated muscles. The TrPs were effectively inactivated by the surgery.

One cannot assume that the presence of radiculopathy activated the TrPs just because they occurred together in the same individual, however, the studies noted above are strongly suggestive. The distinction is clouded by the fact that TrPs activated as satellites of the original pain of radiculopathy may refer pain in patterns that mimic the radicular pain. Nevertheless, one of the authors (RDG) has seen a number of individuals present with acute TrP syndromes in the shoulder or in the hip and lower extremity that respond to manual and injection therapy, only to recur within a day or two, and then return within days to a week or two with a fully developed clinical picture or radiculopathy that was not present before (weakness, altered tendon reflexes, and sensory loss). The findings in these patients indicate that acute radiculopathy can present as myofascial pain symdrome (MPS).

The two conditions may appear as one in the postdisc syndrome but in reality are separate entities. These patients continue to experience pain following a well performed and truly needed laminectomy. They suffer from continuing activity of myofascial TrPs in muscles that refer pain in much the same distribution as that of the previous radicular pain. The postlumbar-laminectomy pain syndrome described by Rubin[225] demonstrates the postdisc syndrome of the lumbar spine. Recurrent disc herniation and postoperative scar tissue formation with root compression must be identified and treated, but even in these cases, the pain often comes from a myofascial TrP. In the lumbar disc syndrome involving the S_1 root, TrPs in the hamstring muscles are commonly the cause of the ongoing pain.

Recognition and inactivation of the myofascial TrPs that remained following a successful laminectomy for nerve root compression has provided complete and lasting relief in many patients.

H. SCREENING LABORATORY TESTS

The following tests are valuable in the detection of perpetuating factors in patients with chronic myofascial pain, or in any patient with myofascial TrPs who responds poorly to specific myofascial therapy. The hematologic profile, blood chemistry profile, and vitamin tests are done routinely. Thyroid tests are done when indicated by history and physical findings.

Hematologic Profile

The erythrocyte sedimentation rate (ESR) is normal in uncomplicated MPS. A

normal ESR helps to eliminate the possibility of a chronic bacterial infection. When elevated, it is nonspecific and may indicate other conditions, such as polymyositis, polymyalgia rheumatica, rheumatoid arthritis, or cancer.

A decreased erythrocyte count, low hemoglobin, and/or microcytosis indicates anemia, which tends to make the muscles hypoxic and to increase TrP irritability. Iron deficiency is identified by a low serum ferritin level. Anemia can be caused by a folate and/or cobalamin deficiency, each of which additionally increases TrP irritability. An increased mean corpuscular volume of >92 fl is suspicious. As it rises from 95 to 100 fl, the likelihood of a folate or a cobalamin deficiency increases.

Eosinophilia may be due to an active allergy, or to infestation with an intestinal parasite, such as *E. histolytica* or a tapeworm.

An increased proportion of mononuclear cells (>50%) may occur because of low thyroid function, or due to active infectious mononucleosis or an acute viral infection.

Blood Chemistry Profile

An automated blood chemistry profile is a useful screening test. Increased serum cholesterol can result from decreased thyroid function, whereas a low serum cholesterol may reflect folate deficiency. Elevated levels of uric acid identify hyperuricemia, which occasionally results in gout. A low serum total calcium suggests a calcium deficiency, but for determination of the adequacy of available calcium, a serum *ionized* calcium measurement is needed.

Low serum potassium can cause muscle cramps and is likely to perpetuate myofascial TrPs.

An elevated fasting blood sugar deserves further investigation to rule out diabetes with a 2-hour postprandial blood glucose or a glucose tolerance test. Measurement of sensory nerve conduction velocities can help to rule out or substantiate diabetic neuropathy.

Vitamin Determination

Serum levels of vitamins B_1, B_6, B_{12}, folic acid, and vitamin C can be enormously valuable in the rational management of patients with myofascial pain syndromes. Abnormally low levels of any of these vitamins perpetuate TrPs. Values in the ***lower quartile*** of normal are less than optimal and are highly suspect as perpetuators of myofascial TrPs. Since a battery of B_{12} and folic acid levels is readily available and not unreasonably expensive, it can be cost-effective in a substantial percentage of patients with *chronic* myofascial pain to obtain this battery *routinely* initially. These patients, because of their chronic TrP problem, are a select group who are more likely than most patients to have vitamin inadequacy.

Thyroid Tests

TSH measures the adequacy of hormone production by the thyroid gland. When the TSH is low, low T_4 levels will identify pituitary failure. The third generation sTSH test and T_3 will evaluate hyperthyroidism, whereas sTSH and free T_4 are used to assess the adequacy of thyroid replacement.

REFERENCES

1. Abt AF, von Schuching S, Enns T: Vitamin C requirements of man re-examined. *Am J Clin Nutr* 12:21-29, 1963.
2. Adams RD, Asbury AK: Diseases of the peripheral nervous system. Chapter 377. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et al.* McGraw-Hill, New York, 1980 (p. 2039).
3. Agudelo CA, Weinberger A, Schumacher HR, *et al.:* Definitive diagnosis of gout by identification of urate crystals in asymptomatic metatarsophalangeal joints. *Arthritis Rheum* 22-559-560, 1979.
4. Aidley DJ: *The Physiology of Excitable Cells.* Cambridge University Press, Cambridge, 1971 (pp. 115, 228).
5. Akerblom B: *Standing and Sitting Posture.* A.B. Nordiska Bokhandein, Stockholm, 1948.
6. Ampola MG, Mahoney MJ, Nakamura E, *et al.:* Prenatal therapy of a patient with vitamin B_{12} responsive methylmalonic acidemia. *N Engl J Med* 293:314-317, 1975.
7. Anderson CE: Vitamins. Chapter 3, In: *Nutritional Support of Medical Practice.* Edited by Schneider HA, Anderson CE, Coursin DB, Harper & Row, Hagerstown, Md. 1977 (pp. 25-27).
8. Anderson R: Ascorbic acid and immune function. In: *Vitamin Ascorbic Acid.* Edited by Counsell JN, Hornig DH. London, 1981.
9. Appledorf H, Newberne PM, Tannenbaum SR: Influence of altered thyroid status on the food intake and growth of rats fed a thiamine-deficient diet. *J Nutr* 97:271-278, 1969.
10. Argov Z Renshaw PF, *et al.:* Effects of thyroid hormones on skeletal muscle bioenergetics. In vivo phosphorous-31 magnetic resonance spectroscopy

study of humans and rats. / *Clin Invest 81:*1695-1701, 1988.

11. Austen KF: Diseases of immediate type hypersensitivity. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et al.* McGraw-Hill, New York, 1980 (pp. 345-347).
12. Avioli LV: Calcium and phosphorous. Chapter 7A. In: *Modern Nutrition in Health and Diseases.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 298, 305).
13. Azuma J, Kishi T, Williams RH, ef *al:* Apparent deficiency of vitamin B„ in typical individuals who commonly serve as normal controls. *Res Commun Chem Pathol Pharmacol* 14:343-348, 1976.
14. Babior BM, Bunn HF: Megaloblastic anemias. Chapter 311. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et al.* McGraw-Hill, New York, 1980 (pp. 1518-1524).
15. Bailey LB, Mahan CS, Dimperio D: Folacin and iron status in low-income pregnant adolescents and mature women. *Am J Clin Nutr* 33:1997-2001, 1980.
16. Baines M: Detection and incidence of B and C vitamin deficiency in alcohol-related illness. *Ann Clin Biochem* 15:307-312, 1978.
17. Baker H, Frank O: Vitamin status in metabolic upsets. *World Rev Nutr Diet* 9:124-160, 1968.
18. Baker H, Frank O, Feingold S, *et al:* Vitamins, total cholesterol, and triglycerides in 642 NY City school children. *Am J Clin Nutr* 20:850-857, 1967.
19. Baker H, Frank O, Hutner SH: Vitamin analyses in medicine. Chapter 20. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 612, 621-624).
20. Baker H, Frank O, Zetterman RK, *et al:* Inability of chronic alcoholics with liver disease to use food as a source of folates, thiamin, and vitamin B₁. *Am J Clin Nutr* 28:1377-1380, 1975.
21. Beal MC: A review of the short-leg problem. *JAOA* 50:109-121, 1950.
22. Beck WS, Goulian M: Drugs effective in pernicious anemia and other megaloblastic anemias. Chapter 51. In: *Drill's Pharmacology in Medicine.* Ed. 4. Edited by Dipalma JR. McGraw & Hill, New York, 1971 (pp. 1062-1074).
23. Beard J, Borel M: Iron deficiency and thermoregulation. *Nutrition Today 23:42-45,* 1988.
24. Beard J, Tobin B, *et al:* Norepinephrine turnover in iron deficiency at three environmental temperatures. *Am J Physiol 255.*R90-R96, 1988.
25. Beard JL; Borel MJ, *et al:* Impaired thermoregulation and thyroid function in iron- deficiency anemia. *Am J Clin Nutr* 52:813-819, 1990.
26. Bendall JR: *Muscles, Molecules and Movement.* American Elsevier Publishing Company, New York, 1969 (p. 162).
27. Bennett RM, Clark SR, *et al.:* IGF-1 assays and other GH tests in 500 fibromyalgia patients [Abstract], /*Musculoske Pain* 3:109, 1995.
28. Berger L, Gerson CD, Yu T: The effect of ascorbic acid on uric acid excretion with a commentary of the renal handling of ascorbic acid. *Am J Med* 62:71-76, 1977.
29. Bernat I: *Iron metabolism.* Plenum Press, New York, 1983.
30. Bezzano G: Effects of folic acid metabolism on serum cholesterol levels. *Arch Intern Med* 124:710-713, 1969.
31. Bishnoi A, Carlson HE, *et ah:* Effects of commonly prescribed nonsteroidal anti- inflammatory drugs on thyroid hormone measurements. *Am J Med* 96:235-238, 1994.
32. Blum A: Do cigarette smokers need vitamin C supplementation? *JAMA* 244:193, 1980.
33. Bochetta A, Bernardi F, *et al.:* Thyroid abnormalities during lithium treatment. *Acta Psychiatr Scand* 83.193-198, 1991.
34. Boni L, Kieckens L, Hendrikx A: An evaluation of a modified erythrocyte transketolase assay for assessing thiamine nutritional adequacy. / *Nutri Sci Vitaminol* 26:507-514, 1980.
35. Botez MI, Cadotte M, Beaulieu R, *et al.:* Neurologic disorders responsive to folic acid therapy. *Can Med Assoc J* 115:217-222, 1976.
36. Botez MI, Peyronnard JM, Bachevalier J, *et al.:* Polyneuropathy and folate deficiency. *Arch Neurol* 35:581-585, 1978.
37. Botez MI, Peyronnard JM, Charron L: Polyneuropathies responsive to folic acid therapy. Chapter 36. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (p. 411).
38. Bothwell TH: Overview and mechanisms of iron regulation. *Nutrition Rev* 53:237-245, 1995.
39. Bourdillon JF: *Spinal Manipulation.* Ed. 2. Appleton-Century-Crofts, New York, 1973 (pp. 39-43, Figs. 5-10).
40. *Ibid.* (pp. 82-86).
41. Brent GA: The molecular basis of thyroid hormone action. *N Engl J Med* 332:847-853, 1994.
42. Brooke MH: *A Clinicians View of Neuromuscular Disease.* Williams & Wilkins, Baltimore, 1977.
43. Bueding E, Stein MH, Wortis H: Blood pyruvate curves following glucose ingestion in normal and thiamine-deficient subjects. / *Biol Chem* 140:697-703, 1941.
44. Calder JH, Curtis RC, Fore H: Comparison of vitamin C in plasma and leucocytes of smokers and non-smokers. *Lancet* 1:556, 1963.
45. Cameron E: Biological function of ascorbic acid and the pathogenesis of scurvy. *Med Hypotheses* 2:154-163, 1976.
46. Cameron E, Pauling L: *Cancer and vitamin C.* Linus Pauling Institute of Science and Medicine, Menlo Park, Calif. 1979.
47. Cameron N: *Personality Development and Psychopathology: A Dynamic Approach.* Houghton Mifflin, Boston, 1963.
48. Canham JE, Baker EM, Harding RS, *et al.:* Dietary Protein-its relationship to vitamin B„ requirements and function. *Ann NY Acad Sci* 166:16-29, 1969 (pp. 16-29).
49. Carmel R, Johnson CS: Racial patterns in pernicious anemia. *N Engl J Med* 298:647- 650, 1978.
50. Carmel R, Sinow RM, *et al.:* Atypical cobalamin deficiency. Subtle biochemical evidence of deficiency is commonly demonstrable in patients without megaloblastic anemia and is often associated with protein-bound cobalamin malabsorption. / *Clin Med* 209:454-463, 1987.

51. Carmel R; Weiner JM *et al.:* Iron deficiency occurs frequently in patients with pernicious anemia. *JAMA* 257:1081-1083, 1987.
52. Carney MW: Psychiatric aspects of folate deficiency. Chapter 42. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (pp. 480-482).
53. Carney MW, Williams DG, Sheffield BF: Thiamine and pyridoxine lack in newly- admitted psychiatric patients. *Br J Psychiatry* 135:249-254, 1979.
54. Cass LJ, Frederik WS, Cohen JD: Chronic disease and vitamin C. *Geriatrics* 9:375- 380, 1954.
55. Chanarin I: *The megaloblastic anemias.* Ed. 2. Blackwell: Oxford, 1979.
56. Charlton RW; Bothwell TH: Iron absorption. *Ann Rev Med* 34:55-68, 1983.
57. Chaudhuri CR, Chatterjee IB: L-Ascorbic acid synthesis in birds: phylogenetic trend. *Science* 164:435-436, 1969.
58. Cheraskin E, Ringsdorf WM Jr: A relationship between vitamin C intake and electrocardiography. / *Electrocardiol* 12:441, 1979.
59. Chu J: Dry needling (intramuscular stimulation) in myofascial pain related to lumbosacral radiculopathy. *EurJPhys Med Rehabil* 5(4):106-121, 1995.
60. Chu J: Twitch-obtaining intramuscular stimulation: its effectiveness in the long-term treatment of myofascial pain related to lumbosacral radiculopathy [Abstract]. *Arch Phys Med Rehabil* 78:1024, 1997.
61. CiaccioEI: The vitamins. Chapter 62. In: *Drill's Pharmacology in Medicine.* Ed. 4. Edited by DiPalma JR. New York, McGraw-Hill, 1971 (pp. 1293-1294).a
62. *Ibid.* (pp. 1282-1284, 1287-1290).b
63. Crook WG: Can what a child eats make him dull, stupid, or hyperactive? *J Learn Disabil* 13:281-286, 1980.
64. Danese MD, Powe NR, *et al.:* Screening for mild thyroid failure at the periodic health exam. *JAMA* 276:285-292, 1996.
65. Davalos A, Fernandez-Real JM, *et al.:* Iron-related damage in acute ischemic stroke. *Stroke* 25:1543-1546, 1994.
66. Dayan CM, Daniels GH: Chronic autoimmune thyroiditis. *NEngl J Med* 335:99-107, 1996.
67. DeMaeyer E, Adiels-Tegman M: The prevalence of anemia in the world. *World Health Statist Q* 36:302-316, 1985.
68. Dempsey WB: Vitamin B_6 and pregnancy. Chapter 12. In: *Human Vitamin B_6 Requirements:* National Academy of Sciences, Washington, 1978 (pp. 202, 203).
69. Dickerson JW: Vitamin requirements in different clinical conditions. *Biblthca Nutr Dicta* 35:44-52, 1985.
70. Diffrient N, Tilley AR, Bardagiy JC: *Humanscale 1/2/3.* Massachusettes Institute of Technology Press, Cambridge, 1974 (pp. 19-22).
71. Dillman E, Johnson DG, ef *al:* Catecholamine elevation in iron deficiency. *Am J Physiol* 237.R297-R300, 1979.
72. Dipalma JR: Vitamin toxicity. *Am Fam Phys* 18:106-109, 1978.
73. Donaldson RM Jr.: Serum B_{12} and the diagnosis of cobalamin deficiency. *N Engl J Med* 299:827-828, 1978.
74. Dong BJ, Hauck WW, ef *al:* Bioequivalence of generic and brand-name levothyroxine products in the treatment of hypothyroidism. *JAMA* 277:1205-1213, 1997.
75. Dreosti IE: Magnesium status and health. *Nutrition Reviews* 53.-S23-S27, 1995.
76. Driskell JA: Vitamin B_{12} status of the elderly. Chapter 16. In: *Human Vitamin B_{12} Requirements.* National Academy of Sciences, Washington, 1978 (pp. 252-255).
77. Driskell JA, Chrisley BM, ef *al.:* Plasma pyridoxal phosphate concentrations of men fed different levels of vitamin B-6. *Am J Clin Nutr* 48:122-126, 1988.
78. Dumitru D: Electrodiagnostic Medicine. Hanley & Balfus, Philadelphia, 1997 (pp. 1083-1084).
79. Ebadi M: Vitamin B_{12} and biogenic amines in brain metabolism. Chapter 8. In: *Human Vitamin B_{12} Requirements,* National Academy of Sciences, Washington, 1978 (pp. 129- 150).
80. Ellis JM, Kishi T, Azuma J, et *al.:* Vitamin B_{12} deficiency in patients with a clinical syndrome including the carpal tunnel effect. Biochemical and clinical response to therapy with pyridoxine. *Res Commun Chem Pathol Pharmacol* 3:743-757, 1976.
81. Ellis JM, Presley J: *Vitamin B_{12}: The Doctor's Report.* Harper & Row, New York, 1973 (pp. 74-78).
82. Ellis JM, Folkers K, ef *al.:* Response of vitamin B-6 deficiency and the carpal tunnel syndrome to pyridoxine. *Proc Natl Acad Sci USA* 79:7494-7498, 1982.
83. Engle WK: Ponderous-purse disease. *NEngl f Med* 299:557, 1978.
84. Erbe RW: Inborn errors of folate metabolism. JV *Engl J Med* 293:753-758, 807-811, 1975.
85. Evans JR, Hughes RE, Jones PR: Some effects of cigarette smoke on guinea-pigs. *Proc Nutr Soc* 26:36, 1967.
86. Festen HP: Intrinsic factor secretion and cobalamin absorption. Physiology and pathophysiology in the gastrointestinal tract. *Scand J Gastroenterol 188(Suppl):l-7* 1991.
87. Finck CA, PD G ef *al.:* Lactic acidosis as a result of iron deficiency. / *Clin Invest* 64.129-137, 1979.
88. Folkers K, Watanabe T, Ellis JM: Studies on the basal specific activity of the glutamic oxaloacetic transaminase of erythrocytes in relation to a deficiency of vitamin B_{12}. *Res Commun Chem Pathol Pharmacol* 17:187-189, 1977.
89. Fong T: Problems associated with megadose vitamin C therapy. *West J Med* 134:264, 1981.
90. Ford LT, Goodman FG: X-ray studies of the lumbosacral spine. South *Med J* 59:1123- 1128, 1966.
91. Fordyce WE: *Behavioral Methods for Chronic Pain and Illness,* C.V. Mosby, Saint Louis, 1976 (pp. 72-73).
92. Foster DW, Rubenstein AH: Hypoglycemia, insulinoma, and other hormone-secreting tumors of the pancreas. Chapter 340. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, et *al.* McGraw-Hill, New York, 1980 (pp. 1758-1762).
93. Fox BA, Cameron AG: *Food Science, Nutrition and Health.* Ed. 6. Edward Arnold, London, 1995.
94. Franzblau A, Rock CL, ef *al:* The relationship of vitamin B6 status to median nerve function and

carpal tunnel syndrome among active industrial workers. *JOEM* 38:485- 491, 1996.

95. Gerwin R: A study of 96 subjects examined both for fibromyalgia and myofascial pain. *JMusculoske Pain* 3.121, 1995.
96. Gerwin RD, Gevirtz R: Chronic myofascial pain: iron insufficieny and coldness as risk factors. / *Musculoske Pain* 3:120, 1995.
97. Ginter E. Ascorbic acid synthesis in certain guinea pigs. *Int J Vitamin Res* 46:173-179, 1976.
98. Ginter E: Chronic marginal vitamin C deficiency: biochemistry and pathophysiology. *World Rev NutrDiet* 33:104-141, 1979.
99. Ginter E: What is truly the maximum body pool size of ascorbic acid in man? *Am J Clin Nutr* 33:538, 1980.
100. Goldsmith GA: Curative nutrition: vitamins. Chapter 7. In: *Nutritional Support of Medical Practice.* Edited by Schneider HA, Anderson CE, Coursin DB. Harper & Row, Hagerstown, MD., 1977 (pp. 103-106).
101. *Ibid.* (pp. 108. 109).
102. *Ibid.* (pp. 113-114).
103. Gould N: Back-pocket sciatica. JV *Engl J Med* 290:633, 1974.
104. Green R, Kinsella LJ: Current concepts in the diagnosis of cobalamin deficiency [Editorial]. *Neurology* 45:1435-1440, 1995.
105. Griner PF, Oranburg PR: Predictive values of erythrocyte indices for tests of iron, folic acid, and vitamin B_{12} deficiency. *Am J Clin Pathol* 70:748-752, 1978.
106. Guard O, Dumas R, Audry D, *et al:* [Clinical and pathological study of a case of subacute combined degeneration of the cord with folic acid deficiency]. *Rev Neurol (Paris)* 137:435-446, 1981.
107. Hall CA: Function of vitamin B12 in the central nervous system as revealed by congenital defects. *Am JAnaesth* 34:121-127, 1990.
108. Hallberg L, Brune M, Erlandsson M, ef *al:* Calcium effect of different amounts on nonheme and heme-iron absorption in humans. *Am J Clin Nutr* 53:112-119, 1991.
109. Harris RI, Beath T: The short first metatarsal, its incidence and clinical significance. *J Rone Joint Surg* 31-A:553-565, 1949.
110. Harris AD, Heatley RV: Nutritional disturbances in Crohn's disease. *Postgrad Med* 59:690-697, 1983.
111. Haskell BE: Analysis of vitamin $B_{„}$. Chapter 4. In: *Human Vitamin R_k Requirements.* National Academy of Sciences, Washington, 1978 (pp. 61, 67).
112. Healton EB, Savage DG, ef *al.:* Neurologic aspects of cobalamin deficiency. *Medicine* 70:229-245, 1991.
113. Herbert V: The 1986 Herman Award Lecture. Nutrition science as a continually unfolding story: the folate and vitamin B-12 paradigm. *Am J Clin Nutr* 46:387-402, 1987.
114. Herbert V: Staging vitamin B-12 (cobalamin) status in vegetarians. *Am J Clin Nutr* 59.-1213S-1222S, 1994.
115. Herbert V: Experimental nutritional folate deficiency in man. *Trans Assoc Am Phys* 75:307-320, 1962.
116. Herbert V: Biochemical and hematologic lesions in folic acid deficiency. *Am J Clin Nutr* 20:562-569, 1967.
117. Herbert V: Drugs effective in megaloblastic anemias; vitamin B_{12} and folic acid. Chapter 64. In: *The Pharmacological Rasis of Therapeutics.* Ed. 4. Edited by Goodman LS, Gilman A. Macmillan, New York, 1970 (pp. 1431-1441).
118. Herbert V: Malnutrition and the immune response. *Infect Dis* 7:4-10, 1977.
119. Herbert V: The nutritional anemias. *Hosp Pract* 15:65-89, 1980.
120. Herbert V, Colman N: Hematological aspects of folate deficiency. Chapter 9. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH, Raven Press, New York, 1979 (pp. 67-72).
121. Herbert V, Colman N, Jacob E: Folic acid and Vitamin B_{12}. Chapter 6J. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 229-255).
122. Hercberg S, Galen P, *et al.:* Essential mineral and trace element nutritive methodology: iron. In: *Nutritional Status Assessment.* Edited by Fidanza F. London, Chapman & Hall, 1991.
123. Herzlich B, Herbert V: Depletion of serum holotranscobalamin II: an early sign of negative vitamin B12 balance. *Lab Invest* 58:332-337, 1988.
124. Hillman RE: Megavitamin responsive aminoacidopathies. *Pediatr Clin North Am* 23:557-567, 1976.
125. Hockertz S, Schettler T, *et al:* Effect of acetylsalicylic acid, ascorbate and ibuprofen on the macrophage system. *Arzneimittel-Forschung* 42.1062-1068, 1992.
126. Hodges RE: Ascorbic acid. Chapter 6K. In: *Modern Nutrition in Health and Disease.* Ed.6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia 1980 (pp. 259-273).
127. Hoffbrand AV, Jackson BF: Correction of the DNA synthesis defect in vitamin B12 deficiency by tetrahydrofolate: evidence in favour of the methylfolate trap hypothesis as the cause of megaloblastic anaemia in vitamin B12 deficiency. *Br J Haematol* 83:643-647, 1993.
128. Hoyumpa AM: Alcohol and thiamine metabolism. *Alcohol Clin Exp Res* 7:11-14, 1983.
129. Hudson OC, Hettesheimer CA, Robin PA: Causalgic backache. *Am J Surg* 52:297-303, 1941.
130. Hughes RE: Nonscorbutic effects of Vitamin C: biochemical aspects. *Proc R Soc Med* 70:86-89, 1977.
131. Hunter R, Barnes J, Oakeley HF, *et al.:* Toxicity of folic acid given in pharmacological doses to healthy volunteers. *Lancet* 1:61-63, 1970.
132. Ianuzzo D, Patel P, *et al:* Thyroidal trophic influence on skeletal muscle myosin. *Nature* 270:74-76, 1977.
133. Ingbar SH, Woeber KA: Diseases of the thyroid. Chapter 335. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et al.* McGraw-Hill Book Company, New York, 1980 (pp. 1696,1698- 1699,1701-1703, 1711).
134. Joosten E, van den Berg A, *et al:* Metabolic evidence that deficiencies of vitamin B-12 (cobalamin), folate, and vitamin B-6 occur commonly in elderly people. *Am J Clin Nutr* 58:468-476, 1993.
135. Judovich B, Bates W: Pain *Syndromes.* Ed. 3. F.A. Davis, Philadelphia, 1949 (pp. 46- 51, Figs. 31-35).

136. Jurell KC, Zanetos MA, *et al:* Fibromyalgia: a study of thyroid function and symptoms. / *Musculoskel Pain* 4:49-60, 1996.
137. Kallner AB, Hartman D, Hornig DH: On the requirements of ascorbic acid in man: steady-state turnover and body pool in smokers. *Am J Clin Nutr* 34:1347-1355, 1981.
138. Kaminski M, Boal R: An effect of ascorbic acid on delayed-onset muscle soreness. *Pain* 50:317-321, 1992.
139. Kariks J, Perry SW: Folic-acid deficiency in psychiatric patients. *Med]Aust* 1:1192- 1195, 1970.
140. Karamizrak SO, Islegen C, *et al:* Evaluation of iron metabolism indices and their relation with physical work capacity in athletes. *Br J Sports Med* 30:15-19, 1996.
141. Karnaze DS, Carmel R: Neurologic and evoked potential abnormalities in subtle cobalamin deficiency states, including deficiency without anemia and with normal absorption of free cobalamin. *Arch Neurol* 47:1008-1012, 1990.
142. Reiser G, Berchtold P, Bolli P, *et al:* Storung der Vitamin B_{12}-Absorption infolge Biguanidtherapie. *SchweizMed Wochenschr* 100:351-353, 1970.
143. Kelley WN: Gout and other disorders of purine metabolism. Chapter 92. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et al.* McGraw-Hill, New York, 1980 (pp. 479-486).
144. Kent S: Vitamin C therapy: colds, cancer and cardiovascular disease. *Geriatrics* 33:91-105, 1978.
145. Kieburtz KD, Giang DW, ef *al:* Abnormal vitamin B12 metabolism in human immunodeficiency virus infection: association with neurologic dysfunction. *Arch Neurol* 48:312-314, 1991.
146. Kim JC: Ultrastructural studies of vascular and muscular changes in ascorbic acid deficient guinea-pigs. *Lab Anim* 11:113-117, 1977.
147. Klee GG, Hay ID: Biochemical thyroid function testing. *Mayo Clin Proc* 69:469-470, 1994.
148. Klein KK: A study of the progression of lateral pelvic asymmetry in 585 elementary, junior and senior high schoolboys. *Am Correct Ther)*'23:171-173,1969.
149. Klein KK, Redler I, Lowman CL: Asymmetries of growth in the pelvis and legs of children: a clinical and statistical study 1964-1967. / *Am Osteopath Assoc* 68:153-156, 1968.
150. Knigge KM, Penrod CH, Schindler WJ: *In vitro and in vivo* adrenal corticosteroid secretion following stress. *Am J Phys* 196:579-582, 1959.
151. Koenig WC Jr, Powers JJ, Johnson EW: Does allergy play a role in fibrositis? *Arch Phys Med Rehabil* 58:80-83, 1977.
152. Krief S, Lonnqvist F, *et al:* Tissue distribution of beta3-adrenergic receptor mRNA in man. / *Clin Invest* 92:344-349, 1993.
153. Langohr HD, Petruch F, Schroth G: Vitamin B_{12} B_2 and B_6 deficiency in neurological disorders. *J Neurol* 225:95-108, 1981.
154. Lehninger AL: *Biochemistry.* Worth, New York, 1970 (p. 204).
155. *Ibid.* (pp. 383, 550).
156. Lerner AM: Infections with herpes simplex virus. Chapter 193. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et al.* McGraw-Hill, New York, 1980 (pp. 847-851).
157. Levine M, Conry-Cantilena C, *et al:* Vitamin C pharmacokinetics in healthy volunteers: evidence for a recommended dietary allowance. *Proc Natl Acad Sci* 93:3704-3709, 1996.
158. Levine M, Hartzell W: Ascorbic acid: the concept of optimum requirements. Third Conference on Vitamin C. *Ann NY Acad Sci* 498:424-444, 1987.
159. Lewis A, Wilson CW: The effect of vitamin C deficiency and supplementation on the weight pattern and skin potential of the guinea-pig (proceedings). *Br/Pharmacol* 67:457P-458P, 1979.
160. Li TK: Factors influencing vitamin B_6 requirement in alcoholism. Chapter 13. In: *Human Vitamin B_6 Requirements.* National Academy of Sciences, Washington, 1978 (p. 210).
161. Lindenbaum J, Rosenberg IH, *et al.:* Prevalence of cobalamin deficiency in the Framingham elderly population. *Am J Clin Nutr* 60:2-11, 1994.
162. Linkswiler HM: Vitamin B_6 requirements of men. Chapter 19. In *Human Vitamin B_6 Requirements.* National Academy of Sciences, Washington, 1978 (pp. 282-288).
163. Lipton MA, Kane FJ Jr: Psychiatry. Chapter 30. In: *Nutritional Support of Medical Practice.* Edited by Schneider HA, Anderson CE, Coursin DB. Harper & Row, Hagerstown, Md., 1977 (pp. 468-469).
164. Loh HS: Screening for vitamin C status. *Lancet* 1:944-945, 1973.
165. Lonsdale D, Shamberger RJ: Red cell transketolase as an indicator of nutritional deficiency. *Am J Clin Nutr* 33:205-211, 1980.
166. Looker AC, Dallman PR, *et al.:* Prevalence of iron deficiency in the United States. *JAMA 277:973-976*, 1997.
167. Louboutin JP, Fichter-Gagnepain V, *et al:* Comparison of contractile properties between developing and regenerating soleus muscleunfluence of external calcium concentration upon the contractility. *Muscle Nerve* 28:1292-1299, 1995.
168. Lowe JC: Thyroid status of 38 fibromyalgia patients: implications for the etiology of fibromyalgia. *Clin Rull Myofasc Ther* 2:36-40, 1996.
169. Lowman CL: The sitting position in relation to pelvic stress. *Physiother Rev* 21:30-33, 1941.
170. Lui NST, Roels OA: Vitamin A and carotene. Chapter 6A. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (p. 154).
171. Maigne R: *Orthopedic Medicine, A New Approach to Vertebral Manipulation,* translated by W.T. Liberson. Charles C Thomas, Springfield, 111., 1972 (pp. 192, 292, 390).
172. *Ibid.* (pp. 392-394).
173. Macdonald VW, Charache S, *et al.:* Iron deficiency anemia: mitochondrial alpha- glycerophosphate dehydrogenase in guinea pig skeletal muscle. *J Lab Clin Med* 205:11- 18, 1985.
174. Marcus M, Prabhudesai M, et *al.:* Stability of vitamin B12 in the presence of ascorbic acid in food and serum: restoration by cyanide of apparent loss. *Am JClin Nutr* 33:137-143, 1980.
175. Martin GR: Studies on the tissue distribution of ascorbic acid. *Ann NY Acad Sci* 92:141-7, 1961.
176. McCombe PA, McLeod JG: The peripheral neuropathy of vitamin B12 deficiency. / *Neurol Sci* 206:117-126, 1984.

177. Meindok H, Dvorsky R: Serum folate and vitamin-B_{12} levels in the elderly. *J Am Geriatr Soc* 18:317-326, 1970.
178. Middaugh LD, Grover TA, Zemp JW: Effects of dietary folic acid reduction on tissue folate concentrations and on neurochemical and behavioral aspects of brain function in adult and developing mice. Chapter 24. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (p. 226, 227).
179. Moldofsky H, Scarisbrick P: Induction of neurasthenic musculoskeletal pain syndrome by selective sleep stage deprivation. *Psychosom Med* 38:35-44, 1976.
180. Moldofsky H, Scarisbrick P, England R, *et al.:* Musculoskeletal symptoms and non- REM sleep disturbance in patients with "fibrositis syndrome" and healthy subjects. *Psychosom Med* 37:341-351, 1975.
181. Mortensen L, Charles P: Bioavailability of calcium supplements and the effect of vitamin D: comparisons between milk, calcium carbonate, and calcium carbonated plus vitamin D. *Am J Clin Nutr* 63:354-357, 1996.
182. Morton DJ: *The Human Foot.* Columbia University Press, New York, 1935 (pp. 156- 157, Figs 76, 77).
183. Morton DJ: Foot disorders in women. / *Am Med Worn Assoc* 10:41-46, 1955.
184. Muller B, Zulewski H, *et al.:* Impaired action of thyroid hormone associated with smoking in women with hypothyroidism. *N Engl J Med* 333:964-969, 1995.
185. Nadler JL, Rude R: Disorders of magnesium metabolism. *Endocrin Metabol Clin North Am* 24:623-641, 1995.
186. Naimark BJ, Ready AE, *et al.:* Serum ferritin and heart disease: the effect of moderate exercise on stored iron levels in postmenopausal women. *Can J Cardiol* 32:1253-1257, 1996.
187. National Research Council, Committee on Dietary Allowances: *Recommended Dietary Allowances.* Ed. 9. National Academy of Sciences, Washington, 1980 (pp. 75-77, 108- 110, 117, 118).
188. *Ibid.* (pp. 125-164).
189. *Ibid.* (pp. 84, 85, 99-102).
190. Neal RA, Sauberlich HE: Thiamin. Chapter 6E. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 191, 193-196).
191. Neeck G, Riedel W: Thyroid function in patients with fibromyalgia syndrome. /*Rheumatol* 39:1120-1122, 1992.
192. Nelson PJ, Pruitt RE, Henderson LL, *et al.:* Effect of ascorbic acid deficiency on the in vivo synthesis of carnitine. *Biochem BiophysActa* 672:123-127,1981.
193. Newham DJ, Jones DA, er *al.:* Repeated high-force eccentric exercise: effects on muscle pain and damage. / *Appl Physiol* 63:1381-1386, 1987.
194. Nichol CJ, Johnson IA: Energy metabolism of fast- and slow-twitch skeletal muscle in the rat: thyroid hormone induced changes. / *Comp Physiol* 342:465-472, 1981.
195. Nichols PJ: Short-leg syndrome. *Br Med J* 1:1863-1865, 1960.
196. Niederwiesner A: Inborn errors of pterin metabolism. Chapter 33. In: *Folic Acid In Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (pp. 351, 354, 364, 365).
197. Norman EJ, Morrison JA: Screening elderly populations for cobalamin (vitamin B12) deficiency using the urinary methylmalonic acid assay by gas chromatography mass spectrometry. *Am J Med* 94:589-594, 1993.
198. Nuviala RJ, Castillo MC, *et al:* Iron nutritional status in female karatekas, handball and basketball players, and runners. *Physiol Behav* 59:449-453, 1996.
199. Nygard O, Nordrehaug JE, *et al:* Plasma homocysteine levels and mortality in patients with coronary artery disease. JV *Engl J Med* 337:230-236, 1997.
200. Paine CJ, Grafton WD, Dickson VL, *et al:* Oral contraceptives, serum folate, and hematologic status. *JAMA* 231:731-733, 1975.
201. Parle JV, Fanklyn JA, *et al:* Prevalence and follow-up of abnormal thyrotropin (TSH) concentrations in the elderly in the United Kingdom. *Clin Endocrinol* 34:77-83, 1991.
202. Parry GJ, Bredesen DE: Sensory neuropathy with low-dose pyridoxine. *Neurology* 35:1466-1468, 1985.
203. Passeri M: [Preventive role of vitamins in some old age diseases (author's translation)]. *Acta Vitaminol Enzymol* 2:147-62, 1980.
204. Pauling L: *Vitamin C and the Common Cold.* W.H. Freeman, San Francisco, 1970.
205. Pelletier O: Vitamin C status of cigarette smokers and nonsmokers. *Am J Clin Nutr* 23:520-524,1970.
206. Pfeiffer CC: *Mental and Elemental Nutrients.* Keats Publishing, New Canaan, Conn., 1975 (pp. 146, 251, 280, 281, 469).
207. Pincus JH: Folic acid deficiency: a cause of subacute combined system degeneration. Chapter 39. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (p. 432).
208. Pincus JH, Reynolds EH, Glaser GH: Subacute combined system degeneration with folate deficiency. *JAMA* 221:496-497, 1972.
209. Plorde JJ: Amebiasis. Chapter 199. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et al.* McGraw-Hill, New York, 1980 (pp. 863-864).
210. Plorde JJ: Minor protozoan diseases. Chapter 205. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et* al. McGraw- Hill, New York, 1980 (pp. 887-888).
211. Plorde JJ: Cestode (tapeworm) infections. Chapter 213. In: *Harrison's Principles of Internal Medicine.* Ed. 9. Edited by Isselbacher KJ, Adams RD, Braunwald E, *et al.* McGraw-Hill, New York, 1980 (pp. 916-917).
212. Pruthi RK, Tefferi A: Pernicious anemia revisited. *Mayo Clin Proc* 69:144-150, 1994.
213. Rajaram S, Weaver CM, *et al:* Effects of long-term moderate exercise on iron status in young women. *Med Sci Sports Exerc* 27:1105-1110, 1995.
214. Randall HT: Water, electrolytes and acid-base balance. Chapter 8. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 368, 378).

215. Redler I: Clinical significance of minor inequalities in leg length. *New Orleans Med Surg f* 104:308-312, 1952.
216. Robbins J, Rail JE, Gorden P: The thyroid and iodine metabolism. Chapter 19. In: *Metabolic Control and Disease.* Ed. 8. Edited by Bondy PK, Rosenberg LE. Saunders, Philadelphia, 1980 (pp. 1333, 1343-1345).
217. Roe DA: *Drug-induced Nutritional Deficiencies.* AVI Publishing, Westport, Conn., 1976 (pp. 7-17, 72, 73, 79-81, 85, 96-99, 150, 151, 160-167, 215-216, 223-227).
218. *Ibid.* (pp. 72, 83, 120, 217).
219. Romano TJ: Magnesium deficiency in patients with myofascial pain. *J Myofasc Ther* 1:11-12, 1994.
220. Romano TJ, Stiller JW: Magnesium deficiency in fibromyalgia syndrome. / *Nutr Med* 4:165-167,1994.
221. Rose DP: Oral contraceptives and vitamin B„. Chapter 11. In: *Human Vitamin B. Requirements.* National Academy of Sciences, Washington, 1978 (pp. 193-201).
222. Rosen NB: Physical medicine and rehabilitation approaches to the management of myofascial pain and fibromyalgia syndromes. *Clin Rheum* 8:881-916, 1994.
223. Rosenberg IH, Dyer J: The prevalence and causes of folic acid deficiency in the United States. Chapter 4. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine,* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (pp. 19-22).
224. Rosenblatt DS, Cooper BA: Methylenetetrahy-drofolate reductase deficiency: clinical and biochemical correlations. Chapter 34. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (p. 389).
225. Rubin D: Myofascial trigger point syndromes: an approach to management. *Arch Phys Med Rehabil* 62:107-110, 1981.
226. Ruff RL, Weissmann J: Endocrine myopathies. *Neurol Clin North Am* 6:575-592, 1988.
227. Runcie J: Folate deficiency in the elderly. Chapter 45. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (pp. 493-499).
228. Russell IJ, Vipraio GA, *et al:* Insulin-like growth factor (IGF1) in fibromyalgia, rheumatoid arthritis, osteoarthritis and healthy normal controls: Roles of diagnosis, age, sex and ethnic origin [Abstract]. *Arthritis Rheum* 35.S160, 1992.
229. Ryschon TW, Rosenstein DL, *et al:* Relationship between skeletal muscle intracellular ionized magnesium and measurements of blood magnesium. / *Lab Clin Med 127:207-* 213, 1996.
230. Sauberlich HE, Canham JE: Vitamin B.. Chapter 61. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 219-225).
231. Sauberlich HE: Implications of nutritional status on human biochemistry, physiology and health. *Clin Biochem* 37.132-142, 1984.
232. Scarlett JD, Read H, *et al:* Protein-bound cobalamin absorption declines in the elderly. *Am J Hematol* 39:79-83, 1992.
233. Schaumberg H, Kaplan J, *et al:* Sensory neuropathy from pyridoxine abuse: a new megavitamin syndrome. *N Engl]Med* 309:445-448, 1983.
234. Schmid A, Jakob E, *et al:* Effect of physical exercise and vitamin C on absorption of ferric sodium citrate. *Med Sci Sports Exerc* 28:1470-1473, 1966.
235. Schneider HA, Anderson CE, Coursin DB: *Nutritional Support of Medical Practice.* Harper & Row, Hagerstown, Md., 1977 (pp. 37, 38, 111, 115-118, 131, 436, 450, 480-482).
236. Shackleton PJ, Fish DI, *et al:* Intrinsic factor antibody tests. / *Clin Pathol* 42:210- 212, 1989.
237. Shane B: Vitamin B„ and blood. Chapter 7. In: *Human Vitamin B. Requirements.* National Academy of Sciences, Washington, 1978 (pp. 115, 122-124).
238. Sharav Y, Tzukert A, Refaeli B: Muscle pain index in relation to pain, dysfunction, and dizziness associated with the myofascial pain-dysfunction syndrome. *Oral Surg* 46:742-747, 1978.
239. Shaw S, Lieber CS: Nutrition and alcoholism. Chapter 40. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 1225,1226).
240. *Ibid.* (p. 1235).
241. Shevell MI Rosenblatt DS: The neurology of cobalamin. *Can J Neurol Sci* 19:472-486, 1992.
242. Shils ME: Magnesium. Chapter 7B. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 315, 317).
243. Shils ME: Nutrition and neoplasia. Chapter 38. In: *Modern Nutrition in Health and Disease.* Ed. 6. Edited by Goodhart RS, Shils ME. Lea & Febiger, Philadelphia, 1980 (pp. 1179, 1180).
244. Shorvon SD, Reynolds EH: Folate deficiency and peripheral neuropathy. Chapter 37. In: *Folic Acid in Neurology, Psychiatry, and Internal Medicine.* Edited by Botez MI, Reynolds EH. Raven Press, New York, 1979 (p. 420).
245. Shorvon SD, Carney MW, *et al:* The neuropsychiatry of megaloblastic anemia. Br *Med J* 283:1036-1038, 1980.
246. Sicuranza BJ, Richards J, Tisdall LH: The short leg syndrome in obstetrics and gynecology. *Am J Obstet Gynecol* 107:217-219, 1970.
247. Simons DG, Travell J: Common myofascial origins of low back pain. *Postgrad Med* 73:66-108, 1983.
248. Singer PA, Cooper DS, *et al:* Treatment guidelines for patients with hyperthyroidism and hypothyroidism. *JAMA* 273:808-812, 1995.
249. Smith JL, Hodges RE: Serum levels of vitamin C in relation to dietary and supplemental intake of vitamin C in smokers and non-smokers. /Inn *NY Acad Sci* 498:144-152, 1987.
250. Smythe HA: Fibrositis and other diffuse musculoskeletal syndromes. In: *Textbook of Rheumatology,* Vol 1. Edited by Kelley WN, Harris ED Jr, Ruddy S, *et al.* W. B. Saunders, Philadelphia, 1981 (p. 489).
251. Solanki DL, Jacobson RJ *et al:* Pernicious anemia in blacks: a study of 64 patients from Washington, DC, and Johannesburg, South Africa. *Am J Clin Pathol* 75:96-99, 1981.
252. Sonkin LS: Myofascial pain due to metabolic disorders: diagnosis and treatment. Chapter 3. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 45-60).
253. Staub JJ, Althaus BU, ef *al:* Spectrum of subclinical and overt hypothyroidism. *Am J Med* 92:631-641, 1992.

254. Stead WW: Tuberculosis. Chapter 156. In: *Harrison's Principles of Internal Medicine.* Ed. 7. Edited by Wintrobe MM, Thorn GW, Adams RD, ef *al.* McGraw-Hill, New York, 1974 (p. 867).
255. Sterling K: Thyroid hormone action at the cell level. *NEngl J Med* 300:117-123, 173- 177, 1979.
256. Sternbach RA: *Pain Patients, Traits and Treatment,* Academic Press, New York, 1974 (pp. 40-51).
257. Sternbach DJ: Stress in the lives of musicians—on stage and off. In: Bejjani FJ, *Current Research in Arts* Medicine. Chicago: A Capella Books, 1993.
258. Surks MI, DeFesi CR: Normal serum free thyroid hormone concentrations in patients treated with phenytoin or carbamazepine. *JAMA* 275:1495-1498 1966.
259. Surks MI, Sievert R: Drugs and thyroid function. *N Engl J Med* 333.1688-1694, 1995.
260. Swanson JW, Kelly JJ, *et al.:* Neurologic aspects of thyroid dysfunction. *Mayo Clin Proc* 56:504-512, 1981.
261. Tallaksen CM, Bohmer T, ef *al:* Concentrations of the water-soluble vitamins thiamin, ascorbic acid, and folic acid in serum and cerebrospinal fluid of healthy individuals. *Am J Clin Nutr* 56:559-564, 1992.
262. Taylor TV, Rimmer S, Day B, ef *al.:* Ascorbic acid supplementation in the treatment of pressure-sores. Lancef 2:544-546, 1974.
263. Tefferi A, Pruthi RK: The biochemical basis of cobalamin deficiency. *Mayo Clin Proc* 69:181-186, 1994.
264. Theuer RC, Vitale JJ: Drug and nutrient interactions. Chapter 18. In: *Nutritional Support of Medical Practice.* Edited by Schneider HA, Anderson CE, Coursin DB. Harper & Row, 1977 (pp. 299, 300,302).
265. Thomson AD, Baker H, Leevy CM: Patterns of ^{35}S-thiamine hydrochloride adsorption in the malnourished alcoholic patient. *J Lab Clin Med* 76:34-45, 1970.
266. Thornton WE, Thornton BP: Folic acid, mental function, and dietary habits. / *Clin Psychiatry* 39:315-319, 322, 1978.
267. Thurnham DI: Red cell enzyme tests of vitamin status: do marginal deficiencies have physiological significance? *Proc Nutr Soc* 40:155-163, 1981.
268. Tichauer ER: Industrial engineering in the rehabilitation of the handicapped. / *Industr Eng* 19:96-104, 1968.
269. Timmerman MG: Medical problems of adolescent female athletes. *Wis Med J 95:* 351- 354, 1996.
270. Toft AD: Thyroxine therapy. *N Engl J Med* 1994 *331:*174-180 (1994).
271. Tomkin GH, Hadden DR, Weaver JA, ef *al.:* Vitamin B_{12} status of patients on long- term metformin therapy. *Rr Med J* 2:685-687, 1971.
272. Travell J: Referred pain from skeletal muscle: the pectoralis major syndrome of breast pain and soreness and the sternomastoid syndrome of headache and dizziness. *NY State J Med* 55:331-339, 1955.
273. Travell J: *Office Hours: Day and Night.* The World Publishing Company, New York, 1968.
274. Travell J: Low back pain and the Dudley J. Morton foot (long second toe). *Arch Phys Med Rehabil* 56:566, 1975.
275. Travell J: Identification of myofascial trigger point syndromes: a case of atypical facial neuralgia. *Arch Phys Med Rehabil* 62:100-106, 1981.
276. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 11:425-434, 1952.
277. Travell JG: The quadratus lumborum muscle: an overlooked cause of low back pain. *Arch Phys Med Rehabil* 57:566, 1976.
278. Uchida T: Overview of iron metabolism. *Int J Hematol* 62.193-202 1995.
279. Utiger RD: Therapy of hypothyroidism. *N Engl] Med* 323:126-127, 1990.
280. Vallance S: Relationships between ascorbic acid and serum proteins of the immune system. *Br Med* /2:437-438, 1977.
281. van der Metz J, Westhuyzen J: The fruit bat as an experimental model of the neuropathy of cobalamin deficiency. *Comp Biochem Physiol* 88:171-177, 1987.
282. Van Itallie TB: Assessment of nutritional status. Chapter 75. In: *Harrison's Principles of Internal Medicine.* Ed. 7. Edited by Wintrobe MM, Thorne GW, Adams RD, ef *al.* McGraw-Hill, New York, 1974 (p. 419).
283. Van Itallie TB, Follis RH Jr: Thiamine deficiency, ariboflavinosis, and vitamin B„ deficiency. Chapter 78. In: *Harrison's Principles of Internal Medicine.* Ed. 7. Edited by Wintrobe MM, Thorne GW, Adams RD, ef *al.* McGraw-Hill, New York, 1974 (pp. 430-432).
284. Vilter RW: Nutritional aspects of ascorbic acid: uses and abuses. *West J Med* 133:485- 492, 1980.
285. Vimokesant SL, Nakornchai S, Dhanamitta S, ef *al.:* Effect of tea consumption on thiamin status of man. *Nutr Rep Int* 9:371-376, 1974.
286. Wakabayashi A, Yui Y, Kawai C: A clinical study on thiamine deficiency. *Jpn Circ J* 43:995-999, 1979.
287. Weeks VD, Travell J: Postural vertigo due to trigger areas in the sternocleidomastoid muscle. / *Pediatr* 47:315-327, 1955.
288. Weiner WJ: Vitamin B„ in the pathogenesis and treatment of diseases or the central nervous system. Chapter 5. In: *Clinical Neuropharmacology,* Vol 1. Edited by Klawans HL. Raven Press, New York, 1976 (pp. 107-136).
289. White JD: No ill effects from high-dose vitamin C. *NEngl J Med* 304:1491, 1981.
290. Williams RJ: *Physicians Handbook of Nutritional Science.* Charles C Thomas, Springfield, 111., 1975 (pp. 48, 70-82).
291. Wilson CM, Kevany JP: Screening for vitamin C status. *BrfPrev Soc Med* 26:53-54, 1972.
292. Wood B, Breen KJ: Clinical thiamine deficiency in Australia: the size of the problem and approaches to prevention. *Med J Aust* 1:461-462, 464, 1980.
293. Wu CM, Chen HH, Hong, CZ: Inactivation of myofascial trigger points associated with lubar radiculopathy: surgery versus physical therapy [Abstract]. *Arch Phys Med Rehabil* 78:1040-1041, 1997.
294. Yao Y, Yao SL ef *al.:* Prevalence of vitamin B12 deficiency among geriatric outpatients. / *Fam Pract* 35:524-528, 1992.

PART 2
HEAD AND NECK PAIN

CHAPTER 5
Overview of Head and Neck Region

By Bernadette Jaeger

with contributions by

David G. Simons and Lois Simons

INTRODUCTION TO PART 2

Part 2 of this manual is concerned with the muscles of the head and neck that refer pain to the uppermost parts of the body. This second of five parts comprises all of the head muscles and most of the neck muscles, including the sternocleidomastoid, trapezius, digastric and other anterior neck muscles, suboccipital, and cervical paraspinal muscles. It excludes the scalene and levator scapulae muscles because they refer pain downward, but these are still important muscles to consider when treating head and neck pain complaints and are included in part three of this volume. Treatment of other head and neck muscles and resolution or control of a particular head and neck muscle dysfunction and/or pain problem may depend on treatment of these latter two muscles as well. For example, it may not be possible to adequately stretch the sternocleidomastoid muscle if there is myofascial trigger point (TrP) involvement of the contralateral levator scapulae. Stretching of the clavicular head of the sternocleidomastoid could cause painful reactive shortening of the contralateral levator scapulae and inhibit a full stretch of the sternocleidomastoid. Also, untreated levator scapulae TrPs may keep upper trapezius TrPs active.

One should keep in mind that TrPs primarily cause increased muscle tension (tonus) that limits its stretch range. Also, TrPs can produce inhibition of muscle function. The overall goal of treatment is to restore normal function.

This chapter is divided into four sections. Section A is a **PAIN GUIDE to INVOLVED MUSCLES** to help the reader determine which muscles to examine, based on the location of the patient's pain. Section B reviews the **DIFFERENTIAL DIAGNOSIS** of head, neck and face pain, including various types of headache and temporomandibular disorders, and reviews recent literature on muscle pain and myofascial TrP pain with respect to its prevalence and presentation in these disorders. Section C presents a **SCREENING EXAMINATION** and rationale for treatment of temporomandibular disorders and a simple method for evaluating and correcting anterior head positioning and poor body mechanics as they relate to myofascial trigger points. Section D presents a **GENERAL TREATMENT APPROACH** that has been shown to be successful for patients with *chronic* head, neck or facial pain caused wholly or in part by myofascial TrPs.

A. PAIN GUIDE TO INVOLVED MUSCLES

This guide lists the muscles that may refer pain to specific areas of the head and neck, as identified in Figure 5.1. This figure is used by locating the region where the patient has pain. Under that regional heading in the pain guide are listed the muscles that may refer pain to that anatomic area. The number in parenthesis following each muscle is the chapter number for that muscle; TrP stands for trigger point.

The muscles listed in boldface type are likely to refer an essential pain pattern to that area. Regular type identifies the muscles that may refer a spillover pattern to the region. The muscles are listed in such a way that, in our experience, the muscle which is a more frequent cause of pain in an area is listed higher than others. However, the nature of the examiner's practice influences the selection of patients and, thus, which muscles are involved most often.

PAIN GUIDE

VERTEX PAIN
- **Sternocleidomastoid (sternal)** (7)
- **Splenius capitis** (15)

BACK-OF-HEAD PAIN
- **Trapezius (TrPJ** (6)
- **Sternocleidomastoid (sternal)** (7)
- **Sternocleidomastoid (clavicular)** (7)
- **Semispinalis capitis** (16)
- **Semispinalis cervicis** (16)
- **Splenius cervicis** (15)
- **Suboccipital group** (17)
- **Occipitalis** (14)
- **Digastric** (12)
- **Temporalis** (TrPJ (9)

TEMPORAL HEADACHE
- **Trapezius (TrPJ** (6)
- **Sternocleidomastoid (sternal)** (7)
- **Temporalis (TrPs$_{1i2>3}$)** (9)
- **Splenius cervicis** (15)
- **Suboccipital group** (17)
- **Semispinalis capitis** (16)

FRONTAL HEADACHE
- **Sternocleidomastoid (clavicular)** (7)
- **Sternocleidomastoid (sternal)** (7)
- **Semispinalis capitis** (16)
- **Frontalis** (14)
- **Zygomaticus major** (13)

EAR AND TEMPOROMANDIBULAR JOINT PAIN
- **Lateral pterygoid** (11)
- **Masseter (deep)** (8)
- **Sternocleidomastoid (clavicular)** (7)
- **Medial pterygoid** (10)

EYE AND EYEBROW PAIN
- **Sternocleidomastoid (sternal)** (7)
- **Temporalis (TrPJ** (9)
- **Splenius cervicis** (15)
- **Masseter (superficial)** (8)
- **Suboccipital group** (17)
- **Occipitalis** (14)
- **Orbicularis oculi** (13)
- **Trapezius (TrPJ** (6)

CHEEK AND JAW PAIN
- **Sternocleidomastoid (sternal)** (7)
- **Masseter (superficial)** (8)
- **Lateral pterygoid** (11)
- **Trapezius (TrPJ** (6)
- **Masseter (deep)** (8)
- **Digastric** (12)
- **Medial pterygoid** (10)
- **Buccinator** (13)
- **Platysma** (13)
- **Orbicularis oculi** (13)
- **Zygomaticus major** (13)

TOOTHACHE
- **Temporalis (TrPsj$_{23}$)** (9)
- **Masseter (superficial)** (8)
- **Digastric (anterior)** (12)

BACK-OF-NECK PAIN
- **Trapezius (TrPJ** (6)
- **Trapezius (TrPJ** (6)
- **Trapezius (TrP$_3$)** (6)
- **Multifidi** (16)
- **Levator scapulae** (19)
- **Splenius cervicis** (15)
- **Infraspinatus** (22)

THROAT AND FRONT-OF-NECK PAIN
- **Sternocleidomastoid (sternal)** (7)
- **Digastric** (12)
- **Medial pterygoid** (10)

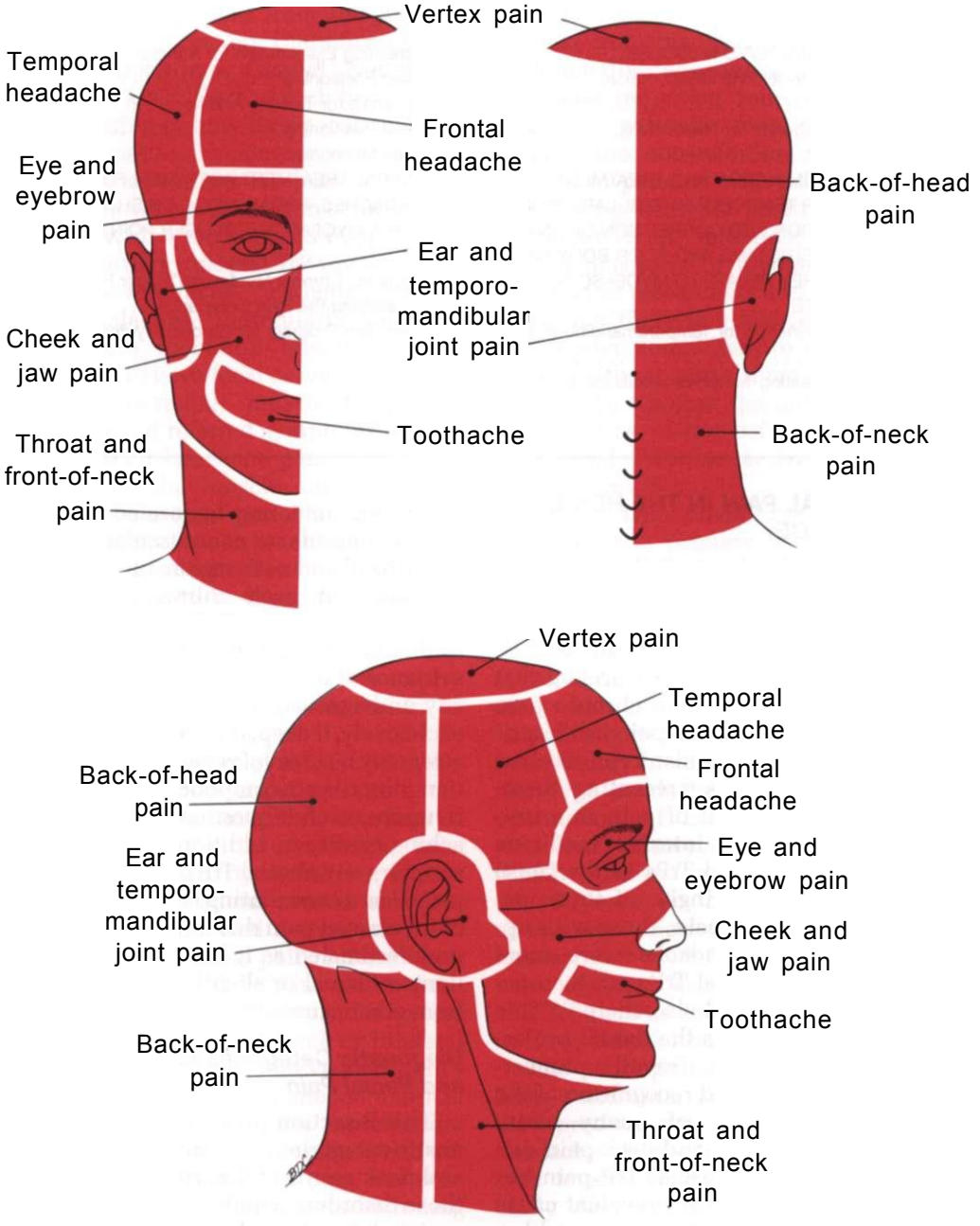


Figure 5.1. Designated areas in the head and neck region to which pain may be referred by myofascial trigger points. See listing of muscles that refer pain to each of these areas.

B. MYOFASCIAL PAIN IN THE HEAD, NECK AND FACE

Myofascial pain due to TrPs is a prevalent cause of pain in all parts of the body and has been reported as a source of pain in numerous medical specialties.[28, 110] While it may no longer be surprising that up to 80% of patients in a chronic pain center have myofascial TrP pain as the primary diagnosis,[28] it was also reported that up to 30% of patients presenting themselves with a complaint of pain in a university based general internal medicine practice had myofascial TrPs as the cause of the pain.[110] Interestingly, of those patients presenting with pain, those with upper body pain or headache were more likely to have myofascial TrP pain than patients with pain located elsewhere.[110] This may explain why it was the dental profession that recognized Dr. Travell's pioneering efforts and promoted recognition of the muscular component of many craniomandibular and head and neck pain disorders. Certainly, myofascial TrP pain has been reported as the most prevalent cause of painful symptoms in temporomandibular (TM) disorders (a term used to describe clinical problems involving the masticatory muscles, temporomandibular joint (TMJ), or both).[31,79,112] Similarly, sufficient evidence exists supporting a substantial role of myofascial TrP pain in chronic tension-type and migraine headaches.[51]

In myofascial pain due to TrPs, the presenting complaint, which is usually a referred symptom, may be located in or about normal muscular or nonmuscular structures. In the head and neck region, the patient may complain of such things as headache, toothache, sinus or TMJ pain, yet clinical evaluation of these areas may not yield any evidence of local pathologic change. In fact, any undiagnosed pain, particularly, but not exclusively, if deep, dull, and aching in character may be of myofascial TrP origin. If a patient describes 2 components to the pain, or if upon careful questioning notes a dull aching quality in addition to other pain descriptors, myofascial TrP pain should be suspected as a contributing factor. The intensity of myofascial pain due to TrPs should not be underestimated as it has been rated by patients as equal or slightly greater than pain from other causes.[110]

Diagnostic Categories for Head, Neck and Facial Pain

This B section presents the various diagnostic categories of chronic orofacial, head and neck pain and describes in more detail those disorders which are likely to have associated myofascial pain. Case examples are included and pertinent literature documenting the role of myofascial TrP pain in mimicking, producing or contributing to many of these painful disorders is reviewed. Diagnostic examination techniques to help the clinician distinguish pain arising primarily from the temporomandibular joint versus myofascial TrPs are included in Section C.

Table 5.1 is adapted from the International Headache Society Classification for Headache Disorders, Cranial Neuralgias and Facial Pain.[81] Next to each broad category is a rating of how likely it is that disorders in a particular category will be associated with myofascial TrP pain.

What follows is a discussion of those pain categories with a moderate to high or very high chance of myofascial TrP involvement.

Migraine Headache. Patients with migraine headaches, particularly migraine without aura (common migraine), have been shown to have focal cervical and masticatory (pericranial) muscle tenderness with associated referred symptoms that reproduced their headache pain.[42,55,66,80,118] These pericranial muscle sites are more tender in migraine patients than nonheadache controls even when the migraine patients are headache-free.[55,66] Increasing headache intensity is associated with increased tenderness of the pericranial muscle sites.[56,61] Injection of these tender sites with saline or lidocaine was shown to produce complete headache elimination in 60% of patients studied.[118]

The tender pericranial muscle sites described and studied in patients with migraine without aura have many characteristics in common with myofascial TrPs. Myofascial TrPs are, by definition, focally tender points in skeletal muscle and produce consistent referred symptoms when palpated. Overlapping pain referral patterns from myofascial TrPs in various pericranial muscles produce a typical migraine picture that can be unilateral or bilateral (Fig. 5.2). Research has shown that *active* TrPs (TrPs causing spontaneous clinical pain, such as headache) are more tender than *latent* TrPs (TrPs quiescent with respect to spontaneous symptoms, but conforming to all other TrP criteria including referred pain with palpation).[52] This is consistent with the observation that the pericranial muscle sites in migraine headache subjects are more tender outside of a headache attack than those of nonheadache controls, and that muscle tenderness increases with increasing headache intensity within an attack.[55] Injection and even dry needling of TrPs has been shown to be effective in reducing or eliminating both the referred symptoms and local tenderness from myofascial TrPs,[33,39,44,63,111] undoubtedly accounting for the 60% headache reduction rate in the study of migraine patients mentioned above.[118] Clearly, the constellation of signs documented in patients with migraine without aura, along with the positive response to treatment with injection, is consistent with a diagnosis of myofascial TrP pain. While most researchers and clinicians now agree that there is a myogenic/myofascial nociceptive component to the pain of migraine without aura, there is still controversy as to whether the muscle is the primary source of the pain, or whether the myofascial TrPs are activated by central mechanisms.[82]

Tension-type Headache. Tension-type headache is a primary headache disorder whose pathophysiology also engenders much debate. These headaches are usually bilateral with a pressing, nonpulsating quality, lasting 30 minutes to 7 days when episodic and may be daily without remission when chronic. While a psychological basis has been entertained,[108]" the predominant theory historically has been that tension-type headaches are due to sustained contraction of cervical and pericranial muscles.[1] However, electromyographic (EMG) studies completed over the last 10-15 years do not support a muscle contraction mechanism in tension-type headaches.[10, 43, 83, 86, 97] Rather, more and more evidence exists implicating myofascial TrP pain as an etiologic source of pain in these headaches.[51,81]

The overlapping pain referral patterns from pericranial and cervical myofascial TrPs produce not only a typical migraine headache distribution, but also a characteristic tension-type headache picture especially if bilateral (Fig. 5.2). Even the "steady, deep aching" quality of myofascial TrP pain is comparable to the "pressing/tightening" quality of tension-type headache described in the International Association for the Study of Headache Classification.[81] Where studies have failed to show any positive correlation between tension-type headache and EMG elevation, they *have* shown a positive correlation with muscle tenderness.[41, 53]

Table 5.1 ***Head, Neck or Facial Pain Disorder****

Disorder	*Probability of Myofascial TrP Pain*
Migraine headache	high
Migraine without aura	
Migraine with aura	
Other	
Tension-type headache	very high
Episodic	
Chronic	
Cluster headache and chronic paroxysmal hemicrania	low to moderate
Miscellaneous headaches, unassociated with structural lesion	low
Cold stimulus headache	
Benign cough headache	
Benign exertional headache	
Orgasmic headache	
Head and neck pain associated with head trauma	moderate to high
Acute post-traumatic head and neck pain	
Chronic post-traumatic head and neck pain	
Head and facial pain associated with vascular disorders	low
Acute ischemic cerebrovascular disease	
Intracranial hematoma	
Subarachnoid hemorrhage	
Giant Cell Arteritis	
Carotid or vertebral artery pain	
Head and facial pain associated with nonvascular intracranial disorders	low
High or low cerebrospinal fluid pressure	
High pressure hydrocephalus	
Intracranial infection or neoplasm	
Head pain associated with substances or their withdrawal	low to high
Acute substance use/exposure (alcohol, caffeine, nitrites, MSG)	
Chronic substance use/exposure (ergotamine, analgesics)	
Acute use withdrawal (alcohol)	
Chronic use withdrawal (ergotamine, caffeine, narcotics)	
Head pain associated with noncephalic infection	low
Viral	
Bacterial	
Other	
Head pain associated with metabolic disorder	low
Hypoxia, hypercapnia or mixed hypoxia and hypercapnia	
Hypoglycemia	
Dialysis	
Other	
Head, neck or facial pain associated with disorders of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures, including the TMJ	high
Cranial neuralgia, nerve trunk pain, and deafferentation pain	low to moderate
Persistent or continuous neuralgias	
Paroxysmal neuralgias	
Head, neck and facial pains not classifiable	**?**
Cervicogenic headache	high

*Adapted from the International Headache Society Classification for Headache Disorders, Cranial Neuralgias and Facial Pain.[81] Next to each broad category is a rating of how likely it is that disorders in a particular category will be associated with myofascial pain.

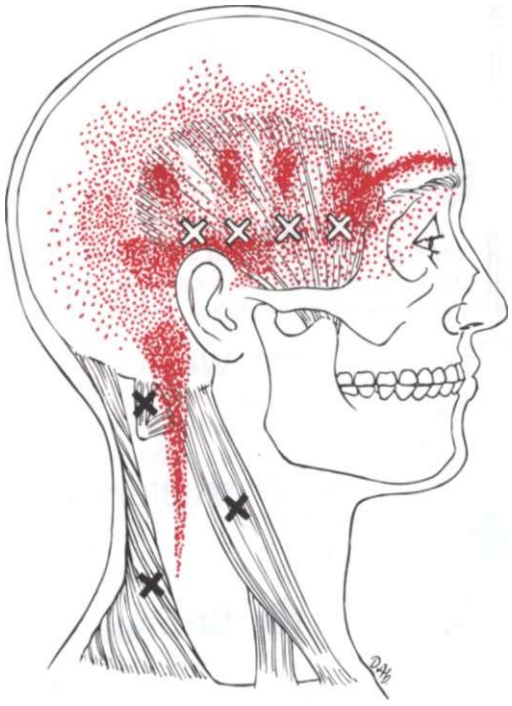


Figure 5.2. Overlapping pain referral patterns *(red)* from myofascial trigger points (Xs) in various masticatory and cervical muscles produce typical unilateral or bilateral migraine or tension-type headache pictures.

Many studies have documented the presence of pericranial muscle tenderness[3,54,61,69,119,123] and referred pain with muscle palpation[54,66] not only in migraine, but also in tension-type headache. As with migraine without aura, there is a positive correlation between the degree of muscle tenderness and the intensity of tension-type headache,[8,60,61] a feature which again correlates with what is known about latent and active myofascial TrPs.[52]

Other characteristics of TrPs that are compatible with tension-type headache are the taut bands which make the muscles feel tense, but which have no observable EMG activity, despite the fact that the TrPs themselves do.[46,106] Myofascial TrP activity increases dramatically in response to psychological stress and diminishes with relaxation;[73] tension-type headaches also worsen with stress and improve with relaxation.[41,94]

It would seem that, in their fascination with central and intracranial neurovascular mechanisms, many headache researchers have failed to acknowledge that there is a myofascial TrP component to tension-type and most migraine headache pain, despite strong and consistent supporting evidence. However, in an effort to account for the varying clinical presentations of headache, and taking into account what is known about the neurovascular mechanisms as well as the "pericranial muscle tenderness," Olesen proposed a model that is supportive of the role of myofascial TrPs in headache. In this model the cranial vasculature and the pericranial muscles (myofascial TrPs) are the 2 primary nociceptive sources and supraspinal (emotional/psychological) factors either enhance or reduce the pain.[82] The final common pathway is through the second order pain transmission neuron upon which, Oleson speculates, the inputs from the primary afferent nociceptors of intracranial and extracranial vasculature, extracranial musculature (myofascial TrPs) and supraspinal "on-off" cells converge. The strength of input from each of the converging neurons determines which headache picture emerges clinically (Fig. 5.3). For example, nociception predominantly from myofascial TrPs will produce a tension-type headache picture. This model explains why some patients have both migraine and tension-type headaches, or why some patients presenting with chronic tension-type headache relate a history of intermittent migraine. It is likely that early identification and treatment of myofascial TrPs in these headache patients will reduce incidence of progression to chronicity.

Because TrPs appear to play an important role in migraine and tension-type headaches, all headache patients should be evaluated for their presence.[59] If found, the treatment regimen should include myofascial TrP pain reduction techniques or a myofascial TrP pain management program. The same treatment strategies used for myofascial TrP pain work well for the reduction of headache, whether migraine or tension-type, when associated with "pericranial muscle tenderness" (myofascial TrPs)[37] *(see* Section D at the end of this chapter).

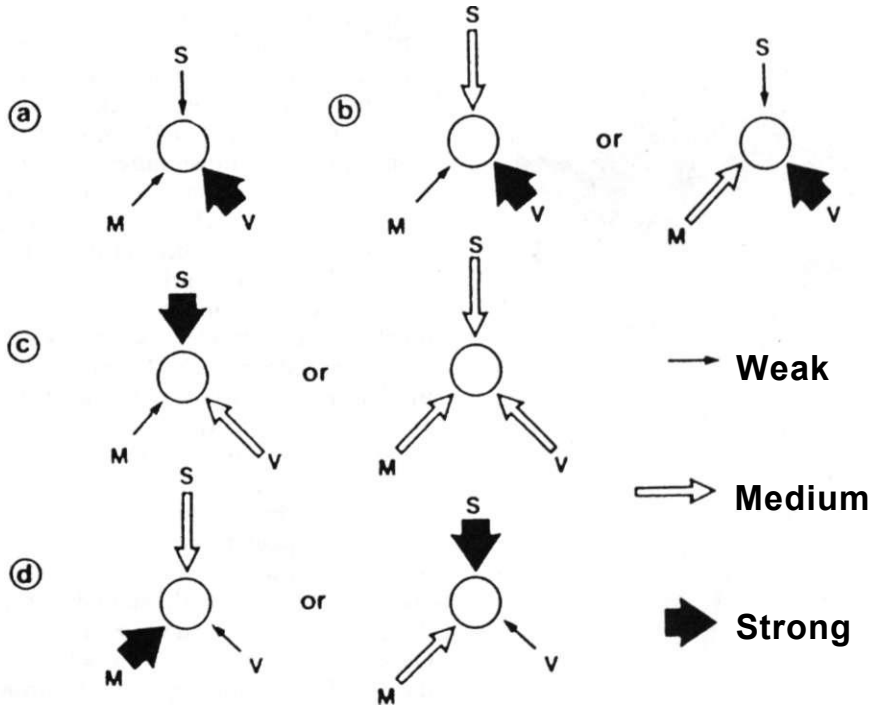


Figure 5.3. Predicted importance of supraspinal, vascular, and myofascial inputs to brain-stem neurons in various forms of migraine and tension-type headache. Some examples of the innumerable modulations of the vascular-supraspinal-myogenic model of migraine and other headaches. S: supraspinal net effect (usually facilitation during headache); *M:* myofascial nociceptive input; *V:* vascular nociceptive input. Thickness of arrows represents relative intensity of input, **a;** migraine aura without headache: despite strong vascular input there is no pain because of small S and M. **b;** migraine with aura: because of stronger supraspinal or myofascial input the subject now suffers from headache, **c;** migraine without aura: the vascular input is not as strong as in migraine with aura, but the headache is no less intense because of a stronger supraspinal facilitation or the combined effects of V and M. The latter case is likely to suffer alternating migrainous or tension-type headaches depending on small shifts in the relative magnitude of M and V. **d;** tension-type headache: M is greater than V, and S is medium or large. (Reprinted with permission from Olesen J. Clinical and pathophysiological observations in migraine and tension-type headache explained by integration of vascular, supraspinal and myofascial inputs. *Pain* 1991;46:125-132.)

Cluster Headaches and Chronic Paroxysmal Hemicrania. Both of these headache types share several features, including location, quality, intensity and unilaterality of the pain, associated autonomic phenomena and an intermittent nature, individual headache attacks ranging from minutes to hours only. Research has focused on autonomic dysfunction, cranial arteries and blood flow, biochemical and neurotransmitter changes, neuroendocrinology, sleep and central mechanisms.[23] Pericranial muscle tenderness or the presence of myofascial TrPs has not been systematically studied in these two headache types and likely reflects a low incidence of occurrence. The intermittent character of these two headache types, coupled with short duration, is probably the reason that myofascial TrPs do not seem to develop. However, this author's experience is that myofascial pain may occur in chronic cluster headache (recurring attacks for over a year without remission longer than 14 days) and may complicate management if not identified and controlled.

Case report: **This was a 57-year-old male with a 38-year history of cluster headaches. Early on, the cluster episodes occurred approximately once every 14-16 months. Upon presentation, the headaches had been occurring chronically without remission for 3 years. The patient was controlling his headaches with 1 to 4 verapamil and 1 to 2 cafergot daily. They always started as a dull suboccipital pain on the left, spreading to involve the left eye. Duration ranged from 75 minutes to 14 hours (not typical of cluster). Associated symptoms included mild nasal stuffiness on the same side. Physical examination was within normal limits except for nonpainful crepitus in the left temporomandibular joint, elevation of the left shoulder girdle, and anterior head positioning. Of greater significance was an active myofascial TrP in the left sternocleidomastoid muscle which referred pain into the left suboccipital region and left maxilla and forehead, as well as inducing a feeling of nasal stuffiness on the left side. Tenderness without pain referral was found in the left suboccipital and upper trapezius muscles. Physical therapy evaluation confirmed underlying stiffness in the upper cervical spinal joints. Diagnosis of cluster variant with cervical musculoskeletal dysfunction and myofascial TrPs as contributing and possibly triggering factors was made. It is likely that the patient was also suffering from analgesic rebound secondary to the cafergot consumption. Treatment was aimed primarily at correction of the musculoskeletal dysfunction with instruction in posture and body mechanics, mobilization of the upper cervical segments, and home stretching exercises for muscles with myofascial TrPs and palpation tenderness. The left sternocleidomastoid muscle was injected with procaine one time. Medications were slowly reduced and discontinued. Within 6 weeks, the patient was experiencing significant reduction in the frequency and intensity of headaches. The patient rated the physical therapy and home exercise program as the most useful components of his treatment. He felt that the stretching exercises gave him control over his headaches as he was often able to abort a headache using these. The headaches reverted back to an episodic cluster pattern and responded well to prophylactic cluster headache medications when they occurred.**

Miscellaneous Headaches, Unassociated with Structural Lesion. These headaches include cold stimulus, benign cough or exertional headache and orgasmic headache. By their very nature these headaches are associated with a specific inciting event, treatment being aimed at eliminating or avoiding the precipitating cause. Because these headaches are relatively infrequent and shortlasting, development of associated myofascial TrPs appears to be rare.

Head and Neck Pain Associated with Head Trauma. A fairly consistent constellation of head pain and other symptoms have been reported following minor closed head injuries with actual cranial impact, or flexion-extension injuries without cranial impact. Painful symptoms are usually in and around the head, neck and shoulders and often appear within the first 24-48 hours, although onset may be days or weeks.[95] Headache is the most common complaint that lasts beyond the normal healing phase of acute soft tissue injury. The mechanism of the head pain, which may mimic *any* of the primary headache disorders, but most commonly tension-type headaches, remains puzzling, but may well have its roots in the posttraumatic activation or development of myofascial TrPs. Other postulated but also unproven etiologic sources for the pain include cervical soft tissue and cervical and temporomandibular joint injuries, along with possible physiological or microstructural disturbances of the brainstem or vestibular apparatus.[95]

Acute muscle overload, such as occurs with flexion-extension injuries, is a well recognized and widely accepted cause for activating myofascial TrPs, although systematic blinded or controlled research studies proving this are lacking and are needed. Posttraumatic myofascial pain has been noted as a source of pain in the head

and neck region.[9,31] and one study documented the frequency of myofascial TrPs in various muscles in 100 consecutive motor vehicle accident victims.[4] The latter study found myofascial TrPs in muscles consistent with the force overload expected from different directions of impact and within the areas the patients complained of pain. Forty-four percent of subjects complained of headache; the semispinalis capitis and splenius capitis were the most commonly involved muscles, regardless of the direction of impact, second only to the quadratus lumborum muscle.[4]

It is interesting to note that the most frequent presentation of posttraumatic headache is "clinically indistinguishable from chronic muscle contraction headache that is unrelated to trauma" (tension-type headache).[117] A logical extrapolation is that posttraumatic headache of this type has myofascial TrPs just as tension-type headaches do. Saper notes, among other pain patterns, that of "myofascial-like pain with TrPs in the occipital, cervical, shoulder (trapezius, supraspinatus) and paraspinal regions."[95] He further recognizes that "Referred phenomena from suboccipital regions to frontal, vertex, or orbital regions have been documented and may account for the frequency of complex pain patterns" in posttraumatic headache. Interestingly, back in 1946, Simons and Wolff observed that injection of local anesthetic into "areas of deep tenderness" eliminated the pain of post-traumatic headache.[108]

Whether a primary or secondary cause of pain in these patients, it is highly likely that myofascial TrPs play a significant role in posttraumatic head and neck pain. Evaluation should include palpation for myofascial TrPs and, when identified, they should be treated with appropriate myofascial TrP pain management strategies (see Section D).

Head Pain Associated with Substances or Their Withdrawal. This category encompasses headaches now coined with the term "analgesic rebound" or "drug induced refractory" headache. These headaches are due to the excessive use of symptomatic medications such as aspirin, acetaminophen, nonsteroidal anti-inflammatory drugs, or ergots. Prophylactic medications no longer are effective. Clinical experience indicates that most patients with analgesic rebound headache have active myofascial TrPs contributing to their pain. However, these seem to resolve in most cases with detoxification from the offending substance and therefore seem to be secondary to the headache caused by the drug dependence. Research studies documenting the prevalence of myofascial pain in this population are lacking, as are studies looking at the efficacy of simple detoxification versus treatment of the myofascial pain versus both. Such studies are urgently needed.

Head, Neck or Facial Pain Associated with Disorder of Cranium, Neck, Eyes, Ears, Nose, Sinuses, Teeth, Mouth, or Other Facial or Cranial Structures, Including the TMJ. This category includes the various organic diseases such as inflammation, infection, degeneration, neoplastic invasion, and obstruction that may affect any of the organs in the craniofacial region, including the TMJ and the head and neck muscles (see Table 5.2).

While the majority of these disorders will present with acute symptomology and respond to appropriate acute disease treatment strategies, it is important to remember that nociceptive input produces secondary reflex muscle contraction and, if prolonged, contributes to the development of myofascial TrPs and associated referred pain.[24] The pain and symptoms arising from the subsequent myofascial TrPs will often persist even if the primary pain source is eliminated.

TEETH, JAWS AND RELATED STRUCTURES. The following is an example of a patient

Table 5.2 ***Organic Disease of Extracranial Structures***

Structures	*Diseases*
Cranial bone	Inflammation
Neck	Infection
Eyes	Degeneration
Ears	Obstruction
Nose and sinuses	Neoplastic invasion
Teeth and related structures	
Temporomandibular joints	
Head and neck muscles	

who had a prolonged painful problem with an upper molar. Despite ultimate resolution of the dental complaint, she began to complain of ipsilateral facial discomfort and ear symptoms which turned out to be myofascial TrP in origin.

Case report: A **39-year-old female presented with a chief complaint of fullness in her left ear and mild aching in her left jaw muscle. Extensive otolaryngologic workup had been normal, but a mild click in the left TMJ prompted a referral to a clinician trained in orofacial pain disorders. Careful questioning elicited a 2-year history of extensive dental work on the patient's upper left second molar including root canal treatment, apicoectomy (surgical removal of the root tip), and finally extraction, with pain on and off in this tooth during this time. Physical examination was most significant for active myofascial TrPs in the left trapezius, sternocleidomastoid, masseter and lateral pterygoid muscles which contributed to the left ear fullness and the aching in her left jaw. The left joint click was of no clinical significance. The patient improved with treatment aimed at resolving the myofascial TrPs. This included instruction in good posture and body mechanics, spray and stretch, self spray and stretch, and TrP injections in the left lateral pterygoid and masseter muscles.**

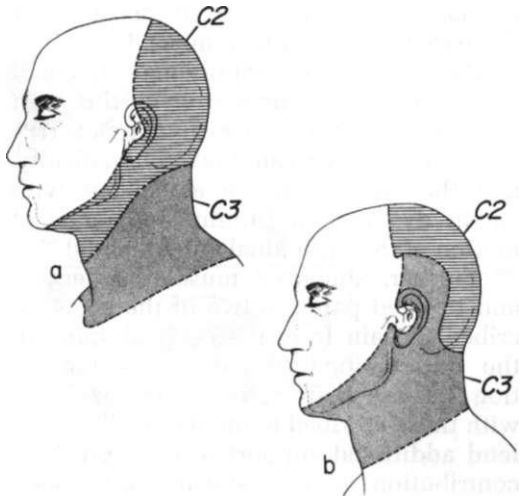


Figure 5.4. Cervical dermatomes, **a** represents the C_2 and C_3 tactile dermatomes as defined by Foerster[29] and **b** represents C_2 and C_3 pain dermatomes as defined by a study by Polleti[87] Not shown here is that C, also provides sensory innervation of the posterior scalp and that pain from C, may be perceived in the retro-orbital area, forehead and temple. Together C_{2}, C_2, and C_3 provide overlapping sensory innervation to the back of the head, lateral scalp, anterolateral neck down to and including the region of the clavicle, parts of the ear, the temporomandibular joint area, and the lower border of the mandible.

NECK. The neck is rife with structures that potentially could and probably do cause various painful conditions,[6,21] but controversy continues to abound as to its contribution to headaches and head pain. Only 2 subcategories for causes of neck pain are officially listed in the International Headache Society classification, namely the "cervical spine," and "retropharyngeal tendinitis."[81] Interestingly, under "cervical spine," inclusion criteria cite pain that *"project(s)* to forehead, orbital region, temples, vertex or ears," in addition to a local neck or occipital pain distribution. Further inclusion criteria require at least *one* of either: diminished cervical range of motion, abnormal cervical "muscle contour, texture, tone or response to active and passive stretching and contraction," or *"abnormal tenderness of neck muscles."* Radiologic studies should reveal some obvious pathology, abnormal posture or reduced range of motion.[81]

The cervical dermatomes include the back of the head, parts of the ear, the TMJ, and lower border of the mandible (Fig. 5.4). Rarely, nerve root irritation or entrapment may cause pain to be experienced in these dermatomal projections.[6] But what about the projection of pain to the forehead, orbit, temples, vertex or ears described in the International Headache Classification?[81]

The primary afferent nociceptors of the trigeminal nerve synapse in the nucleus caudalis of the spinal trigeminal tract. The nucleus caudalis descends as low as C_3-C_4 in the spinal cord. Many nociceptors from the deep cervical structures synapse on the same second-order pain transmission neurons as the trigeminal nerve.[58] Convergence and central modulation at these locations of nociceptive input such as that from

myofascial TrPs can readily account for these referred pain phenomena.[26,45,75,103]

Myofascial TrPs certainly may be one of the nociceptive sources causing this pain referral, especially since myofascial TrPs develop or are activated posttraumatically, and they appear to be associated with spinal dysfunction (diminished range of motion of the individual spinal joints).[50,64,102] Further, abnormal muscle tenderness and referred pain are two of the signs ascribed to pain from the cervical spine in the International Headache Classification.[81] These characteristics are consistent with those ascribed to myofascial TrPs and lend additional support to their probable contribution to cervical pain and associated headaches.

TEMPOROMANDIBULAR JOINTS. The TMJs, located anterior to the ears bilaterally, represent the articulation between the lower jaw and the cranium. They are such unique, complex, bilateral articulations that a brief review of functional anatomy and basic biomechanics is merited to make discussion of TM joint disorders easier to understand.

ANATOMY: The TMJs are bilateral, compound, synovial joints, with dense, nonvascular fibrous connective tissue covering the articular surfaces, located on the mandibular condyle and the glenoid fossa of the temporal bone (Fig. 5.5). This is in contrast to most synovial joints which have hyaline cartilage covering the articular surfaces. The fibrous tissue surface, a phylogenetic difference, allows for remodeling in response to stress, something hyaline cartilage cannot do. Interposed between the articular surfaces is an articular disc also composed of dense nonvascular fibrous tissue. The articular disc is tightly bound to the lateral and medial poles of the condyle and attaches anteriorly to the joint capsule. Posteriorly, the disc continues as a thick double layer of vascularized connective tissue which splits and, superiorly becomes a fibroelastic layer attaching to the posterior aspect of the glenoid fossa and inferiorly continues as a fibrous layer attaching to the posterior aspect of the condylar neck. Between the layers is highly vascular and innervated loose connective tissue that attaches to the posterior wall of the joint capsule. The disc essentially divides the joint into upper and lower compartments and functions as a third bone in the articulation allowing ginglymo-arthrodial (hinge-sliding) movements.

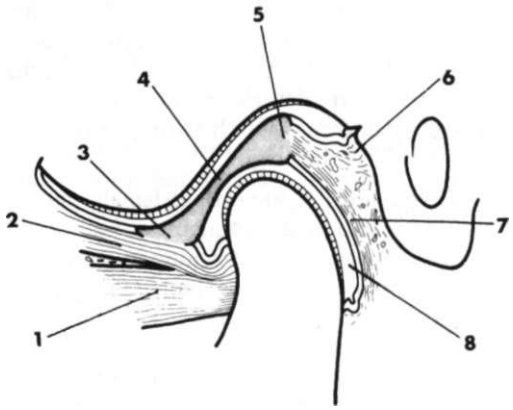


Figure 5.5. Sagittal diagram of the medial third of the TM joint depicting the following structures: (1) inferior portion of and (2) superior portion of the lateral pterygoid muscle; (3) anterior band of, (4) central portion of, (5) and posterior band of the articular disc; (6) superior lamina of the posterior attachment, (7) inferior lamina of the posterior attachment, (8) lower synovial space. Left side of figure is anterior. (Reprinted with permission from Solberg WK. Temporomandibular disorders. *Br Dent J* 1986.)

Inferior and posterior dislocation of the condyle is limited by the fibrous joint capsule and a thickened anterolateral and lateral portion of the joint capsule, called the temporomandibular ligament. Stability during movement is provided by the posterior temporalis and inferior head of lateral pterygoid muscles. For a more complete review of anatomy and biomechanics of the TMJ, the reader is referred to Sarnat and Laskin,[96] Bell,[5] Solberg and Clark,[115] and others.[16,77]

BIOMECHANICS: The morphology and structural arrangement of a joint dictates its movement. The TMJ is considered a compound joint because it has two parts that move in different ways. The articular disc is a key factor in its biomechanics. The condyle articulates against disc for mostly hinge type movement during early jaw opening (20-30 mm). The disc and condyle then function together to glide

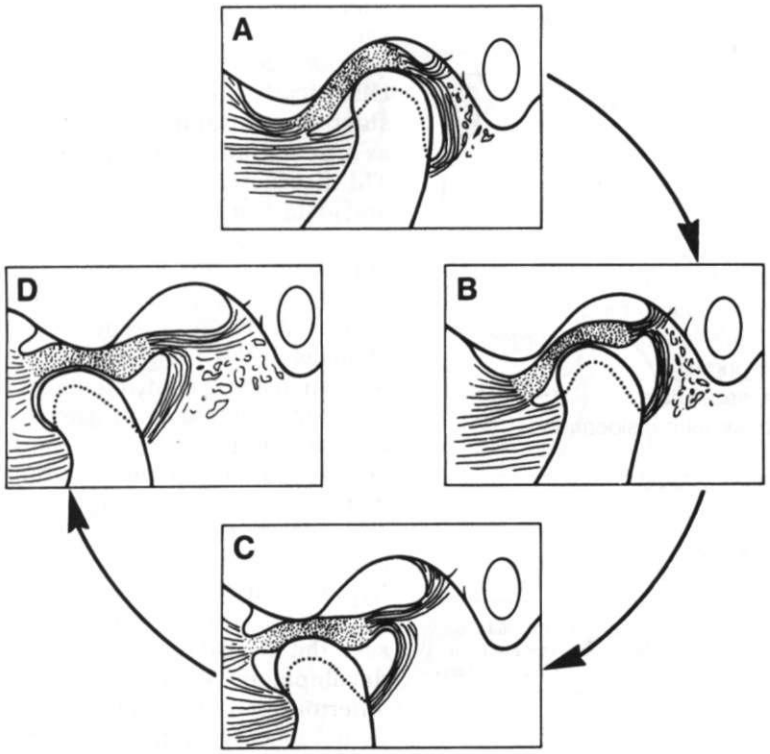


Figure 5.6. Normal temporomandibular joint function during opening movement, as seen by arthrography. The disc is the stippled structure between the condyle below, and the temporal bone above. **A,** mandible in the closed position. **B-D,** progressive stages of opening. The disc slides forward with the condyle as it translates to, and sometimes over, the articular eminence. The superior lamina of the posterior attachment becomes stretched, the inferior lamina does not. (Reproduced with permission from Solberg WK, Clark GT. *Temporomandibular Joint Problems: Biologic Diagnosis and Treatment.* Quintessence, Chicago, 1980:73.)

down the articular eminence for full jaw opening (Fig. 5.6).

BIOMECHANICS IN INTERNAL DERANGEMENTS: The term "internal derangement" applies to all joints and encompasses those disorders causing mechanical interferences to normal joint function. In TMJs, this involves primarily displacement and distortion of the articular disc, as well as remodeling of the articular surfaces, and joint hypermobility.[113] Many of the articular disorders affecting TMJs involve abnormal or restricted range of motion and noise, but are relatively painless. These include the congenital or developmental disorders, disc derangement disorders, osteoarthritis and ankylosis listed in Table 5.3. Any pain associated with these disorders is usually momentary and associated with pulling or stretching of ligaments. In the case of ankylosis, pain ensues if the mandible is forcibly opened beyond adhesive restrictions. Forcible opening can cause acute inflammation. Primary or secondary osteoarthritis, unless accompanied by synovitis, is also associated with minimal pain or dysfunction,[79] although crepitus and limited range of motion may be present. While clicking, irregular condylar movement and locking are the most common and early signs of internal derangement, it is only when accompanied by pain from inflammation or capsular pain with function that we see the development of myalgia, myofascial TrPs and referred symptoms.

Clicking occurs when there is anteromedial displacement of the articular disc,

Table 5.3 *Temporomandibular Joint Articular Disorders**

Congenital or developmental disorders
Aplasia
Hypoplasia
Hyperplasia
Neoplasia
Disc derangement disorders
Disc displacement with reduction
Disc displacement without reduction
Osteoarthritis (non-inflammatory disorders)
Osteoarthritis: primary
Osteoarthritis: secondary
Temporomandibular joint dislocation
Ankylosis
Fracture (condylar process)
Inflammatory disorders
Capsulitis/Synovitis
Polyarthritides

•Adapted from the American Academy of Orofacial Pain.[72] The classification is intended to be used as the TM joint sub-grouping in the International Headache Society classification outlined at the beginning of this chapter.

which the condyle must override to reach its normal position for full mouth opening (Fig. 5.7). The clicking sound is caused by the impact of the disc-condyle complex against the articular eminence.[113] Clicking is usually reciprocal, a second less pronounced click occurring as the condyle slips off of the disc again on closing. Clicking may progress to locking, where the disc no longer reduces onto the condyle for translation, and the joint movement is essentially blocked by the folded and deformed articular disc (Fig. 5.8). Since both clicking and locking are usually painless problems, patients often present themselves for evaluation only after they no longer can consistently reduce a locking joint themselves, the chief complaint being restriction, not pain. Pain may ensue however, if disc displacement continues.[113] Jaw opening may actually improve over time, but may be attended by increased pain on functioning as inflammation and osteoarthritis accompany the internal derangement. Chronic internal derangements tend to have a self-limiting course of 3-5 years[91] and since many are painless or silent, people often do not choose to seek care.[113]

ROLE OF OCCLUSION IN TM DISORDERS: Historically the dental profession has considered occlusal disharmony and variation as a potentially primary etiologic factor in TM disorders. However, literature reviews and data from recent studies do not support occlusion as a significant etiologic component to TM disorders.[70,89,90,120] Even loss of molar support, which seems to correlate with the occurrence of osteoarthritic changes in the TMJ, has no identifiable effect when age is controlled for, since loss of teeth and incidence of osteoarthritis both increase with age.[121,122] Nor do 4-6 mm changes in occlusal vertical dimension (the distance between a point on the maxilla and one on the mandible when the teeth are in occlusion) cause masticatory muscle hyperactivity or other TM disorder symptoms.[93] Logistic regression analysis to assess the contribution of occlusion to the development of TM disorders found that anterior open bite (when the anterior teeth do not meet when the posterior teeth are in occlusion) correlated with osteoarthritis and myofascial pain due to TrPs. However, the authors of this study felt that the occlusal changes in osteoarthritis are probably secondary to the joint changes and not etiologic.[90] In contrast, the association of myofascial TrP pain to anterior open bite in the absence of osteoarthritis was puzzling. It was unclear whether the anterior open bite preceded the TrP pain or whether the presence myofascial TrPs caused the change in occlusion.

The contribution of occlusion to the *etiology* of TMJ disorders and myofascial pain due to TrPs remains unclear, controversial, and needs research investigation. However, TM disorder patients with complaints or signs of occlusal alteration, should routinely be examined for masticatory muscle TrPs, since unilateral shortening of masticatory muscles due to TrPs may shift the mandible causing an easily reversible occlusal change. Masticatory muscle myofascial TrPs should be inactivated prior to initiating any prosthodontic treatment.

IMPACT OF TMJ DISORDERS ON MYOFASCIAL TRPS: Nonpainful joint disorders in and of themselves rarely cause myofascial TrPs to

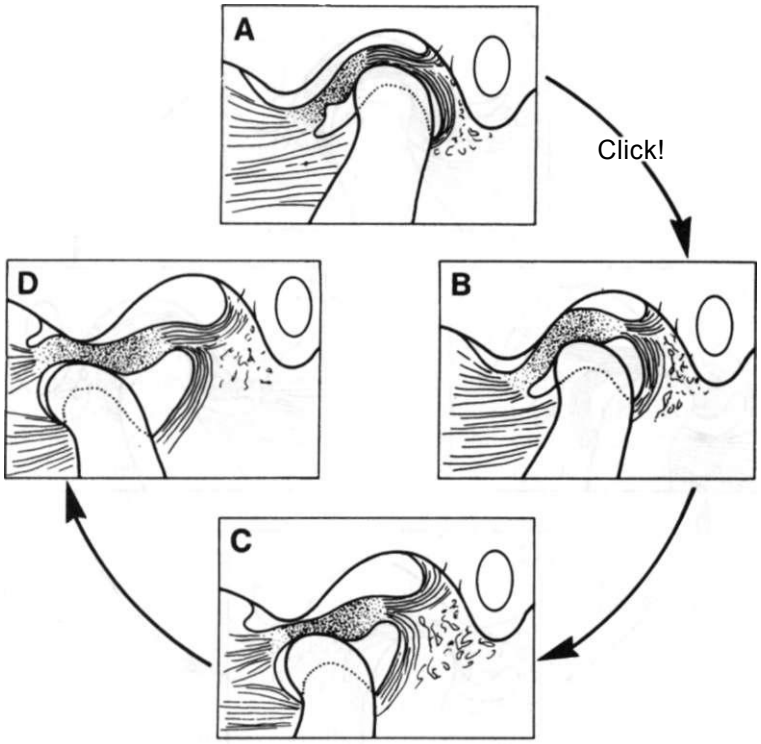


Figure 5.7. Mechanism of early *click* due to slight anterior displacement of the articular disc. **A,** rest position. **B,** as the condyle begins to translate forward, it must override a thickness of posterior disc material, causing a click. This seats the condyle in the central, thin part of the disc. **C** and **D,** after the click, mandibular opening and translation of the condyle proceed with apparently normal disc mechanics. (Reproduced with permission from Solberg WK, Clark GT. *Temporomandibular Joint Problems: Biologic Diagnosis and Treatment.* Quintessence, Chicago, 1980:75.)

develop. It is the acute inflammatory processes listed in Table 5.3, which may intermittently or persistently accompany chronic joint conditions, that tend to herald the onset of myofascial TrPs. Acute inflammation intrinsic to the joint or acute stages of arthritis are the usual causes of pain emanating from the joint itself. In a study conducted at the University of Minnesota TMJ and Facial Pain Clinic, doctors evaluated 296 consecutive patients with chronic head and neck pain complaints.[31] Only 21% of these patients had a temporomandibular joint disorder as the primary cause of pain. In all 21% the joint disorder included an inflammation of the TMJ capsule or the retrodiscal tissues. This type of pain is characteristically periarticular and aching in quality and will respond to acute pain management therapies *[see* Section C). However, since these disorders are almost always accompanied by reflex muscle splinting, spasm or pain, it is common to see the development of myofascial TrPs, especially if the inflammation is prolonged or recurrent. Myofascial pain due to TrPs was the primary diagnosis in 55.4% of the patients in the Minnesota study, almost 3 times the incidence of primary joint pain. Nonpainful internal derangements of the TMJs were felt to be a perpetuating factor to the myofascial TrPs in 30.4%.[31] Considering this data, it is important to make a distinction between true temporomandibular joint pain, myofascial pain due to TrPs alone, and myofascial pain due to TrPs that is being perpetuated by a noninflammatory or intermittently inflammatory joint condition. Treatment priorities will be affected accordingly. In order to determine the extent of joint involvement, a simple TMJ screening examination is described in Section C of this chapter.

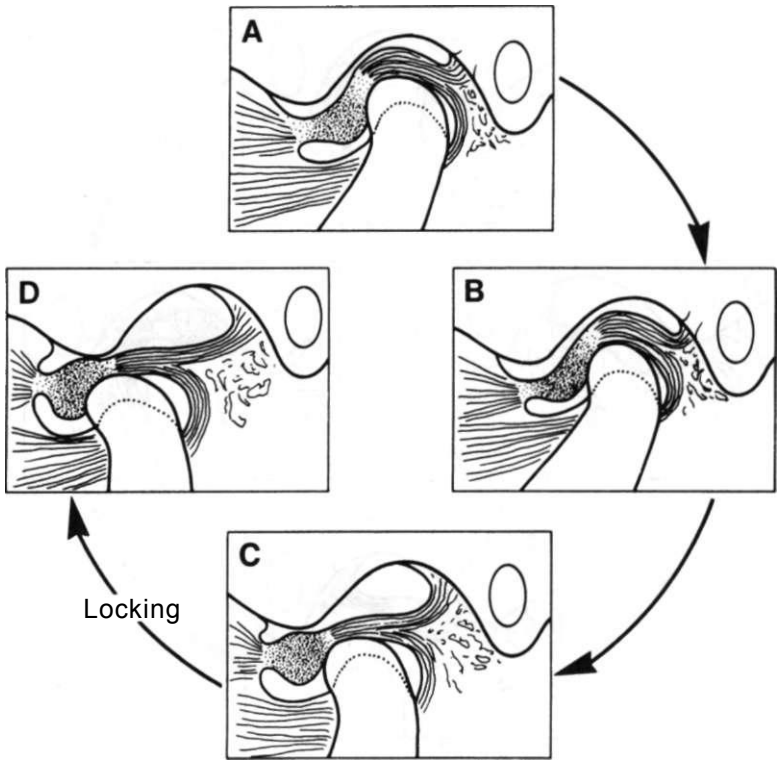


Figure 5.8. Mechanism of blocking mandibular depression at one point due to marked anterior displacement of the articular disc. **A,** rest position. **B,** as the condyle translates forward, it impinges on the disc, but is unable to ride over it. **C** and **D,** this blocks full forward translation, and thereby, full jaw opening, (Reproduced with permission from Solberg WK, Clark GT. *Temporomandibular Joint Problems: Biologic Diagnosis and Treatment.* Quintessence, Chicago, 1980:77.)

The following is a typical case example of a patient who had an acute exacerbation of a chronic TMJ condition followed by persistent symptoms due to myofascial TrPs.

Case report: A **47-year-old man with a long history of painless internal derangement of both TMJs presented with an acute left TMJ inflammation. This was conservatively treated with rest and anti-inflammatory medications. Severe symptoms subsided, but the patient continued to complain of persistent mild "aching of the left jaw" and ringing in his left ear especially with clenching. Careful history and examination revealed that the pain was no longer specifically over the joint, but was actually inferior and anterior to the left TMJ over the masseter muscle. Range of motion of the TMJ had increased from 41 to 47 mm and the joint was non-tender to palpation. Palpation of the masseter muscle, particularly the deep fibers, reproduced the patient's current symptoms. Myofascial TrPs in this part of the masseter muscle have been reported to cause unilateral tinnitus and accounted for the high pitched sound the patient complained of with clenching. The nonastute clinician may direct his energies towards treating the TMJs, especially since there is definite internal derangement bilaterally, worse on the left. Unfortunately, the source of the pain is now from masseter myofascial TrPs and not the joint. Treatment must begin with inactivating the TrPs.**

MASTICATORY, HEAD AND NECK MUSCLES. Muscle and other soft tissue disorders are the most common source of pain in the general population.[57] Much controversy still exists with nomenclature and on what

the different sources of muscle pain are and how to define and accurately differentiate between the various clinical presentations such as splinting or spasm or localized myalgia or myofascial pain due to TrPs. This area of controversy and confusion has very recently been thoroughly reviewed and greatly clarified.[76, 107] Certainly, in chronic pain settings, including university based TMJ and Craniofacial Pain clinics, myofascial pain due to TrPs, as defined in this text, is the most prevalent cause of painful symptoms.[28,31,110,112]

The dental literature is brimming with material discussing the role of the TMJ and the associated masticatory musculature in the production of various painful conditions now typically referred to under the umbrella term "TMD" or "temporomandibular disorders." Unfortunately, to this day, many terms relating to myofascial pain due to TrPs and TMD are used interchangeably and definitions of myofascial pain vary depending on who is writing the article, chapter, or book.

For example, years ago Laskin[62] coined the term "myofascial pain dysfunction" or "MPD" syndrome, requiring only *one* of the following symptoms to make the diagnosis: unilateral pain in or around the ear or preauricular area, masticatory muscle tenderness, painful TMJ noises, limited or deviating jaw opening. Objective findings were required to be negative: no radiographic evidence of TMJ disease and no tenderness on palpation of the TMJ via the auditory meatus. Clearly, this vague and very broad list of inclusion criteria resulted in the use of this term as a catch all diagnostic category for any patient with facial pain of unknown origin. It has also led to a misunderstanding of myofascial pain due to TrPs as it is defined today.[104,105] Many physicians and dentists alike, still insist on calling it *myofacial* pain, and think of it as a myalgia of the facial or masticatory muscles. Others feel it is a syndrome that involves some internal derangement of the TMJ plus associated local muscle soreness. But myofascial pain due to TrPs is not limited to the head and neck region, nor is it primarily related to TMJ problems or TMD.

Even as recently as 1992, the term "myofascial pain" was used by Dworkin and his colleagues to describe any facial pain complaint accompanied by masticatory muscle tenderness to palpation with or without limited range of motion of the jaw.[18] No mention is made of focal muscle tenderness, palpable nodules and taut bands within the muscle, or patient recognition of referred painful symptoms, the key diagnostic criteria for myofascial pain due to TrPs as described here. The use of the term "myofascial pain" for what essentially amounts to masticatory muscle allodynia, was proposed by very respected researchers in the area of TMD. They propose using this definition as research diagnostic criteria. Although the authors admit that the choice of the term "myofascial pain" as opposed to myalgia, fibromyositis or fibromyalgia, was rather arbitrary as a muscle pain descriptor, this definition will probably perpetuate broad misuse and misunderstanding of myofascial pain due to TrPs and related terms for years to come.

Multiple definitions of the same term and use of different terms to define the same clinical phenomenon obviously cause significant problems in interpreting research data, comparing studies or simply understanding what different authors may be describing or treating. In view of this, the following clinical diagnostic criteria for myofascial pain due to TrPs are reiterated and their use is encouraged. These diagnostic criteria have been successfully used in previous studies[52,92,110] and are a reasonable step towards separating simple local muscle tenderness (allodynia) from myofascial pain due to TrPs for research purposes.

For a complete list of diagnostic criteria for myofascial TrPs, see Table 2.4B. The diagnosis of myofascial pain due to TrPs depends on, at the very least, the presence of all of the following:

1. Regional or local pain situated in any structure of the body, typically with a deep, aching quality.
2. Presence of a focally tender spot in a taut band of skeletal muscle (the TrP), usually but not invariably, distant from or outside of the clinical pain site.
3. The application of 2-4 kg/cm^2 of pressure on the TrP will reproduce the clinical pain complaint within 10 seconds.[45a]
4. Diminished range of motion of the involved muscle due to pain.

Cranial Neuralgias, Nerve Trunk Pain and Deafferentation Pain. This group of pains encompasses those disorders involving nerve injury or dysfunction of the sensory component of any of the cranial or cervical nerves. This is in contrast to the normal transmission of nociceptive information along these primary afferent nociceptors. In general, this classification of pains can be divided into two main groups, persistent or continuous, and paroxysmal, based on their temporal pattern (*see* Table 5.4).

Table 5.4 ***Cranial Neuralgias***

Persistent/Continuous
Post herpetic neuralgia
Post traumatic neuralgia
Anesthesia dolorosa
Neuritis
Paroxysmal
Trigeminal neuralgia
Glossopharyngeal neuralgia
Nervus intermedius neuralgia
Superior laryngeal neuralgia
Occipital neuralgia
Neuroma

PERSISTENT NEURALGIAS. The persistent neuralgias produce unremitting pain which, by its very nature, may be accompanied by prolonged reflex muscle contraction and postural strain as the patient avoids movements that may trigger or intensify the neuritic pain. This type of cumulative microtrauma is a suspected precursor to the development of myofascial TrP pain.[24] Patients with post herpetic neuralgia, for example, will inevitably complain of the burning, tingling, dysesthetic pain associated with nerve dysfunction, but also often relate a deep, aching component to their pain, which is characteristic of musculoskeletal and myofascial TrP pain.[27] Clinically, examination of these patients reveals that many of them do have active myofascial TrPs contributing to their pain, although blinded or controlled research studies documenting this have not been published. One paper does report the presence of intercostal muscle TrPs, following acute herpes zoster of the intercostal nerves, that responded well to TrP injections.[11] Because of the prolonged suffering postherpetic neuralgia causes in a predominantly elderly population, it would be useful to determine how much of the persistent pain is actually myofascial TrP in origin. Systematic studies are needed to determine the prevalence of myofascial TrPs in this type of patient and, if significant, whether treatment of the myofascial TrP component is necessary once the neuropathic pain resolves or is controlled, or whether there is any clinical benefit to treating only the myofascial TrP component, especially if the neuropathic component is poorly controlled.

PAROXYSMAL NEURALGIAS. The paroxysmal neuralgias are less likely to be accompanied by myofascial pain due to TrPs because of the very brief and intermittent nature of the pain. Unpublished data from a UCLA study looking at 36 trigeminal neuralgia patients failed to show any direct association of trigeminal neuralgia with myofascial pain due to TrPs.[34] What may be seen is the emergence of a "new pain" that is myofascial TrP in origin and results from repeated muscle splinting against neuralgic pain paroxysms. Consider the following case.

Case report: **A 63-year-old female presented with classic left sided second and third division trigeminal neuralgia. She was started on slowly increasing doses of carbamazepine (Tegretol) to control the pain paroxysms, but returned the following week complaining of a new pain in the tip of her chin on the left side. The neuralgic pains were improved but not completely controlled yet. The new pain had a deep aching character and was fairly constant and continuous. The patient was seen bracing herself against the neuralgic pain by tipping her head to the side of the pain and raising her left shoulder. Careful examination revealed an active myofascial TrP in the belly of the left sternocleidomastoid muscle that intensified her chin pain when palpated. Injection of the sternocleidomastoid TrP with 0.5% procaine, followed by stretching, immediately relieved the chin pain which did not return. Good control of the neuralgia was finally achieved with daily doses of 1200 mg of Tegretol.**

While it is rare to see myofascial pain with most of the paroxysmal cranial neuralgias, it has been documented as actually mimicking occipital neuralgia.[36] The Headache Classification Committee of the International Headache Society describes the pain of occipital neuralgia as a paroxysmal stabbing but notes that aching may occur between paroxysms.[81] Classic descriptions of occipital neuralgia have documented the pain as being both paroxysmal and continuous with burning and aching qualities.[7,40,47] Radiation of pain to the frontal region is common. These descriptions are consistent with both neuropathic and musculoskeletal pain (burning/stabbing and aching respectively) and myofascial pain due to TrPs (aching with referred symptoms). In addition to occasionally being a purely myofascial problem,[36] the occipital nerve may become entrapped by taut muscle bands associated with myofascial TrPs as it passes through the semispinalis capitis muscle. This would account for the aching pain and referred frontal symptoms (myofascial TrP pain) as well as the neuritic pain from entrapment. The Headache Classification Committee has noted that "occipital neuralgia must be distinguished from the occipital referral of pain from the atlantoaxial or upper zygapophyseal joints or from *tender TrPs in neck muscles or their insertion."*[81]

Since classic treatments for true occipital neuralgia often involve invasive and irreversible surgical techniques, the prudent clinician will always rule out myofascial TrPs first. If encountered, competent myofascial pain management should precede any definitive neuroablative treatment. Effective treatment of the myofascial pain due to TrPs may also concurrently resolve related neuropathic pain by relieving associated nerve compression. The need for surgery is then obviated.

Head and Neck Pains Not Classifiable. This category officially contains "any type of headache which does not fulfill criteria for one of the disorders described in [the International Headache Society classification]."[81] One such headache is cervicogenic headache which was first described by Sjaastad and his colleagues in 1983,[109] but has not yet found a formal place in the headache classification. Cervicogenic headaches are described as predominantly unilateral fronto-temporal headaches with otherwise migraine-like characteristics. Distinguishing features include consistent unilaterality, precipitation with neck movement or *pressure on certain tender spots in the neck,* and associated shoulder and arm pain with neck stiffness. Onset is often preceded by trauma.

Debate continues as to whether cervicogenic headaches are a distinct headache entity and, if so, what the mechanism of pain is. Cervicogenic headaches have many features in common with migraine without aura, tension-type headache and posttraumatic headache. One unifying feature is the presence of myofascial TrPs in all of these headache types. That patients with cervicogenic headaches have myofascial TrPs that reproduce their headache pain was documented in an evaluation of 11 patients diagnosed by Sjaastad himself as fulfilling the criteria for cervicogenic headaches.[50] Other authors cite the presence of a "trigger point" as a diagnostic feature in cervicogenic headache.[30, 85] This "trigger point" is described as being "a circumscribed hypersensitive skin and *muscle* spot with a reduced pain threshold"[85] (a description consistent with but not sufficient to diagnose myofascial TrPs), or as being located over specific anatomical sites in the neck or nuchal line without specifically implicating muscle, nerve or bone.[30]

The fact that many cervicogenic headache patients have a history of trauma supports the idea of unilateral cervical soft tissue injury, protective muscle splinting and subsequent myofascial TrP development. Studies have documented that most cervicogenic headache patients have reduced segmental cervical spine mobility.[84,109] It has been proposed that reduced mobility may be due to a fibrous "fixation of tissue" between the cervical joints (an intraoperative observation made by Gronbaek[38]), and that the "fixation of tissue" may accompany healing following cervical trauma. However, unless muscle tension and shortening due to TrPs has been eliminated, it is hazardous to assume fibrous fixation. For instance, conservative treatment of a small group of cervicogenic headache patients

using myofascial pain management strategies was successful at significantly reducing both the frequency and intensity of these headaches.[50] As with occipital neuralgia, many of the classic treatments for cervicogenic headaches involve invasive neuroablative techniques or surgery, best avoided if effective conservative treatments are available. It is unfortunate that the treatable headache of so many patients is considered "unclassifiable" because the examiner needs to learn how to find and diagnose myofascial TrPs.

C. SCREENING EXAMINATION AND TREATMENT RATIONALE FOR TEMPOROMANDIBULAR DISORDERS; EVALUATION AND CORRECTION OF ANTERIOR HEAD POSITIONING AND POOR BODY MECHANICS AS THEY RELATE TO MYOFASCIAL TRIGGER POINTS

This section reviews screening examinations for TMJ disorders and anterior head positioning, along with basic treatment or corrective strategies for each. In addition, screening for and correcting poor body mechanics is also reviewed. However, regardless of what the preliminary diagnosis might be, or even if there are positive findings on TMJ screening exam, it is important to remember that a systematic and thorough examination of *all* of the head and neck muscles looking for active and latent myofascial TrPs is essential for complete evaluation of any persistent or chronic head and neck pain complaint. This textbook provides the information needed for examination of each of the head and neck muscles in the individual muscle chapters. With time and practice, the clinician can become very proficient at systematically examining and recording the sensitivity and referred pain, dysfunction, or other symptoms from each individual muscle. Regardless of diagnosis, myofascial pain due to TrPs is likely to contribute to and complicate the clinical picture and management of most chronic pain complaints *[see* Section B of this chapter).

Screening Examination for Temporomandibular Joint Disorders

Of course, the most important part of any diagnostic effort is obtaining a good history of the complaint(s); this will often be enough to make a fairly accurate preliminary determination of the probable cause. Once it is clear that the patient may be suffering from either a TM *joint* disorder, or myofascial pain due to *TrPs,* or a combination of the two, the following examination techniques will help delineate the extent of TMJ involvement.

Joint Capsule Tenderness.

EXAM. Since pain emanating from the TMJ itself is almost always associated with inflammation of the joint capsule or retrodiscal tissues, the simplest test is to palpate the joints to determine whether or not the tenderness of an acute inflammation is present. For the TMJs, this involves two different palpations. The first is over the lateral poles which are found just anterior to the tragus of the ear where one can feel the joint move when the mouth is opened and closed. This tests for capsular inflammation. The second involves placing a finger in each external auditory meatus to access the posterior superior part of the joint where potentially inflamed retrodiscal tissues are.

Palpation of the lateral poles is accomplished by simultaneously applying pressure to both joints with the tip of the index fingers anterior to the tragus of the ear. Firm palpation may be *uncomfortable,* but is only *painful* if the joint capsule is inflamed. Simultaneous palpation allows the patient to compare one side to the other (Fig. 5.9).

Palpation of the retrodiscal tissues is achieved by placing the little fingers just inside each ear and gently pressing down on top of the joint. A normal joint may exhibit discomfort with this palpation but should not be painful (Fig. 5.10).

An interesting observation is the complaint of persistent periarticular TMJ pain without true joint inflammation. In this situation, any tenderness to joint palpation is relatively mild compared to that typically seen with the acute inflammatory conditions. Rather, there are masseter, pterygoid or sternocleidomastoid muscle TrPs causing referred pain to the joint with associated secondary referred cutaneous and deep tissue hypersensitivity.[25,75,79] Diagnosis can be confirmed with spray and stretch

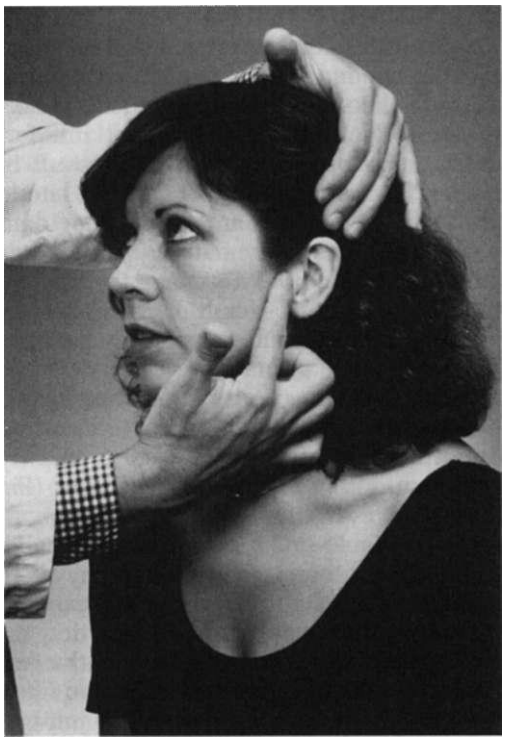


Figure **5.9.** Palpation of the lateral pole of the TMJ condyle is accomplished by applying pressure to the joint with the tip of the index finger anterior to the tragus of the ear. Simultaneous palpation of both joints is useful to allow the patient to compare pain or tenderness of one side to the other.

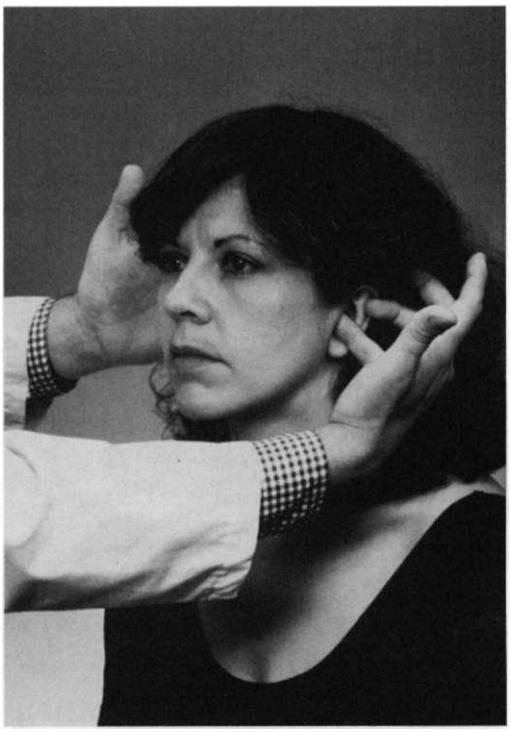


Figure 5.10. Palpation of the retrodiscal tissues of the TMJ is achieved by placing the little fingers just inside each ear and gently pressing down on top of the joint. Bilateral palpation is useful to allow comparison of pain or tenderness from one side to the other.

of the involved muscles. The spray alone can reduce both the referred joint pain and the referred hypersensitivity, and the stretch helps to inactivate the TrP cause of the tenderness.

CLINICAL SIGNIFICANCE. Presence of acute inflammatory TMJ pain is reason to refer the individual to a dentist trained in orofacial pain and TM disorders. Resolution of the joint inflammation will certainly be essential for resolution of any concurrent masticatory muscle myofascial TrPs. The pain from a hot joint will restrict any masticatory muscle stretching and TrPs will recur secondary to the central excitatory effects from the nociceptive source. Palliative care is essential to calm the joint down. One can start to manage any myofascial TrPs while instituting palliative joint care by simultaneously educating the patient in good posture and body mechanics *(see* Section D), having them reduce or eliminate damaging oral habits such as gum chewing, fingernail biting, pen chewing etc, and teaching them gentle stretches for the cervical muscles, if there is no cervical joint pathology to contraindicate stretching. Once the joint inflammation is under control, masticatory TrPs can be addressed, if still necessary. Once acute inflammatory conditions have been ruled out, the remaining tests will help determine the extent of TMJ internal derangement, if any exists.

Joint Sounds.

EXAM. While many TMJ disorders are accompanied by some variation of joint sounds, there is as yet no reproducibly reliable test or instrument to examine for these.[14] The most commonly used test is fingertip palpation; some clinicians augment this test with auscultation using a

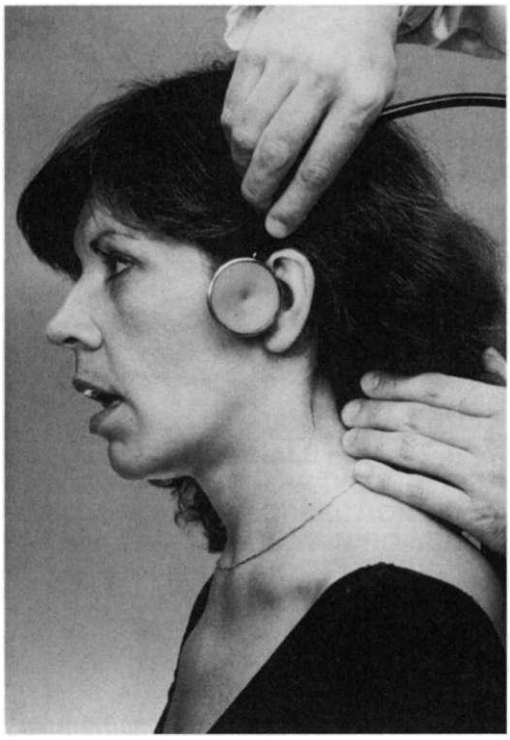


Figure 5.11. Auscultation of the TMJ for joint sounds using a stethoscope.

stethoscope (Fig. 5.11). Both techniques are only marginally reliable for research purposes,[20] but are widely used clinically.

Palpation involves placing the pad of the index fingers over each TMJ (just anterior to the tragus of the ear) while the patient opens and closes their mouth. A normal joint will be essentially silent and move smoothly. Crepitation (rough, sandy or diffuse noise or vibration) is usually a sign of degenerative joint changes (osteoarthrosis). Discrete clicks and pops may represent a mechanical problem with the disc, or more localized disc and articular surface abnormalities. The timing, quality and intensity of joint noises helps to define the type and severity of joint involvement, if any.[14] A loud discrete click on opening, followed by a quieter, less intense click on closing (called a reciprocal click) is typical of an anteriorly displaced disc with reduction (see Fig. 5.7). The location of the opening click is usually at wider jaw opening than the closing click which often occurs just before the teeth come together. Discrete clicks that occur at the same point on opening and closing probably represent discrete disc and articular surface abnormalities. Not all intra-articular interferences with joint movement will result in noise. Occasionally only a brief lateral shift in the mandible or condyle is evident on exam.[15] The presence of joint sounds alone, however, does not mean that the patient has a TM disorder. Many people have joint sounds without any sign of true joint disease.[14]

Auscultation using a stethoscope placed lightly over each TMJ while the patient opens and closes, may be used to amplify joint sounds for clinical purposes (Fig 5.11). However, the technique is only moderately reliable (50-65% agreement) using trained examiners, even when they were using a split stethoscope with two earpieces and one diaphragm.[19]

Because the mandible connects the two TMJs, transfer of vibration and sound often makes it difficult to assess which joint, if only one, is causing the noise or irregular movement. Sometimes the patient clearly senses which joint is involved. If not, another method involves continuing to palpate the lateral poles of the joints while the patient moves his or her jaw to the left and to the right without opening more than 1 or 2 mm. While it is generally accepted that an involved right joint will click or crepitate with jaw movements to the left and vice versa, this method of examination showed unacceptable between-examiner agreement for research purposes when studied.[14]

CLINICAL SIGNIFICANCE. Painless internal derangements are *not* a contraindication for treatment of myofascial TrPs. Treatment of myofascial TrPs in the masticatory elevator muscles typically involves stretching which requires wide jaw opening. In general, stretching is good for the joints, and wide jaw opening should not be discouraged.[114] Even clicking joints may be stretched ***unless:***

1. The click is painful,
2. There are significant episodes of locking (frequent episodes of inability to open the mouth without manipulation first), or
3. The patient relates a significant history of open dislocations.

The only caveat with wide jaw opening when there is a painless click in the joint is that the condyle should be on the disc before wide opening is attempted (i.e., it should click first).

On the other hand, if the patient complains of pain with clicking or an increasing frequency of locking closed, they should be evaluated by a dentist trained in orofacial pain and TM disorders.

Mandibular Range of Motion

EXAM. The normal *minimum* interincisal range of jaw opening is generally accepted to be between 36-44 mm,[79] with a maximum normal range of motion of up to 60 mm. A quick screening test for normal jaw opening is to ask the patient whether he or she can fit the first two knuckles of the nondominant hand between the incisor teeth. In the absence of internal derangement and elevator muscle myofascial trigger points, all normal people can do this, some can fit three knuckles. For a reproducible numerical value, interincisal opening should be measured with a sterilized millimeter ruler.[18] It is useful to use a ruler where zero is directly at one end of the ruler without any indentation space. Place the "0" end on top of one of the lower central incisors and measure to the incisal edge of the corresponding upper central incisor (Fig. 5.12). Always measure between the same central incisors in order to be able to compare measurements from one time to the next. This is a very reliable and reproducible clinical measure and "represents the gold standard for evaluating mandibular movement."[14]

Clinically three vertical measurements are useful: maximum comfortable opening, full unassisted opening (active range of motion) and assisted opening (passive range of motion).[18] The first is the pain-free range of motion and should be at least 36-44 mm.[79] Ask the patient to open up to the point at which he or she first experiences pain and measure. Then ask the patient to open as wide as possible regardless of pain and measure. Lastly, test the end feel of the joint and measure the passive range of motion by gently trying to open the jaw further. Place a thumb on the upper incisors and a middle finger on the lower incisors and *gently* pry the jaws apart.

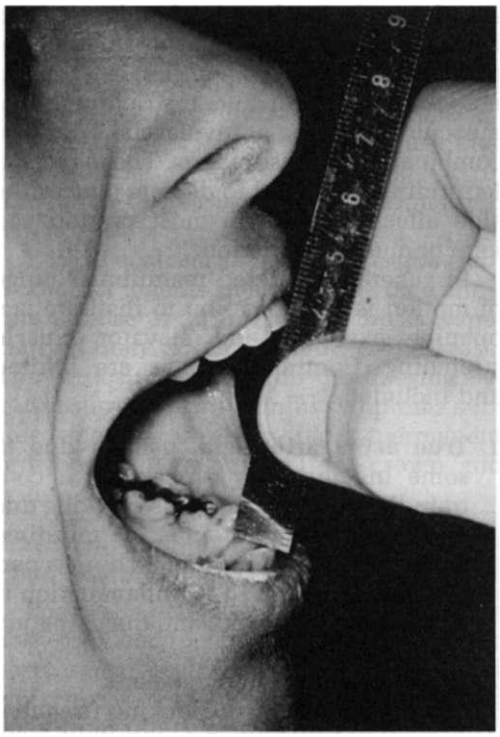


Figure 5.12. Measuring interincisal opening with a millimeter ruler. Place the "0" end of a sterilized millimeter ruler that has no indentation space on top of one of the lower central incisors and measure to the incisal edge of the corresponding upper central incisor.

While some feel that this test has poor reliability and may be difficult to interpret,[65] the following delineate clinical differences worth noting. A normal joint will have 1-2 mm of "give." Restriction of oral opening due to muscle splinting may result in a relatively dramatic increase in jaw opening with this maneuver, although the patient may complain of pain. Muscular restriction may also cause tremor and reflex contraction against the opening pressure. Restriction of oral opening due to mechanical obstruction or ankylosis in the TMJ will typically result in a hard end feel and no increased range.

CLINICAL SIGNIFICANCE. Hypermobility of the TMJ (jaw opening at or beyond the upper limits of normal, that is, greater than 60 mm) or a significant history of open dislocations are indications for *caution* with as-

sisted stretch. On the other hand, *restricted* mandibular opening indicates internal derangement or ankylosis of the TMJs, tightness of the joint capsule, restriction due to muscular splinting or myofascial TrPs, or a combination of these factors. The mandibular midline will tend to deviate toward the side affected with the most pronounced joint or muscle restriction.

In general, restricted mandibular range of motion is an indication to institute jaw opening exercises and elevator muscle stretching. Contraindications are limited and include:

1. **True acute arthralgia,** usually due to some inflammatory process, is a contraindication to excessive stretching due to pain and reflex muscle splinting. Once this has resolved, stretching is permissible if needed. TMJ inflammation is determined by history and the palpation exam described above.
2. ***Painful* internal derangement.**
3. **Significant history of locking** (frequent episodes of inability to open the mouth without manipulation first).

If a patient exhibits a limited range of mandibular motion and little is gained by muscle stretching, the TMJ capsule(s) may be tight. Mobilization of the TM joints may be accomplished by holding the jaw with the thumb behind the lower incisors and gently pulling the jaw forward without opening. The muscles must be relaxed to allow this movement, and this can be facilitated by having the patient gently rest his or her upper teeth on the operator's thumb nail. Once in protrusion, the thumb can be placed on the occlusal surface of the second molar on the side to be mobilized. A *gentle* downward pumping motion distracts the joint. Following this the thumb is placed lingual to the last molar and lateral forces are *gently* applied. If joint capsule tightness (or loss of joint play) is causing the restricted range of motion, these maneuvers should increase active opening by at least 5-10 mm.[114] Reduced mandibular range of motion that responds neither to spray and stretch, nor to joint mobilization, may indicate TMJ ankylosis or an anteriorly displaced disc without reduction. In this case referral to a dentist trained in TM disorders is strongly recommended.

Mandibular Path of Opening and Closing

EXAM. Without measuring, observe the path of opening and closing, looking for deflections and deviations from a straight path: these may be indicative of either mechanical problems within the joint or muscle incoordination or unilateral shortening.

CLINICAL SIGNIFICANCE. The jaw will tend to deflect toward the side affected with an internal derangement or ankylosis restricting range of motion of the individual joint, or to the side with elevator muscle shortening or elevator muscle myofascial TrPs. This sign alone, in the absence of inflammation or painful internal derangement, is not a contraindication for the treatment of myofascial TrPs. However, a significantly restricted mandibular range of motion (less than 36 mm) along with deflection to one side, and a hard end feel, may be indicative of either unilateral ankylosis or an anteriorly displaced disc without reduction. This situation merits an evaluation by a specialist in TM disorders, although basic myofascial TrP pain management strategies, such as good posture and body mechanics, cervical stretching (Section D) may certainly be instituted right away if desired.

Rationale for Treating Temporomandibular Disorders

When a history yields a complaint of pain localized to the TMJ or pain which started with an episode involving the TMJ, *and* screening examination yields significant positive joint findings, the patient should be referred to a dentist who specializes in the treatment of orofacial pain and temporomandibular disorders. The following summarizes very briefly a rationale for treatment of TM joint complaints.

Palliative Care. Palliative care is appropriate in acutely painful disorders such as capsulitis, synovitis, or during acute stages of arthritis. Palliative care includes, but is not limited to, placing the patient on a soft diet with instructions to reduce all abusive oral or jaw habits, prescribing a 7-10 day course of anti-inflammatory medications, and recommending placement of a cold pack or ice over one or both joints (10 minutes on, 10 minutes off) 2-3 times per day.

Definitive Therapy

ELIMINATION OF CAUSE. In the treatment of painful TMJ disorders, as with the treatment or management of pain due to myofascial TrPs, the clinician and patient both must take into account the patient's role in controlling causative and perpetuating factors, such as posture, body mechanics, functional demands and emotional tension. It is extremely important to educate the patient about all aspects of his or her disease and to enlist the patient's help and compliance prior to initiating therapy. Predisposing factors to TMJ disease include skeletal and craniofacial disharmonies, abnormal biomechanical loading (such as might occur with significant occlusal change or loss), and chronic microtrauma (such as may occur with bruxism, chronic clenching or excessive gum chewing). Precipitating factors include macrotrauma, emotional tension, arthritis, or any source of chronic deep pain input.

SYMPTOM RESOLUTION AND STABILIZATION OF INJURED PARTS. Any structural changes which have occurred in the TMJ will not resolve spontaneously even with elimination of etiologic factors. Remodeling does occur because of the fibrous tissue that covers the articular surfaces, but joint biomechanics may be compromised indefinitely and patients must be advised of this. However, it is important to remember that altered joint biomechanics and joint noises are common and usually painless. Patients must understand that they have an active role in achieving and maintaining a painfree state.

USE OF INTRAORAL ORTHOSES. Consideration to occlusal appliance therapy is given to those patients who have painful internal derangements and/or relate a history indicative of significant parafunction (such as chronic or frequent daytime clenching, nocturnal bruxism, focally painful joints or temples on awakening, or have other abusive oral habits such as gum chewing or finger nail biting), or who, on exam, have evidence of notable occlusal wear (such as lock and key patterns of the anterior teeth with excursive movements or flattening of molar cusps) and myalgia. While experimental evidence suggests that an occlusal appliance is nonspecific in its action,[68,69] it will, at the very least, protect the teeth from further damage and has been shown to offset muscle fatigue due to nocturnal bruxism.[13,116] The latter effect is likely due to the temporary alteration in muscle activity patterns.[12,78,101,116] Daytime use may be beneficial if the patient needs a reminder to decrease other deleterious oral habits.

Loss of occlusal support in one quadrant is another potential factor that may indicate the use of an intraoral orthosis, especially if there is symptomatic TMJ disease. The orthosis can provide the missing occlusal support until painful symptoms subside enough to allow more permanent prosthodontic reconstruction (crown and bridge or dentures).

Screening Examination for Anterior Head Positioning

Posture is defined as "the attitude of the body."[17] Good posture is when the body parts, muscles and bones, are aligned and work together in harmony, protecting the body from injury or progressive deformity, regardless of attitude. Poor posture is basically a bad, but correctable habit resulting in misalignment of various body parts. These body parts are at higher risk for injury or pain due to the increased strain misalignment places on the supporting structures.

The normal spine has two lordotic curves, one in the C-spine area and one in the lumbar region, when viewed from the side. Kyphosis occurs through the thoracic region. A plumb line from the external auditory meatus should pass through the shoulder and hip and end slightly anterior to the ankles. From the front, the head should be centered, the shoulders should be level, and the clavicles essentially parallel to the ground. Interestingly, the head is not perfectly balanced on top of the cervical spine. Rather, it's weight is slightly anterior to the center of gravity, accounting for the large posterior cervical muscles required to hold the head up and the rather tiny anterior neck muscles.

While there are several postural parameters that can be assessed for clinical completeness, the only one that will be discussed in this chapter is anterior head positioning because of its significant contributions to the perpetuation of myofascial

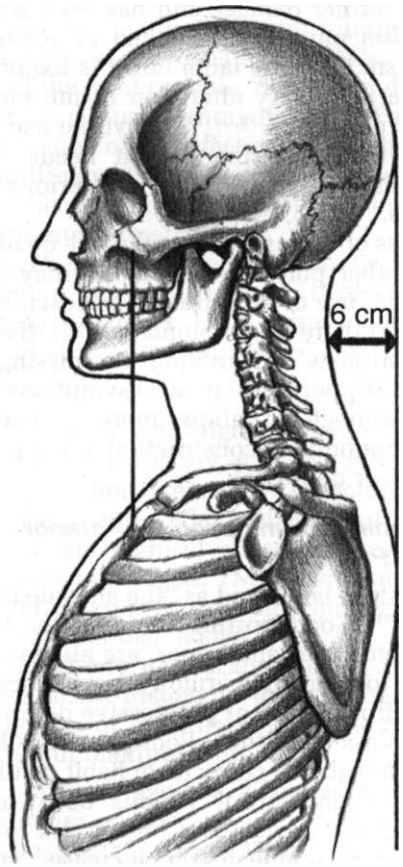


Figure 5.13. The normal head and neck posture illustrated in this figure minimizes demands on jaw and neck muscles. It shows the zygomatic (cheek) bone aligned over the manubrium of the sternum. The illustrated measure of head posture relates to a real or imaginary plumb line that just touches the kyphotic *curve* of the thoracic spine. A distance of 6 cm (2 3/8 inches) between the plumb line and the depth of the cervical lordosis is considered to be normal.

TrPs in the head, neck and shoulder muscles, as well as certain TM joint disorders.

Anterior Head Position.

EXAM. Assessment of anterior head position is probably the single most useful postural parameter in a patient with head and neck pain complaints. Looking at the patient from the side, place a real or imaginary plumb line on a tangent to the crest of the kyphotic curve of the thoracic spine. With a ruler, measure the distance from this line to the depth of the cervical curve. This measurement should be approximately 6 cm *(see* Fig. 5.13).

CLINICAL SIGNIFICANCE. A measurement of less than 6 cm is an indication of loss of cervical lordosis, frequently seen posttraumatically. Measurements in excess of 6 cm indicate anterior head positioning. Anterior head positioning occurs with rounded shoulders. The result is that the suboccipital, posterior cervical, upper trapezius and splenius capitis muscles contract and shorten to bring the head into extension to allow the eyes to gaze forward. Although the cervical spine can be hyperextended when a forward head position is present, it more often shows a loss of the normal lordosis with relative flattening of the curve. In this position the sternocleidomastoid muscle works at a mechanical disadvantage and becomes overloaded, as does the splenius cervicis. These muscles frequently become involved with TrPs when a forward head position is present. In addition to extra muscular work, forward head positioning also places an extra strain on the occipitoatlantal junction since the occiput is in an extended position relative to C_1. This increases the chances of compression pathology in this region. Anteriorly, the suprahyoid and infrahyoid muscles are placed in a stretch position creating increased elastic tension downward on the mandible, hyoid bone and tongue. As a result the mandibular elevator muscles reflexly contract to counteract the mouth opening forces from the suprahyoid and infrahyoid muscles. This reflex contraction results in increased EMG levels in the elevator muscles as well as increased intra-articular pressure in the TMJs.

Aside from the potential for compression of nerve roots, zygapophyseal facets and the posterior portions of the bodies of the cervical vertebrae from the attendant upper cervical extension, the increased muscular effort caused by anterior head positioning is a powerful perpetuating factor to myofascial TrPs in the cervical, masticatory, and upper shoulder girdle muscles. The resulting increased intra-articular pressure in the TMJs may contribute to the development of early clicking especially if the disc is already slightly thinned posteriorly.

Anterior head positioning is not only a problem with standing, but also occurs while sitting and while changing position *(see* Chapter 41, Section C). There are multiple situations throughout the day which

will aggravate anterior head position. History taking during initial evaluation can be extremely useful in terms of identifying poor body mechanics that may be perpetuating myofascial TrPs. The most common problem areas are described later in this section under "Body Mechanics".

Rationale for Treatment of Anterior Head Positioning

Restoration of normal posture, particularly normal head positioning, is the crucial first step in the management of almost any chronic head and neck pain condition. This is because myofascial TrPs are almost always a contributing, if not causative factor, and anterior head positioning perpetuates these.

Exercises to Achieve Good Posture To be maximally effective, all posture exercises must be repeated frequently throughout the day. A good rule of thumb is to repeat the exercises at least every 1-2 hours which translates into a minimum of 6 times per day. It is better to do one posture exercise 6 different times per day than to do 6 repetitions once. There is little point in performing 6 posture exercises in the morning and then walking around in poor posture for the rest of the day.

The following complete posture exercise has a shoulder posture component and a head posture component that together are designed to restore normal shoulder position and thoracic kyphosis as well as normal head position. All exercises require properly coordinated breathing to be optimally effective.

SHOULDER POSTURE. This exercise will bring abducted and protracted, rounded shoulders back and down while stretching the pectoralis muscles and strengthening the upper back muscles (Fig. 5.14). Have the patient:

Stand with his or her feet about 4 inches apart, arms at the sides, thumbs pointing forward.
Tighten the buttocks to stabilize the lower back.
Rotate his or her arms and shoulders *out* and *back* (thumbs pointing back) while inhaling, squeezing the shoulder blades together in the back.
Maintain this position while pulling the shoulders down and exhaling.
Hold this position while breathing normally and correcting the head posture which is described next.

Another stretching exercise for the pectoral muscles is illustrated in Figure 42.9.

HEAD POSTURE. This exercise is designed to correct the anterior head position and should be performed in conjunction with the previous shoulder posture exercise. Once shoulder posture has been corrected, have the patient *gently* move his or her head back to bring the ears in line with the shoulders (also known as axial extension). This must be accomplished without moving the nose up or down and without opening the mouth (Fig. 5.15).

Patients should hold the correct postural position for at least 6 seconds while breathing normally. Once complete, they should be advised to relax, but remain in good posture. They must not collapse into habitual poor posture. If the improved postural position feels uncomfortable or military, have the patient shift his or her body weight from the heels onto the balls of the feet. This moves the head backward over the shoulders as a counterweight and straightens the lines of weight bearing *(see* Fig. 41.4).

In addition to the posture exercise described above, patients should be taught correct tongue position. Correct tongue position with the teeth apart, relaxes the mandibular elevator muscles and eliminates clenching.

TONGUE POSITION. Patients should be instructed to keep the tongue on the roof of the mouth where it ends up when they say the letter "N" or the word "Boston". This will place the tongue on the roof of the mouth behind, but not touching, the upper teeth, with the teeth slightly apart. If possible, patients should bring their lips together and breathe through the nose.

Body Mechanics

Body mechanics is defined as: "the application of kinesiology to use of the body in daily life activities and to the prevention and correction of problems related to posture."[17] There are many situations that occur on a day to day basis that place the body, various joints and the muscles in positions requiring extra strain and work. The

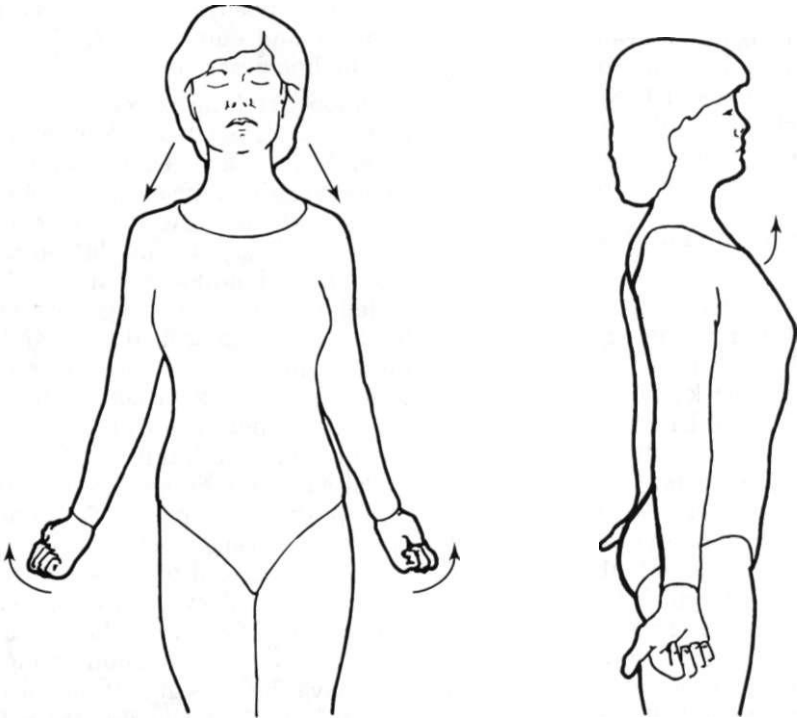


Figure 5.14. Shoulder posture exercise. To be performed in combination with the "Head posture exercise," Figure 5.15.
The patient should: 1. Stand with his or her feet about 4 inches apart, arms at the sides, thumbs pointing forward.
2. Tighten the buttocks to stabilize the lower back.
3. Rotate the thumbs, arms and shoulders *out* and *back* while inhaling, squeezing the shoulder blades together in the back.
4. Maintain this position while pulling the shoulders down and exhaling.
5. Hold this position while breathing normally and correcting the head posture described in Figure 5.15.

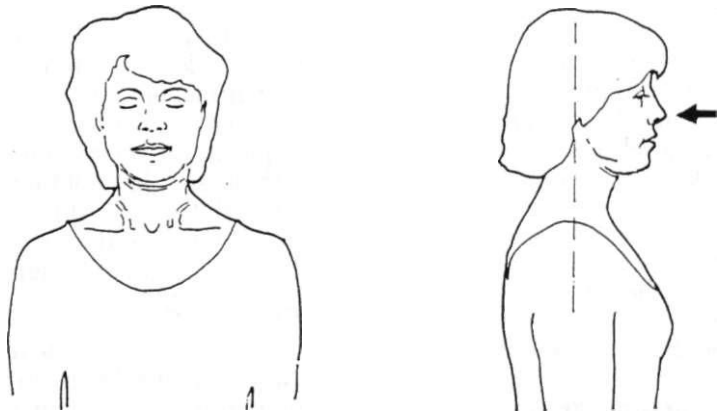


Figure 5.15. Head posture exercise. This exercise is designed to correct the anterior head position and should be performed in conjunction with the previous shoulder posture exercise. Once shoulder posture has been corrected, have the patient *gently* move the head back to bring the ears in line with the shoulders (also known as axial extension). This must be accomplished without moving the nose up or down and without opening the mouth.

best example is anterior head positioning described above, but this does not only occur with standing, but may be aggravated while sitting in a car or at a desk or in front of a computer, or while eating dinner or watching TV. Other common habits, such as sleeping posture and telephone use also deserve investigation in the chronic head and neck pain patient with or without myofascial TrPs.

The initial patient interview is a good time to gather basic information on body mechanics that may be aggravating joint and muscle pain. It is useful to ask about the amount of time spent at any one activity as well, since the longer someone is in a suboptimal position, the more problems it can produce.

Sleeping Posture.

HISTORY. Ask patients whether they sleep on the back, side or stomach. Then ask about how many pillows they use, and whether they are thick or thin, synthetic or down or foam and whether or not their bed is soft or firm.

CLINICAL SIGNIFICANCE. The best sleeping posture is on the back (supine) in a firm bed with adequate support of the cervical lordosis. Soft beds cause strain on all of the muscles and ligaments and should be replaced with a firm mattress, if possible. A plywood bed board almost as big as the mattress, placed between the mattress and the bed spring also may help. Alternatively, several separate boards **1.3** cm **(1/2** in) thick and **15-20** cm **(6-8** in) wide, cut three-quarters of the length of the mattress, may be placed lengthwise, but not crosswise, to correct the hammock-like sag of a soft bed. Support of cervical lordosis while sleeping on ones back can be easily achieved by using a soft pliable pillow under the head and neck and bringing the two comers up over the shoulders *(see* Fig. **7.7A).**

Patients should be discouraged from stomach (prone) sleeping with the head turned to one side as this places undue strain on the cervical joints and muscles. One solution for devout stomach sleepers is to have them use pillows under the chest to minimize head rotation. Another is to try and break the habit by tying a sheet around the waist with the knot on the stomach. Patients with lumbar disc dysfunction who may have been instructed to lie prone as part of a therapeutic program should place a pillow under the chest to minimize head/neck rotation, and they can use a pad under the forehead for support without rotation.

Side sleepers should concentrate on having sufficient pillow support between the head and neck, not the shoulder *(see* Fig. **7.7C),** and a bed that support the spine in a neutral position. For some patients, foam pillows should be avoided because their springiness aggravates TrP symptoms especially in the upper trapezius and sternocleidomastoid muscles.

Car Posture.

HISTORY. How many hours per day does the patient spend in a car? What kind of lumbar support is in the car? Usually there is little or none, or the patient uses it incorrectly, if at all.

CLINICAL SIGNIFICANCE Inadequate lumbar support causes loss of lumbar lordosis which results in collapse of the chest, forward rounding of the shoulders and extension of the head in an anterior position *(see* Fig **41.4).** Since many cars have "bucket" seats or little or no lumbar support, most people spend many hours per day peering over the dashboard in forward head posture along with all of the attendant muscle and joint strain this causes (Fig. **5.16).**

Using a lumbar support of some kind is *essential* whenever riding in a car whether as driver or passenger. This can be in the form of a built-in inflatable lumbar support available in many car seats now, a towel roll (described below) or can be more sophisticated to include such products as a McKenzie Lumbar Roll, SACRO-EASE or ACCU-BACK. If a towel roll or McKenzie lumbar roll is to be used, it should not be discarded in the back seat of the car upon getting in!

A firmly rolled towel provides a desirable combination of firmness and resilience for use as a lumbar support. It should be *approximately* **12** inches wide and **3-4** inches in diameter and should be placed in the back at the height about where a belt would normally go *(see* Figs. **16.4D** and **41.4).** It is important that the size of the roll be an appropriate fit for the

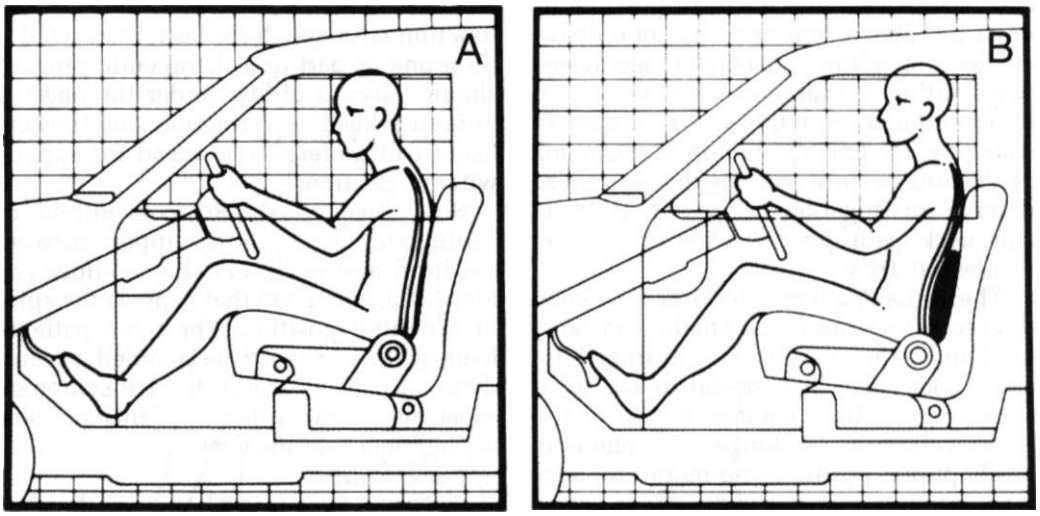


Figure 5.16. Car posture. A. Inadequate lumbar support in most car seats results in loss of lumbar lordosis which causes collapse of the chest, forward rounding of the shoulders and extension of the head in an anterior position. B. Use of lumbar support corrects this situation.

individual using it. The towel roll can be used as a more permanent lumbar support, either in the car, at home or in the office, by slipping it into an attractive cover and tying it to the seat. This will make it easier to use and improve compliance.

Office Habits.

HISTORY. How many hours per day do patients spend sitting at a desk, at a computer, reading or writing? What kind of chairs do they use? Do their feet reach the floor? How high is the computer monitor? Is there glare on the screen? How high is the keyboard?

CLINICAL SIGNIFICANCE. Once again, inadequate lumbar support will result in anterior head positioning. In an extensive study to determine what chair design causes minimum muscular stress, as measured electromyographically when typing, Lundervold[67] found that the chair should have: a backrest with a backward slope, a seat which is slightly hollowed out at the bottom to allow room for the buttocks, no casters, and firm upholstery. Seat height should be low enough so that the feet rest flat on the floor without compression of the thigh by the front edge of the seat. A foot rest may be used, if necessary, to avoid underthigh compression. The lower edge of the back rest is positioned to support that part of the lumbar spine which flexes the most when bending forward, and the upper edge of the backrest should reach high enough to cover and support at least the inferior angles of the scapulae.

Computer monitors that are too high or too low, or with glare on the glass, cause abnormal cervical postures while trying to read the screen. Patients must be instructed to correct these problems. A phone book can raise a computer screen that is too low, seating adjustments may correct a screen that is too high, and special screen covers can cut unnecessary glare.

If working on a computer, the keyboard should be kept as close to *lap level* as possible. If no keyboard table is available, it is better to have the keyboard on the knees than on a desk. Similarly, if typing, the undersurface of the typing table should fit just above the knees so that the arms and shoulders do not need to be raised to reach the typewriter keys.

If reading or writing, the chair should be pulled as close to the desk as possible. The work should be kept as close to the body as

possible and the *eyes* instead of the head should be turned down to look at the work *[see* Fig. 16.4). An inclined surface or a lapboard facilitates erect posture.

TV and Reading Habits.

HISTORY. How many hours per day do patients spend watching TV or reading? What position are they in for these activities and on what kind of furniture: sitting or lying on a soft or firm sofa, in bed, on a chair, on the floor? Does watching the TV require them to turn the head?

CLINICAL SIGNIFICANCE. *In addition* to the general features of chair design described above, the following are important considerations for chairs to be used for reading, socializing or watching TV, (but not for eating):

1. The *back of the chair* should slope *25-30 degrees back* from the vertical so that the hips do not need to slide forward for comfort.
2. The chair must have *armrests* that are high enough to provide support for the elbows without causing the shoulders to hike up. Without armrests, there will be a tendency to cross the arms in front of the chest for comfort. This causes the muscles across the front of the chest to shorten and rounds the shoulders forward.

Telephone Use.

HISTORY. Which side does the patient hold the phone on? How many hours per day does the patient spend answering and speaking on the phone? Does the patient cradle the phone between the ear and the shoulder when he or she needs to write something down?

CLINICAL SIGNIFICANCE. If answering the phone is part of a desk job, the phone should be in close proximity to the person to prevent repetitive reaching. When answering the phone, the phone should be hand held and not propped between the shoulder and the ear; this prevents muscle shortening and repetitive or prolonged compression of cervical joints. Speaker phones or headsets are essential if answering phones comprises a large part of the daily activities.

Chapter 41, Section C of this volume includes additional consideration of posture and body mechanics in relation to *movement* and *change* of position.

D. GENERAL TREATMENT APPROACH FOR CHRONIC HEADACHES, FACIAL, NECK OR SHOULDER PAIN WITH A MYOFASCIAL TRIGGER POINT COMPONENT

Acute vs. Chronic Myofascial Trigger Point Pain

Acute Single Muscle Syndromes. Myofascial TrP pain is a regional pain syndrome that may affect single muscles, especially in situations with a clearly defined etiologic incident such as localized trauma or infection. Acute single muscle syndromes often revert spontaneously to pain-free latent TrPs, but are subject to reactivation. Typically, if recognized early, single muscle myofascial TrPs respond easily to appropriate TrP release techniques and recurrence is rare. Failure to correctly diagnose myofascial TrPs when they first develop and become symptomatic, sets the stage for development of secondary and satellite TrPs in muscles in the pain referral sites and in synergistic and antagonistic muscles, greatly complicating the clinical picture and its treatment. Acute myofascial TrP pain syndromes become chronic myofascial pain syndromes through failure to resolve the acute problem promptly.

All of the relevant information for TrPs in specific muscles, including the referred pain pattern, symptoms, what activates or perpetuates them, how to examine for them, the differential diagnosis, TrP release and injection, and corrective actions, are detailed in the individual muscle chapters in this volume and, for the lower torso, pelvis, and lower limbs, in volume II. Once the pain picture becomes more complex, with multiple TrPs, overlapping pain patterns, and numerous perpetuating factors, acute single muscle pain treatment strategies will be much less effective and a chronic pain management approach needs to be instituted.

Chronic Myofascial TrP Pain. Chronic pain syndromes become complex, involving all aspects of the patient's life and, with rare exceptions, include a significant, if not dominant myofascial TrP component.[28] Effective resolution requires attention to all

aspects of involvement in a comprehensive, team-based program. Isolated attention to individual TrPs early in treatment can be helpful to demonstrate to the patients and reassure them that their problem has a basic organic cause which they can do something about, but it will require a comprehensive approach to unravel all the intertwining components.

For successful management of chronic myofascial TrP pain, one of the most important concepts is to teach patients that they can be in control of their pain in the long run. They must learn to live with their muscles by learning about them and respecting how they work. In this type of management program, the clinician's role is nor just to treat the pain for the patient as a service, but rather, to primarily teach and demonstrate to patients what they can and must do for themselves. Oftentimes, in primary care situations, just instituting correction of basic posture and body mechanics alone can go a long way in reducing pain frequency and intensity.

The program outlined here is designed for the individual physician or dentist with room for inclusion of other health care providers. Patients with complex pain complaints and multiple perpetuating factors may require more expertise than the individual practitioner has to offer in order to appropriately manage different components and perpetuating factors of the patient's pain. For example, psychological perpetuating factors, such as moderate to severe depression or anxiety, may require referral to a psychologist or psychiatrist; dental perpetuating factors, such as a painful internal derangement, or clenching or bruxing requiring construction of an intraoral orthosis, may require referral to a dentist trained in orofacial pain.

Quantifying the Pain Experience

Since pain, regardless of etiology, is a subjective experience that is communicated to us only through words and behaviors, measuring pain is extremely difficult. Unlike measuring blood pressure, temperature, or erythrocyte sedimentation rate, it is difficult to quantify the intensity of pain an individual is experiencing. There are several physiologic and psychologic factors that will influence the intensity of pain perceived. Other cognitive, behavioral, and learning factors will affect how this pain is communicated.

Nonetheless, measuring pain is important, not just for studying pain mechanisms in a laboratory, but also in order to assess treatment outcome. To this end a number of instruments have been developed and tested for their reliability and validity in measuring different aspects of the pain experience. The clinician is encouraged to use one or both of the following subjective pain measures prior to treatment, during and after the 6-week program in order to better assess progress. These scales are in addition to monitoring objective changes on physical exam, such as increased range of motion of the neck or jaws, or decreased TrP tenderness as measured with a pressure algometer *(see* Chapter 2, Section B).

Visual Analog Scales. *A* visual analog scale is an unmarked line which represents a continuum of a particular experience such as pain. The most common scale used for pain is a 10 cm line, either horizontal or vertical, with perpendicular stops at the ends. The ends are anchored by "No pain" and "Pain as bad as imaginable" (Fig. 5.17). Numbers should *not* be used along the line to ensure a better, less biased distribution of pain ratings; otherwise a disproportionately high frequency of 5's and 10's will be chosen." Patients or subjects are asked to place a slash mark somewhere along the line to indicate the intensity of their current pain complaint. For scoring purposes, a millimeter ruler is used to measure along the line and obtain a numerical score for the pain ratings. Most people understand this scale quickly and can easily rate their pain. Children as young as 5 years of age are able to use this scale.[98] The reliability and validity for measuring pain relief has been demonstrated.[48,88]

The use of the scale should be clearly explained to the patient or subject. For treatment outcome measures, relief scales (line anchored with "no pain relief" and "complete pain relief") may be superior to asking absolute pain intensity.[49] Similarly, if a pain intensity visual analog scale is used, patients or subjects may be more accurate if they are allowed to see their previous scores as opposed to being blind.[100]

Figure 5.17. Visual analog scale. May be oriented horizontally or vertically. Note lack of numbers

Caution is advised with photocopying as this process usually lengthens the line and introduces error.

McGill Pain Questionnaire. The McGill Pain questionnaire (Table **5.5)** is a verbal pain scale that uses a vast array of words commonly used to describe a pain experience. Different types of pain, different diseases and disorders have different qualities of pain. It is the quality of pain that gives the most important clues to the possible etiology of a chronic pain complaint. Thus, qualitative sensory descriptors are invaluable in providing key clues to possible diagnoses. Similarly, patients use different words to describe the affective or emotional component of their pain. In order to facilitate the use of these words in a systematic way, Melzack and Torgerson set about categorizing many of these verbal descriptors into classes and subclasses designed to describe these different aspects of the pain experience. In addition to words describing the sensory qualities of pain, affective descriptors including such things as fear and anxiety, and evaluative words describing the overall intensity of the pain experience were included.[74]

The words are listed in **20** different categories (Table **5.5).** They are arranged in order of magnitude from least intense to most intense, and are grouped according to distinctly different qualities of pain. The patients or subjects are asked to circle only one descriptor in each category, *if the* category contains a word that applies to them.

The first **10** categories represent different sensory descriptors that cover various temporal, spatial, pressure, and thermal qualities of pain. The next **5** categories are affective or emotional descriptors; category **16** is evaluative (i.e., how intense is the pain experience); and the last **4** categories are grouped as miscellaneous.

In order to score the questionnaire, the words in each category are given a numerical value. The first word in each category ranks as **1,** the second as **2** etc. The scores for each category are added up separately for the sensory, affective, evaluative and miscellaneous groupings. Then the total number of words chosen is also noted. By using this questionnaire it is possible to get a sense of the quality of a patient's pain complaint **(categories *1—10),* its intensity (category 16),** and also the amount of emotional or psychological overlay accompanying the pain (categories **11-15).** Changes in a patient's pain experience can be monitored by administering the questionnaire at various time points during treatment and follow up.

Treatment Program for Chronic Myofascial Pain

The following represents a comprehensive **6**-week treatment outline. It can be used for any patient with chronic head, neck or shoulder pain in which myofascial TrPs have been diagnosed by a thorough history and TrP oriented physical examination, whether they play a primary or secondary role. This includes patients with any of the diagnoses described in Section B of this chapter, as well as some patients with shoulder girdle pain complaints due to myofascial TrPs, particularly when this program is used in combination with treatment outlined in individual muscle chapters of this volume. The program provides a systematic structure for addressing almost all of the common perpetuating factors to upper quarter myofascial TrP pain and leaves room for consideration of individual muscle needs as well. The program has been shown to be effective in a retrospective study of **25** chronic myofascial head and neck pain patients.[37] With patient compliance, pain intensity and medication consumption decreased dramatically and significantly pre- to posttreatment. Presumably because of the self-efficacy model advocated and the behavioral changes initiated by this program, the patients were able to maintain their lowered pain and medication intake levels up to **12** months

Table 5.5 ***McGill Pain Questionnaire***

Some of the words below describe your present pain. Circle only one word in each of the 20 groups *if* the group contains a word that describes your pain. Leave out any group that is not suitable.

1	2	3	4
Flickering	Jumping	Pricking	Sharp
Quivering	Flashing	Boring	Cutting
Pulsing	Shooting	Drilling	Lacerating
Throbbing		Stabbing	
Beating		Lancinating	
Pounding			
5	**6**	**7**	**8**
Pinching	Tugging	Hot	Tingling
Pressing	Pulling	Burning	Itchy
Gnawing	Wrenching	Scalding	Smarting
Cramping		Searing	Stinging
Crushing			
9	**10**	**11**	**12**
Dull	Tender	Tiring	Sickening
Sore	Taut	Exhausting	Suffocating
Hurting	Rasping		
Aching	Splitting		
Heavy			
13	**14**	**15**	**16**
Fearful	Punishing	Wretched	Annoying
Frightful	Gruelling	Blinding	Troublesome
Terrifying	Cruel		Miserable
	Vicious		Intense
	Killing		Unbearable
17	**18**	**19**	**20**
Spreading	Tight	Cool	Nagging
Radiating	Numb	Cold	Nauseating
Penetrating	Drawing	Freezing	Agonizing
Piercing	Squeezing		Dreadful
	Tearing		Torturing

From Melzack R. The McGill pain questionnaire: major properties and scoring methods. Pain 1975;1:275.

posttreatment at which point study follow up ceased.

Week 0 "Set up the plan."

EDUCATION. Patients must be educated about the causes of their pain, whether there are single or multiple diagnoses, and all causative and perpetuating factors to each must be explained in detail. Ultimately, patients must understand that improvement of their pain depends on their compliance to a home program of self-help exercises and healthy living practices designed to reduce or eliminate the majority of the perpetuating factors to their pain. *All perpetuating factors must be identified and the treatment plan must include a means of controlling as many of these as possible [see* Chapter 4 and individual muscle chapters). Once patients understand that they have an active role in their recovery, a treatment program can begin.

CHANGE TO TIME CONTINGENT MEDICATION. Patients consuming daily analgesics for their pain should be placed on a *time contingent,* and not pain contingent medication schedule. This is a well accepted strategy for reducing the behavioral reinforcement of medication use and provides a basis for *systematic reduction of pain medication* over time.[2,32] In addition, time contingent medication dosing eliminates the problem of needing higher doses and more time to reduce pain levels and prevents the cycle of under-medication and pain alternating with overmedication and drug toxicity.[2] In simple cases, where the patient is taking only one kind of analgesic, they can be started on the same dose of the same medication that they are taking at the start of treatment, only in divided regular doses. If they are taking several different kinds of analgesic drugs, these should be consolidated into one type of analgesic if possible. The medications are consumed on the agreed upon schedule regardless of pain levels. Dosages are reduced at least 20% per week *until the patient is drug free.* Medication management becomes more complicated in patients taking drugs from multiple families (e.g., nonsteroidal, opioid, barbiturate, benzodiazepine, etc.) or exhibiting drug seeking behavior, although the same basic principles can be applied. It is outside the scope of this text to discuss in-depth chronic pain management techniques. Similarly, strategies for medication changes in patients with analgesic rebound headaches are described in detail elsewhere,[35,71] even though many of these patients also have myofascial TrPs.

START HOURLY DIARIES. Depending on the complexity of the pain complaint(s), the use of hourly pain diaries may or may not be necessary. However, use of diaries is extremely useful to determine pain patterns, alleviating and aggravating factors to the pain, medication use, exercise frequency and activities. A chart is devised with at least 4 columns: one for time of day, one for pain levels rated on a scale of 0-10, one for medication use, and one for major activity for the hour. Patients are asked to fill them out hourly, but no less than every 3 hours. If they wait until the end of the day and fill them out retrospectively, the accuracy will diminish dramatically: memory for pain is influenced by the pain the patient is experiencing at the time they are filling out the form.[22]

SCHEDULE ALL APPOINTMENTS. Patients should commit to seeing the clinician on a regular time contingent schedule. These appointments should be scheduled out in advance once per week for 6 consecutive weeks if at all possible. In addition to removing the behavioral reinforcement that accompanies seeing the doctor only when they have pain, this weekly schedule allows for regular follow up to monitor progress and compliance, reinforce posture and exercises, reduce medications and address and control other perpetuating factors.

At this time, other aspects of the treatment plan should be discussed and arranged. These may include physical therapy, visits with a psychologist or to attend a stress management class, or appointments for construction of an intra-oral orthosis.

START VITAMIN OR THYROID SUPPLEMENTS. If initial history and physical, followed by appropriate lab testing, indicates the need for vitamin or thyroid supplementation, these should be initiated now *[see* Chapter 4, Sections C and D).

Week 1 "Address Mechanical Perpetuating Factors."

REVIEW COMPLIANCE. At every appointment it is important to review compliance to instructions from the previous week. Only with compliance to behavioral changes and exercises will patients start to feel better. The clinician must make sure that the patient has contacted other members of the treatment team, if indicated, and has scheduled the appropriate appointments.

CHECK MEDICATIONS. The clinician must review medication intake and patient compliance to the time contingent schedule. Appropriate adjustments can be made at this point, if **necessary.**

REVIEW DIARIES. If the patient **is using** diaries, it will be easy to assess any patterns relating to pain, medication intake, and activities. Based on the information gleaned from the diaries, the clinician can

help the patient problem solve and make suggestions for coping.

ADDRESS SLEEP DISTURBANCE. If the patient relates a history of sleep disturbance, this must be addressed and corrected. Simple sleep hygiene measures such as eliminating caffeine, alcohol, chocolate and exercise in the evening, and ensuring that the room is quiet and without disturbances, may suffice. Tricyclic antidepressant agents in low doses **(10—75** mg) are also extremely useful, both for sleep and for pain.

CORRECT LEG LENGTH DISCREPANCY. For assessment of leg length discrepancy and corrective measures see Chapter **4,** Section B of this volume and Chapter **4** in volume II of the *Trigger Point Manual.*

POSTURE AND BODY MECHANICS. This is the time to educate the patient about the importance of good posture and body mechanics. Giving them an understanding of the complex changes that occur with anterior head positioning will encourage compliance to good posture.

TEACH POSTURE EXERCISES. See under Section C of this chapter. Emphasis on correct breathing and relaxation while performing *all* posture and stretching exercises is *essential* because reduction in stress has been shown to directly reduce TrP EMG activity.[73] Many patients who have been given exercises without emphasis on breathing in the past will very clearly state that they feel a much greater benefit from the exercises with correct breathing than without. Correct breathing and not rushing promotes relaxation, which reduces TrP activity and allows better stretching.

TEACH CORRECT BODY MECHANICS. See under Section C of this chapter and Section C of Chapter **41.**

Week 2 "Increase Home Exercise Program. "

REVIEW COMPLIANCE. Review compliance to instructions from the previous week; check that the patient is performing the posture exercises correctly and has made the suggested improvements in their body mechanics.

CHECK MEDICATIONS. Determine intake and compliance to time contingency. Reduce medication intake by at least **20%.**

REVIEW DIARIES. See Week **1.**

TEACH CERVICAL STRETCHING EXERCISES. General cervical stretching exercises for improved range of motion are very useful for releasing latent or active myofascial TrPs in the cervical and upper shoulder girdle muscles which in turn will reduce secondary TrPs in the masticatory muscles and other sites of pain referral (see Fig. **16.11** and Fig. **17.7).** Use of appropriate breathing *(see* Chapter **20,** Section **14** and Chapter **45)** is extremely important and serves the secondary, but very important function of helping the patient to take a break and relax. Patients who do this correctly will often admit that the relaxation seems to help them more than the actual stretches. This is likely because relaxation reduces TrP activity.[73]

The following basic principles apply to all stretching exercises:

1. Good posture is essential before starting to stretch. Therefore, patient should be instructed to do the POSTURE EXERCISES *first,* then sit down in good posture, before beginning to stretch.
2. The patient should be sitting in a chair that is comfortable. The chair should support the patient's weight so that he or she can relax more muscles and get a better stretch.
3. Patients should inhale slowly and deeply at the beginning of each stretch. As they exhale, they should allow their muscles to relax and lengthen. Encourage them to relax and feel the stretch with each subsequent exhalation.
4. Instruct patients to avoid overstretching. Smooth, easy, gentle motions are best. They must not rush and they must never jerk or pull the muscle.
5. One or two different stretches, *with coordinated breathing and relaxation,* should be performed every **1-2** hours throughout the day. It is unrealistic to expect a patient to perform all of the exercises they might be given every **1-2** hours or **6** times per day; however, they can alternate the various exercises and perform at least one or two at any given time fairly easily. The advantage of this is **2**-fold: improved compliance *and* regular breaks for relaxation, the added benefit of stretches *with appropriate breathing.*

START WALKING OR OTHER AEROBIC EXERCISE PROGRAM. Patients with chronic pain often are deconditioned, as they have avoided exercise and activities that might aggravate their pain, as well as sometimes suffering from varying degrees of depression. Encouraging some form of aerobic exercise, which can start as simply as brisk walking for 10 minutes 3 times per week, will have many positive effects. This is an area where pain diaries come in handy to help the patients determine where they might be able to fit a modest exercise program into their daily routines.

TRIAL OF SPRAY AND STRETCH OR OTHER TRP RELEASE TECHNIQUE. If time permits it may be useful at this point to determine the patient's response to TrP release. If the patient responds positively, appropriate self-help techniques can be taught at subsequent appointments. Similarly, this is often a point at which "re-proving" to the patient that the pain is indeed (largely, if not completely) due to myofascial TrPs, reinforces their compliance to the various aspects of the program.

Week 3 "Teach Self Spray-and-Stretch or other TrP Release Techniques."

REVIEW COMPLIANCE. Review compliance to instructions from previous week and check to see that the patient is performing appropriate posture and cervical stretching exercises correctly. It is particularly important to encourage patients to *slow down* and *breathe* correctly, as they often will be rushing to finish.

CHECK MEDICATIONS. See previous week.

REVIEW DIARIES. See previous week.

REVIEW PROGRESS. Review progress with other health care professionals if this is part of the treatment plan.

SELF STRETCH OF KEY MUSCLES. Teach patients how to self stretch 1 or 2 key muscles that are related to his or her chief complaint (refer to Guide Chapter for individual muscles in Parts 2-5).

These should also be taught *with coordinated breathing* and be performed alternating with the posture and general cervical stretches every 1-2 hours.

INCREASE AEROBIC EXERCISE PROGRAM. If walking, encourage patients to increase the amount of time they are walking by 5 minutes or so, or increase the frequency of exercise from 3 to 4 times per week, or increase their distance by walking faster for the same amount of time. Patients should *not* increase speed and distance simultaneously.

Weeks 4 and 5 "Are We Ready For or Do We Need Trigger Point Injections?"

Continue as above stressing a self help model. Reinforce compliance. Acknowledge good, well behaviors and ignore the bad as much as possible.

TEACH MORE SELF STRETCH EXERCISES AS INDICATED.

EVALUATE THE NEED FOR TRP INJECTIONS OR OTHER TRP RELEASE TECHNIQUES. Clinicians may be surprised to find that if patients are compliant to the self-help and general healthy living strategies taught to them in the first 3 weeks of the program, painful TrP symptoms will have subsided substantially. Many TrPs will now be latent and not causing any painful clinical symptoms. The number of active, bothersome TrPs may only number one or two. These can be injected if the patient desires, or other TrPs release techniques specific for the muscle involved may be more effective now and can be incorporated into the patient's home program.

Week 6 "Reevaluate." After 6 weeks, patients should be reevaluated to determine progress. Reevaluation should include objective as well as subjective measures and should not be based only on the patients' verbal reports that they are feeling better or not. Two reliable subjective pain scales, the visual analog scale and the McGill Pain Questionnaire, were discussed above. Diaries, if used, can also provide information on the changes in the subjective experience of pain.

Objective measures include changes in physical exam such as improvement in posture and anterior head positioning, cervical and mandibular range of motion, TrP tenderness as measured with pressure algometry *[see* Chapter 2, Section B), medication intake, activity levels and attitude.

If compliance is good and progress is poor, it is time to reevaluate the perpetuating factors. If most of the identifiable perpetuating factors have been controlled and the patient still complains of the same or similar intensity of pain as when the treatment program started, the patient must be

reevaluated for other organic disease. Consider the following case.

> *Case report: A* 76-year-old man presented himself with a chief complaint of pain in the pharynx and soft palate. He was referred after two separate medical evaluations by ear, nose and throat specialists, including magnetic resonance images (MRIs) of the head and neck that had been within normal limits. Myofascial TrPs that reproduced his pain were found in the medial pterygoid muscles bilaterally. The patient had an astounding anterior head position of 15 cm.
>
> A 6-week treatment program similar to the one described above was instituted and included posture exercises, correction of body mechanics, self spray and stretch to the jaw elevator muscles and TrP injections of the medial pterygoid muscles. The patient was extremely compliant, improving his anterior head posture to 8 cm. His wife helped him with home spray and stretch. TrP injections gave him good relief, but the pain always returned. After 6 weeks, subjective pain measures were essentially unchanged, despite good control of the obvious perpetuating factors. The patient was referred back to another ear, nose and throat specialist, who on visual inspection of the larynx was now able to identify a patch of dysplastic tissue which on biopsy turned out to be squamous cell carcinoma.

If the patient's progress is good, and further improvement is likely with continued treatment, an additional 2-4 weeks can be added, followed by another reevaluation. A tapering follow up schedule, dropping the patient from weekly to biweekly visits, then to every 3 or 4 weeks, followed by every 3 months for a year, is recommended once improvement is satisfactory.

REFERENCES

1. Ad Hoc Committee on Classification of Headache. *JAMA* 279:717-718, 1962.
2. Max MB, Payne R, Shapiro B, *et al.: Principles of Analgesic Use in the Treatment of Acute Pain and Cancer Pain.* Ed 3. Skokie, IL, American Pain Society, 1992.
3. Atkinson R, Appenzeller O: Headache. *Postgrad Med J* 60:841-846, 1984.
4. Baker BA: The Muscle Trigger: Evidence of Overload Injury. / *Neurol Orthoped Med Surg 7(1):35-44,* 1986.
5. Bell WE: *Clinical Management of Temporomandibular Disorders.* Yearbook Medical Publishers, Inc. 1982.
6. Bland JH: *Disorders of the Cervical Spine: Diagnosis and Medical Management.* W.B. Saunders Company, Philadelphia, 1987.
7. Bogduk N, Lance JW: Pain and pain syndromes including headache, Ch. 8. In: *Current Neurology.* Edited by Appel H. Wiley Medical Publications, New York, 1981.
8. Bovim G: Cervicogenic headache, migraine, and tension-type headache. Pressure-pain thereshold measurements. Pain *51:*169-173, 1992.
9. Braun B, DiGiovann A, Schiffman E, *et al:* A crosssectional study of temporomandibular joint dysfunction in post-cervical trauma patients. / *Craniomandib Disord Oral Facial Pain 6(1):24-31,* 1992.
10. Chapman SL: A review and clinical perspective on the use of EMG and thermal biofeedback for chronic headaches. *Pain* 27:1-43, 1986.
11. Chen SM, Chen JT, Wu YC, *et al.:* Myofascial Trigger Points in Intercostal Muscles Secondary to Herpes Zoster Infection to the Intercostal Nerve. *Arch Phys Med Rehabil 77S61,* 1996.
12. Clark GT: Occlusal therapy: occlusal appliances. In: *The President's Conference on the Examination, Diagnosis, and Management of Temporomandibular Disorders.* Edited by Laskin DM, Greenfield W, Gale E, et *al.* American Dental Association, Chicago, 1983, pp. 137-146.
13. Clark GT, BeemsterboerPL, Solberg WK, etai.:Nocturnal electromyographic evaluation of myofascial pain dysfunction in patients undergoing occlusal splint therapy. *J Am Dent Assoc* 99:607-611, 1979.
14. Clark GT, Delcanho RE, Goulet JP: The utility and validity of current diagnostic procedures for defining temporomandibular disorder patients. *Adv Dent Res 7(2)*$7- 112, 1993.
15. Clark GT, Merrill RL: Diagnosis and nonsurgical treatment of internal derangements. In: *The Temporomandibular Joint: A Biological Basis for Clinical Practice.* Ed. 4. Edited by Sarnat BG, Laskin DM. WB Saunders Co, Philadelphia, 1992.
16. Clark GT, Seligman DA, Solberg WK, *et al.:* Guidelines for the examination and diagnosis of temporomandibular disorders. / *Am Dent Assoc 106:75-78,* 1983.
17. *Dorland's Illustrated Medical Dictionary.* Ed. 25. WB Saunders, Philadelphia, 1974.
18. Dworkin SF, LeResche L: Research diagnostic criteria for temporomandibular disorders: Review, criteria, examinations and specifications. / *Craniomand Disord Facial Oral Pain* 6:301-355, 1992.
19. Dworkin SF, LeResche L, DeRouen T: Reliability of clinical measurement in temporomandibular disorders. *Clin J Pain* 4:89-99, 1988.
20. Dworkin SF, LeResche L, DeRouen T, Von Korff M: Assessing clinical signs of temporomandibular disorders: Reliability of clinical examiners. / *ProsthetDent* 63:574-579, 1991.
21. Edmeads J: Headaches and head pains associated with diseases of the cervical spine. *Med Clin North Am* 62:533-544, 1978.

22. Eich E, Reeves JL, Jaeger B, *et al:* Memory for pain: Relation between past and present pain intensity. Pain 23:375-379, 1985.
23. Ekbom K, Hardebo JE, Waldenlind E: Mechanisms of cluster headache. In *Basic Mechanisms of Headache.* Edited by Olesen J, Edvinsson L. Elsevier Science Publishers B.V., Amsterdam, 1988, pp. 463-476.
24. Fields H: *Pain.* McGraw-Hill Information Services Company, Health Professions Division, New York, 1987, pp. 213-214.
25. *Ibid.* (p. 84).
26. *Ibid.* (p. 91).
27. *Ibid.* (p. 215).
28. Fishbain DA, Goldberg M, Meagher BR, *et al:* Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. *Pain* 26:181-197, 1986.
29. Foerster O: The dematomes in Man. *Brain* 56:1-38, 1932.
30. Fredriksen TA, Hovdal H, Sjaastad O: "Cervicogenic headache": Clinical manifestation. *Cephalalgia* 7:147-160, 1987.
31. Fricton J, Kroening R, Haley D, *et al.:* Myofascial pain and dysfunction of the head and neck: A review of the clinical characteristics of 164 patients. *Oral Surg Oral Med Oral Pathol* 60:615-623, 1985.
32. Fordyce WE, Steger JC: Chronic pain. In: *Behavioral Medicine: Theory and Practice.* Edited by Pomerleau OF, Brady JP. Williams & Wilkins, Baltimore, 1979.
33. Frost FA, Jesson B, Siggaard-Andersen J: A control, double-blind comparison of mepivacaine injection versus saline injection for myofascial pain. *Lancet* 2:8167-8168, 1980.
34. Graff-Radford SB: Personal communication.
35. Graff-Radford SB, Bittar G: The use of methylergonovine (Methergine) in the initial control of drug induced refractory headache. *Headache* 33,7j:390-393, 1993.
36. Graff-Radford SB, Jaeger B, Reeves JL: Myofascial pain may present clinically as occipital neuralgia. *Neurosurgery* 29:610-613, 1986.
37. Graff-Radford SB, Reeves JL, Jaeger B: Management of head and neck pain: The effectiveness of altering perpetuating factors in myofascial pain. *Headache* 27:186-190, 1987.
38. Gronbaek E: Cervical anterolateral microsurgery for headache. In: *Updating in Headache.* Edited by Pfaffenrath V, Lundberg PO, Sjaastad O. Springer Verlag, Berlin, 1985, pp. 17-23.
39. Hameroff SR, Crago BR, Blitt CD, *et al.:* Comparison of bupivacaine, etidocaine, and saline for trigger-point therapy. *Anesth Analg* 60:752-755, 1981.
40. Hammond SR, Danta G: Occipital neuralgia. *Clin Exp Neurol* 25:258-279, 1978.
41. Hatch JP, Moore PJ, Cyr-Provost M, ef *al.:* The use of electromyography and muscle palpation in the diagnosis of tension-type headache with and without pericranial muscle involvement. *Pain 49:175-*178, 1992.
42. Hay KM: Pain thresholds in migraine. *Practitioner* 222:827-833, 1979.
43. Haynes SN, Cuevas J, Gannon LR: The psychophysiological etiology of muscle- contraction headache. *Headache* 22:122-132, 1982.
44. Hendler N, Fink H, Long D: Myofascial Syndrome: Response to trigger point injections. *Psychosomatics* 24:990-999, 1983.
45. Hoheisel U, Mense S, Simons DG, *et al:* Appearance of new receptive fields in rat dorsal horn neurons following noxious stimulation of skeletal muscle: a model for referred muscle pain? *Neuroscience Letters* 253:9-12, 1993.
45a. Hong CZ, Chen YN, Twehous D, *et al.:* Pressure threshold for referred pain by compression on the trigger point and adjacent areas. / *Musculoske Pain 4(3)m-79,* 1996.
46. Hubbard DR, Berkoff GM: Myofascial Trigger Points Show Spontaneous Needle EMG Activity. *Spine* 28:13;1803-1807, 1993.
47. Hunter CR, Mayfield FH: Role of the upper cervical roots in the production of pain in the head. *Am J Surg* 78:743-751, 1949.
48. Huskisson EC: Visual analog scales. In. *Pain Measurement and Assessment.* Edited by Melzack R. Raven Press, New York, 1973, pp. 33-37.
49. Huskisson EC: Measurement of pain. *Lancet 2:117-*131, 1974.
50. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 9:157-164, 1989.
51. Jaeger B: Tension-type headache and myofascial pain. In: *Orofacial Pain and Temporomandibular Disorders.* Edited by Fricton JR, Dubner RB. Raven Press, New York, 1995, pp. 205-213.
52. Jaeger B, Reeves JL: Quantification of changes in myofascial trigger point sensitivity with the pressure algometer. *Pain* 27:203-210, 1986.
53. Jaeger B, Reeves JL, Graff-Radford SB: A psychophysiological investigation of myofascial trigger point sensitivity vs. EMG activity and tension headache. *Cephalalgia 5(Suppl* 3J.68-69, 1985.
54. Jaeger B, Skootsky SA, Cueva LA: Myofascial pain is common in tension-type headaches. *Proceedings of the American Pain Society* :43, 1991.
55. Jensen K, Tuxen C, Olesen J: Pericranial muscle tenderness and pressure pain threshold in the temporal region during common migraine. Pain *Supplement* 4.-S574, 1987.
56. Jensen K, Bulow P, Hansen H: Experimental tooth clenching in common migraine. *Cephalalgia* 5:245-251, 1985.
57. Koch H: The management of chronic pain in office-based ambulatory care. National Ambulatory Medical Care Survey. Advance Data from Vital and Health Statistics. No. 123. DHHS Pub. No.(PHS)84-1250. Public Health Service, Hyattsville, MD, 1986.
58. Kunc A: Significant factors pertaining to the results of trigeminal tractotomy. In: *Trigeminal Neuralgia.* Edited by Hassler R, Walker AE. Stuttgart, Georg Thieme Verlag, 1970, pp. 90-100.
59. Langemark M, Jensen K: Myofascial mechanisms of pain. In: *Basic Mechanisms of Headache.* Edited by Olesen J, Edvinsson L. Elsevier Science Publishers B.V., Amsterdam, 1988, pp. 331-341.
60. Langemark M, Jensen K, Jensen, TS, *et al.:* Pressure pain thresholds and thermal nociceptive thresholds in chronic tension-type headache. *Pain* 38:203-210, 1989.

61. Langemark M, Olesen J: Pericranial tenderness in tension headache. *Cephalalgia* 7:249-255, 1987.
62. Laskin DM: Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 79:147-153, 1969.
63. Lewit K: The needle effect in the relief of myofascial pain. *Pain* 6:83-90, 1979.
64. Lewit K: Muscular pattern in thoraco-lumbar lesions. *Manual Med* 2:105-107, 1986.
65. Lobbezoo-Scholte AM, De Wijer A, Steenks MH, et al: Interexaminer reliability of six orthopedic tests in diagnostic subgroups of craniomandibular disorders. / *Oral Rehabil* 22:273-285, 1994.
66. Lous I, Olesen J: Evaluation of pericranial tenderness and oral function in patients with common migraine, muscle contraction headache and combination headache. *Pain* 22:385-393, 1982.
67. Lundervold A: Electromyographic investigations during sedentary work, especially typing. *Br fPhys Med* 24:32-36, 1951.
68. Lundh H, Westesson PL: Long term follow-up after occlusal treatment to correct abnormal temporomandibular joint disc position. *Oral Surg Oral Med Oral Pathol* 67:2-10, 1989.
69. Lundh H, Westesson PL, Kopp S, *et al.:* Anterior repositioning splint in the treatment of temporomandibular joints with reciprocal clicking: Comparison with a flat occlusal splint and an untreated control group. *Oral Surg Oral Med Oral Pathol* 60:131-136, 1985.
70. Magnusson T, Carlsson GE, Egermark I: Changes in clinical signs of craniomandibular disorders from the age of 15 to 25 years. / *Orofacial Pain* 8:207-215, 1994.
71. Matthew NT, Kurman R, Perez F: Drug induced refractory headache - clinical features and management. *Headache 30(* 10J.-634-638, 1990.
72. McNeill *C: Temporomandibular Disorders: Guidelines for Classification, Assessment, and Management.* Quintessence, Chicago, 1993.
73. McNulty WH, Gewirtz RN, Hubbard DR, *et al.:* Needle electromyographic evaluation of trigger point response to a psychological stressor. *Psychophysiology* 32:313-316, 1994.
74. Melzack R, Torgeson WS: On the language of pain. *Anesthesiology* 34:50, 1971.
75. Mense S: Referral of muscle pain: new aspects. *Am Pain SocJ* 3:1-9, 1994.
76. Mense S, Simons DG, Russell J: *Muscle Pain.* Williams and Wilkins, 1999.
77. Nassif J, Hilsen K: Screening for temporomandibular disorders: History and clinical examination. / *Prosthodont* 2:42-46, 1992.
78. Okeson JP: The effects of hard and soft splints on nocturnal bruxism. *f Am Dent Assoc* 224:788-791, 1987.
79. Okeson JP (ed): *Orofacial Pain. Guidelines for Assessment, Diagnosis, and Management.* American Academy of Orofacial Pain. Quintessence Publishing Co, Chicago, 1996.
80. Olesen J: Some clinical features of the acute migraine attack. An analysis of 750 patients. *Headache* 28:268-271, 1978.
81. Olesen J: Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalalgia 8(Suppl 7),* 1988.
82. Olesen J: Clinical and pathophysiological observations in migraine and tension-type headache explained by integration of vascular, supraspinal and myofascial inputs. *Pain* 46:125-132, 1991.
83. Peterson AL, Talcott GW, Kelleher WJ, *et al:* Site specificity of pain and tension in tension-type headaches. *Headache* 35f2j:89-92, 1995.
84. Pfaffenrath V, Dandekar R, Mayer ET, *et al:* Cervicogenic headache: Results of computer-based measurements of cervical spine mobility in 15 patients. *Cephalalgia* 8:45-48, 1988.
85. Pfaffenrath V, Dandekar R, Pollmann W: Cervicogenic headache-The clinical picture, radiological findings and hypotheses on its pathophysiology. *Headache* 27:495-499, 1987.
86. Piloff H: Is the muscular model of headache still viable? A review of conflicting data. *Headache* 25:186-198, 1984.
87. Poletti CE: C2 and C3 pain dermatomes in man. *Cephalalgia* 22:155-159, 1991.
88. Price DD, McGrath PA, Ralfi R, *et al.:* The validation of visual analogue scale measures for chronic and experimental pain. Pain 27:45-56, 1983.
89. Pullinger AG, Seligman DA: Trauma history in diagnostic groups of temporomandibular disorders. *Oral Surg Oral Med Oral Pathol* 72:529-534, 1991.
90. Pullinger AG, Seligman DA, Gornbein JA: A multiple logistic regression analysis of the risk and relative odds of temporomandibular disorders as a function of common occlusal features. / *Dent Res* 72:968-979, 1993.
91. Rasmussen OC: Description of population and progress of symptoms in a longitudinal study of temporomandibular arthropathy. *Scand J Dent Res* 89:196-203, 1981.
92. Reeves JL, Jaeger B, Graff-Radford SB: Reliability of the pressure algometer as a measure of trigger point sensitivity. *Pain* 24:313-321, 1986.
93. Rivera-Morales WC, Mohl ND: Relationship of occlusal vertical dimension to the health of the masticatory system. / *Prosthet Dent* 65:547-553, 1991.
94. Sandrini G, Antonaci F, Pucci E, *et al:* Comparative study with EMG, pressure algometry and manual palpation in tension-type headache and migraine. *Cephalalgia* 34:451-457, 1994.
95. Saper JR, Silberstein S, Gordon CD, et *al: Handbook of Headache Management.* Williams & Wilkins, Baltimore, 1993.
96. Sarnat BG, Laskin DM (eds): *The Temporomandibular Joint: A Biological Basis for Clinical Practice.* Ed. 4. WB Saunders Co, Philadelphia, 1992.
97. Schoenen J, Gerard P, De Pasqua V, *et al:* EMG activity in pericranial muscles during postural variation and mental activity in healthy volunteers and patients with chronic tension-type headache. *Headache* 32(5j:321-324, 1991.
98. Scott J, Ansell BM, Huskisson EC: The measurement of pain in juvenile chronic polyarthritis. *Ann Rheum Dis* 36:186-187, 1977.
99. Scott J, Huskisson EC: Graphic representation of pain. *Pain* 2:175-184, 1976.
100. Scott J, Huskisson EC: Accuracy of subjective measurements made with or without previous scores: An important source of error in serial measurements of subjective states. *Ann Rheum Dis* 38:558-559, 1979.

101. Shan SC, Yun WH: Influence of an occlusal splint on integrated electromyography of the masseter muscle. / ***Oral Rehabil*** 38:253-256, 1991.
102. Simons DG: Myofascial pain syndromes of head, neck and low back. In: ***Pain Research and Clinical Management,*** Vol. 3. Edited by Dubner R, Gebhart GF, Bond MR. Elsevier Science and Publishers, New York, 1988.
103. Simons DG: Neurophysiological basis of pain caused by trigger points. ***Am Pain Soc J*** 3:17-19, 1994.
104. Simons DG: Myofascial pain syndrome: One term but two concepts; a new understanding. / ***Musculoske Pain*** 3(3J;7-13, 1995.
105. Simons DG: Clinical and etiological update of myofascial pain from trigger points. / ***Musculoske Pain*** 4(3/2j:93-121, 1996.
106. Simons DG, Hong CZ, Simons LS: Prevalence of spontaneous electrical activity at trigger spots and control sites in rabbit muscle. / ***Musculoske Pain*** 3:35-48, 1995.
107. Simons DG, Mense S: Understanding and measurement of muscle tone as related to clinical muscle pain. Pain 75:1-17, 1998.
108. Simons DJ, Wolff HG: Studies on headache: mechanisms of chronic post-traumatic headache. ***Psychosom Med*** 8:227, 1946.
108a. Sjaastad O. So-called "tension headache:"A term in need or revision? ***CurrMed Res Opin*** 6:41-54,1980.
109. Sjaastad O, Saunte C, Hovdal H, ***et al.:*** "Cervicogenic" headache. An hypothesis. ***Cephalalgia*** 3:249-256, 1983.
110. Skootsky SA, Jaeger B, Oye RK: Prevalence of myofascial pain in general internal medicine practice. ***West J Med*** 353:157-160, 1989.
111. Sola AE, Kuitert MC: Myofascial trigger point pain in the neck and shoulder girdle. ***Northwest Med*** 54:980-984, 1955.
112. Solberg WK: Myofascial pain and dysfunction. In: ***Clinical Dentistry.*** Edited by Clark JW. Harper & Row, Publishers, Inc, Hagerstown, MD, 1976.
113. Solberg WK: Temporomandibular disorders. ***Br Dent J***1986.
114. Solberg WK: Personal communication, 1997.
115. Solberg WK, Clark GT: ***Temporomandibular Joint Problems. Biologic Diagnosis and Treatment.*** Quintessence Publishing Co, Chicago, 1980:69-91
116. Solberg WK, Clark GT, Rugh JD: Nocturnal electromyographic evaluation of bruxism patients undergoing short term splint therapy. / ***Oral Rehabil*** 2:215-223, 1975.
117. Speed WG: Posttraumatic headache. In: ***The Practicing Physician's Approach to Headache.*** Ed. 4. Edited by Diamond S, Dalessio DJ. Williams & Wilkins, Baltimore, 1986.
118. Tfelt-Hansen P, Lous I, Olesen J: Prevalence and significance of muscle tenderness during common migraine attacks. ***Headache*** 23:49-54, 1981.
119. Tunis MM, Wolff HG: Studies on headache. Cranial artery vasoconstriction and muscle contraction headache. ***Arch Neurol Psychiatry*** 73:425-434, 1954.
120. Verdonck A, Takada K, Kitai N, ***et al.:*** The prevalence of cardinal TMJ dysfunction symptoms and its relationship to occlusal factors in Japanese female adolescents. / ***Oral Rehabil 21*** .-687-697, 1994.
121. Whittaker DK, Jones JW, Edwards PW, ***et al.:*** Studies on the temporomandibular joints of an eighteenth century London population (Spitalfields). / ***Oral Rehabil*** 3 7:89- 97, 1990.
122. Widmalm SE, Westesson PL, Kim IK, ef ***al.:*** Temporomandibular joint pathology related to sex, age and dentition in autopsy material. ***Oral Surg Oral Med Oral Pathol*** 78:416-425, 1994.
123. Wolff HG: ***Headache and Other Head Pain.*** Oxford University Press, New York, 1963:582-616.

CHAPTER 6
Trapezius Muscle

HIGHLIGHTS: The trapezius is tripartite. The upper, middle and lower trapezius fibers have different fiber directions and often different functions. Therefore, in this chapter they are often considered as separate muscles. **REFERRED PAIN** arises as often from trigger points (TrPs) in the upper trapezius as in any other muscle of the body. The TrPs in the upper trapezius fibers characteristically refer pain and tenderness along the posterolateral aspect of the neck, behind the ear and to the temple. The TrPs in the lower trapezius refer pain and tenderness mainly to the posterior neck and adjacent mastoid area, suprascapular region, and interscapular region. The less common middle trapezius TrPs project pain toward the vertebrae and to the interscapular region. **ANATOMY:** the paired trapezii form a diamond shape that extends in the midline from the occiput above to T_{12} below. It reaches anteriorly to include the lateral one-third of the clavicle, laterally to include the acromion, and posteriorly throughout the length of the spine of the scapula. **INNERVATION** is provided by the spinal part of the accessory nerve (cranial nerve XI), which supplies mainly motor fibers, and by the second to fourth cervical nerves, which supply mainly sensory fibers to the muscle. **FUNCTION** of the upper trapezius is to draw the clavicle (and indirectly the scapula) backwards and raise them by rotating the clavicle at the sternoclavicular joint. The upper trapezius also complements the serratus anterior in rotation of the scapula so that the glenoid fossa faces upward. The lower trapezius stabilizes the scapula for this rotation. The middle trapezius strongly adducts the scapula, stabilizing traction forces. **SYMPTOMS** involve primarily pain referred in characteristic patterns, with relatively little limitation of motion. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the upper trapezius depend, in part, on such skeletal variations as a lower limb-length inequality, a small hemipelvis, or short upper arms. Activation very commonly results from the stress of sustained elevation of the shoulders, as when holding a telephone receiver without elbow support, or working at a high keyboard with inadequate armrests. Acute trauma, as in a "whiplash" from the side, and chronic trauma, as in compression of the muscle by tight bra straps or a misfitting heavy coat, can activate trapezius TrPs. Middle and lower trapezius TrPs are often perpetuated by tight pectoral muscles that must be released. **PATIENT EXAMINATION** reveals that active rotation of the head and neck toward the opposite side is painful at nearly full range, and sidebending to the opposite side is moderately restricted. **DIFFERENTIAL DIAGNOSIS:** pain arising from trapezius TrPs commonly leads to a mistaken diagnosis when a TrP origin of the pain is not investigated. Related articular dysfunctions are found in the cervical or the thoracic spine depending on which part of the trapezius muscle harbors the TrPs. **TRIGGER POINT RELEASE** in this muscle emphasizes release techniques that minimize forceful stretching, especially avoiding it for the middle and lower parts of the trapezius muscle. **TRIGGER POINT INJECTION** of the upper trapezius is done from the front with the patient supine, whereas the other trapezius TrPs are best approached from behind with the patient lying on the opposite side. Trapezius TrPs usually respond well to local injection if tight pectoral muscles have been released. **CORRECTIVE ACTIONS** for body asymmetry and short upper arms include compensating lifts or pads. Misfitting furniture should be modified or replaced. The muscle should be unloaded of unnecessary stress by proper positioning, and the patient should practice an appropriate exercise program at home to control the activity of trapezius TrPs.

1. REFERRED PAIN
(Figs. 6.1-6.4)

The authors have found that the trapezius is probably the muscle most often beset by myofascial trigger points (TrPs), as have other clinicians.[26, 45, 64, 80, 103] It is a frequently overlooked source of temporal[76] and cervicogenic[33] headache. Six trigger regions with distinctive pain patterns are found in the upper, middle, and lower portions of the trapezius; two are located in each portion. A seventh TrP, probably a skin TrP, refers a non-painful autonomic response. The TrPs are numbered in their approximate order of prevalence.

Central TrP_1 in the upper trapezius is apparently the most frequently identified myofascial TrP location in the body, although a latent TrP in the third finger extensor may be more common.[21] The upper trapezius TrP was clearly the most commonly identified in a survey of 200 healthy asymptomatic young adults.[80] This TrP_1 makes a significant contribution to the facial pain of the myofascial pain-dysfunction syndrome as described by Laskin,[51] which was widely recognized by the dental profession,[5,20,78,102] and would now be considered one of the many different kinds of craniomandibular disorders. This (pain-dysfunction) syndrome is a largely outmoded concept that should be replaced with specific diagnoses that identify the pain as having a muscular origin that is referred to the head, or, less commonly, from a painful dysfunction of the temporomandibular joint.[36]

Upper Trapezius Fibers
(Fig. 6.1)

TrP_1. This central TrP can be found in *the midportion of the anterior border of the* upper trapezius and involves the most vertical fibers that attach anteriorly to the clavicle. In our experience, TrPs in this area consistently refer pain unilaterally upward along the posterolateral aspect of the neck to the mastoid process, and are a major source of "tension neckache" (Fig. 6.1), as others also have reported.[57,104] The referred pain, when intense, extends to the side of the head, centering in the temple and back of the orbit;[48,105] in addition, it may include the angle of the jaw,[61,89,91,93,95,107] also described as the region of the masseter.[10] Occasionally, pain extends to the occiput, and rarely, some pain is referred to the lower molar teeth. When referred pain from upper trapezius TrPs overlaps with referred pain from myofascial TrPs in other muscles (namely the sternocleidomastoid, suboccipital, and temporalis muscles), the resulting overlap can produce a typical tension-type headache.[36] (see Fig. 5.2). *Pain referred from TrP1 may occasionally* appear in the pinna, but not deep inside the ear. Stimulation of this TrP by needling and injection has initiated referred vasomotor effects in the homolateral and opposite ear.[88, 93, 94]

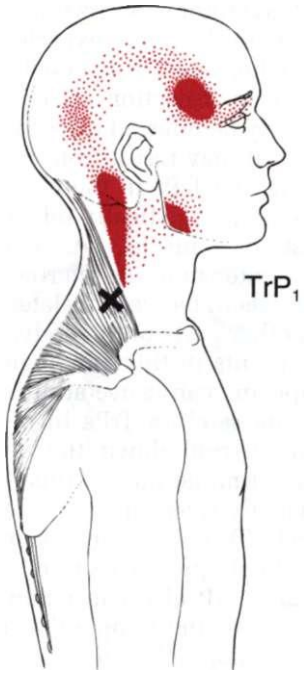


Figure 6.1. Referred pain pattern and location **(X)** of central trigger point 1 in the middle of the most vertical fibers of the upper part of the trapezius muscle. *Solid red* shows the essential referred pain zone while the *stippling* maps the spillover zone.

Other authors describe a similar postauricular pain pattern,[22,46,69] including one in children.[4] A shoulder component of the pain[23,44] is to be expected when the underlying supraspinatus muscle also harbors active TrPs.[52] Occasional reports[22,27] associate TrP activity of the upper trapezius

fibers with symptoms of dizziness or "vertigo," and with dizziness experienced momentarily when the TrP is penetrated by a needle during injection. This postural dizziness may be referred directly from the trapezius or it may result from reflex stimulation of active TrPs in the clavicular division of the synergistically related sternocleidomastoid muscle. A comparable secondary extension of referred pain is sometimes seen between related muscle groups in other parts of the body.

Trigger points in this TrP_1 region of the upper trapezius can cause additional pain by activating satellite TrPs in other muscles. Pain referred down the arm in response to stimulation of this trapezius TrP[83] is usually referred from satellite scalene muscle TrPs. Similar "extension" of the referred pain pattern caused by this upper trapezius TrP also can come from its satellite TrPs in the temporalis, masseter, splenius, semispinalis, levator scapulae, and rhomboid muscles.[30]

When patients had both neck pain and shoulder pain, Sola and Kuitert[79] found that levator scapulae and infraspinatus TrPs were more frequently the cause than were trapezius TrPs.

Experimental injection of the upper trapezius with hypertonic saline in 14 normal subjects induced pain at the base of the neck in all but one subject, projected pain to the same side of the face or head in 12 subjects, and decreased the skin temperature that overlapped the area of referred pain in 6 subjects.[83]

TrP_2 (Fig. 6.2). The location of central TrP_2 is caudal and slightly lateral to TrP_1. The TrP_2 region is located in the middle of the more nearly-horizontal fibers of the upper trapezius. The referred pain pattern of this TrP lies slightly posterior to the essential cervical reference zone of TrP_1, blending with its distribution behind the ear (Fig. 6.2).

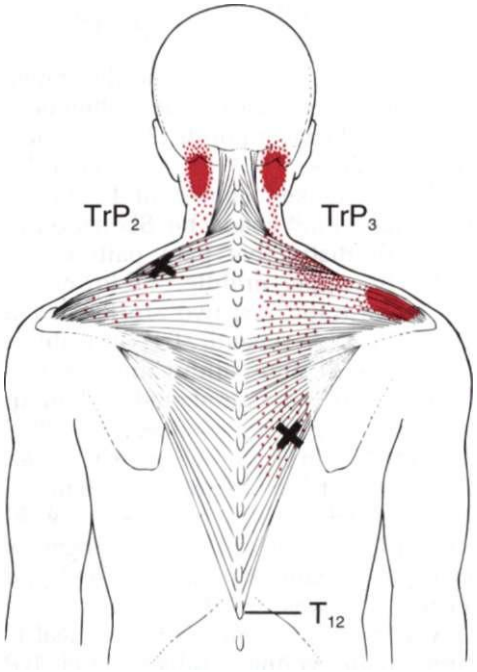


Figure 6.2. Left side of figure shows referred pain pattern and location **(X)** of central trigger point 2 in the middle of the more horizontal fibers of the **upper** part of a left trapezius muscle. Right side of figure shows referred pain pattern and location *(X)* of central trigger point 3 in a right **lower** trapezius; this is likely to be a key TrP that induces satellite TrPs in the region to which it refers pain in the upper part of the trapezius muscle. (Conventions are as in Fig. 6.1).

Lower Trapezius Fibers

TrP_3 (Fig. 6.2). This central TrP of the lower trapezius is very common, very important, and frequently overlooked. It is located in the midfiber region usually near the lower border of the muscle and refers pain severely to the high cervical region of the paraspinal muscles, to the adjacent mastoid area and to the acromion (Fig. 6.2).[91] It also refers an annoying deep ache and diffuse tenderness over the suprascapular region.[105] This tenderness is described by the patient as a "soreness," and the patient tends to rub the tender region. Such referred diffuse tenderness should not be mistaken for the focal tenderness of a TrP. However, TrP_1 and TrP_2 in the upper trapezius do often develop as satellites within this zone of pain and tenderness that is usually referred from lower trapezius TrP_3. Satellite TrPs can be distinguished from simple referred tenderness by the palpable nodule and taut band, local twitch response, sharply localized spot tenderness, induction of referred pain by pressure on the nodule, and by some restriction of neck rotation to the opposite side.

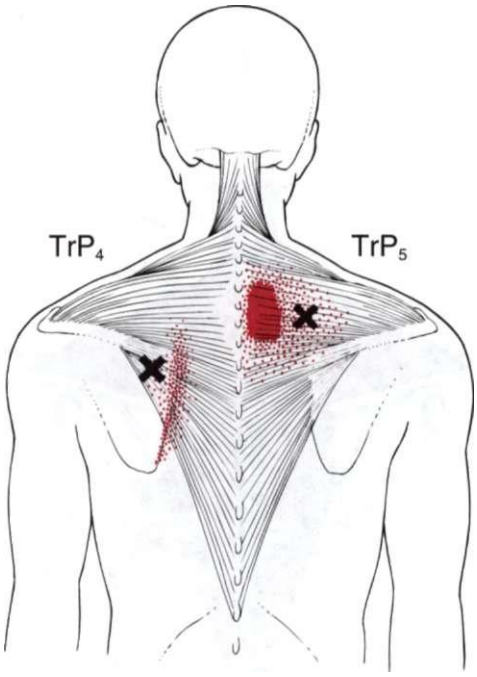


Figure 6.3. Left side of figure shows referred pain pattern and location **(X)** of attachment trigger point 4 in the region of lateral attachment of the left lower trapezius. This tender location is likely a region of enthesopathy at the end of the taut bands associated with a central trigger point 3 (shown for the other side of the body in Fig. 6.2.). Right side of figure shows referred pain pattern and a typical location **(X)** of a central trigger point 5, which is found in the midfiber region of the middle trapezius. (Conventions are as in Fig. 6.1).

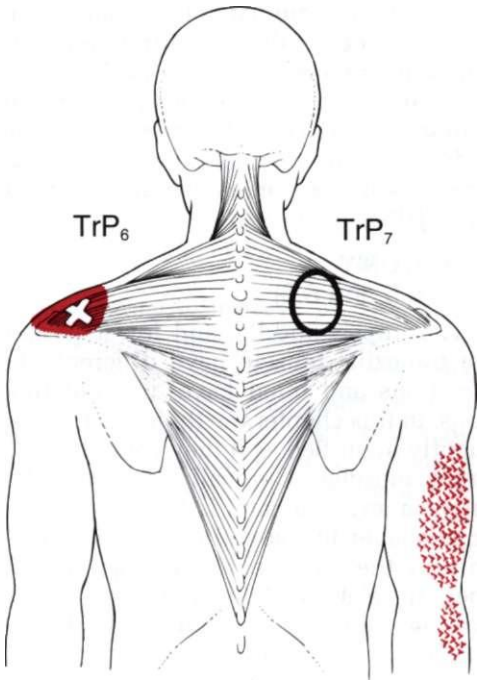


Figure 6.**4.** Referred pain pattern and location **(X)** of attachment trigger point 6 at the lateral attachment region of the left middle trapezius. Tenderness in this region is likely enthesopathy at the end of the taut bands associated with a central trigger point in the middle trapezius (Shown on the right side of Fig. 6.3) (Conventions are as in Fig. 6.1). Trigger point 7 on the right lies within the *encircled area* over the middle trapezius and identifies the location where one sometimes finds a skin trigger point. The zone to which it can refer pilomotor activity, or "gooseflesh," is identified on the right upper limb by red ">" symbols.

TrP_4 (Left side of Fig. 6.3). This attachment TrP refers a steady burning pain downward along, and medial to, the vertebral border of the scapula. This TrP_4 is most likely a region of enthesopathy secondary to a central TrP_3 and should recover following inactivation of the TrP_3 that is causing it.

Middle Trapezius Fibers

TrP_5 (Right side of Fig. 6.3). This group of central TrPs may occur midfiber anywhere in the middle part of the trapezius muscle. They refer superficial burning pain medially, concentrated between the TrP and the spinous processes of the C_7 to T_3 vertebrae.

TrP_6 (Left side of Fig. 6.4). This attachment TrP is found near the acromion in the region of the musculotendinous junctions of middle trapezius fibers and refers aching pain to the top of the shoulder, or acromion. The tenderness in this location is most likely caused by enthesopathy secondary to a central TrP_3 of the middle trapezius near the region of the oval portrayed on the right side of Figure 6.4 or as shown on the right side of Figure 6.3.

TrP_7 (Fig. 6.4). *A superficial* TrP that is most likely a skin TrP rather than a myofascial TrP sometimes occurs within the area encircled in Figure 6.4. It can produce a disagreeable "shivery" sensation with pilomotor erection (gooseflesh) on the lateral

aspect of the homolateral arm and sometimes also of the thigh, as a referred autonomic phenomenon. The referred activity sometimes may be induced merely by stroking the skin over the trigger area. This is insufficient stimulation to induce referred pain from either central or attachment TrPs.

2. ANATOMY
(Figs. 6.5 and 6.6)

The upper, middle, and lower parts of the trapezius muscle have different fiber directions and often have different functions. In this chapter the three parts are frequently identified as if they were three different muscles. Clinically, the boundary between any two parts is frequently indistinguishable by palpation and is defined only by the location of the attachment of fibers in relation to the spinous processes, scapular spine, acromion, and clavicle. When the right and left trapezius muscles are viewed together from the rear, they appear to have a large diamond shape. Together, the fibers of both upper trapezii are shaped like a coat hanger.

Upper Trapezius Fibers (Figs. 6.5 and 6.6)

The upper (superior) fibers arise from the medial third of the superior nuchal line. In the midline, they arise from the ligamentum nuchae (Fig. 6.5). The fibers converge laterally and forward and attach to the posterior border of the lateral third of the clavicle (Fig. 6.6).

A careful anatomical analysis of the direction of fibers in the upper trapezius[40] revealed that, contrary to the impression given by most authors on the subject, none of the superior (upper) trapezius fibers are in a position to exert a direct upward force on the clavicle, and therefore not on the scapula. The few thin fibers that have a vertical orientation from the superior nuchal line swing around the neck and pass almost horizontally, only slightly downward, before attaching to the clavicle.

Johnson *et al.*[40] reported the transversely oriented fascicles in this superior part of the trapezius as arising from the lower half of the ligamentum nuchae and inserting into the lateral third of the clavicle. The

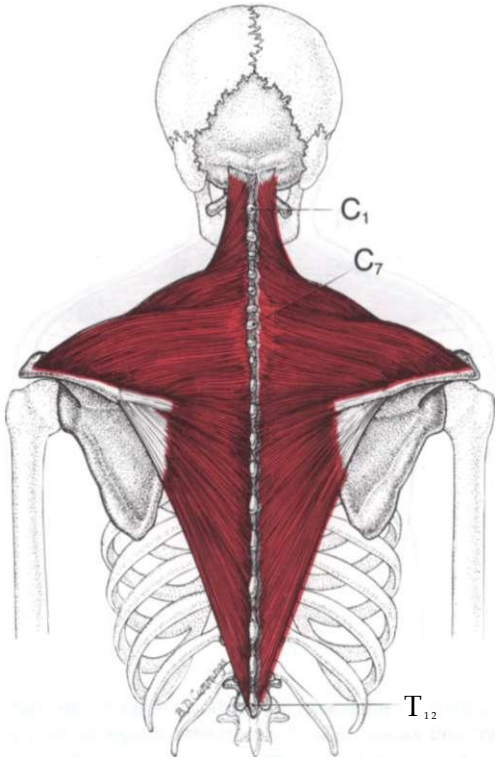


Figure 6.5. Attachments of the right and left trapezius muscles, rear view. The midline trapezius attachments extend from the occiput to the T_{12} spinous process.

larger fascicles of upper trapezius fibers run nearly horizontal (at an elevation of < 20°) and are in a position to draw the lateral end of the clavicle medially and upward by swinging it around its attachment at the sternoclavicular joint. Through this rotation of the clavicle about the sternoclavicular joint, these upper trapezius fibers can raise the clavicle and (indirectly through the acromioclavicular joint) the scapula.

Middle Trapezius Fibers (Fig. 6.5)

These nearly horizontal fibers attach ***medially*** to the spinous processes and interspinous ligaments of the C_6 through T_3 vertebrae, and ***laterally*** to the medial margin of the acromion and superior lip of the spine of the scapula (Fig. 6.5). Johnson *et al.*[40] considered the middle part of the trapezius to consist of those fascicles from

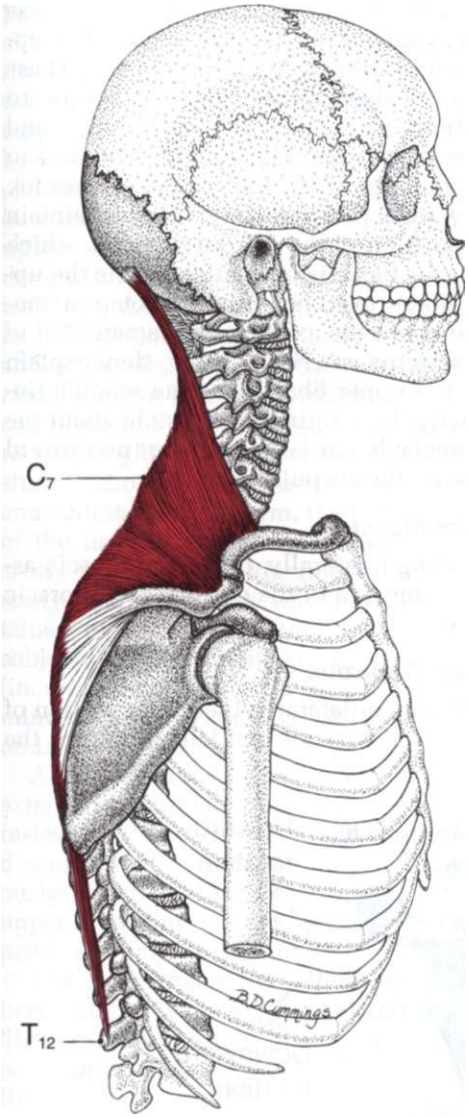


Figure 6.6. Attachments of the right trapezius muscle, side view. The longest, most vertical fibers (ones that cross the greatest number of joints) are the fibers most likely to develop TrPs.

C_7 and T_1, with the C_7 fascicle attaching to the acromion and the T_1 fascicle reaching the spine of the scapula.

Lower Trapezius Fibers (Fig. 6.5)

Fibers from this fan-shaped part of the muscle attach ***medially*** to the spinous processes and interspinous ligaments of approximately the T_4 through T_{12} vertebrae. ***Laterally,*** they converge and attach in the region of the tubercle at the medial end of the spine of the scapula just lateral to the lower attachment of the levator scapulae muscle (Fig. 6.5). Johnson *et al.*[40] considered the lower part of the trapezius to consist of those fascicles from spinous processes starting at T_2.

Supplemental References

Additional illustrations of this muscle show the back view,[1, 11,12, 17,62,71, 82, 85] the side view,[2, 68, 86] and an anomalous subtrapezius muscle.[17] One nearly back view shows the direction of middle and lower trapezius muscle fibers when the arm is abducted to 90°.[47]

3. INNERVATION

Motor innervation of the trapezius is supplied by the spinal portion of the accessory (spinal accessory) nerve (cranial nerve XI). The trapezius portion of the motor nerve arises within the spinal canal from ventral roots, usually of the first five cervical segments; it ascends through the foramen magnum and exits the skull *via* the jugular foramen to supply, and sometimes to penetrate, the sternocleidomastoid muscle. The nerve then joins a plexus deep to the trapezius.

The plexus is joined by fibers (primarily sensory) from spinal nerves C_2, C_3 and C_4; together, they supply both the motor and sensory innervation to the trapezius muscle.[11, 56]

Eleven of 13 patients with radical neck dissections for cancer that included sacrifice of both the accessory nerve and the cervical plexus[81] presented electromyographic (EMG) evidence of variable partial denervation of the trapezius muscle. This suggests that there is a supplemental (apparently thoracic) motor supply to all three parts of the trapezius muscle in many individuals. This means that compression of these fibers also may predispose to the development of trapezius TrPs. A study of 54 radical neck dissections[50] found that approximately two-thirds of the patients retained some degree of motor supply to the trapezius muscle and that an ensuing

shoulder-arm-syndrome varied from severe complaints to no complaint.

4. FUNCTION
(Fig. 6.7)

Summarizing earlier descriptions of trapezius effects on scapular motions (see Fig. 6.7 for definitions): elevation of the scapula activates both upper and middle trapezius fibers; adduction activates all of its fibers but depends primarily on the middle fibers; depression employs the lower fibers;[106] rotation of the glenoid cavity involves chiefly the upper fibers when rotation is upward, and the lower fibers when rotation is downward.[47,73]

Johnson *et al.*[49] in a report of a biomechanical and anatomical analysis of the trapezius muscle, state that the essentially transverse orientation of the upper and middle trapezius fibers allows them to draw the clavicle, acromion, and spine of the scapula backwards and medially (aided by the lower, or thoracic, fibers) and propose that any upward action of the thin superior (nuchal) portion would be dissipated in the cervical fascia before these fibers reached the clavicle (which they approach in almost a horizontal plane). These authors[49] suggest that, in regard to upward rotation of the scapula, the upper and lower fibers participate in different ways in conjunction with the serratus anterior. They state that the lower fibers maintain the position of the deltoid tubercle, which becomes the axis of rotation, while the upper fibers exert an upward rotation moment about the axis to complement that of the serratus anterior. Further, they explain that the upper fibers raise the scapula (indirectly) by rotating the clavicle about the sternoclavicular joint and exert no upward force on the scapula.

Entire Muscle

Acting bilaterally, the entire muscle assists extension of the cervical and thoracic spine.[47]

Upper Trapezius

Acting unilaterally, the upper portion of the muscle extends and laterally flexes the

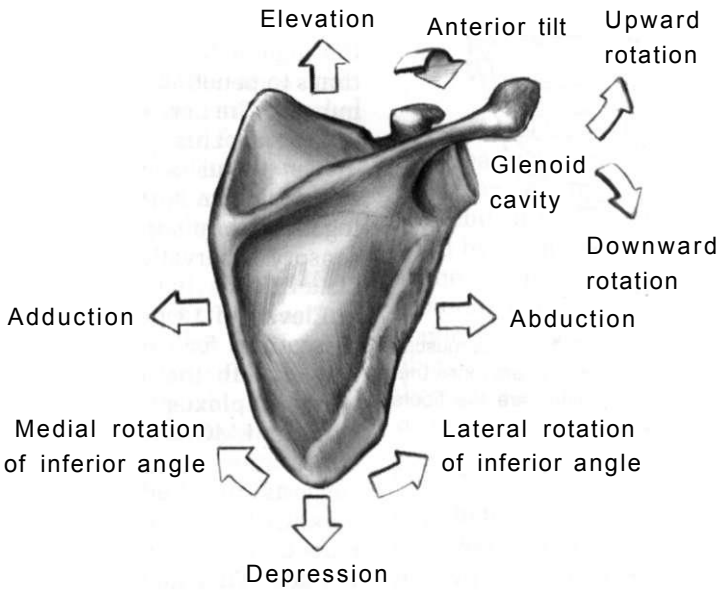


Figure 6.7. Illustration of terms used to describe movements of the right scapula, as seen from behind. Anterior tilt applies to the upper border of the scapula. Upward and downward rotation refers to direction of movement of the glenoid cavity. Medial and lateral rotation refers to direction of movement of the inferior angle. Adduction is scapular movement in a medial direction (toward the vertebral column), and abduction is movement of the scapula as a whole in a lateral direction (away from the vertebral column). (Redrawn from Kendall FP, McCreary EK, Provance PG. *Muscles, Testing and Function.* Ed. 4. Baltimore: Williams & Wilkins, 1993:282.)

head and neck toward the same side, and aids in extreme rotation of the head so that the face turns to the opposite side.[3,16,47] It can draw the clavicle (and *indirectly* the scapula also) backwards and can raise them by rotating the clavicle at the sternoclavicular joint.[40] It usually helps (but can be trained not) to carry the weight of the upper limb (indirectly through the shoulder girdle) during standing, or to support a weight in the hand with the arm hanging.[3] In conjunction with the levator scapulae and upper digitations of the serratus anterior, the upper trapezius provides the upper component of the force couple necessary to rotate the glenoid fossa upward.[3,33,73] One study[34] showed that during both flexion and abduction of the arm, the EMG activity of the upper trapezius increased progressively and became vigorous. In another study, when the arm was actively maintained in 90° of abduction, all 7 healthy subjects showed significant EMG evidence (increased amplitude) of fatigue within 1 minute and on average, in less than 30 seconds.[28]

Acting bilaterally, the upper fibers may extend the head and neck, but only against resistance.[73,106] A respiratory function was demonstrated by stimulation,[16] but is seriously questioned.[31,56] Recruitment of the upper trapezius for respiration likely depends strongly on circumstances.

The mechanism by which the nearly horizontally oriented upper trapezius fibers can be effective in assisting the serratus anterior muscle is well explained.[40] By exerting a medially directed force on the clavicle, which must rotate around the sternoclavicular joint, it effectively draws the lateral end of the clavicle (to which it attaches) medially and upward. The resulting elevated position of the acromion transfers much of the weight being carried by the humerus to the sternoclavicular joint as a compressive force relieving the cervical spine of compression. You can demonstrate this on yourself by palpating the thick bundle of muscle fibers forming the lower portion of the upper trapezius as they attach to the lateral end of the clavicle while elevating your shoulder against resistance. The orientation of the fibers is nearly horizontal rather than vertical.

To the extent that the upper trapezius raises the lateral end of the clavicle, it (indirectly) also would raise the scapula.

Middle Trapezius

Because of its intermediate position, the middle trapezius has two distinctly different functions. The more superior middle trapezius fibers that attach to the acromion assist in adducting the scapula, and after upward rotation has been initiated it can serve as part of the force couple that upwardly rotates the scapula,[40] assisting the upper trapezius and serratus anterior. The more inferior fibers that attach to the spine of the scapula are more horizontal and effectively adduct the scapula (i.e., move it toward the midline), as reported by others.[16,47,73]

Lower Trapezius

The lower fibers adduct the scapula and are said by most authors to depress the scapula and to rotate the glenoid fossa upward.[3,47,73] However, Johnson *et al.*[40] in their biomechanical analysis of the relative locations of the scapular attachment of the lower trapezius fibers and the center of rotation of the scapula make it clear that these lower fibers are in no position to contribute any net torque about the axis to help rotate the glenoid fossa upward. This would be primarily a serratus anterior function complemented by the upper trapezius. Initially, the center of rotation of the scapula is essentially where the lower trapezius fibers attach at the deltoid tubercle of the spine of the scapula.[40] As the scapula rotates, the center of rotation migrates toward the acromioclavicular joint so that the center of rotation, the attachment point of the fibers, and the fiber direction form essentially a straight line producing no effective rotational moment. The authors concluded that the middle and lower trapezius fibers serve to *stabilize* the position of the scapula while other muscles rotate it. During the movement, the lower trapezius fibers would show EMG activity, but not for the reason previously assumed.

Keyboard Operation. Lundervold[58-60] studied conditions that increased EMG activity (and therefore the likelihood of activating TrPs) in the upper trapezius

muscle by monitoring the muscle with surface electrodes while the subjects were using a typewriter. Muscular activity increased markedly when the subject: sat in a tense upright posture instead of a relaxed, well-balanced position;[59, 60] sat without a firm back support;[60] typed with the keyboard elevated;[58,59] was tired;[58] or was untrained.[60] An increased rate of striking one key increased sharply the amplitude and duration of the bursts of trapezius activity and decreased the silent period between bursts.[60]

Sports. **Comparison of EMG activity in the upper trapezius muscles of normal competitive swimmers and those with a painful shoulder[77] showed basically the same pattern of peak activity between the pull-through and the recovery phases in both groups. However, the EMG activity in swimmers with a painful shoulder was more uniform, persisting at a moderate level when the normal activity practically disappeared, and the EMG of the painful shoulders did not reach as high a peak. There was no indication the painful shoulders were examined for TrPs, which can cause marked inhibition and distortion of normal motor coordination.**

EMG monitoring of the upper, middle, and lower trapezius fibers with surface electrodes was performed during 13 sports activities, including right-handed overhand throws, underhand throws, tennis, golf, and 1-foot jumps in basketball.[7] All records showed the motor unit activity on the left side to be equal to, or greater than, that on the right side, predominantly in the middle and lower trapezius fibers.[7] The recording of the basketball throw showed this left-sided effect most strongly.

Driving. **In a study of subjects driving an automobile simulator, the upper trapezius was found to contract only weakly, but more actively than the middle and lower portions of the muscle.[43]**

5. FUNCTIONAL UNIT

The paired trapezius muscles are synergistic with each other for extension of the head, neck, or thoracic spine, and during symmetrical upper limb activities.

Unilaterally, the different parts of the muscle (with different fiber direction) are synergistic with each other for scapular adduction and rotation.

Upper Trapezius

This part of the muscle acts synergistically with the sternocleidomastoid for some head and neck motions. It is an antagonist to the levator scapulae during scapular rotation. During abduction of the arm, the rotation of the scapula (in part by the trapezius) is synergistic with the glenohumeral movement produced by the supraspinatus and deltoid muscles. This coordinated movement during elevation of the arm is identified as the "scapulohumeral rhythm."[39]

Middle Trapezius

These nearly horizontal fibers act synergistically with the rhomboid muscles to adduct the scapula. By fixing (stabilizing) the scapula, the middle fibers also are synergistic with the deltoid, supraspinatus, and long head of the biceps brachii in elevation of the arm at the shoulder joint. These trapezius fibers are antagonists to all but the most caudally directed fibers of the pectoralis major muscle.

Lower Trapezius

In stabilizing the axis of rotation of the scapula, these fibers are synergistic with the lower part of the serratus anterior (and with the *upper* fibers of the trapezius) in upward rotation of the glenoid fossa of the scapula.

6. SYMPTOMS

Upper Trapezius

TrP_1. When TrP_1 is active, the patient usually has severe posterolateral neck pain that often is constant and usually is associated with temporal headache on the same side (Fig. 6.1). Occasionally, pain is projected to the angle of the jaw. The patient is likely to be misdiagnosed as having cervical radiculopathy, or atypical facial neuralgia.

TrP_2. TrP_2 causes similar neck pain, but usually without headache (Fig. 6.2). Pain on motion, due to upper trapezius TrPs alone, occurs only when the head and neck are almost fully rotated actively to the opposite side,[93] which contracts the muscle in a shortened position. With very active upper trapezius TrPs, and with *additional* involvement of the levator scapulae or

splenius cervicis muscles, the patient may develop an acute "stiff neck."[67, 87, 92] This painfully limits rotation of the head toward the same side, which elongates the upper trapezius.

Activity of TrP_1 and TrP_2 may cause intolerance to the weight of heavy clothing, such as a misfitting heavy overcoat, that rests on the trapezius (coat-hanger muscle) at the angle and back of the neck, instead of on the acromion processes.

Middle Trapezius

TrP_5. TrP_5 causes the patient to complain of burning interscapular pain (Fig. 6.3).

TrP_6. Trigger area 6, which probably represents an attachment TrP, produces more localized pain and tenderness over the acromion (Fig. 6.4), making the shoulder intolerant of pressure from a well fitted heavy coat, or from a ponderous purse[18] carried on a shoulder strap.

TrP_7. Trigger area 7 may be associated with spontaneous episodes of a "queer shivery feeling" with pilomotor erection (gooseflesh) on the anterolateral surfaces of the homolateral arm, and sometimes of the thigh (Fig. 6.4). The feeling produced by this referred autonomic response is described as "like shivers running up and down the spine" when chalk or a fingernail scrapes across a blackboard.

Lower Trapezius

TrP_3 and TrP_4. Central TrP_3 and attachment TrP_4 cause suprascapular, interscapular, acromial, and/or neck pain with little, if any, restriction of neck motion (Figs. 6.2 and 6.3). TrP_3 is often the "joker" responsible for persistent upper back and neck pain after the active TrPs in the upper trapezius and other shoulder and neck muscles have been eliminated. This TrP_3 is often a key TrP that induces satellite TrPs in upper back and neck muscles.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

In any part of the trapezius, TrPs may be activated by sudden trauma, such as falling off a horse, falling down steps, or suffering a cervical flexion-extension injury ("whiplash") in an auto accident[57] and may be perpetuated by the mechanical and systemic factors considered in Chapter 4. Examination of 37 patients with "whiplash" for active TrPs[31] revealed that 35 (95%) of them had active TrPs in the upper trapezius, but only two (5%) had active TrPs in the lower trapezius.

Upper Trapezius

Its function of neck stabilization is commonly overloaded by tilting of the shoulder-girdle axis due to a lower limb-length inequality or small hemipelvis (body asymmetry). The limb asymmetry tilts the pelvis laterally, which bows the spine into a functional scoliotic curve and, in turn, tilts the shoulders, causing one to sag. The upper trapezius must work constantly to keep the head and neck vertical and the eyes level. A walking cane 12-15 cm (5 or 6 in) too long tilts the axis of the shoulder girdle and causes a similar trapezius problem by forcing the shoulder up on the side of the cane. A cane is properly fitted if, with the shoulders level, the elbow bends 30-40% with the cane held beside the foot.[29]

The normally minimal antigravity function of the upper trapezius is overstressed by any position or activity in which the trapezius helps to carry the weight of the arm for a prolonged period: telephoning or sitting without armrest support, particularly when the upper arms are congenitally short; holding the arms elevated to reach a high keyboard or a high drawing board;[59] or working with sewing material on the lap with the elbows unsupported.

The upper trapezius may be strained by obvious acute gross trauma but, more often, it is strained by chronic injury due to overload or microtrauma that may not be so obvious. Such injury can be caused by clothing and accessories, by pressure from tight narrow bra straps supporting large breasts, by the shoulder strap of a ponderous purse[18] or of a heavy backpack, or by a heavy coat. It also may be caused by a sustained load in habitual elevation of the shoulders, as an expression of anxiety or other emotional distress, during long telephone calls, playing the violin, or by rotation of the head *far* to one side in a fixed position (holding the head turned to converse with a person seated at the side, or sleeping prone with the head strongly rotated).

Occupational overload is receiving increasingly serious attention. However, the

important contribution of TrPs as a major cause of the pain is not yet generally recognized. In a prospective study of employees,[98] the authors recorded the EMG activity of the upper (acromial) fibers of the middle trapezius doing repetitive tasks. Elevated static and mean EMG activity levels and fewer EMG gaps of at least 0.6 sec duration correlated significantly with future complaints of neck and shoulder pain. These subjects were not examined for TrPs, but chronic overload such as this without adequate periods of relief activates TrPs. A similar prospective 1-year study of 30 female packers doing repetitive light work[97] revealed that within one year, 17 of the 30 developed sufficient work-related trapezius myalgia to be classified as patients, with a median time of onset of 26 weeks. The authors did not address the cause of the pain, which was likely TrPs in many subjects. Another similar EMG study compared office workers and production workers[39] and concluded that EMG findings alone did not discriminate those workers likely to develop muscle pain symptoms. It was apparent that an important factor was not considered, and that the presence of latent TrPs in the upper trapezius at the onset of employment was not included in the study.

Biopsies from the upper part of trapezius muscles of 10 patients with work-related chronic trapezius myalgia[55] showed larger type I fibers, and lower levels of adenosine triphosphate and phosphocreatine in type I and II fibers than control subjects. Although these patients apparently were not examined for myogelosis or TrPs, the enlargement of type I fibers is characteristic of myogelosis and TrPs[74] and the histochemical changes are compatible with the energy crisis that has been shown to be associated with areas of myogelosis (TrPs).[8]

Other factors may activate upper trapezius TrPs. Armrests that are too *high* push the scapulae up and shorten the upper trapezius for long periods. The muscle's accessory function of head rotation can be overstressed by the quick repetitive movement of flicking long hair out of the eyes.

Upper trapezius TrPs may be activated by, and remain as sequelae to, cervical radiculopathy.[57]

Middle Trapezius

This part of the muscle also becomes overloaded when the arm is held up and forward for a long time. Sustaining this position also overloads the pectoralis major fibers, which are prone to develop latent (painless) TrP activity that increases their tension, pulling the arm and scapula forward. Then, the antagonistic middle trapezius fibers become overstretched and weakened by this unrelenting abduction of the scapula and protraction of the shoulder. This can result in a round-shouldered posture. The middle trapezius (and rhomboid) muscle fibers may be overloaded and may then develop active TrPs that cause pain.

These middle trapezius fibers are subject to strain when the driver of a car holds the hands on top of the steering wheel, again, in a round-shouldered position.

Lower Trapezius

The lower fibers are strained during prolonged bending and reaching forward while sitting (to reach the desk when the knees lack space under its surface) and by supporting the chin on the hand, while resting the elbow on the front of the chest because armrests are missing.[90]

8. PATIENT EXAMINATION

After establishing the event(s) associated with the onset of pain complaint, the clinician should make a detailed drawing representing the pain described by the patient. If the drawing is in the style of the pain patterns published in this volume, it can be very useful for monitoring patient progress. The appropriate body forms are found in Figures 3.2-3.4.

Lower trapezius weakness, which can occur from inhibition by its own TrPs or from other sources, may allow the scapula to ride up and tilt downward anteriorly (forward and downward tilt of coracoid process), and may lead to adaptive shortening of the pectoralis minor muscle. The resultant position of the scapula and "round-shouldered" posture can be seen by the examiner.

The TrPs in the upper fibers of the trapezius can restrict arm abduction at its full range by the effect on upward rotation of the scapula. The upper trapezius is characterized as tending to be hyperactive and

tense while the lower trapezius is prone to inhibition and weakness.[25,38,54] These patterns may relate to reflex responses to TrPs in functionally related muscles, a subject that deserves experimental investigation.

The examiner should assess joint play in the sternoclavicular, acromioclavicular, and glenohumeral joints. These necessary accessory joint movements were described by Mennell.[65]

Upper Trapezius

The patient with an active TrP_1 or TrP_2 in the upper trapezius, especially one who has short upper arms or who sits without an armrest, tends to fold the arms across the chest and to cradle the chin in one hand. This patient may be seen to rub the trapezius muscle and to keep moving the head as if trying to stretch the muscle. He or she is likely to present an apparently elevated shoulder on the side of the thickened, tense upper trapezius with a slight tilt of the neck toward the more affected side.

When the trapezius alone is involved, there is minimal limitation of head and neck rotation. The most restricted movement is lateral flexion of the head and neck (sidebending) away from the involved upper trapezius. Passive sidebending may be reduced to 45°, or less. When sidebending is tested and the head is then rotated toward the side of the involved muscle, the patient may feel increased tension and/or referred pain along the side of the neck. Neck flexion is only slightly restricted, as is arm abduction due to the painfully restricted upward rotation of the scapula. Active rotation of the head to the *opposite* side is usually painful at the extreme range of motion, since the muscle contracts strongly in this most shortened position. Active rotation to the *same* side is usually pain free, unless either the levator scapulae on the same side, or the opposite upper trapezius, also harbor TrPs.

If active TrPs also are present in the levator scapulae muscle, head and neck rotation to the painful side is markedly restricted, so that the patient tends to hold the neck stiff and turns the body.

Middle Trapezius

The patient with pain arising from the middle trapezius is likely to have a round-shouldered posture secondary to shortening of the antagonistic pectoralis major and/or minor muscles due to their active or latent TrPs. The strong pectoral muscles exhaust the weaker middle trapezius fibers in their futile effort to adduct the scapulae and maintain a normal posture.

When the skin overlying an active TrP_7 (Fig. 6.4) is lightly stroked, a visible wave of pilomotor activity (gooseflesh, an autonomic response) may be seen to spread homolaterally down the arm and sometimes over the outer aspect of the thigh. The patient is aware of a queer, creeping sensation in the skin.

Lower Trapezius

Active TrPs in these lower fibers may affect upward rotation of the scapula because of impairment of their stabilization function.

If the lower trapezius is inhibited and weak from the activity of TrPs, the scapula may be elevated and the upper part tilted forward (coracoid process tilted forward and downward), and the patient will exhibit a round-shouldered posture.

9. TRIGGER POINT EXAMINATION (Fig. 6.8)

To determine the most useful diagnostic criteria for TrPs, Gerwin *et al.*[21] tested the reliability with which four experienced physicians following a *3-hour training session* could identify five characteristics of TrPs in five pairs of muscles (one was the upper trapezius) in 10 subjects. Four criteria are highly reliable in this muscle: the detection of spot tenderness, palpation of a taut band, the presence of referred pain, and reproduction of the subject's symptomatic pain (agreement 90% to perfect and kappa 0.61 to 0.84). Identification of a local twitch response (LTR) by manual palpation was unreliable in this muscle. However, when present, an LTR is a strong confirmatory finding, and is especially valuable when needling TrPs therapeutically. With a few hours of adequate training, experienced clinicians who use the four reliable criteria can identify TrPs in this muscle with a high degree of confidence.

Upper Fibers

TrP_1. With the patient supine, or possibly seated, the muscle is placed on moderate slack by bringing the ear slightly toward the shoulder on the same side (Fig.

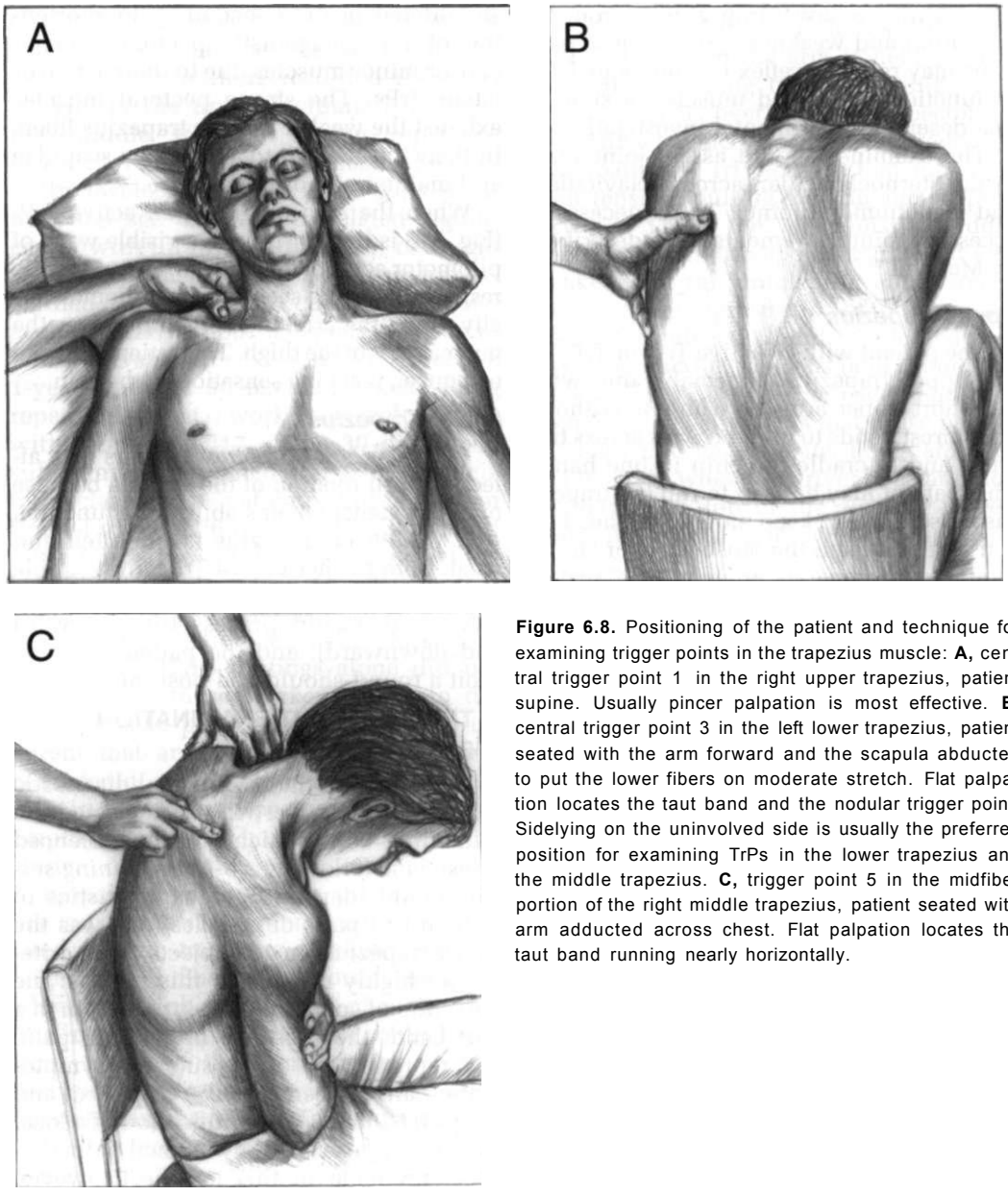


Figure 6.8. Positioning of the patient and technique for examining trigger points in the trapezius muscle: **A,** central trigger point 1 in the right upper trapezius, patient supine. Usually pincer palpation is most effective. **B,** central trigger point 3 in the left lower trapezius, patient seated with the arm forward and the scapula abducted to put the lower fibers on moderate stretch. Flat palpation locates the taut band and the nodular trigger point. Sidelying on the uninvolved side is usually the preferred position for examining TrPs in the lower trapezius and the middle trapezius. **C,** trigger point 5 in the midfiber portion of the right middle trapezius, patient seated with arm adducted across chest. Flat palpation locates the taut band running nearly horizontally.

6.8A). In a pincer grasp, the entire mass of the free margin of the upper trapezius is lifted off the underlying supraspinatus muscle and apex of the lung. Then the muscle is firmly rolled between the fingers and thumb to palpate for a nodule and firm bands to locate the spot tenderness of TrP_1. This manual technique has been illustrated previously.[66,91,93] Sustained compression of the TrP often evokes pain referred to the neck, occiput, and temple, as also observed by Patton and Williamson.[70]

On the other hand, the pain caused by loading the muscle (abducting the arm above 90%) is prevented by firm pressure on the muscle with the palm of the hand during abduction.[44] Some of this pain also may be due to TrPs in the underlying

supraspinatus muscle, which more directly contributes the power to abduct the arm.

TrP_2. This TrP may be identified by a similar pincer technique in deeper fibers inferior to TrP_1 if the patient has mobile connective tissue. Patients with firmer tissue require flat palpation. TrP_2 is located at the level of the C_5 to C_6 spinous processes approximately halfway between the acromion and the spinous processes. Webber" and Long[57] also identified the location of this TrP.

Middle and Lower Fibers

For examination of the remaining trapezius TrPs, the patient sits with the arms folded across the front of the body to abduct the scapulae, as in Figure 6.8B and C, and "humps the back" to flex the dorsal spine. Cross-fiber palpation identifies taut bands in the muscle by rolling them against the underlying ribs. The firm bands usually exhibit visible local twitch responses to snapping palpation of the TrP.

TrP_3. This *lower* trapezius central TrP (CTrP) usually lies in the lateral margin (the most inferior fibers) close to where the fibers cross the medial border of the scapula, or sometimes at or below the level of the inferior angle of the scapula (right side of Fig. 6.2). This TrP_3 sometimes feels like a button or nodule within the taut band and is easily missed if slack in the muscle has not been eliminated by the patient leaning forward, as shown in Figure 6.8B.

TrP_4. This attachment TrP (ATrP) is found in the region of the lateral musculotendinous junction of the *lower* trapezius near where it attaches to the deltoid tubercle of the spine of the scapula (left side, Fig. 6.3). Tenderness in this region is likely to be enthesopathy secondary to taut bands associated with central TrP_3. Except for its location at the end of the muscle rather than in the muscle belly, this ATrP can appear confusingly similar to a CTrP. However, it has a different cause for the local sensitization of nociceptors. There may be a palpable tender area of induration at the end of the palpable taut band that is responsible for this local tissue reaction to stress. The tenderness may be circumscribed to a limited region, compression may elicit referred pain that the patient recognizes, and needling the region of tenderness may elicit an LTR.

TrP_5. This central TrP of the *middle* trapezius is located by flat palpation midfiber (right side of Fig. 6.3) in the nearly horizontal fibers about 1 cm (1/2 in) medial to the scapular attachment of the levator scapulae (Fig. 6.8C).

TrP_6. Finding this less common attachment TrP requires flat palpation in the lateral attachment region of the middle trapezius (left side of Fig. 6.4). Tenderness of this ATrP is likely caused by enthesopathy at the end of a taut band associated with the midfiber $CTrP_5$ and is identified as described above for $ATrP_4$.

TrP_7. This infrequent trigger point lies superficially over the midmuscle region of the middle trapezius (right side of Fig. 6.4). This trigger area may be stimulated by pinching it through the skin, or it may be stimulated directly by penetrating it with a needle. It very likely is a skin TrP rather than a myofascial TrP.

Other Trigger Points

When patients have pain and deep tenderness referred to the suprascapular region, but do not have active trapezius TrPs, the responsible TrPs are likely to be found in the levator scapulae or scalene muscles.

10. ENTRAPMENT

In one autopsy study of 40 greater occipital nerves,[6] the nerve emerged from just below the occiput through the trapezius muscle in 45% of cases and through the underlying semispinalis capitis muscle in 90% of cases (*see* Fig. 16.5). The nerve can be entrapped as it emerges through the semispinalis capitis when that muscle becomes taut due to more caudal TrPs at the mid-cervical level (see Section 10 of Chapter 16). The trapezius itself has not been found to entrap the nerve, but may contribute a shearing stress.

When the (spinal) accessory nerve emerges through the sternocleidomastoid muscle, the trapezius muscle may be weakened by entrapment of its motor nerve fibers between taut bands of sternocleidomastoid fibers.[68]

11. DIFFERENTIAL DIAGNOSIS

In addition to or instead of trapezius TrPs, the patient with head and neck pain may have: a temporomandibular disorder with or without associated TrPs of masticatory muscles, related TrPs in other muscles, articular dysfunctions of the cervical spine, and/or any of the diagnoses listed below. Patients with chronic axial pain that includes multiple regional involvement should be examined for tender points diagnostic of fibromyalgia.

Other Diagnoses

Referred pain to the head from TrPs in several masticatory and neck muscles (including the upper trapezius) at the same time are easily (and frequently) mistakenly diagnosed as **tension headache.**[36,37] Pain originating from TrPs in the upper trapezius and the splenius capitis muscles can confusingly simulate **occipital neuralgia**[34] and **cervicogenic headache.**[35] Myofascial pain from any part of the trapezius muscle (often in combination with other muscles) can lead to the diagnosis of **chronic intractable benign pain of the neck and/or back,**[75] which according to the authors, is nearly always of myofascial origin and therefore does not stand up as a valid diagnosis. Shoulder pain from the lower trapezius TrP may be mistaken for **bursitis.**[100]

Related Trigger Points

In the presence of **upper trapezius** TrPs, associated TrPs are likely to develop in the functionally related levator scapulae and contralateral trapezius muscles, and also in the ipsilateral supraspinatus and rhomboid muscles. Satellite TrPs may appear in the temporalis and occipitalis muscles, which lie within the zones of pain referred from TrPs in the upper trapezius. Hong[30] identified a number of satellite TrPs that were inactivated by simply inactivating key TrPs in the upper trapezius. The satellite TrPs appeared in the temporalis, masseter, splenius, semispinalis, levator scapulae and rhomboid minor muscles.

When the **middle trapezius** is involved, the pectoral muscles and the paraspinal group in the region of the T_1-T_6 vertebrae commonly have associated TrPs.

On the other hand, satellite TrPs may arise in the upper trapezius as the result of key TrPs in another muscle. The TrPs in the **lower trapezius** are prone to act as key TrPs and induce satellite TrPs in the upper trapezius, and sometimes in the levator scapulae and the posterior cervical muscles. For this reason, one should routinely check the lower trapezius for TrPs, especially when the upper trapezius TrPs respond poorly to treatment. Keep in mind, however, that a TrP in the lower trapezius may itself be a satellite of a key TrP in the latissimus dorsi muscle.

Related Articular Dysfunctions

Upper Trapezius. The symptoms caused by upper trapezius TrPs may be closely associated with and confusingly similar to somatic or articular dysfunctions below the C_2, C_3, and C_4 vertebrae. Commonly, one or more of these restricting articular dysfunctions and upper trapezius TrPs coexist, and both must be treated.

Treatment by the muscle energy technique[25] (contract-relax) not only corrects the restricting dysfunction but also has an element of muscle re-education and avoids high velocity maneuvers. If associated key and residual TrPs remain they should be released promptly. This combined approach is often effective when either alone was not.

Hypermobility of the C_4 segment has been observed clinically to be associated with the trapezii. Joint stress that causes radiating pain can involve the trapezius secondarily, and the muscle often becomes hyperirritable and develops TrPs. An upper trapezius source of pain may be differentiated from a joint source by testing for pain on sidebending of the cervical spine and then: 1) Passively support the patient's upper limb and sidebend the cervical spine again. If the pain is markedly reduced or absent, the problem may be in the trapezius. 2) Apply pressure downward on the shoulder (as in lengthening the upper trapezius). If there is an increase in pain, the upper trapezius may be the source of the problem. If *neither* of these tests changes the pain, the cervical joints (perhaps C_4) may be the problem.

Treatment for TrPs is discussed in the next section. Hypermobility is treated with

appropriate isometric (stabilizing) exercises to the neck and with maintenance of a neutral position of the cervical spine during introduction of progressive upper limb activities. It is good to remember that a hypermobile segment may be adjacent to *hypomobile* segments which need to be released.

Middle Trapezius. The cervicothoracic junction is a troublesome transitional vertebral area that commonly develops dysfunctions, primarily of C_6, C_7, T_1, and occasionally T_2. Commonly these dysfunctions are associated with adduction of the scapulae and elevation of the first rib on the same side. Myofascial release of the shortened middle trapezius muscles is applied toward abduction of the scapulae bilaterally with the patient supine.

Lower Trapezius. Articular dysfunctions associated with interscapular pain and lower trapezius TrPs[54] may extend from T_4 to T_{12}. However, there is usually a central painful segment near T_6 or T_7 which is the primary structural dysfunction that must be treated along with inactivation of the TrPs.

12. Trigger Point Release
(Figs. 6.9, 6.10, 6.11)

Correction of poor posture (particularly "round-shouldered" posture with an excessive forward-head position) and maintenance of good posture are primary in any treatment approach, both for initial relief of pain and for lasting relief. Refer to Chapter 5, Section C, and Chapter 41, Section C for discussions of posture and body mechanics.

The upper trapezius is generally recognized as prone to hyperactivity and increased tension whereas the lower trapezius tends to be just the opposite, inhibited, weak and overstretched.[25, 38, 54] Understandably, because of its dual function, the middle trapezius cannot be unambiguously assigned to either category. Therapy that depends primarily on stretch can be counterproductive in muscles prone to inhibition and weakness. Therefore, we emphasize for both the middle and lower parts of the trapezius the application of massage to the taut band, TrP pressure release, and indirect techniques,[32,41,42] carefully avoiding forceful and excessive stretch. Vapocoolant or icing can precede any of these applications.

Upper Trapezius
(Fig. 6.9)

Essentially the same release procedure is used for both TrP_1 and TrP_2. To perform the **spray-and-release** technique on the right upper trapezius, the patient sits in an armchair, leans back comfortably and fully relaxes, with the arm on the involved side supported on a pillow. The operator guides the patient's head to laterally tilt toward the contralateral side with the head slightly flexed and the face turned slightly toward the involved right side (Fig. **6.9A).** At the same time, the operator applies the vapocoolant spray in parallel sweeps from the acromion to the mastoid area, behind the ear, around to the temple, and sometimes to the jaw (particularly if that area is included in the patient's pattern of referred pain). The operator takes up slack with the guiding hand as it develops in the muscle.

Following the spray phase, the operator applies a gentle myofascial release technique that requires the ability to feel the increased resistance as muscle elongation encounters a barrier and to feel when the barrier releases. The patient abducts the right scapula by placing the arm forward on the pillow. The operator (Fig. **6.9B)** stabilizes the patient's head position with one hand and with the other hand (right hand in this case) takes up any slack in the muscle by gently pressing laterally and downward on the scapula. Release is augmented by having the patient coordinate downward eye motion and slow exhalation with relaxation, and coordinate upward eye motion and slow inhalation with gentle contraction of the muscle against the operator's right hand. Respiratory augmentation is more effective, particularly in this muscle, if the patient emphasizes diaphragmatic breathing and avoids paradoxical respiration. Postisometric relaxation for the upper trapezius is illustrated and described in detail by Lewit.[53]

The patient should have good elbow support during the procedure and during moist heat application following the release procedure; the armrests of the chair should carry the weight of the patient's arm (see Fig. **6.13A** and **D).**

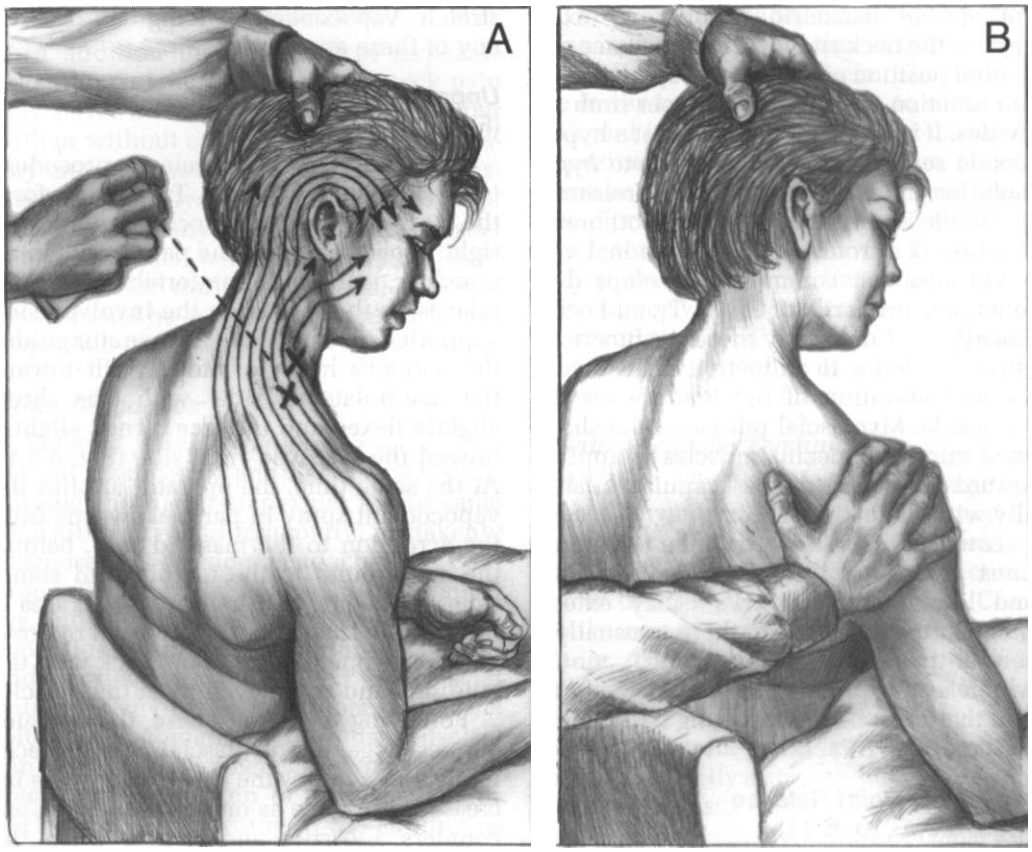


Figure 6.9. Spray and stretch-release of trigger points in the right upper trapezius muscle. **A,** The patient, seated in a relaxed position, places the right arm on a pillow for support. The operator first applies successive sweeps of vapocoolant over the spray pattern *(arrows)* while guiding and supporting the patient simultaneously to lean the head away from the muscle being treated, rotate the face gently toward the side of the involved muscle, and rock the head forward *without* flexing the cervical spine. **B,** The patient next places the arm of the involved side slightly forward on the pillow to abduct the scapula. The operator uses the left hand to *stabilize* the head position and takes up any slack in the muscle by gently pressing laterally and downward on the scapula as the muscle tension releases. Postisometric relaxation makes an effective addition to this release (see text).

Spray and stretch should also always be applied to the *contralateral* trapezius to prevent activation of any TrPs in it due to unaccustomed shortening when the involved muscle is stretched to its maximum normal length. Others have reported stretch and spray to be effective for this muscle.[19, 107]

When patients present with the sudden onset of a severe stiff neck involving multiple neck muscles (e.g. upper trapezius, levator scapulae, sternocleidomastoid, and posterior cervical) in painful spasm, the neck may be completely immobilized. The spasm must be relieved before attempting other therapy. Any attempt to release the muscles by lengthening them only aggravates the symptoms. Application of a comfortable high voltage galvanic stimulation sufficient to fatigue the muscles, can relax them and relieve the painful spasm.

Middle Trapezius
(Fig. 6.10)

In the case of middle trapezius TrPs the importance of *checking* both *pectoral*

muscles for tightness (and TrPs) cannot be overemphasized. Most commonly, the middle trapezius stretch-weakness and TrPs are from overload and are secondary. Unless the tightness of the anterior muscles causing the problem is effectively addressed, the patient will continue to have trouble. It is not unusual for the trouble-making pectoral TrPs to be latent and producing shortening, but not a source of pain in their own right. The symptoms that they cause appear secondarily in the overloaded posterior muscles.

The **spray-and-release** technique for **middle** trapezius TrPs begins with the patient sidelying or semiprone and the involved (right) side uppermost (Fig. 6.10A). The right upper limb is elevated 90° (in line with the muscle fibers being released) and dropped forward off the edge of the treatment table to take up slack in the muscle by abducting the scapula. The spray is applied from the lateral attachment point, over the TrP region and over all the middle trapezius fibers, following the fibers medially and covering the referred pain zone, overlapping the lower trapezius to some extent. Frequently the entire trapezius is involved and spray should then start from the lowermost fibers at the level of T_{12}, fanning upward and laterally to cover all three parts of the muscle and the referred pain zones.

Release of the middle trapezius fibers continues with the operator using one hand to stabilize the patient's midthoracic spine and placing the other hand over the scapula to take up slack as the muscle relaxes (Fig. 6.10B). Augmented postisometric relaxation is accomplished by asking the patient to "Look up to the right and breathe in. Now look to your left and *slowly* breathe out. Relax and let your arm drop toward the floor." With the patient in this position, gravity resists the middle trapezius contraction and then assists the relaxation. This procedure can be repeated two or three times to fully release the muscle fibers. The operator's hand guides the scapula in line with the fibers being released (applying light resistance for the contraction phase only if needed), and takes up the slack in the muscle.

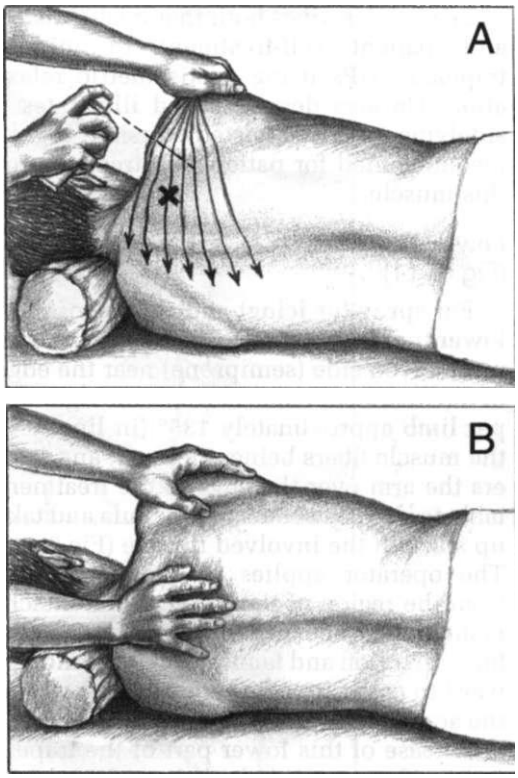


Figure 6.10. Spray and release of trigger points in the **middle** part of the right trapezius muscle. **A,** application of spray with the patient lying on the uninvolved (left) side. **B,** release of middle trapezius fibers. See text for details.

In addition to this technique, the middle trapezius responds well to TrP pressure release by the operator or self-release by the patient using a cold tennis ball (see Fig. 18.4); it also responds well to local massage of the taut band in the region of the TrP. Another effective release for the middle and lower trapezius fibers is the scapular mobilization technique described and illustrated in Figure 18.3. The release procedure is followed promptly by full active range of motion and the application of moist heat.

For TrP_7, the spray is applied over the trigger area and the muscle fibers, continuing the sweeps down over the lateral aspect of the arm to cover the major "gooseflesh" reference zone. This TrP may require injection to completely inactivate it.

Lewit[54] describes both therapist-assisted and patient self-treatment of middle trapezius TrPs using postisometric relaxation. He also describes and illustrates a sidelying gravity-assisted version highly recommended for patient self-treatment of this muscle.

Lower Trapezius
(Fig. 6.11)

For **spray (or icing) and release** of right **lower** trapezius TrPs, the patient lies on the uninvolved side (semiprone) near the edge of the treatment table, elevates the right upper limb approximately 135° (in line with the muscle fibers being released), and lowers the arm over the edge of the treatment table to slightly abduct the scapula and take up slack in the involved muscle (Fig.6.11). The operator applies the spray upward from the region of the T_{12} vertebral attachment of the trapezius, following the muscle fiber direction and fanning laterally and upward to cover its pain reference zone from the acromion to the occiput (Fig. 6.11 A).

Release of this lower part of the trapezius can be accomplished through postisometric relaxation of the involved muscle fibers, with the vapocoolant being applied only while the patient is exhaling and relaxing the muscle. Figure 6.11 B illustrates a bimanual release technique which can incorporate postisometric relaxation; the operator asks the patient to look up to the right, inhale, and then look down to the left and exhale slowly, relaxing completely and letting the arm drop toward the floor. Since gravity can assist release of the lower trapezius, it is not necessary for the operator to apply pressure against the patient's scapula; however, the operator's touch can guide and encourage appropriate contraction and relaxation.

The release procedure is followed promptly by full active range of motion and the application of moist heat. It is usually best also to treat the contralateral trapezius, which must balance the released tension of the treated muscle.

Since the lower trapezius is often weak, the aim is not primarily stretch but rather release of tension in the taut band. Toward this aim, the operator can apply TrP pressure release and/or deep local massage of the nodular TrP. The patient can apply self-TrP pressure release by lying on a tennis ball that is positioned to press on the TrPs.

The lower part of the trapezius is often the key to successful treatment of the upper trapezius, levator scapulae and some neck extensor muscles; these muscles lie in the pain reference zone of the lower trapezius and may develop satellite TrPs to the key lower trapezius TrP. The lower trapezius itself (and by extension the above-mentioned muscles) may develop pain and TrPs due to TrP tension in the antagonistic pectoralis major *[see* Chapter 42) and pectoralis minor *[see* Chapter 43). When the pectoral muscles are involved, their full normal rest length must be restored in order for the lower trapezius to be relieved of overload, and then the lower trapezius may need to be strengthened.

Each release procedure is followed promptly by full active range of motion and moist heat to the treated region.

Lewit[54] describes a strengthening exercise to restore normal muscle balance and correct fixation of the scapula rather than describing a release technique. However, before beginning such a strengthening exercise which can be very helpful, any TrPs in the lower trapezius should first be inactivated.

Scapular mobility, as well as joint play in the sternoclavicular and acromioclavicular joints, should be restored if restricted.

13. TRIGGER POINT INJECTION
(Fig. 6.12)

The fibers of any part of the trapezius should be injected for TrPs only if spot tenderness is observed in a palpable nodule or taut band and the patient's pain is reproduced by digital compression of the tender spot. Eliciting an LTR by snapping palpation helps to confirm the diagnosis but is too unreliable to be included as a necessary finding. However, it is an important guide to effective placement of the needle during injection or dry needling. Injection is always followed at once by slow active full range of motion.

TrP_1, and rarely TrP_2, of the upper trapezius are injected from an anterior approach with the patient supine, using 0.5% procaine solution.[89,91] Other trapezius TrPs

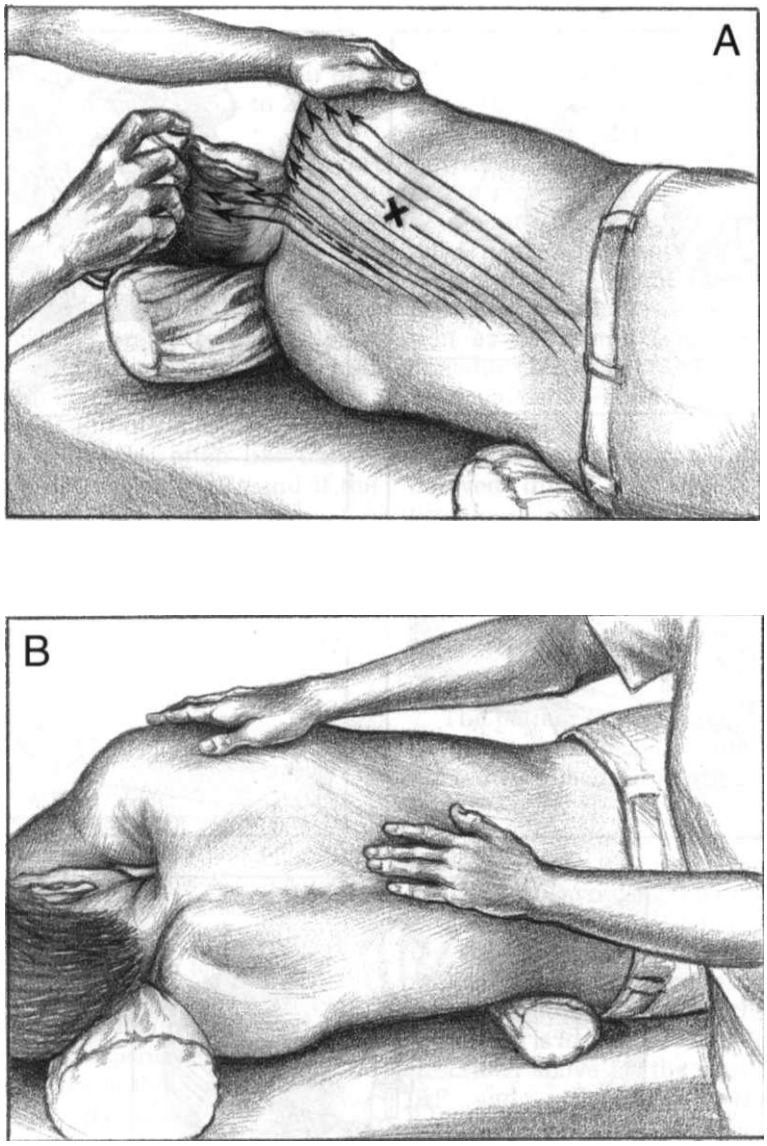


Figure 6.11. Spray and release of trigger points in the **lower** part of the right trapezius muscle. **A,** application of spray with the patient lying on the uninvolved (left) side. **B,** bimanual release of the lower trapezius fibers. See text for details.

are injected with the patient lying on the uninvolved side with the back toward the operator or lying prone.

Upper Trapezius

(Fig. 6.12, TrP_1 and TrP_2)

For injection of the more anterior central TrP_1, the patient lies supine with the shoulder on a pillow to slacken that part of the muscle (Fig. 6.12, TrP_1). The muscle is held firmly in a pincer grasp to precisely locate the TrPs for injection and to lift the muscle off underlying structures. The needle tip is directed upward across the muscle mass that is held between the digits (Fig. 6.12, TrP_1) to avoid any possibility of penetrating the apex of the lung.

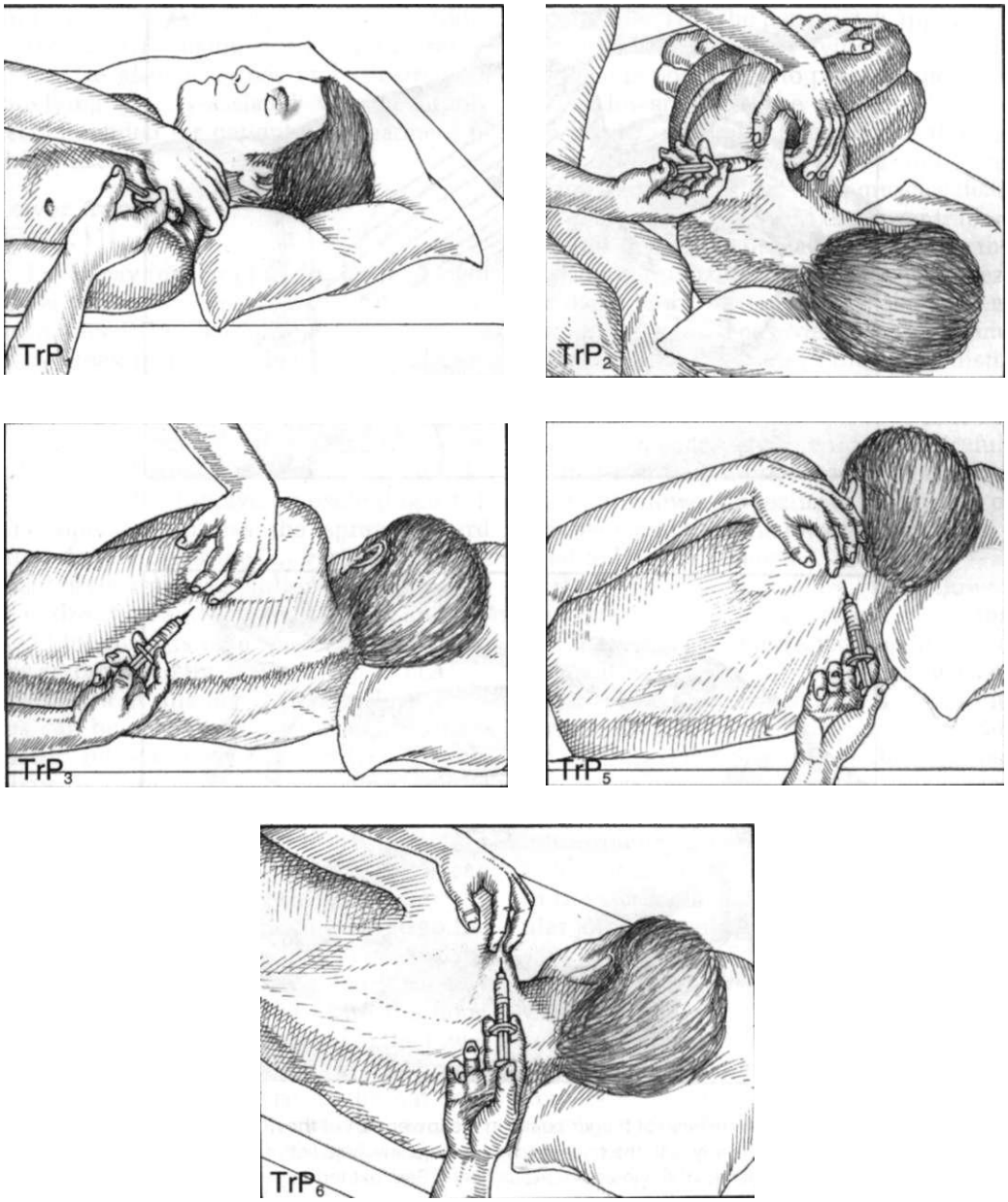


Figure 6.12. Patient position and injection technique for trigger points in the left trapezius muscle. To inject central *TrP*$_1$, the patient lies supine for the anterior approach to the upper trapezius, to avoid penetrating the apex of the lung. To inject central *TrP*$_2$ the patient lies on the right side for the posterior approach to the left upper trapezius, with the muscle lifted off the apex of the lung. To inject central *TrP*$_3$ in the lateral border of the lower trapezius, the patient lies on the opposite side. The needle is aimed at a rib, to avoid penetrating an intercostal space. The Hong technique for holding the syringe (see Chapter 3, Section 13) is recommended for this location. The central *TrP*$_5$ in the middle trapezius midfiber region close to the vertebral border of the scapula is injected with the patient lying on the opposite side. The Hong technique also is recommended for injection here. The attachment *TrP*$_6$ is injected along the lateral musculotendinous junction of the middle trapezius with the patient lying on the other side.

Injection of a trapezius TrP identified as TrP_1 in 20 patients with pain in the masseter region significantly reduced their perceived pain from a mean of 5.6 to 2.8 on a 10 point scale.[10]

Except in thin patients, the more posterior and inferior central TrP_2 is best approached by positioning the patient on the uninvolved side (Fig. 6.12, TrP_2); the needle is again directed upward away from the lung. To avoid penetrating too deeply when injecting TrP_2 in patients with loose skin, the operator's finger can be inserted under the front margin beneath the muscle, between the TrP and the chest.

A supraspinatus TrP often lies underneath the upper trapezius TrP_2, and if the deeper TrP is penetrated by the needle, the patient may report referred pain felt in the mid-deltoid region. Other authors have described and illustrated a similar technique for injecting TrP_2.[49,72]

> To relieve neck and back pain, Trommer and Gellman[96] infiltrated with procaine what they construed to be 15 intracutaneous TrPs *overlying* the upper trapezius. Occasionally, one sees cutaneous TrPs that refer pain like muscular TrPs. It also is possible that they relieved the pain by infiltrating the area of referred pain and referred tenderness as described by Weiss and Davis,'"[1] and by Theobald.[84] As noted above, pain and tenderness are often referred to this area from TrPs in the lower trapezius. In this case, the patient is more likely to experience lasting relief if the active TrPs in the lower trapezius that are causing the referred pain are injected, rather than the skin over the upper trapezius where the pain is felt.

Middle Trapezius

(Fig. 6.12, TrP_5 and TrP_6)

The patient lies on the opposite side with the hand placed on the thigh, or between the knees, to stabilize the scapula. Central TrP_5 (right side of Fig. 6.3) may or may not be over the scapula. If not (Fig. 6.12, TrP_5), the needle must be directed at an acute angle to the skin to ensure not penetrating to the level of the ribs. Needle contact with an active locus of the TrP is confirmed by the occurrence of a local twitch response. Coincidental penetration of TrP_7 (Fig. 6.4) while going through the skin to inject TrP_5 may surprise the patient by setting off waves of "gooseflesh."

Attachment TrP_6 (left side of Fig. 6.4) is in the region of the lateral musculotendinous junction of the middle trapezius and most likely represents enthesopathy secondary to a TrP_5 (right side of Fig. 6.3). Inactivation of this attachment TrP by injection as illustrated (Fig. 6.12, TrP_6) will expedite clearing of the trigger area tenderness, but relief is not likely to be lasting if the source of the enthesopathy, central TrP_5, is not inactivated. If the response is delayed, this is a situation where a one-time local injection of dilute steroid at the attachment TrP may be appropriate and helpful. Steroid is not recommended for the injection of central TrPs.

Lower Trapezius

(Fig. 6.12, TrP_3)

The patient lies on the uninvolved side. To locate and inject TrP_3, the scapula is abducted by placing the arm in front of the body in order to place the lower trapezius on a moderate stretch (Fig. 6.12, TrP_3). Care is taken to aim the needle toward an underlying rib, avoiding the intercostal space.

Attachment TrP_4 (Fig. 6.3, left side) overlies the scapula in the region of the lateral musculotendinous junction of the lower trapezius along the root of the spine of the scapula. It is identified and injected much as described above for the similar attachment TrP_6, and as illustrated for that attachment TrP (Fig. 6.12, TrP_6). To inject this TrP, the needle is aligned with the lateral fibers of the muscle and directed toward the shoulder.

14. CORRECTIVE ACTIONS
(Figs. 6.13, 6.14, and 6.15)

Upper Trapezius

Body Structure. A lower limb-length inequality or a small hemipelvis, as described in Section 7 above, must be corrected (*see* Fig. 48.9C and Fig. 48.10C and **D** in this volume, and Chapter 4 of Vol. 2).

When the patient's upper arms are short in relation to torso height, they do not reach the armrests of most chairs (Fig. 6.13C); this imposes sustained gravity

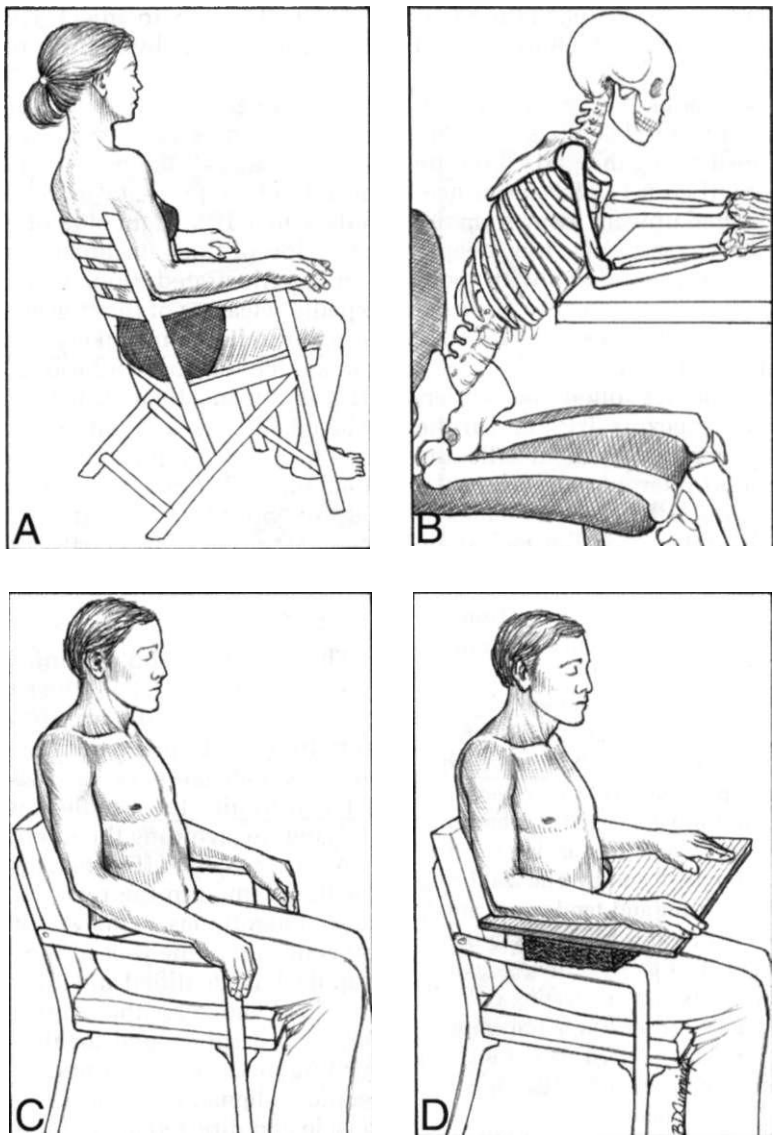


Figure 6.13. Short upper arms: the problem and its solution. **A,** the elbows of a person with average length of the upper arms are well supported in a properly designed chair; the armrest surface is usually about 23 cm (9 inches) above the seat bottom and the backrest overlaps the scapulae by several centimeters (an inch or two). **B,** skeleton with short upper arms demonstrates the strained posture that results from that structural inadequacy. **C,** the elbows of the patient with short upper arms are unsupported in another chair with the usual design similar to that in **A.** The dangling elbows overload the upper trapezius muscles. **D,** the needed elbow support can be provided by raising the armrest height with cellulose sponges, or plastic foam pads, glued beneath a writing board.

stress on the trapezius muscles. The Boston rocker has high armrests designed for nursing mothers, and is well suited to persons with short upper arms. Figure 6.13D illustrates another solution. An average armrest height of 21.6 cm (8.5 in.), measured from the compressed seat, satisfies most people.[15] Pads made from cellulose kitchen sponges or plastic foam may be covered and attached to the armrests, or may be mounted underneath a writing board that rests on the armrests and raises the board to the desired height for elbow support. The patient must learn to adapt any and all chairs that are used.

Relief from Postural and Activity Stress. No patient with TrPs in the upper trapezius should sleep on a foam rubber pillow; its springiness aggravates TrP symptoms. When traveling, the patient may need to take along a nonspringy comfortable pillow from home to avoid this hazard.

Antigravity stress on the upper trapezius in normally proportioned individuals is corrected by selecting chairs with armrests of the correct height to provide elbow support (Fig. 6.13A), or by building up the height of the armrests, if they were designed too low (Fig. 6.13D).[90] Dentists, secretaries, draftsmen, writers and seamstresses, for instance, should arrange their seating to provide suitable elbow support. Every seated person benefits by learning to distinguish between chairs that fit and chairs that enforce poor posture which abuses the muscles.[90]

Patients who are intensely preoccupied with what they are doing are prone to lose track of time and maintain an undesirable posture. This can happen while engrossed at a computer or leaning forward over a desk for a prolonged period while writing. These individuals can relieve muscle tension every 20 or 30 min, without interrupting the train of thought, by setting an interval-timer for that length of time and placing it across the room. Then they must get up and can stretch while they walk to turn off the buzzer and reset the timer.

For secretaries, a common source of gravity stress is a keyboard set so high that they hold the shoulders in an elevated position for the fingers to reach the keyboard conveniently. Excessive sustained EMG activity of the upper trapezius is eliminated by lowering the keyboard.[5]" If the keyboard support cannot be lowered sufficiently, the height of the seat should be raised until the forearms are horizontal, which relieves the trapezius muscles. Several centimeters (an inch or more) of folded newspapers or a magazine may be placed on the rear two-thirds of the seat bottom; the front third of the seat is not raised, thus avoiding underthigh compression. This slopes the seat forward and has the advantage of opening the angle at the hips and knees. If this raises the seat so much that the feet no longer rest flat on the floor, a small footrest is required. With video terminals, the copy should be placed as close beside the screen as possible. Placing the copy flat beside the keyboard should be avoided.

If the keyboard height is properly adjusted but the individual leans forward away from the backrest, the upper trapezius muscles may still be overloaded. Leaning back against the backrest of a chair so that it supports the scapulae can provide much relief. The individual must lean back and allow the shoulders to drop down so the backrest supports them. In most chairs, a small cushion for lumbar support facilitates good posture. Chapter 41, Section C includes additional suggestions for the correction of poor posture.

Muscles are more tolerant of prolonged activity if they have frequent short breaks permitting relaxation. A few cycles of active range of motion makes the break more effective. In the case of the upper trapezius, this may be achieved by slowly rotating the shoulders in a full circle several times, first in one direction and then in the other direction.

Many men (and women also when pants with pockets are fashionable) intuitively relieve upper trapezius muscle strain by standing (Fig. 6.14) or walking with hands in the pockets. This method of relief is recommended for persons prone to develop upper trapezius TrPs.

For patients who have long conversations on the telephone, a speaker phone relieves the neck and arm muscles from the strain of holding a handset.

Holding the steering wheel of a car by holding on the sides of the wheel or on top

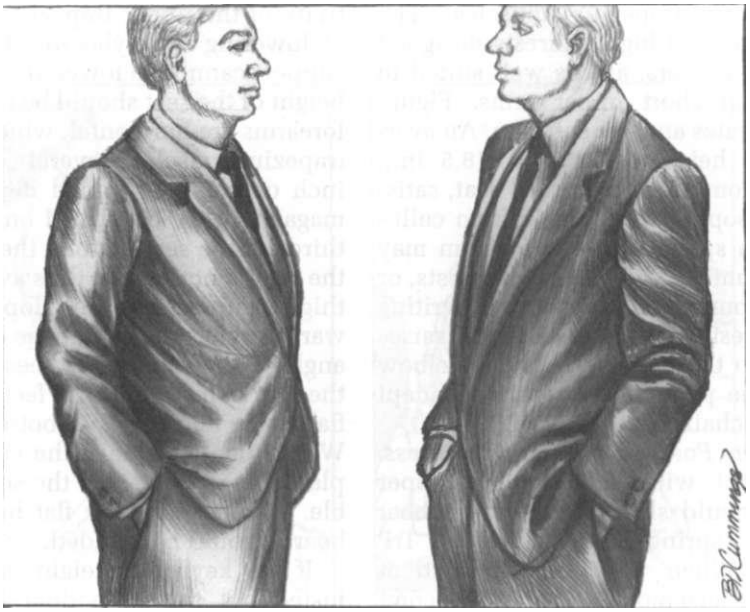


Figure 6.14. Common hands-in-pockets posture that helps to relieve strain on the upper trapezius muscles.

of it without armrests for long periods of time can seriously overload the upper trapezius (and levator scapulae) muscles. Holding the wheel with one hand at the bottom and with the forearm supinated and resting on the thigh allows emergency maneuvering and provides trapezius relief on that side.

When conversing with someone, the patient should turn his or her chair to face the other person, or turn the entire body and not just the head. The rotational stress of frequently flicking long hair out of the face is readily solved by a hair clasp or a haircut.

It is best to try to avoid sleeping prone when the trapezius is involved with TrPs. If one does sleep prone, a pillow placed under the shoulder and chest on the same side to which the face is turned helps to reduce rotation of the neck. A semiprone position, achieved by flexing the knee and hip of the side toward which the face is turned, also helps by partly rotating the torso.

A walking cane, when positioned beside the leg, should be long enough so that the elbow is bent 30-40°,[29] and does not require persistent elevation of the shoulder and scapula when used *[see* Fig. 19.3).

Relief from Constriction. Objectionable pressure on the trapezius by a thin, tight bra strap should be relieved by wearing a wider, nonelastic bra strap, and/or by slipping a soft plastic shield under the strap to distribute the pressure.[14] Sliding the strap laterally to rest on the acromion relieves pressure on the muscle. A strapless bra that constricts too tightly around the ribs may cause comparable pressure activation of TrPs in the latissimus dorsi, serratus anterior, or serratus posterior inferior muscles.

A shoulder-strap purse should be slung over the opposite acromion (not resting on the trapezius muscle). The shoulder-strap should be wide and its length adjusted to let the purse fit into the hollow of the waist. This lets the weight of the purse rest partly on the iliac crest when the purse is pressed against the side by the elbow. Whenever feasible, it is best for the muscles to hang the purse from a belt.

A heavy coat that rests on the upper trapezius, rather than on the acromion to the side, should be avoided; shoulder pads inserted in the coat can properly redistribute its weight.

Tension Release Exercises. Two exercises are helpful to maintain full length

and normal tension of the upper trapezius. One is the self-stretch technique for the upper trapezius described and illustrated in Figure 16.1IB. Lewit[54] describes in detail another release of this muscle in the seated position using postisometric relaxation.

Active Exercise. Two of the safest general conditioning exercises to help shoulder muscles that include the trapezius are swimming and jumping rope while progressing forward. Jogging tends to aggravate trapezius TrPs.

Middle Trapezius

When the arm must be held out in front of the body for long periods of time, some form of elbow rest should be devised.

The Middle-trapezius Exercise (Fig. 6.15) is tailored to maintain full active range of motion in both the middle and lower trapezius muscles. The patient is instructed as follows: Lie supine on the floor. Place the elbows, forearms and palms of the hands together in front of the abdomen (Fig. 6.15A). Keep the elbows tightly to-

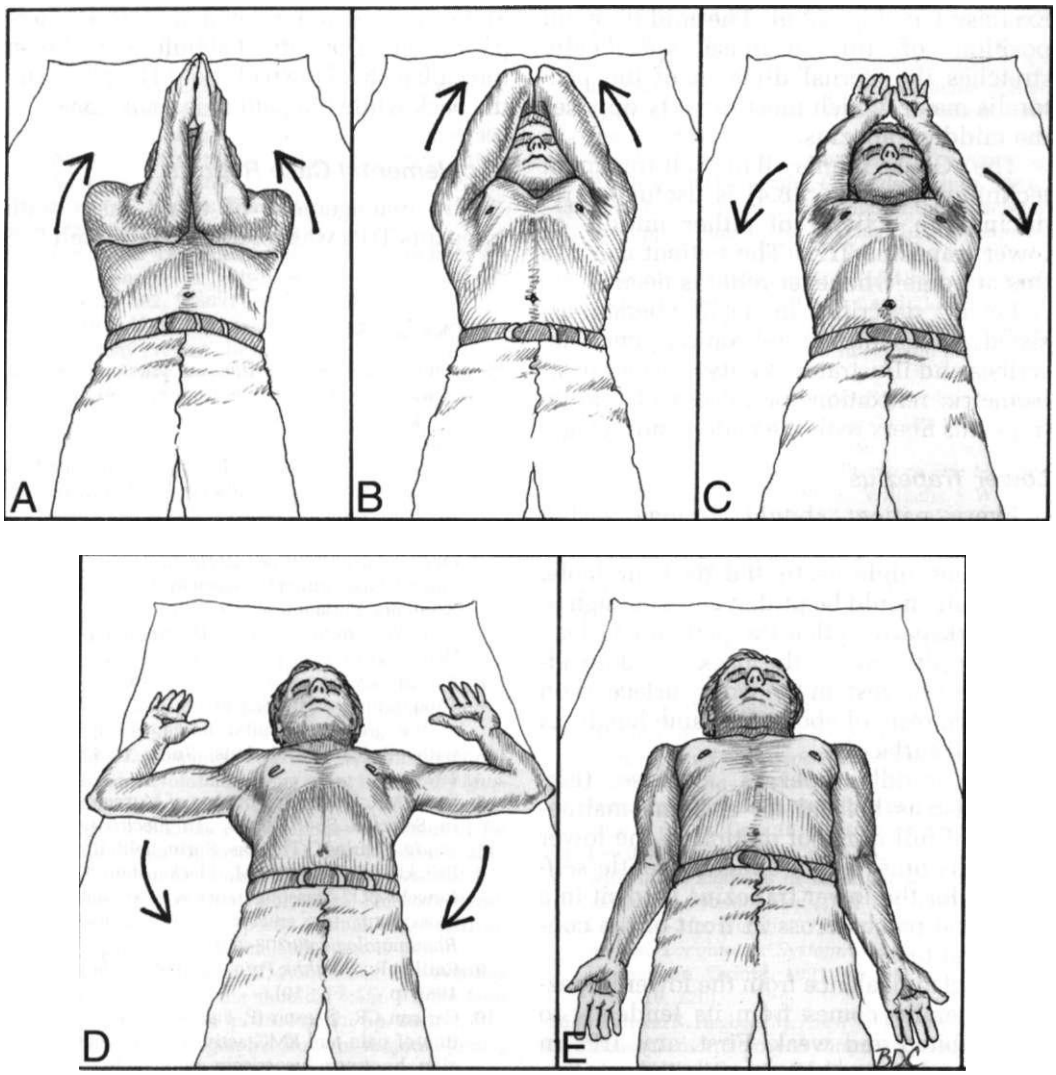


Figure 6.15. The Middle-trapezius Exercise helps to maintain full range of motion in the middle and lower parts of the trapezius muscle by abducting and rotating the scapulae. Movements progress from **A** through **E.** When completed, the patient pauses, breathes deeply to relax, and repeats the sequence, (See Section 14 for a full description),

gether as long as possible while raising the forearms over the face (Fig. 6.15B). Then, drop the forearms past the ears to the floor (Fig. 6.15C). Keeping the back of the elbows and wrists in contact with the floor, swing the arms down against the sides of the body (Fig. 6.15D and E). Pause and relax, while taking several slow deep breaths. Repeat the cycle.

The antagonistic pectoralis major fibers usually are in need of stretching when the middle trapezius harbors active TrPs. These pectoral fibers are passively stretched by doing the In-doorway Stretch Exercise *[see* Fig. 42.9). The middle hand position of this exercise specifically stretches the sternal division of the pectoralis major, which most directly opposes the middle trapezius.

The Cold Tennis Ball self-treatment technique *[see* Fig. 18.4) is useful for reducing the activity of either middle or lower trapezius TrPs. The patient can use this at home whenever relief is needed.

Lewit[84] describes in detail operator-assisted postisometric relaxation, and describes and illustrates gravity-assisted postisometric relaxation for release of middle trapezius fibers with the patient sidelying.

Lower Trapezius

Every patient should arrange seated workspace that provides adequate room for the knees underneath the desk or table. The chair should be pulled close enough to the workspace so that the patient can lean back firmly against the backrest; both elbows should rest on the work surface or on short armrests of about the same height as the desk surface.

The middle-trapezius exercise (Fig. 6.15) also is helpful for at-home maintenance of full range of motion in the lower trapezius muscle. An effective gentle *self-stretch* for the lower trapezius is to sit in a chair and reach across in front of the contralateral toes.

Muscle imbalance from the lower trapezius generally comes from its tendency to be inhibited and weak. First, any TrPs in the muscle must be inactivated. Active strengthening exercises specifically for the middle and lower trapezius can be done in the prone position, placing the arm out horizontally (for the middle trapezius) and lifting the arm up off the surface. For the lower trapezius the arm is elevated up toward the ear (in line with the muscle fibers) and then lifted off the surface. Biofeedback through surface EMG can assure the clinician and the patient that the weak muscle is indeed being activated appropriately and efficiently during a progressive strengthening program.

When using a moist heating pad or hot pack for relief of pain referred from TrPs in the lower fibers of the trapezius, the patient should apply the heat to the mid-back area where the TrPs are located, rather than solely to the suprascapular region and neck where pain is felt. The patient should *never* lie on the pad; instead the pad should be placed on the back while the patient is semiprone.

Supplemental Case Reports

The management of three cases with trapezius TrPs was reviewed by Travell.[87,88]

REFERENCES

1. **Agur AM:** *Grant's Atlas of Anatomy.* **Ed. 9. Williams & Wilkins, Baltimore, 1991:381 (Fig. 6.32).**
2. *Ibid.* **p. 555 (Fig. 8.4).**
3. **Basmajian JV, DeLuca CJ:** *Muscles Alive.* **Ed. 5. Williams & Wilkins, Baltimore, 1985 (p. 263, 265, 267, 268, 426).**
4. **Bates T: Myofascial pain. Chapter 14. In:** *Ambulatory Pediatrics II: Personal Health Care of Children in the Office.* **Edited by Green M, Haggerty RJ. W.B. Saunders, Philadelphia, 1977 (pp. 147-148).**
5. **Bell WE:** *Orofacial Pains-Differential Diagnosis.* **Denedco of Dallas, Dallas, 1973 (p. 97).**
6. **Bovim G, Bonamico L, Fredriksen TA,** *et ah:* **Topographic variations in the peripheral course of the greater occipital nerve. Autopsy study with clinical correlations.** *Spine* **26(4j:475-478, 1991.**
7. **Broer MR, Houtz SJ:** *Patterns of Muscular Activity in Selected Sport Skills, An Electromyographic Study.* **Charles C Thomas, Springfield, 111., 1967.**
8. **Briickle W, Suckfull M, Fleckenstein W,** *et al:* **Gewebe-p02-Messung in der verspannten Riickenmuskulatur (m. erector spinae).** *Zeitschrift fiir Rheumatologie* **49.208-216, 1990**
9. **Cailliet R:** *Shoulder Pain.* **F.A. Davis, Philadelphia, 1966 (p. 22, Fig. 19).**
10. **Carlson CR, Okeson JP, Falace DA,** *et al.:* **Reduction of pain and EMG activity in the masseter region by trapezius trigger point injection.** *Pain* **55(3j:397-400, 1993.**
11. **Clemente CD:** *Gray's Anatomy,* **Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 513, 514, 1189; Fig. 6-42).**

12. Clemente CD: *Anatomy,* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 523).
13. *Ibid.* (Figs. 61, 576).
14. De Silva M: The costoclavicular syndrome: a "new cause". *Annals of the Rheumatic Diseases* 45:916-920, 1986.
15. Diffrient N, Tilley AR, Bardagjy JC: *Humanscale 1/2/3.* MIT Press, Cambridge, 1974.
16. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 3-5).
17. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (pp. 344-352, Figs. 43 and 47).
18. Engle WK: Ponderous-purse disease. *N Engl J Med 299:557,* 1978.
19. Gardner DA: The use of ethyl chloride spray to relieve somatic pain. *J Am Osteopath Assoc* 49:525-528, 1950 (Case 4).
20. Gelb H: Patient evaluation. Chapter 3. In: *Clinical Management of Head, Neck and TMJ Pain and Dysfunction.* Edited by Gelb H. W.B. Saunders, Philadelphia, 1977 (p. 73).
21. Gerwin RD, Shannon S, Hong CZ, er *al:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
22. Good MG: What is "flbrositis"? *Rheumatism* 5:117-123, 1949 (pp. 119-121, Fig 2).
23. Good MG: The role of the skeletal muscle in the pathogenesis of diseases. *Acta Med Scand* 238:285-292, 1950 (Fig. 3, Case 2).
24. Graff-Radford SB, Jaeger B, Reeves JL: Myofascial pain may present clinically as occipital neuralgia. *Neurosurgery* 29f4j:610-613, 1986.
25. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 146, 147, 454, 488).
26. Gutstein M: Diagnosis and treatment of muscular rheumatism. *Br J Phys Med* 2:302-321, 1938 (pp. 310, 311).
27. Gutstein-Good M: Idiopathic myalgia simulating visceral and other diseases. *Lancet 2:326-* 328,1940.
28. Hagberg M: Electromyographic signs of shoulder muscular fatigue in two elevated arm positions. *Am J Phys Med* 60(3J:111-121, 1981.
29. Hoberman M: Crutch and cane exercises and use. Chapter 10. In: *Therapeutic Exercise.* Ed. 3. Edited by Basmajian JV. Williams & Wilkins, Baltimore, 1978 (p. 239).
30. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *JMusculoskePain* 2(2j:29-59, 1994.
31. Hong CZ, Simons DG: Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. *JMusculoske Pain* 2(2J;89-131, 1993.
32. Hoover HV: Functional technic. In: *Yearbook, Academy of Applied Osteopathy.* Carmel, CA, American Osteopathic Association, 1958, (pp. 47-51).
33. Inman VT, Saunders JB, Abbott LC: Observations of the function of the shoulder joint. / *Bone Joint Surg* 26.1-30, 1944 (p. 25, Fig. 31; pp. 26, 27).
34. Ito N: Electromyographic study of shoulder joint. / *Jpn Orthop Assoc* 54:1529-1540, 1980.
35. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 9.157-164, 1989.
36. Jaeger B: Differential diagnosis and management of craniofacial pain. Chapter 11. In: *Endodontics.* Ed. 4. Edited by Ingle JI, Bakland LK. Williams & Wilkins, Baltimore, 1994 (pp. 550-607).
37. Jaeger B, Reeves JL, GraffGreenman-Radford SB: A psychophysiological investigation of myofascial trigger point sensitivity vs. EMG activity and tension headache. *Cephalalgia 5(Suppl 3):68,* 1985.
38. Janda V: Evaluation of muscular imbalance. Chapter 6. In *Rehabilitation of the Spine: A Practitioner's Guide.* Edited by Liebenson C. Williams & Wilkins, Baltimore, 1996 (pp. 97-112).
39. Jensen C, Nilsen K, Hansen K, *et al.:* Trapezius muscle load as a risk indicator for occupational shoulder-neck complaints. *Int Arch Occup Environ Health* 64f6j:415-423, 1993.
40. Johnson G, Bogduk N, Nowitzke A, *et al.:* Anatomy and actions of the trapezius muscle. *Clin Biomech* 9:44-50, 1994.
41. Johnston WL: Functional Technique. Chapter 57. In *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997 (pp. 795-808).
42. Jones LH: *Strain and Counterstrain.* The American Academy of Osteopathy, Colorado Springs, 1981.
43. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving, I-III *Ergonomics* 28:375-388, 1975 (p. 381).
44. Kelly M: New light on the painful shoulder. *Med J Aust* 2:488-493, 1942 (Cases 1 and 2).
45. Kelly M: Some rules for the employment of local analgesic in the treatment of somatic pain. *Med f Aust* 2:235-239, 1947.
46. Kelly M: The relief of facial pain by procaine (novocaine) injections. *J Am Geriatr Soc* 2 2:586-596, 1963 (Table 1, Fig. 4, Case 3).
47. Kendall FP, McCreary EK, Provance PG: *Muscles, Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 282).
48. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (p. 98).
49. Kraus H: Trigger points. *NY State J Med* 73f22j:1310-1314, 1973.
50. Krause HR: Shoulder-arm-syndrome after radical neck dissection: its relation with the innervation of the trapezius muscle. *Int f Oral Maxillofac Surg* 22(5j:276-279, 1992.
51. Laskin DM: Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 79: 147-153, 1969.
52. Lange M: *Die Muskelharten (Myogelosen).* J.F. Lehmanns, Munchen, 1931 (p. 129, Fig. 40b; p. 93, Case 3; p. 118, Case 15; p. 130, Case 21).
53. Lewit K: Postisometric relaxation in combination with other methods of muscular facilitation and inhibition. *Manual Med* 2:101-104, 1986.
54. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 24, 195, 196, 207, 208, 219, 220).
55. Lindman R, Hagberg M, Angqvist KA, ef *al.:* Changes in muscle morphology in chronic trapezius myalgia. *Scand J WorkEnviron Health* 27:347-355,1991.
56. Lockhart RD, Hamilton GF, Fyfe FW: *Anatomy of the Human Body.* Ed. 2. J.B. Lippincott, Philadelphia, 1969 (pp. 318, 321).

57. Long C II: Myofascial pain syndromes: Part II—Syndromes of the head, neck and shoulder girdle. *Henry Ford Hosp Med Bull* 4:22-28, 1956.
58. Lundervold AJ: Occupation myalgia. Electromyographic investigations. *Acta Psychiatr Neurol* 26:360-369, 1951.
59. Lundervold AJ: Electromyographic investigations during sedentary work, especially typewriting. *Br J Phys Med* 24:32-36, 1951.
60. Lundervold AJ: Electromyographic investigations of position and manner of working in typewriting. *Acta Physiol Scand 24(Suppl):* 84, 1951 (pp. 26, 27, 94, 95, 97, 126, 129).
61. Marbach JJ: Arthritis of the temporomandibular joints. *Am Fam Phys* 29:131-139, 1979 (p. 136).
62. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy,* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 119).
63. *Ibid.* (pg. 46).
64. Melnick J: Trigger areas and refractory pain in duodenal ulcer. *NY State J Med* 57:1073- 1076, 1957.
65. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Co, Boston, 1964.
66. Michele AA, Davis JJ, Krueger FJ, *et al.:* Scapulocostal syndrome (fatigue-postural paradox). JVY *State J Med* 50.1353-1356, 1950 (p. 1355, Fig. 4).
67. Modell W, Travell JT, Kraus H, *et al:* Contributions to Cornell Conferences on Therapy. Relief of pain by ethyl chloride spray. *NY State J Med* 52:1550-1558, 1952.
68. Motta A, Tainiti G: Paralysis of the trapezius associated with myogenic torticollis. *Ital J Orthop Traumatol* 3:207-213, 1977.
69. Pace JB: Commonly overlooked pain syndromes responsive to simple therapy. *Postgrad Med* 58:107-113, 1975 (Fig. 4).
70. Patton IJ, Williamson JA: Fibrositis as a factor in the differential diagnosis of visceral pain. *Can Med Assoc J* 58:162-166, 1948 (Case 1).
71. Pernkopf E: Arias *of Topographical and Applied Human Anatomy,* Vol 2. W. B. Saunders, Philadelphia, 1964 (p. 33, Fig. 27).
72. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (see pp. 300- 303).
73. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 146-150).
74. Reitinger A, Radner H, Tilscher H, *et al:* Morphologische Untersuchung an Triggerpunkten [Morphologic study of trigger points]. *Manuelle Medizin* 34:256-262, 1996.
75. Rosomoff HL, Fishbain DA, Goldberg M, *et al:* Physical findings in patients with chronic intractable benign pain of the neck and/or back. *Pain* 37:279-287, 1989.
76. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Rehabil* 62:107-110, 1981.
77. Scovazzo ML, Browne A, Pink M, *et al.:* The painful shoulder during freestyle swimming. *Am J Sports Med 19(6):577-582,* 1991.
78. Sharav Y, Tzukert A, Refaeli B: Muscle pain index in relation to pain, dysfunction, and dizziness associated with the myofascial pain-dysfunction syndrome. *Oral Surg* 46:742- 747, 1978.
79. Sola AE, Kuitert JH: Myofascial trigger point pain in the neck and shoulder girdle. *Northwest Med* 54:980-984, 1955.
80. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med* 34:585-590, 1955.
81. Soo KC, Guiloff RJ, Oh A, *et al.:* Innervation of the trapezius muscle: a study in patients undergoing neck dissections. *Head Neck* 22(6j:488-495, 1990.
82. Spalteholz W: *Handatlas der Anatomie Des Menschen,* Ed. 11, Vol. 2, S. Hirzel, Leipzig, 1922 (pp. 302, 303, Fig. 380).
83. Steinbrocker O, Isenberg SA, Silver M, *et al:* Observations on pain produced by injection of hypertonic saline into muscles and other supportive tissues. / *Clin Invest* 32:1045- 1051,1953 (Fig. 2).
84. Theobald GW: The role of the cerebral cortex in the perception of pain. *Lancet* 2:41-47, 94-97,1949 (p. 41, Fig. 3).
85. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul, Ed. 2, Vol. 1. Macmillan, New York, 1919 (Fig. 507).
86. *Ibid.* (Fig 534).
87. Travell J: Rapid relief of acute "stiff neck" by ethyl chloride spray. / *Am Med Worn Assoc* 4:89-95, 1949 (Cases 2 and 4).
88. Travell J: Basis for the multiple uses of local block of somatic trigger areas (procaine infiltration and ethyl chloride spray). *Miss Valley Med J* 72:13-22, 1949 (Case 3).
89. Travell J: Pain mechanisms in connective tissues. In: *Connective Tissues, Transactions of the Second Conference, 1951.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1952 (pp. 94-96, Figs. 28 and 29).
90. Travell J: Chairs are a personal thing. *House Beautiful,* pp. 190-193, (Oct.) 1955.
91. Travell J: Symposium on mechanism and management of pain syndromes. *Proc Rudolf Virchow Med Soc* 26:128-136, 1957 (Figs. 1 and 2).
92. Travell J: Temporomandibular joint pain referred from muscles of the head and neck. / *Prosthet Dent* 20:745-763, 1960 (Figs. 1 and 2).
93. Travell J: Mechanical headache. *Headache* 7:23-29, 1967 (Fig. 1).
94. Travell J, Bigelow NH: Role of somatic trigger areas in the patterns of hysteria. *Psychosom Med* 9:353-363, 1947.
95. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 2 2:425-434, 1952.
96. Trommer PR, Gellman MB: Trigger point syndrome. *Rheumatism* 8:67-72, 1952 (Case 7).
97. Veiersted KB, Westgaard RH: Development of trapezius myalgia among female workers performing light manual work. *Scand J Work Environ Health* 29:277-283, 1993.
98. Veiersted KB, Westgaard RH, Andersen P: Electromyographic evaluation of muscular work pattern as a predictor of trapezius myalgia. *Scand J Work Environ Health* 29:284- 290, 1993.
99. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J AO A* 72:697-710, 1973 (Fig. 28, No. 2).

100. Weed NK: When shoulder pain isn't bursitis. The myofascial pain syndrome. *Postgrad Med 74(3):W\-W2,* 1983.
101. Weiss S, Davis D: The significance of the afferent impulses from the skin in the mechanism of visceral pain. Skin infiltration as a useful therapeutic measure. *Am J Med Sci* 176:517-536, 1928.
102. Wetzler G: Physical therapy. Chapter 24. In: Diseases *of the Temporomandibular Apparatus.* Edited by Morgan DH, Hall WP, Vamvas SJ. C.V. Mosby, St. Louis, 1977 (p. 355).
103. Williams HL, Elkins EC: Myalgia of the head. *Arch Phys Ther* 23.14-22, 1942 (p. 19).
104. Winter Z: Referred pain in fibrositis. *Med Rec 157:34-37,* 1944.
105. Wyant GM: Chronic pain syndromes and their treatment. II. Trigger points. *Cart Anaesth Soc J* 26:216-219, 1979 (Case 1, Fig. 1).
106. Yamshon LJ, Bierman W: Kinesiologic electromyography: II. The trapezius. *Arch Phys Med Rehabil* 29:647-651, 1948.
107. Zohn DA: *Musculoskeletal Pain.* Ed. 2. Little, Brown & Company, Boston, 1988 (Figs. 9- 2B and 12-1).

CHAPTER 7
Sternocleidomastoid Muscle

HIGHLIGHTS: The sternocleidomastoid is an amazingly complex muscle that frequently contains multiple trigger points (TrPs) in its sternal division, in its clavicular division, or in both. Although the name sternomastoid is in current use, we prefer the name sternocleidomastoid to recognize the equal significance of the *clavicular* division. **REFERRED PAIN** from these two anatomically and functionally different divisions presents quite different patterns. In each division, TrPs also evoke different autonomic phenomena or proprioceptive disturbances. The sternal division may refer pain to the vertex, to the occiput, across the cheek, over the eye, to the throat, and to the sternum. With clavicular division TrPs, patients commonly experience frontal headache and earache, whereas sternal division TrPs give rise to eye and face pain likely to be diagnosed as "atypical facial neuralgia." Referred autonomic phenomena from the sternal division involve the eye and sinuses, while from the clavicular division they are more likely to concern the forehead and ear, including dizziness related to disturbed proprioception and spatial perception. **ANATOMY:** Both divisions of one muscle attach to the head at the mastoid process and along the superior nuchal line. The more superficial, anterior, and diagonal sternal division attaches below to the sternum, while the deeper clavicular division attaches posterior and lateral to it onto the clavicle. **FUNCTION** of one muscle alone includes rotating the face to the contralateral side and tilting it up toward the ceiling. Together, the paired sternocleidomastoid muscles flex the head and neck and act as auxiliary muscles of inhalation. They function to checkrein (control) posterior movement of the head and neck. **SYMPTOMS** of postural dizziness and imbalance may prove even more incapacitating than head pain referred from TrPs in this muscle. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are commonly initiated by an episode of mechanical overload and perpetuated by persistent overload caused by structural inadequacies of the body, or by paradoxical breathing. **TRIGGER POINT EXAMINATION** is most effective if pincer palpation is used to encircle each division separately in order to carefully examine each division for palpable bands, tender TrPs and local twitch responses. **TRIGGER POINT RELEASE** techniques are specific to each division because of the markedly different positions of the head that fully lengthen each division. **TRIGGER POINT INJECTION** is relatively simple and safe when properly done, but sometimes produces considerable postinjection soreness and distressing referred autonomic and proprioceptive phenomena. **CORRECTIVE ACTIONS** to secure lasting relief usually require identification of, and structural compensation for, congenital body inadequacies, such as lower limb-length inequality (LLLI), a small hemipelvis, or relatively short upper arms, and also may require correction of poor posture and systemic perpetuating factors. Lasting relief also may require modification of daily activities, for example, prolonged or frequent telephoning.

1. REFERRED PAIN (AND CONCOMITANTS) (Fig. 7.1)

The sternal and clavicular divisions of this muscle have their own characteristic referred pain patterns and concomitants.[65,66,69,72] As a rule neither division refers pain to the neck, both refer pain to the face and cranium. The face pain referred from trigger points (TrPs) in this muscle is frequently the basis for the diagnoses of "atypical facial neuralgia,"[69] tension headache,[27,35,39] and cervicocephalalgia.[42] The pain and the autonomic or proprioceptive components referred from TrPs in this muscle are widely recognized by the dental profession as a significant component of common facial pain complaints.[48,57]

The pain pattern referred from the sternocleidomastoid muscle in children is similar to that in adults.[1,5]

Williams and Elkins[76] remarked that myalgia of the head is accompanied by circumscribed tender regions in the neck muscles at their attachments to the cranium. They reported inducing referred head pain by applying digital pressure to these tender muscles and by injecting hypertonic salt solution into them, location unspecified. We find attachment TrPs (ATrPs) where the sternocleidomastoid muscle attaches to the mastoid process. These ATrPs are likely enthesopathy secondary to central TrPs (CTrPs) in the muscle belly.

Sternal Division (Fig. 7.1 A)

Pain. An attachment TrP (ATrP) at the *lower end* of the sternal division may refer pain downward over the upper portion of the sternum (Fig. 7.1A). This is the only downward reference of pain from this muscle.[65,69] True trigeminal facial neuralgia is not accompanied by sternal pain, which, when also present, suggests the sternocleidomastoid myofascial syndrome.

When this ATrP is present in the lowest part of the sternal division, those fibers may merge with a slip of the inconstant sternalis muscle. Occasionally, mechanical stimulation of this sensitive area may be associated with a paroxysmal dry cough.

At the *midlevel* of the sternal division, TrPs refer pain homolaterally, arching across the cheek (often in finger-like projections) and into the maxilla, over the supraorbital ridge and deep within the orbit (Fig. 7.1A).[77] The aching quality of the pain described by patients is similar to the deep pain described by Kellgren,[37] following injection of small amounts of hypertonic saline into the muscles. The TrPs along the inner margin at the midlevel of this division refer pain to the pharynx and to the back of the tongue during swallowing[7] (which causes "sore throat") and to a small round area at the tip of the chin.[69] Marbach[43] shows a similar pattern that includes the cheek, temporomandibular joint and mastoid areas.

The TrPs located toward the *upper end* of the sternal division are more likely to refer pain to the occipital ridge behind, but not close to the ear, and to the vertex of the head like a skull cap, with scalp tenderness in the pain reference zone.

Concomitants. Autonomic concomitants of TrPs in the sternal division relate to the homolateral eye and nose.[65,69] Eye symptoms include excessive lacrimation, reddening (vascular engorgement) of the conjunctiva, apparent "ptosis" (narrowing of the palpebral fissure) with normal pupillary size and reactions, and visual disturbances. The "ptosis" is due to spasm of the orbicularis oculi muscle, rather than to weakness of the levator palpebrae muscle. The spasm is apparently caused by referred increased excitability of the motor units of this muscle. The patient may have to tilt the head backward to look up, because of inability to raise the upper eyelid. Visual disturbances can include not only blurring of vision,[63,65] but also dimming of perceived light intensity.[70] Sometimes coryza and maxillary sinus congestion develop on the affected side.

In our experience, unilateral deafness in a few patients with no complaint of tinnitus, has been traced to TrPs in the sternocleidomastoid muscle. Wyant[77] attributed tinnitus in one patient to TrPs in either the sternocleidomastoid, upper trapezius, or cervical paraspinal muscles. Travell[65] has noted the association of unilateral tinnitus with a TrP in the deep division of the masseter muscle. Generally, we associate tinnitus with the deep part of the masseter muscle rather than the sternocleidomastoid. One reliable patient reported a crackling sound in the homolateral ear, which was reproduced by pinching the superficial fibers of the sternal division at its midlevel.

Clavicular Division (Fig. 7.1 B)

Pain. Myofascial TrPs in the midfiber part of this division refer pain to the frontal area and when severe, the pain extends across the forehead to the other side (crossed reference),[64,65] which is very unusual for TrPs. The *upper part* of this division is likely to refer pain homolaterally deep into the ear and to the posterior auricular region (Fig. 7.1B). These TrPs sometimes refer poorly localized pain to the cheek and molar teeth on the same side.[69]

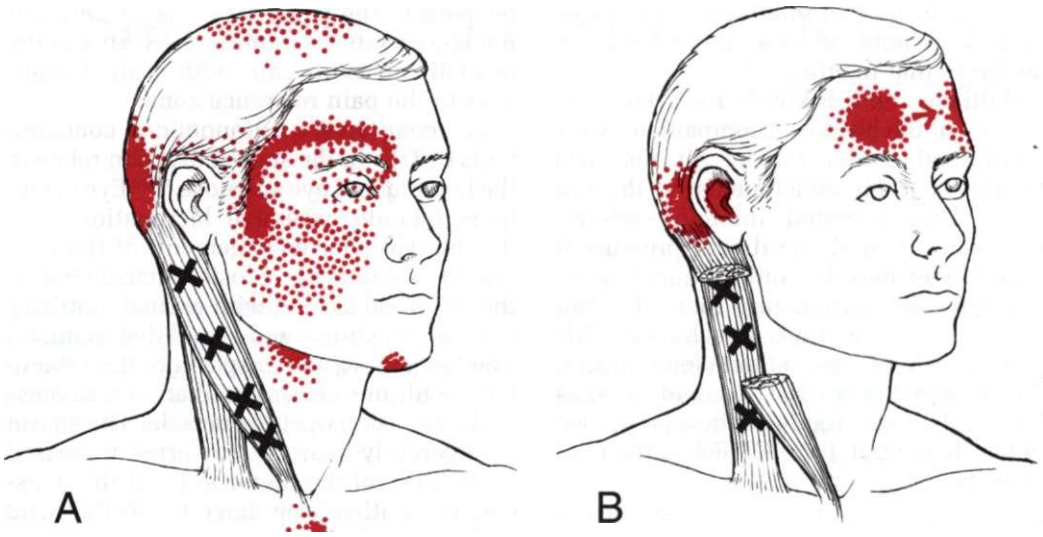


Figure 7.1. Referred pain patterns *(solid red* shows essential zones and *stippling* shows the spillover areas) with location of common trigger points (Xs) in the right sternocleidomastoid muscle. A, the sternal (more anterior and more superficial) division. B, the clavicular (more posterior and deeper) division.

Concomitants. Proprioceptive concomitants of TrPs in the clavicular division[63, 74] relate chiefly to spatial disorientation. Patients complain of postural dizziness (in the form of a disagreeable movement or sensation within the head),[38] and less often, of vertigo (the sensation of objects spinning around the patient, or of the patient spinning).[63,74] During severe attacks,[74] syncope following sudden turning of the head may be due to stretch-stimulation of active TrPs in the clavicular division. Episodes of dizziness lasting from seconds to hours are induced by a change of position that requires contraction of the sternocleidomastoid muscle, or that places it on a sudden stretch. Disequilibrium may occur separately from, or be associated with postural dizziness and may cause sudden falls when bending or stooping, or ataxia (unintentional veering to one side when walking with the eyes open).[62] The patient is unable to relate the vertigo or dizziness to a particular side of the head, even though it can be shown to depend on trigger mechanisms in only one sternocleidomastoid muscle. Postural responses are exaggerated in some patients; when looking up, they feel as if they will "pitch over backwards," and when glancing down, they tend to fall forward. The illusion of a tilted bed is not rare. Nausea is common, but vomiting is infrequent. Dimenhydrinate (Dramamine) may relieve the nausea, but not the dizziness. Good[25] attributed symptoms of dizziness to TrPs in either the sternocleidomastoid or the upper trapezius muscles. We have observed this symptom only from the former, although both muscles are commonly involved together.

These symptoms apparently derive from a disturbance of the proprioceptive contribution of this neck muscle to body orientation in space.[17] In man, the sternocleidomastoid is apparently one of the chief muscular sources of proprioceptive orientation of the head.[64] Experiments in monkeys[16, 17] established that the function of the labyrinths is confined to orienting the head in space, while the neck proprioceptive mechanisms are concerned with orienting the head in relation to the body.

Abolition of either of these systems produces spatial disorientation that is similar in form and magnitude.[16]

When objects of equal weight are held in the hands, the patient with unilateral TrP involvement of the clavicular division may exhibit an abnormal Weight Test. When asked to judge which is heaviest of two objects of the same weight that look alike but may not be the same weight (two vapocoolant dispensers, one of which may have been used) the patient will evidence dysmetria by underestimating the weight of the object held in the hand on the same side as the affected sternocleidomastoid muscle. Inactivation of the responsible sternocleidomastoid TrPs promptly restores weight appreciation by this test. Apparently, the afferent discharges from these TrPs disturb central processing of proprioceptive information from the upper limb muscles as well as vestibular function related to neck muscles.

Mechanical stimulation of active TrPs in the clavicular division also can refer the autonomic phenomena of localized sweating and vasoconstriction (blanching and thermographic cooling) to the frontal area of referred pain.

2. ANATOMY (Fig. 7.2)

Caudally the sternocleidomastoid muscle consists of two divisions: the sternal (more medial, more diagonal, and more superficial) and the clavicular (lateral and deeper). ***Cephalad,*** the two divisions blend to form a common attachment on the mastoid process (Figs. 7.2 and 20.7). The relative size of the two divisions and the space between them at the clavicle are variable.

Sternal Division

These fibers attach ***below*** to the anterior surface of the manubrium sterni. They attach ***above*** to the lateral surface of the mastoid process and to the lateral half of the superior nuchal line of the occipital bone (Fig. 7.2). The variable sternalis muscle may extend downward over the anterior chest, appearing like a continuation of the sternal division of the sternocleidomastoid (see Chapter 44).

Clavicular Division

This division attaches ***below*** to the superior border of the anterior surface of the clavicle along its medial third. It attaches ***above*** to the same bony structures as does the sternal division (Fig. 7.2).

Radziemski *et al.*[53] examined the distribution of muscle spindles in 16 human fetal sternocleidomastoid muscles and found that the greatest concentration of spindles was in the middle third, a few spindles were located in the cranial and sternal attachments of the sternal division, and that no spindles were observed in the clavicular division. In transverse sections, spindles were mainly located in the periphery of the muscle, particularly on the anterior surface. See Figure 2.31 for an example of spindle distribution in the sternocleidomastoid muscle.

Biopsies of a sternocleidomastoid muscle in 6 cancer patients[9] showed a nearly 2:1 ratio of fast twitch type II fibers to slow twitch type I fibers. This distribution is expected in a muscle that is exposed to intermittent loads but is not accustomed to prolonged sustained contraction.

SUPPLEMENTAL REFERENCES

Other authors illustrate this muscle well in the front view,[15,46,59] in a nearly front view,[13] and in the side view.[12, 22,45,60] The sternocleidomastoid is presented in cross section in Figure 7.6C, in Figure 16.8, and by others.[23]

3. INNERVATION

The motor fibers of the sternocleidomastoid muscle (and some of the trapezius) have an unusually close association with the brain stem which helps to account for its remarkable functional concomitants. They pass through the cervical portion of cranial nerve XI (the accessory nerve). These motor fibers of the cervical portion arise within the spinal column from the ventral roots (motor fibers) of the upper five cervical segments and *ascend,* entering the skull through the foramen magnum to join the cranial portion of the accessory nerve.[12,50] Together, they exit the skull in close association with the vagus nerve through the jugular foramen. The cranial portion of the accessory nerve provides the

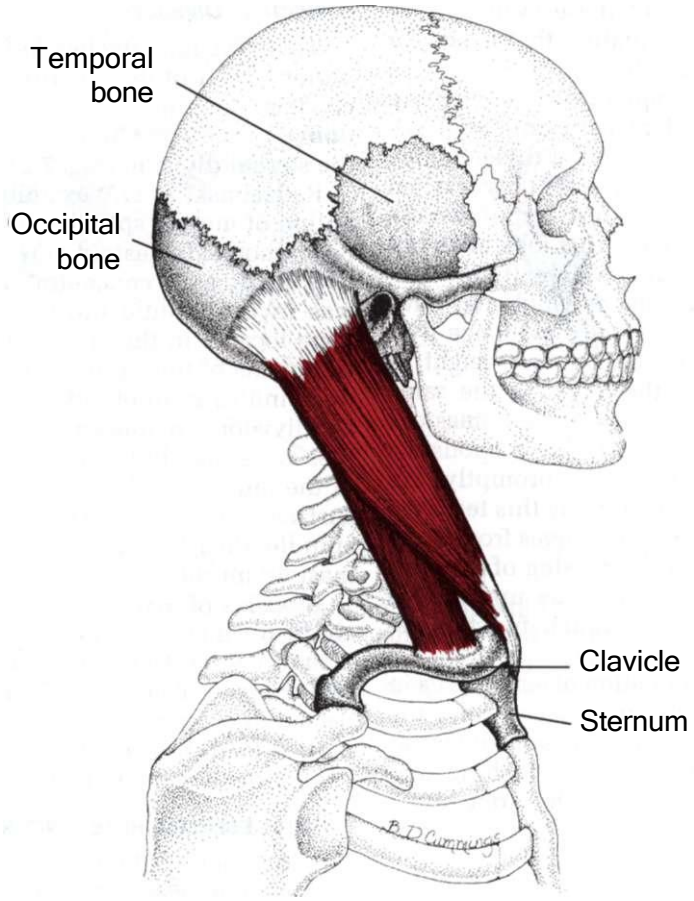


Figure 7.2. Attachments of the two divisions of the sternocleidomastoid muscle (*dark red).* The sternal division is more anterior, more diagonal, and more superficial than the clavicular division. The bones to which the muscle attaches show *darker stipples.*

motor innervation of several throat muscles and may also contribute motor fibers to the sternocleidomastoid muscle. Branches of the accessory nerve innervate the sternocleidomastoid muscle as the nerve passes through first the sternal head and then the clavicular head[56] on its way to innervate the upper portions of the trapezius muscle.

Surgical evidence[29] indicated that, in at least 9 of 15 patients, a functionally significant number of motor fibers originated as part of the vagus nerve intracranially but crossed over into the accessory nerve in the jugular foramen and supplied the sternocleidomastoid muscle.

The lower cervical nerve fibers of the cervical portion of the accessory nerve are largely sensory.[22] Central connections of the spinal nerve fibers include the pyramidal tract and the medial longitudinal fasciculus for the coordination of head and eye movement.[26]

4. FUNCTION

Both Muscles Together

1. Acting bilaterally, the sternocleidomastoid muscles flex the neck and pull the

head forward, bringing the chin onto the chest,[4,36,54] unless the head is strongly extended initially.[20]

2. On upward gaze, the muscles checkrein hyperextension of the neck. They also resist forceful backward movement of the head, which can occur when an unprotected passenger is riding in an auto that is struck from the rear ("whiplash").
3. Together with the trapezius, the two sternocleidomastoid muscles help to stabilize and fix the position of the head in space when the mandible moves during talking and chewing.
4. By strongly lifting the upper anterior rib cage, the muscles act as important auxiliary muscles of inhalation,[4,11,20,36,54] but only when the head and neck are in the erect or hyperextended position, and not when the neck is flexed.
5. Clinically, the sternocleidomastoid muscles participate in the act of swallowing[7] *(see* Sternocleidomastoid Compression Test in Section 8 of this chapter).
6. The sternocleidomastoid muscles contribute to spatial orientation, weight perception, and motor coordination. Experimental loss of sensory input at C_1, C_2, and C_3 results in spatial disorientation, imbalance and motor incoordination in monkeys and baboons.[16,17]

One Muscle

1. Acting unilaterally, the sternocleidomastoid muscle rotates the face toward the contralateral side and tilts it upward.[4,36,54]
2. Acting with the upper trapezius, the sternocleidomastoid muscle side-bends the cervical column, drawing the ear down to the shoulder on the same side.[4,36,54]
3. Acting with the scalene and trapezius muscles of the same side, the sternocleidomastoid muscle helps to compensate for the head tilt that is due to tilting of the shoulder-girdle axis, which, in turn, is often caused by the functional scoliosis associated with a lower limb-length inequality (LLLI), small hemipelvis, and/or quadratus lumborum TrPs.

Sports

During *right-handed* sport activities, the greatest electromyographic activation of the sternocleidomastoid was seen in the *left* muscle during the tennis serve, a golf swing, and during a jump on one foot in volleyball.[8]

5. FUNCTIONAL UNIT

One sternocleidomastoid muscle is synergistic with its homolateral upper trapezius during active lateral bending of the head and neck toward the same side, and also when checkreining lateral bending toward the opposite side. Together, both sternocleidomastoid muscles in their entirety are synergistic in checkreining hyperextension of the head and neck. Likewise, they are synergistic with the scalene muscles bilaterally during vigorous chest breathing (inhalation).

The sternal division on each side acts as an antagonist to the opposite muscle for head rotation.

The platysma, a skin muscle that overlies the sternocleidomastoid, may develop TrPs in relation to involvement of the sternocleidomastoid.

6. SYMPTOMS

Contrary to expectation,[39] neck pain and stiffness are generally not prominent features of sternocleidomastoid TrPs.[10,61] The patient may complain of "soreness" in the neck on rubbing these muscles, but the symptom is often disregarded, sometimes because the TrP nodules and tenderness are mistakenly attributed to lymphadenopathy ("glands"). Surprisingly, the patient with sternocleidomastoid TrPs prefers to lie on the side of the sore muscle if a pillow is adjusted to support the head so that the area of referred tenderness in the face does not bear weight. This muscle may add an additional component to the "stiff neck" syndrome,[61] which is primarily due to TrP activity in the levator scapulae, posterior cervical and trapezius muscles. If sternal division TrPs are sufficiently active they may cause tilting of the head to the same side as the TrPs because of pain on attempting to hold the head upright.[1] "Tension headache" is the diagnosis often given to the patient with the myofascial pain syndrome of the sternocleidomastoid.[34,35,39] The patient may be aware of ipsilateral sweating of the fore-

head, reddening of the conjunctiva and tearing of the eye, rhinitis, and apparent "ptosis" (narrowing of the palpebral fissure). Blurred or possibly double vision is sometimes reported; the pupils react normally. For the referred pain distribution and concomitants of sternocleidomastoid TrPs, see Section 1.

Rarely do sternocleidomastoid TrPs cause a complaint of restricted neck movement, although some limitation at the extremes of neck rotation, flexion and extension may be noted on careful examination.

Sternal Division

Pain referred from the sternal division may occur independently of pain referred from the clavicular division.[69] Sternal division pain involves chiefly the cheek, temple and orbit, as described in Section 1.

Autonomic phenomena referred from TrPs in this division, such as profuse tearing of the eye, is more distressing to some patients than pain. Rather than blurring and dimming of vision, the patient may be most aware of a visual disturbance when viewing strongly contrasting parallel lines, such as a Venetian blind. Narrowing of the palpebral fissure can be a prominent feature on the side of active TrPs in the sternal division.

Clavicular Division

Any one of the three major symptoms produced by TrPs in the clavicular division, namely, frontal headache, postural dizziness or imbalance, and dysmetria (disturbed weight perception)[28] may dominate the clinical picture. The pain is described in Section 1.

The dizziness is postural and occurs with changing loads on the muscle. Hyperextension of the neck and overstretching of the muscle, caused for example by lying without a pillow on a hard X-ray or examining table, may precipitate an attack of dizziness. Active TrPs in the clavicular division may contribute to seasickness or car sickness. Patients may complain of a "sick stomach" with nausea and anorexia that leads to a poor diet. The patient is likely to experience dizziness when turning over in bed at night, and should learn to roll the head on the pillow without lifting the head. During the day, transient loss of equilibrium is likely to follow vigorous quick rotation of the head and neck. During an acute attack of this postural dizziness, a person suddenly has serious difficulty driving an automobile. The car may veer, too. This may be a significant undocumented factor in some traffic accidents.[68]

Loss of equilibrium also may follow sustained tilting of the head to one side, as when holding a telephone receiver to the ear, or bird-watching with binoculars. The disturbed proprioception causing postural dizziness may be more disabling than the head pain coming from this muscle. These symptoms may appear in any combination, or all can appear together.

In a few patients, hearing was impaired unilaterally due to active TrPs in the clavicular division on the same side. Tinnitus has rarely been found to originate from TrPs in the sternocleidomastoid, but is likely to originate in TrPs of the deep division of the masseter muscle.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS (Fig. 7.3)

A posture or activity that activates a TrP, if not corrected, can also perpetuate it. In addition, many structural and systemic factors will perpetuate a TrP that has been activated by an acute or chronic overload (see Chapter 4). Excessive forward-head posture shortens the sternocleidomastoid muscle and activates (and strongly perpetuates) TrPs in it. Another postural source of activation and perpetuation is sitting with the head turned to the side for prolonged periods, for example when watching television or while talking to another person. Sleeping on two pillows (for example, to improve "sinus drainage") flexes the neck and shortens the sternocleidomastoid muscles, which tends to activate their TrPs. If the head must be elevated, it is advisable to place blocks under the legs at the head of the bed to tilt the bed frame, rather than to use extra pillows (*see* Chapter 20).

Mechanical Stress

Sternocleidomastoid TrPs are frequently activated during an episode of mechanical overload, for instance, by protracted neck

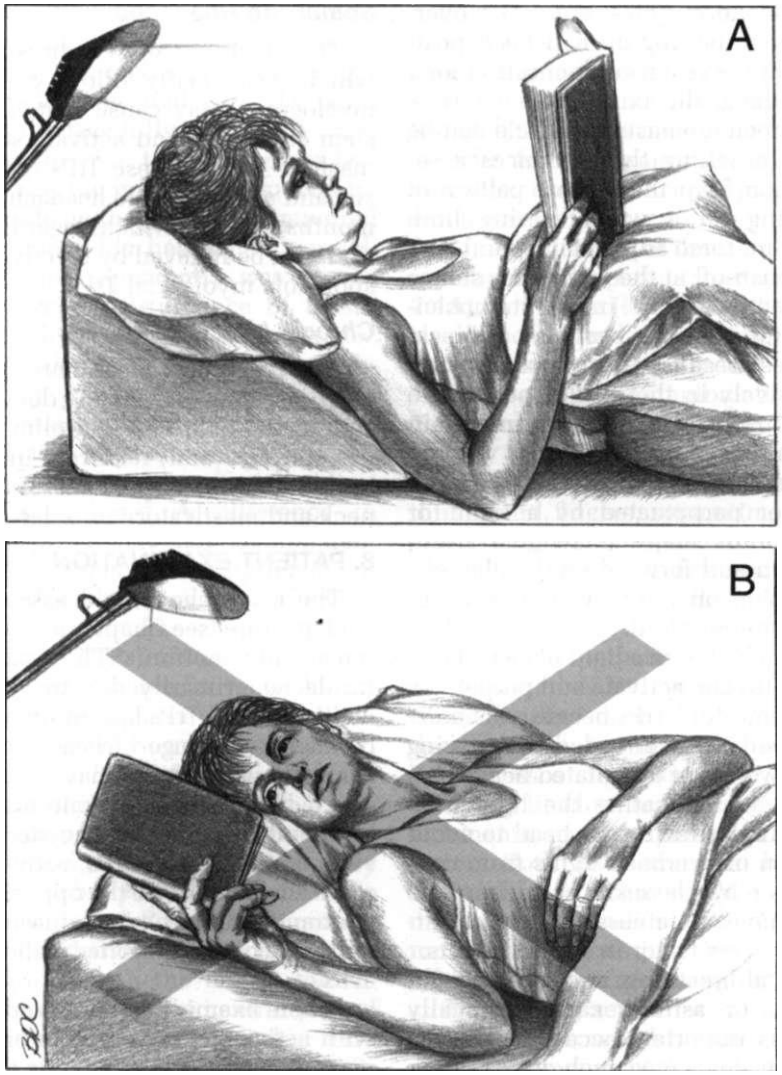


Figure 7.3. Desirable and troublesome (*red* X) head and neck positioning while reading in bed. A, desirable position with correct lighting and effective head support. **B,** undesirable position with inadequate head support and book to one side that can activate and perpetuate TrPs due to sustained contraction and overload, particularly in the uppermost sternocleidomastoid muscle.

extension in overhead work (painting a ceiling, writing on a blackboard, hanging curtains, sitting in a front-row seat in a theater with a high stage), by overuse in sports (wrestling), or by accidental injury (a fall on the head, "whiplash" in a motor vehicle accident[3]).

One common source of chronic postural stress that can activate and/or perpetuate sternocleidomastoid TrPs is deformity or injury that restricts upper limb movement and requires awkward compensatory neck positioning. Another is a structural inadequacy, such as a relatively short leg or small hemipelvis, both of which produce a functional scoliosis and shoulder-girdle tilting (see Figs. 48.9 and 48.10). The sternocleidomastoid muscles, in conjunction

with the scalene muscles, are easily overloaded by maintaining normal head position to level the eyes in compensation for a tilted shoulder-girdle axis.

The sternocleidomastoid muscle can be affected by anything that produces a severe deviation from the normal pattern of gait. Limping on a weightbearing limb (with resultant torso adjustments) and lack of normal push-off at the end of the stance phase can activate TrPs in the sternocleidomastoid (and levator scapulae and scalene muscles) because those muscles contract excessively in their reflex attempt to "help the movement" and/or maintain equilibrium.

Sternocleidomastoid TrPs can be activated and/or perpetuated by a tight (or tense) pectoralis major (clavicular head) pulling down and forward on the clavicle, putting tension on the clavicular head of the sternocleidomastoid.

Reading in bed with a light placed at one side (Fig. 7.3B) can activate and perpetuate sternocleidomastoid TrPs because the muscle on one side is overloaded by carrying most of the weight of the rotated head. This is corrected by relocating the light (Fig. 7.3A). Cocking or tilting the head to avoid the reflection of overhead lights from contact lenses or eyeglasses,[67] or to improve hearing in one-ear deafness, has been a critical muscle-stress factor in some patients.

Paradoxical breathing, a chronic cough, emphysema, or asthma can chronically overload this important accessory muscle of respiration. An acute cough due to upper respiratory infection can activate sternocleidomastoid TrPs and cause a frightful headache with every coughing spell.

Patients may acutely overstress the sternocleidomastoid by the hauling and pulling associated with horseback riding and the handling of horses. Pressure applied to TrPs in this neck muscle by a tight shirt collar or necktie can distressingly induce their referred pain pattern.

Hangover Headache

The "morning-after" hangover headache from alcoholic overindulgence may represent referred pain from activated sternocleidomastoid TrPs.[61] This kind of hangover pain may be quickly relieved by stretch and spray of the affected muscles.

Spinal Tap Headache

The leakage of cerebrospinal fluid, which occasionally follows a spinal tap or myelogram, may cause irritation of brain stem structures and activate sternocleidomastoid TrPs.[21] These TrPs may then persist and cause chronic headache for weeks, months or years, which, regardless of duration, can be relieved by inactivating the responsible myofascial TrPs.

Chronic Infection

Any regional focus of chronic infection, such as sinusitis or a dental abscess, should be identified and eliminated. Herpes simplex (oral) recurrent infection may be a stubborn perpetuator of TrPs in the neck and masticatory muscles.

8. PATIENT EXAMINATION

The examiner should assess head and neck posture (see Chapter 5, Section C) and range of motion. The patient with headache primarily due to active sternocleidomastoid TrPs has minimal restriction of the active range of head and neck motion. Active flexion may be slightly restricted (lacking about one finger breadth between the chin and the sternum). With sufficiently painful TrPs, active rotation is reduced about 10° to the opposite side. The contracting sternocleidomastoid apparently becomes inhibited reflexly by the TrPs.

When examining the standing patient with active sternocleidomastoid TrPs, one may observe a discrepancy in the length of the lower limbs. If the discrepancy is less than 6 mm (0.25 in), the shoulder opposite to the short leg usually sags, whereas in a patient with 1.2 cm (0.5 in) or more of leg-length disparity, the shoulder is more likely to droop on the same side as the short leg.

Signs of autonomic concomitants may be evident in the pain reference zones, as noted in Section 1. The patient with dizziness and disequilibrium due to TrPs in the clavicular division has neither a Romberg's sign nor nystagmus. With this type of myofascial disequilibrium, the patient cannot walk in a straight line toward a point across the room where he or she fixes the gaze. The path veers to one side, usually to the side of active TrPs in the clavicular division.

One man, wearing a stereophonic headset, was aware of markedly decreased hearing in the right ear, on the same side as the active sternocleidomastoid TrPs. He found that turning the face fully to the right, and then dipping the chin to the shoulder (actively stretching the involved sternocleidomastoid muscle on the side of his impaired hearing), restored his hearing to normal. This hearing loss apparently was due to TrP-induced reflex disturbance of tensor tympani muscle tension on the same side.

It is readily demonstrated that when objects of equal weight are placed in the hands of a patient with unilateral clavicular division TrPs that are sufficiently active, the object held on the affected side is perceived as lighter.[28] A difference in weight perception may not be apparent when TrP involvement of the sternocleidomastoid muscles is bilateral, as is often the case.

Central TrPs (CTrPs) of the sternal division, the TrPs responsible for "sore throat" (referred pharyngeal pain during swallowing) show a positive Sternocleidomastoid Compression Test. To perform this test, the sternocleidomastoid muscle is held firmly in a pincer grasp, as for examination, and the tender region immobilized by steadily compressing the belly of the muscle while the patient swallows.[7] Superficial pressure also may be effectively applied over the muscle by picking up the largest fold of skin possible overlying the central part of the muscle and squeezing the skin very firmly while the patient swallows. If TrPs are responsible for the throat pain, and if the muscle or skin is held tightly enough, swallowing usually becomes pain free.

The patient may cough in response to palpation of a TrP near the sternal attachment of the muscle. The complaint of a persistent dry, tickling cough should alert one to examine the patient in the region of both sternal attachments for this "cough" TrP.

9. TRIGGER POINT EXAMINATION (Fig. 7.4)

Gerwin, *et al.*[24] found that the most reliable examination criteria for making the diagnosis of TrPs were the identification of a taut band by palpation, the presence of spot tenderness in the band, the presence of referred pain, and reproduction of the patient's symptomatic pain. Identification of a local twitch response (LTR) by palpation was unreliable in some muscles. The sternocleidomastoid muscle was not tested, but is likely one of the more reliable muscles for this examination. An LTR is a valuable objective confirmatory finding when present.

For examination of the sternocleidomastoid muscle, the patient may be seated (Fig. 7.4A), or supine (Fig. 7.4B). The muscle is slackened somewhat by tilting the patient's head so as to bring the ear toward the shoulder on the symptomatic side (Fig. 7.4B) and, if necessary, by turning the face slightly away from the muscle to be examined. The muscle is encircled between the thumb and fingers, separating it from the underlying structures in the neck (as in Fig. 7.6C). The digits first encircle the entire muscle near its midbelly and then examine separately the deep and superficial divisions for palpable bands,[39] deep tenderness, and LTRs. Snapping a band between the fingers at the TrP regularly produces a visible twitch response, which may be seen as a slight jerk of the head. The TrPs may lie close to the upper or lower attachments, or at the midlevel of either division. Both divisions must be examined thoroughly. Attachment TrPs at the proximal and distal ends of this muscle near its musculotendinous junctions may be more effectively examined using flat palpation.

A prickling sensation in the face, over the mandible, which is the characteristic referred response of TrPs in the overlying platysma muscle, may inadvertently be triggered while palpating the sternocleidomastoid muscle *(see* Fig. 13.1). This may startle and concern the patient, especially if this unexpected sensation is not explained.

10. ENTRAPMENT

When the spinal accessory nerve (cranial nerve XI) penetrates the sternocleidomastoid muscle en route to the trapezius muscle, myogenic torticollis due to contracture of the sternocleidomastoid muscle can cause paresis of the trapezius muscle on the same side.[49]

11. DIFFERENTIAL DIAGNOSIS

The signs and symptoms caused by sternocleidomastoid TrPs confusingly mimic

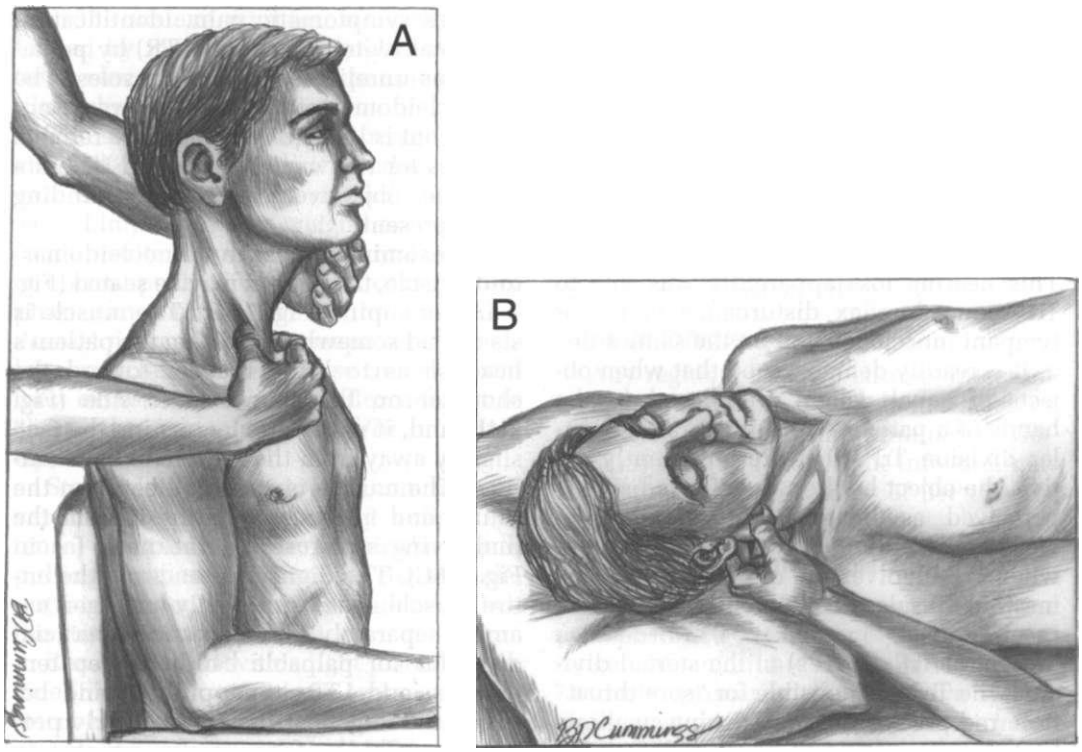


Figure 7.4. Examination of the sternocleidomastoid muscle is most effective using pincer palpation for both divisions and may be done with the patient seated or supine, A, examination of the lower end of the sternal division, with the patient seated. B, examination of the deeper clavicular division, with the patient supine and the head tilted toward the same side to slacken the muscle and permit the examiner's fingers to reach between it and underlying structures.

many kinds of headache, dizziness caused by vestibular dysfunction, and atypical facial neuralgia. The effects of these TrPs must be distinguished from Meniere's disease, tic douloureux, and congenital as well as spasmodic torticollis.

The head pain referred from sternocleidomastoid TrPs is readily mistaken for vascular headache or atypical facial neuralgia.[69] The pain from sternocleidomastoid TrPs can mimic true trigeminal neuralgia in distribution, and can mimic the arthritic pain of the sternoclavicular joint.[55] In a study of cervicogenic headache,[34] 91% of the 11 patients had a sternocleidomastoid TrP that contributed significantly to their pain.

Unlike Meniere's disease, symptoms and signs arising from myofascial TrPs in the clavicular division are rarely associated with unilateral deafness. The patient shows a normal calorimetric test and a negative Romberg's sign, the pupils are normal, there is no nystagmus, and no neurological deficit. Nystagmus and a positive Romberg's sign should alert one to a possible neurological lesion. Consciousness is unimpaired. These features distinguish the myofascial syndromes from more serious conditions like tic douloureux, Meniere's disease, cerebellopontine tumors, intracranial vascular lesions, inflammation of the labyrinth, hemorrhage into the pons, and petit mal epilepsy. The symptom of vertigo usually implies neurological disease and causes the sensation of the patient's spinning, or of the environment revolving around the patient.[18] Vertigo should be distinguished from postural dizziness; the latter is a nonspecific feeling of disorienta-

tion, as some patients say, a "swimming in the head." The patient's imbalance due to myofascial TrPs may mimic ataxia.

Dizziness due to vestibular disease is identified by nystagmus and other tests of vestibular function. The nonvestibular sources of dizziness include ear wax that touches the tympanic membrane, stenosis of the internal carotid artery, which may be detected by listening for a bruit over the bifurcation of the carotid artery or higher in the neck, hypertension, intracranial aneurysm or tumor, or a subclavian steal syndrome with reverse vertebral artery flow. Dizziness has been reported as an early sign of multiple sclerosis in children,[42] as a side effect of quinine,[73] as the result of postural hypotension due to excessive dosage of antihypertensive medication,[75] or due to adrenocortical insufficiency with failure of the orthostatic reflex response. The patient's blood pressure should be taken supine, sitting, and standing.

The facial grimace of *tic* douloureux clearly distinguishes this neurological disease from atypical facial neuralgia and from pain due to TrPs in the sternal division of the sternocleidomastoid.[69]

When autonomic symptoms are due to myofascial TrPs in the sternal division, the absence of miosis and enophthalmus, and the presence of a ciliospinal reflex rule out a Horner's syndrome. The eye symptoms must be distinguished also from paralysis of the extraocular muscles and from conversion hysteria.

The symptoms of "stiff neck"[41,61,67] due to myofascial TrPs, which develop in otherwise normal muscles during or after childhood, are easily distinguished from congenital torticollis, which is characterized by fibrosis and structural shortening of one sternocleidomastoid muscle from infancy.[33,47] Spasmodic or paroxysmal torticollis (wry neck) is a clonic or tonic contraction of cervical muscles due to organic disease or dysfunction of the nervous system and not to conversion hysteria as evidenced by changes in brain-stem auditory-evoked potentials.[19] In one study the abnormality of somatosensory evoked potentials suggested a lesion of the basal ganglia or their connections with the supplementary motor area.[44] Biopsies from both heads of 9 sternocleidomastoid muscles of children with idiopathic torticollis showed much more severe denervation and necrosis of the clavicular head of the muscle than of the sternal head.[56] The authors suggested that the chronic spasm of the sternal head, which the accessory nerve penetrated before reaching the clavicular head, caused severe compromise of both the nerve and vascular supply. This resulted in the focal myopathy and necrosis of the clavicular head.

Symptomatically, idiopathic torticollis merges into torsion dystonia of the neck, and the muscles involved become hypertrophied. Spasmodic torticollis may be inhibited by exerting slight pressure against the jaw on the side to which the head is rotated. Dystonic movement ceases during sleep. Clonic jerks are particularly common in hysterical patients.[6] Spasmodic torticollis in infancy[58] and spasmus nutans[30] are described as self-limited conditions of infancy or childhood, characterized by a head tilt that is strongly suggestive of sternocleidomastoid TrP dysfunction and may include a significant myofascial component.

Related Trigger Points

When TrPs are present in one sternocleidomastoid muscle, they usually are found also in the opposite muscle. The scalene muscles also tend to develop TrPs, especially if the sternocleidomastoid has been affected for a period of time, usually several weeks. If the neck motion (rotation) is "stiff," TrPs may be present in the levator scapulae, trapezius, splenius cervicis, and other posterior neck muscles.[61]

An anomalous sternalis muscle may develop satellite TrPs as a result of primary TrPs in the lower end of the sternal division. Such satellite TrPs in the sternalis refer pain deep under the sternum and across the upper pectoral region to the arm on the same side (see Chapter 44). The pectoral muscles, in turn, may develop another set of satellite TrPs. The masseter, temporalis, orbicularis oculi and frontalis muscles tend to develop satellite TrPs, since they also lie within pain reference zones of the sternocleidomastoid muscle TrPs. These

muscles, as well as a painful temporomandibular joint, may not respond to treatment and "settle down" until the key sternocleidomastoid is effectively released. Hong[31] demonstrated that sternocleidomastoid TrPs can act as key TrPs to satellite TrPs in the temporalis, masseter, and digastric muscles. He found that inactivation of a key TrP inactivated its satellite TrP without further treatment of it.

12. TRIGGER POINT RELEASE (Fig. 7.5)

For lasting relief, mechanical perpetuating factors such as forward-head posture and round-shouldered posture must be corrected (see Chapters **5** and **41,** Section C).

To release sternocleidomastoid trigger points (TrPs) using a **spray-and-stretch** approach, the patient first sits comfortably and relaxed in a low-backed firm-seated armchair with the fingers of each hand hooked under the chair seat or under the thigh. A small hemipelvis, if present, should be corrected by leveling the patient's pelvis with an ischial lift before starting treatment (see Fig. **48.10D).** When multiple neck muscles harbor TrPs, spray-and-stretch techniques are applied first to release TrPs in the trapezius and levator scapulae muscles (see Figs. **6.9-6.11** and **19.5)** to ensure sufficient range of head and neck rotation for a full passive stretch of the sternal division of the sternocleidomastoid. It may be necessary to alternate treatment between the clavicular division of the sternocleidomastoid and the scalene muscles in order to obtain the full range of motion of both muscles. To help the patient relax the neck muscles, the patient's head may be cradled in the operator's hand, with the head resting against the operator's arm or chest. The patient is encouraged to rest the weight of the head on the operator and to use full slow diaphragmatic breathing, which also assists relaxation.

The **clavicular division** of the muscle is gradually released by guiding the head posteriorly and away from the involved side, and rotating it so that the face turns away from the involved side (Fig. **7.5A),** as also illustrated by Zohn.[78] Immediately preceding and during this movement, the vapocoolant spray or icing is applied in slow parallel sweeps from the muscle's lower attachment on the clavicle, upward to its upper attachment on the mastoid process and over the occiput. The sweeps are continued behind the ear and across the forehead to cover the pain reference zones (shown in Fig. **7.5A**).[68] The operator takes up slack in the muscle as it develops. Release of the muscle is enhanced by including postisometric relaxation coordinated with diaphragmatic respiration so that relaxation occurs with exhalation following the application of intermittent cold (see Section **14).**

For referred pain deep in the ear, not relieved otherwise, one sweep of vapocoolant should cross and enter the auditory canal, after warning the patient first. This produces a very startling experience and should not be done accidentally or without warning. In **1902,** Politzer[51] recommended brief spraying of the tympanic membrane with ethyl chloride for relief of pain due to acute otitis media.

Tightness of the **sternal division** of the muscle is released by smoothly coordinating the application of vapocoolant upward over the neck (Fig. **7.5B)** with gentle rotation of the head toward the same side. As rotation is completed, the chin is slowly tipped downward toward the acromion (Fig. **7.5C)** while the stream of vapocoolant continues upward over the head and behind the ear. This head motion moves the occiput attachment of the muscle as far as possible from its sternal attachment. The movement elevates the occiput and mastoid process to secure maximal stretch on the muscle (Fig. **7.5C).** While thus stretching the sternal division, sweeps of the spray are applied upward from the sternal attachment around the neck, covering the muscle to the mastoid region and occiput. Each rotation is carefully coordinated with a sweep of the spray to stay ahead of the rotation movement and assure access to the skin on the neck as the head rotates. Additional sweeps of vapocoolant cover the cheek and forehead pain reference zones. The clinician should be sure to keep spray out of the patient's eye by having the patient close the eye tightly, and by directing the spray away from the eye. Extra protection can be provided by placing a gauze

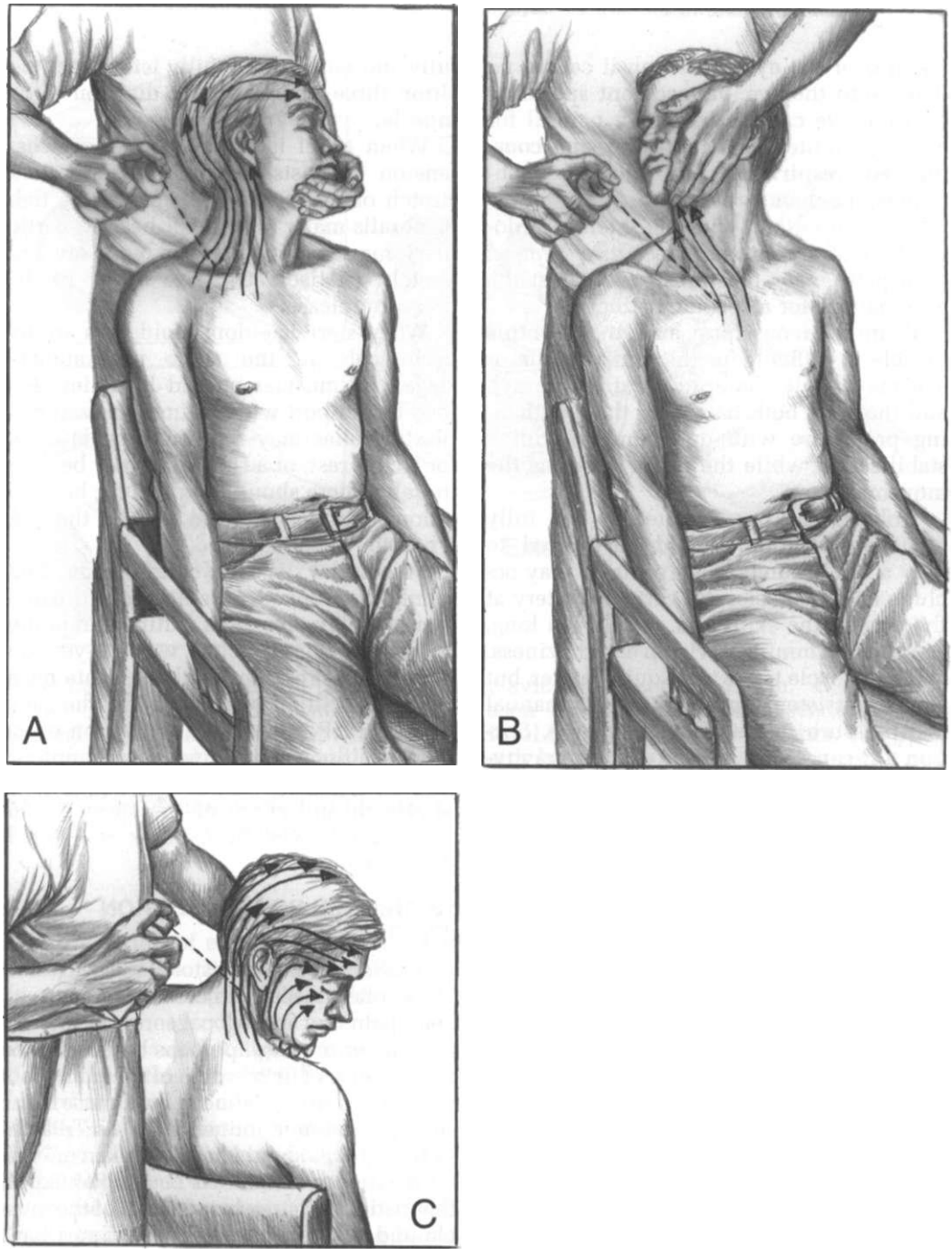


Figure 7.5. Release positions and spray patterns *(arrows)* for the two divisions of the right sternocleidomastoid muscle. The direction for application of vapocoolant or icing is shown by the arrows. **A,** stretch position and spray pattern for the clavicular division. **B,** start of stretch and spray for the release of the sternal division. **C,** the second phase of release of the right sternal division completes coverage of the spray pattern in this position of full release with the head turned 90% and the face tilted downward. The patient's eye can be protected by covering it with a gauze pad or by having the patient close the eyes. For patients with asthma or other respiratory problems, inhalation of the spray vapors should be avoided by applying the spray only while the patient breathes out.

patch over the eye. Although it causes no damage to the eye, vapocoolant splashing into the eye can be extremely painful for several minutes. **Contract-relax and coordinated respiration** are valuable techniques to enhance release.

It is important with the sternocleidomastoid to spray over the *entire* referred pain pattern for that muscle, not just the pain pattern for a specific patient.

If simultaneous spray and stretch of this muscle is difficult for the clinician, he or she may apply the spray first (prespray), and then use both hands for the lengthening procedure with one hand providing stabilization while the other performs the movement.

Holding the head rotated in the fully stretched position should be limited to only a few seconds. This position may occlude an atherosclerotic vertebral artery at the base of the skull and, if held too long, can cause dimming of vision and dizziness.

This muscle tends to be quite tender, but gentle persistent application of bimanual TrP pressure release *(see* Chapter 3, Section 12) can also be effective. The gravity-assisted release recommended by Lewit is described in Section 14 as part of the home program and can be demonstrated to the patient as part of the initial treatment.

A TrP release technique is always applied to both the right and left sternocleidomastoid muscles. The increased range of head rotation achieved by releasing the sternocleidomastoid on one side is likely to induce reactive cramping of the suddenly shortened contralateral muscle. This can cause afterpain and dizziness, due to activation of latent contralateral TrPs by this unaccustomed shortening. Also, a few sweeps of the spray are applied downward over the sternal and pectoral areas. If this is not done, palpation or treatment of very irritable TrPs in the sternocleidomastoid muscles may activate preexisting latent TrPs in the sternalis and pectoralis muscles and, within minutes or hours, produce an attack of chest pain.

After application of spray and stretch, moist heat is applied at once over treated muscles, followed in a few minutes by the most important step, active movement of the head slowly back and forth through the fully shortened to the fully lengthened position three times for each division of the muscle.

When relief is not complete, or when tension persists following spray and stretch of the sternocleidomastoid, a tight pectoralis major (clavicular head in particular) may require release by spray and stretch, postisometric relaxation, or TrP pressure release.

When sternocleidomastoid TrPs are hyperirritable in the acute posttraumatic stage, the muscles should be relieved of load by support without immobilization; a plastic collar may be worn upside down for a chin rest, or a soft collar may be worn loosely. There should be room for head rotation, with space at the side for the chin when the head is turned.

Myofascial TrPs in children are commonly overlooked unless a skilled examiner is looking for them. Aftimos[1] reported 5 subjects, one of which was a 7-year-old child, who presented with an **acute onset** of painful tilting of the head to the same side as a TrP in the sternal division of the sternocleidomastoid muscle. Symptoms were completely relieved by application of stretch and spray of the sternocleidomastoid followed by a home self-stretch program.

13. TRIGGER POINT INJECTION (Fig. 7.6)

The sternocleidomastoid trigger points (TrPs) often react to injection therapy with head pain and more local soreness than do most other muscles, perhaps because of the multiplicity of TrPs, some of which remain active in spite of treatment, or because of the strong autonomic influences of its TrPs. Injection of TrPs should be undertaken only after maximum benefit has been obtained for that patient by stretch and spray of the muscle and other TrP release techniques have been tried. If the patient must take a trip, or is committed to activity immediately afterward, then it is wise to stretch and spray the muscle and to defer its injection. The muscle on only ONE SIDE is injected during one visit. Any TrPs on the other side should be injected only after any reaction to the previous injection has subsided and if the injected TrP sites showed substantial improvement.

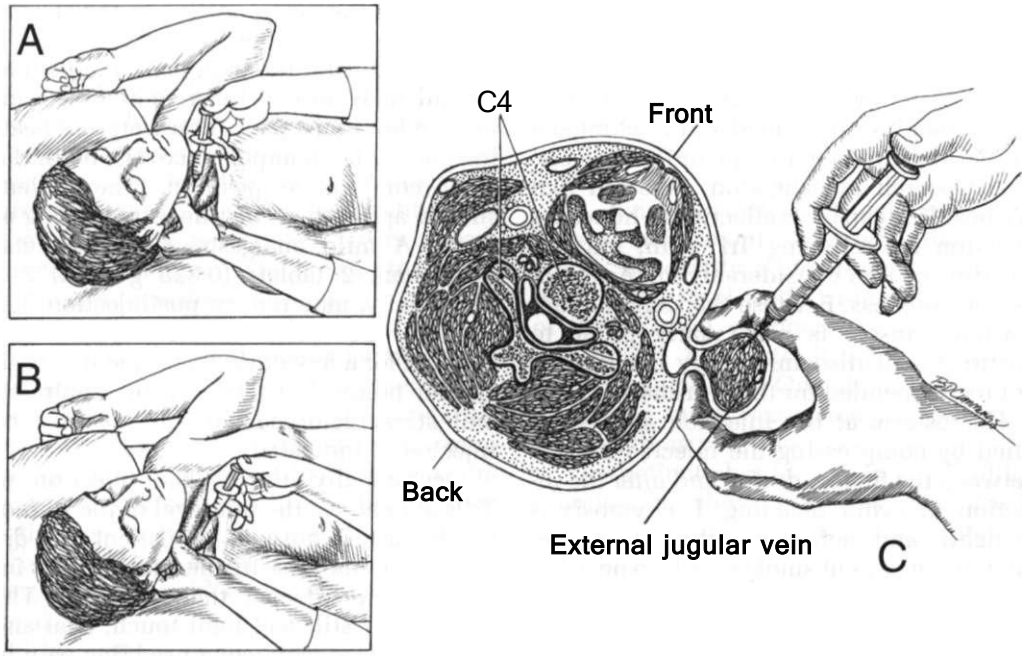


Figure 7.6. Injection of central trigger points in the right sternocleidomastoid into the midregion of the muscle belly, with the patient supine, head tilted toward the same side as the affected muscle and the face turned away. A, midportion of the sternal (superficial) division. B, midportion of the clavicular (deep) division. C, injection seen in anatomical cross section, at the level of the 4th cervical vertebra. The operator has grasped both divisions using pincer palpation and pulled the muscle away from underlying neurovascular structures.

For injection of either division, the patient lies supine (Fig. 7.6A and B). The muscle is slackened by tilting the ear toward the shoulder on the affected side with the face turned slightly upward and to the opposite side; the pillow is placed under the shoulder of the affected side to lift the chest and further slacken the muscle. To inject the deeper clavicular division (Fig. 7.6B), the entire muscle should be encompassed by the examiner's thumb and fingers and lifted off the underlying blood vessels, nerves and scalene muscles (Fig. 7.6C).

The course of the external jugular vein is outlined by blocking the vein with a finger just above the clavicle. When the midlevel of the muscle is being injected, the vein can be shifted either laterally or medially by the finger to avoid penetrating it. The vein is illustrated in Figure 20.8A.

A 22- to 27-gauge needle (preferably 25-gauge), that is 3.8 cm (1.5 in) long, is selected. Penetration of the needle into the TrP at the precise point of maximal tenderness is confirmed by an LTR and/or by local pain with projection of the expected pattern of referred pain. Through a single skin puncture, multiple needling with continuous injection of 1 or 2 ml of 0.5% procaine solution can be carried out until pain and LTRs are no longer elicited by the probing needle.[38,64] Hong[31] described a similar but more sophisticated technique of "fast in, fast out" that is very effective and likely reduces muscle trauma due to injection. Then, with the needle held just under the skin, the muscle can be palpated for any residual firm bands that still harbor TrPs which are still tender and capable of LTRs. If such TrPs are present, further probing with the needle should inactivate

them. Usually, TrPs in the superficial, more medial sternal division are inactivated first (Fig. 7.6A), then the TrPs in the deeper and more posterior clavicular division (Fig. 7.6B). Rachlin[52] described and illustrated a similar injection technique for this muscle.

A research investigation[32] showed that dry needling can be as effective as lidocaine injection for relieving TrP pain, but dry needling caused considerably more postinjection soreness. Because the sternocleidomastoid muscle is particularly prone to posttreatment discomfort, dry needling is not recommended for this muscle.

Hemostasis at the injection site is applied by compressing the injected muscle between the fingers *during* and *after* the injection to avoid bleeding. Ecchymosis is unsightly and increases postinjection soreness. If the patient smokes, or is exposed to smoke, the diet should be supplemented with ascorbic acid, three daily doses of 500 mg each, for at least 3 days before injection. He or she should be very strongly encouraged to avoid exposure to smoke.

After injection, a hot pack is applied over the muscle at once, while the patient lies on the treated side with a pillow between the head and shoulder to lift the chin and place the sternocleidomastoid muscle in a neutral position *(see* Fig. 7.7C). After a few minutes of moist heat, the muscle is checked again for tenderness and LTRs. The muscle is then stretched and sprayed essentially as in Figure 7.5. If spot tenderness in the muscle has not been eliminated, TrP pressure release with the muscle on a partial stretch is employed to inactivate residual TrPs while some local procaine effect remains.

The patient is taught how to use the uninjected contralateral sternocleidomastoid muscle when lifting the head to rise from the supine position. Turning the face to the ipsilateral side relieves the recently injected muscle of possible strain until the local tissue soreness from needling has disappeared, which may require several days.

After the treatment, a soft cervical collar, worn loosely, may be helpful to support the head and inhibit sudden rotary and side motions while the patient is riding in a car as a passenger. Otherwise, a pillow may be placed between the patient's head and the car window to support the head from the side and to rest the sternocleidomastoid muscle.

At home after the treatment, the patient should relax in bed for a short time and, using a hot moist pack (or a wetproof heating pad with a dampened cover), lie in the most comfortable position. The patient should apply the moist heat on retiring at night. A mild analgesic, such as acetaminophen, 2 tablets (0.325 g each) 2-3 times daily, may reduce postinjection discomfort. Strenuous activity should be avoided for a few days. Subsequently, with similar precautions, TrPs in the contralateral sternocleidomastoid muscle may be injected, if indicated.

Occasionally, during the injection of TrPs at or above the midlevel of the sternocleidomastoid muscle, the patient may describe a numbness in the face, which involves tissue deeper than the skin. The patient can still feel light touch, heat and cold, and also may feel a prickling pain in the angle of the jaw, cheek, and pinna of the ear. These symptoms may be due to procaine infiltration of the posterior branch of the greater auricular nerve, which loops around and traverses the face of the sternocleidomastoid muscle.[2] If this nerve is blocked by 0.5% procaine solution, the sensation of numbness disappears in 15 or 20 min, as the local anesthetic effect dissipates.

It is rarely necessary to infiltrate the attachment TrP at the inferior end of the clavicular division of the muscle since this is in the musculotendinous junction region and the tenderness is most likely enthesitis secondary to midbelly TrPs, which are the ones that must be inactivated. If this part of the muscle is injected, it must be kept in mind that this location overlies the apex of the lung and, therefore, must be injected with care to avoid penetrating the lung and causing a pneumothorax.

14. CORRECTIVE ACTIONS (Fig. 7.7)

Avoidance of Postural Strain

Excessive forward-head posture needs to be corrected (see Chapters 5 and 41, Section C). The head needs to be erect, balanced, and *not* projected forward during sitting and standing. To stand correctly, the patient should transfer the body weight

from the heels toward the balls of the feet, shifting forward from the ankles. The arms and shoulders should hang loosely.

Revision of the patient's chair may be required to eliminate a headrest that pushes the head forward. A lumbar pillow is often essential to restore the normal lordosis that promotes erect posture. Nearsightedness should be corrected, since it favors a head-forward posture, which shortens the sternocleidomastoid muscles.

A person with sternocleidomastoid TrPs should not sit with the body facing in one direction while looking in another direction for a prolonged period; this rotation leads to neck muscle problems. For example, when one needs to direct the eyes toward another person for extended conversation or toward a television set for a prolonged time, either the chair or the person's body should be turned, *not* just the head.

The patient with sternocleidomastoid TrPs should be taught, when lifting the head from the supine position, to turn it and unload the affected muscle by using the opposite, less involved sternocleidomastoid, assisting it by lifting some of the weight of the head with the hand. Similarly, the patient may wish to rotate the head slightly when doing a Sit-back or Sit-up Exercise. When turning over in bed at night, the patient should roll the head on the pillow, ***not*** lift the head. With bilateral involvement, the patient may need to slide out of bed from the prone position, thus avoiding overload of these anterior neck muscles on either side.

A small pillow behind the neck produces moderate (normal) cervical lordosis, and a side pillow limits head rotation and sidebending at night. The patient should tuck the corner of the side pillow between the shoulder and chin (Fig. **7.7A** and **C),** NOT under the shoulder (Fig. **7.7B** and **D).** The latter arrangement causes prolonged shortening of the anterior neck muscles on the underside during sleep.

The muscles supporting the head are abused when the bed lamp is placed at one side of the bed (Fig. 7.3B). The light should be located directly overhead, on the headboard (Fig. 7.3A), on the wall, or suspended from the ceiling.

The patient should hold the telephone receiver in one hand, not between the head and shoulder, and at intervals, use the opposite hand to hold the receiver (not changing ears); this varies the tilt of the head. If a patient does much telephoning, use of an executive (speaker) telephone or use of a headset is recommended instead of a handset.

A patient with sternocleidomastoid TrPs should avoid swimming the crawl stroke, especially if breathing is done by turning the head to the side opposite that of the affected sternocleidomastoid muscle, which contracts it strongly in the shortened position. One should also limit prolonged neck extension in overhead work such as painting.

The patient should ***not*** do head-rolling exercises as these exercises can readily over-stretch affected muscles, catching them off guard.

Compensation for Body Asymmetry

An LLLI or a small hemipelvis that tilts the shoulder-girdle axis should be corrected by suitable lifts (see Chapter 4 and Figs. 48.9 and 48.10).

Appropriate Pillow Support for Sleeping

At night, the sternocleidomastoid muscle is especially vulnerable to the jiggling and vibratory movements caused by a foam pillow, a rubber pillow, or other springy pillow, often prescribed to eliminate feather allergens. A number of nonallergenic plastic fillers are now available. Symptoms may recur within a day or two with reactivation of sternocleidomastoid TrPs, if the patient again sleeps on a bouncy foam pillow. The comfortable and protective home pillow should go along on trips, whenever it may be needed.

Avoidance of Constriction

Pressure on the sternocleidomastoid muscles and activation of TrPs may be caused by tightness of the shirt collar. The examiner's finger should fit comfortably inside the collar, not only when the patient is looking straight ahead, but also when the head is turned, which increases the diameter of the neck inside the collar. Cinching the necktie too tightly should be avoided.

Elimination of Chronic Infection

Sources of chronic infection or infestation, as listed in Chapter 4, should be identified and eliminated.

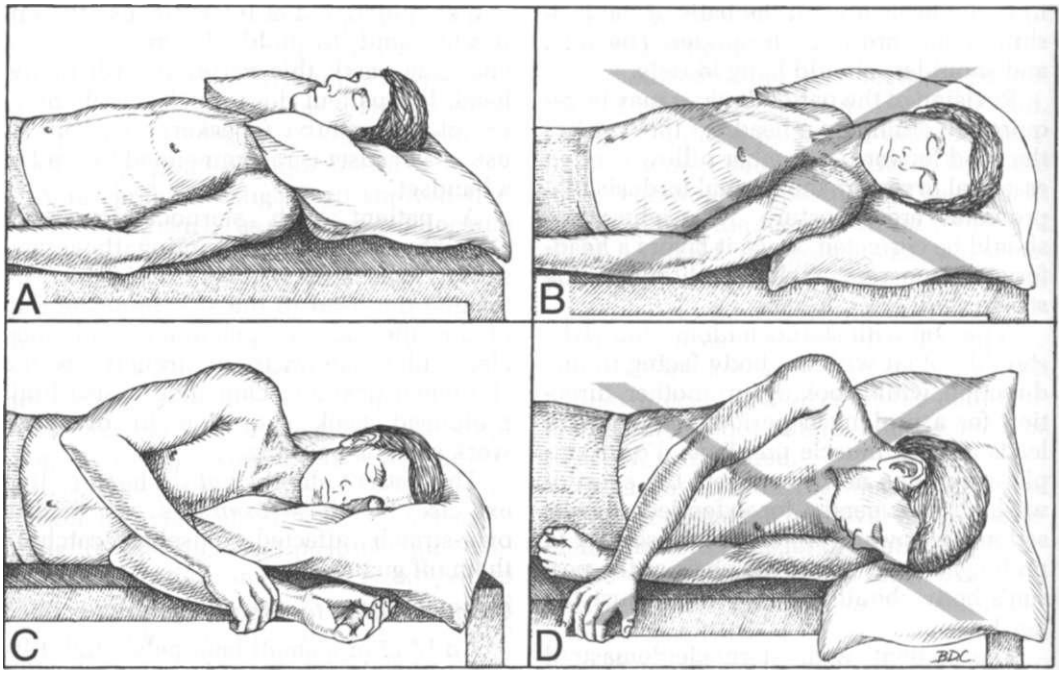


Figure 7.7. Positioning of the pillow to promote relief of the sternocleidomastoid myofascial syndrome, illustrating correct and wrong *(red* **Xs)** positions. **A,** *correct* position, patient supine with the corners of the pillow tucked between the chin and shoulders. **B,** *wrong* position, patient supine, with the pillow *under* the shoulders. **C,** *correct* position, patient side-lying, with the pillow between the head and shoulder. **D,** *wrong* position, patient side-lying, with the chin in the hollow of the shoulder and the pillow under the shoulder, placing the sternocleidomastoid and scalene muscles in a shortened position.

Exercise

For a home stretch exercise, the patient does the Sidebending Neck Exercise, by bringing the ear to the shoulder first on one side, then on the other, while lying supine *(see* Fig. 20.14). When performing the Indoorway Stretch Exercise for pectoralis TrPs, the patient who also has sternocleidomastoid TrPs must not project the head forward. Looking down shortens the sternocleidomastoid muscles, aggravating their TrP activity.

The patient should breathe with the chest and diaphragm correctly coordinated, not in a paradoxical manner *(see* Fig. 20.15 and Chapter 45). The habit of correct diaphragmatic breathing should be established by an exercise program, if the patient has this problem.

Lewit[40] illustrated and described a gravity-induced postisometric relaxation technique suitable for a home program for release of TrPs in the clavicular division of the sternocleidomastoid muscle. The supine patient rests the head over the edge of the table and turns the face to one side, chin supported by the edge of the table acting as a fulcrum. The patient looks up with the eyes only, and takes in a slow, deep breath using diaphragmatic (abdominal) breathing. This effort lightly activates the uppermost sternocleidomastoid muscle. During slow exhalation, the patient looks down and relaxes, allowing the head to drop slightly, elongating the sternocleidomastoid muscle with each breath.

SUPPLEMENTAL REFERENCES, CASE REPORTS

The total management of patients with sternocleidomastoid TrPs has been detailed in case reports.[63,69,71,74]

REFERENCES

1. Aftimos S: Myofascial pain in children. *N Z Med J 102(874):440-441,* 1989.
2. Alberti PW: The greater auricular nerve. *Arch Otolaryngol* 76:422-424, 1962.
3. Baker B: The muscle trigger: evidence of overload injury. *J Neurol Orthop Med Surg* 7:35- 43, 1986.
4. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 426, 466, 467).
5. Bates T: Myofascial pain. Chapter 14. In: *Ambulatory Pediatrics II. Personal Health Care of Children in the Office.* Edited by Green M, Haggerty RJ. W.B. Saunders, Philadelphia, 1977 (pp. 147-148).
6. Brain WR, Walton JN: *Brain's Diseases of the Nervous System.* Ed. 7. Oxford University Press, New York, 1969 (pp. 517, 541-543).
7. Brody SI: Sore throat of myofascial origin. *Milit Med* 129:9-19, 1964.
8. BroerMR, Houtz SJ: *Patterns of Muscular Activity in Selected Sports Skill.* Charles C. Thomas, Springfield, 111., 1967.
9. Brando K, Dahl HA, Teig E, et al.: The human posterior cricoarytenoid (PCA) muscle and diaphragm. *Acta Otolaryngol (Stockh) 102:474-481,* 1986.
10. Brudny J, Grynbaum BB, Korein J: Spasmodic torticollis: treatment of feedback display of the EMG. *Arch Phys Med Rehabil* 55:403-408, 1974.
11. Campbell EM: Accessory muscles, Chapter 9. In: *The Respiratory Muscles, Mechanics and Neutral Control.* Ed. 2. Edited by Campbell EM, Agostoni E, Davis JN. W.B. Saunders, 1970 (pp. 183-186).
12. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 457, 1189, 1205).
13. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 576).
14. *Ibid.* (Figs. 578, 579).
15. *Ibid.* (Figs. 583-585).
16. Cohen LA: Body orientation and motor coordination in animals with impaired neck sensation. *Fed Proc* 18:28, 1959.
17. Cohen LA: Role of eye and neck proprioceptive mechanisms in body orientation and motor coordination. *) Neurophysiol* 24:1-11, 1961.
18. Denny-Brown DE: Neurologic aspects of vertigo. *N Engl J Med* 241:144, 1949.
19. Drake ME Jr: Brain-stem auditory-evoked potentials in spasmodic torticollis [Abstract], *Arch Neurol 45{2):174-175,* 1988.
20. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 479).
21. Dunteman E, Turner S, Swarm R: Pseudo-spinal headache. *Reg Anesth* 21(4j:358-360, 1996.
22. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (p. 236).
23. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 12-19, 23-31).
24. Gerwin RD, Shannon S, Hong CZ, et al.: Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
25. Good MG: Senile vertigo caused by curable cervical myopathy, *f Am Geriatr Soc* 5:662- 667, 1957.
26. Goss CM: *Gray's Anatomy.* Ed. 29. Lea & Febiger, Philadelphia, 1973 (pp. 944, 945).
27. Gutstein M: Diagnosis and treatment of muscular rheumatism. *Br J Phys Med* 1:302-321,1938 (p. 311).
28. Halpern L: Biological significance of head posture in unilateral disequilibrium. *Arch Neurol Psychiatr* 72:160-180, 1954 (Case 3).
29. Hayward R: Observations on the innervation of the sternomastoid muscle. / *Neurol Neurosurg Psychiatry 49(8)351-953,* 1986.
30. Hoefnagel D, Biery B: Spasmus nutans. *Dev Med Child Neurol* 10:32-35, 1968.
31. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. / *MusculoskePain 2(l):29-59,* 1994.
32. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Rehabil* 73:256-263, 1994.
33. Horton CE, Crawford HH, Adamson JE, ef *al.:* Torticollis, *South Med J* 60:953-958, 1967.
34. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 9:157-164, 1989.
35. Jaeger B: Differential diagnosis and management of craniofacial pain. Chapter 11. In: *Endodontics.* Ed. 4. Edited by Ingle JI, Bakland LK. Williams & Wilkins, Baltimore, 1994 (pp. 550-607).
36. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 80, 81, 344).
37. Kellgren JH: Deep pain sensibility. *Lancet* 1:943-949, 1949.
38. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (pp. 97, 104, 105).
39. Lange M: *Die Muskelharten (Myogelosen).* J.F. Lehmanns, Munchen, 1931 (pp. 88, 89, Fig. 30).
40. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (p. 197).
41. Llewellyn LJ, Jones AB: *Fibrositis.* Rebman, New York, 1915 (pp. 201, 203).
42. Long C, II: Myofascial pain syndromes: Part II—Syndromes of the head, neck, and shoulder girdle. *Henry Ford Hasp Med Bull* 4:22-28, 1956 (pp. 23).
43. Marbach JJ: Arthritis of the temporomandibular joints. *Am Fam Physician* 19:131-139,1979 (Fig. 9D).
44. Mazzini L, Zaccala M, Balzarini C: Abnormalities of somatosensory evoked potentials in spasmodic torticollis. *Movement Disord* 9(4j:426-430, 1994.
45. McMinn RM, Hutchings RT, Pegington J, Abrahams P: *Color Atlas of Human Anatomy,* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 39).
46. *Ibid.* (pp. 41, 116).
47. Middleton DS: The pathology of congenital torticollis. *Br] Surg* 18:188-204, 1930.
48. Mikhail M, Rosen H: History and etiology of myofascial pain-dysfunction syndrome. / *Prosthet Dent* 44:438-444, 1980.
49. Motta A, Trainiti G: Paralysis of the trapezius associated with myogenic torticollis. *Ital] Orthop Traumatol* 3:207-213, 1977.
50. Netter FH: *Nervous System.* Volume 1 of *The CIBA Collection of Medical Illustrations.* CIBA Pharmaceutical Company, New Jersey, 1972 (pp. 42, 43).
51. Politzer A: *A Textbook of Diseases of the Ear.* Ed. 4. Lea Bros & Co., Philadelphia, 1902 (p. 642).

52. Rachlin ES: Injection of specific trigger points. Chapter 10. In: ***Myofascial Pain and Fibromyalgia.*** Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 ***(see*** p. 295).
53. Radziemski A, Kedzia A, Jakubowicz M: Number and localization of the muscle spindles in the human fetal sternocleidomastoid muscle. ***Folia Morphol (Warsz) 50(l-2):65-70,*** 1991.
54. Rasch PJ, Burke RK: ***Kinesiology and Applied Anatomy.*** Lea & Febiger, Philadelphia, 1967 (pp. 231, 233, 258).
55. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. ***Arch Phys Med Rehabil*** 62:111-114, 1981 (Tables 1 and 2).
56. Sarnat HB, Morrissy RT: Idiopathic torticollis: sternocleidomastoid myopathy and accessory neuropathy. ***Muscle Nerve*** 4:374-380, 1981.
57. Sharav Y, Tzukert A, Refaeli B: Muscle pain index in relation to pain, dysfunction, and dizziness associated with the myofascial pain-dysfunction syndrome. ***Oral Surg*** 46.742- 747, 1978.
58. Snyder CH: Paroxysmal torticollis in infancy. ***Am J Dis Child*** 217:458-460, 1969.
59. Spalteholz W: ***Handatlas der Anatomie des Menschen.*** Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 270).
60. Toldt C: ***An Atlas of Human Anatomy,*** translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 292).
61. Travell J: Rapid relief of acute "stiff neck" by ethyl chloride spray, ***f Am Med Worn Assoc*** 4:89-95,1949.
62. Travell J: Pain mechanisms in connective tissue. In: ***Connective Tissues, Transactions of the Second Conference, 1951.*** Josiah Macy, Jr. Foundation, New York, 1952 (pp. 86-125).
63. Travell J: Referred pain from skeletal muscle: pectoralis major syndrome of breast pain and soreness and sternomastoid syndrome of headache and dizziness. ***NY State J Med*** 55:331-339, 1955.
64. Travell J: Symposium on mechanism and management of pain syndromes. ***Proc Rudolf Virchow Med Soc*** 16:128-136, 1957 (pp. 4, 5, Figs. 2, 3).
65. Travell J: Temporomandibular joint pain referred from muscles of the head and neck. / ***Prosthet Dent*** 10:745-763, 1960.
66. Travell J: Mechanical headache. ***Headache*** 7:23-29, 1967.
67. Travell J: ***Office Hours: Day and Night.*** The World Publishing Company, New York, 1968 (p. 271).
68. ***Ibid.*** (pp. 293-294).
69. Travell J: Identification of myofascial trigger point syndromes: a case of atypical facial neuralgia. ***Arch Phys Med Rehabil*** 62:100-106, 1981.
70. Travell J, Bigelow NH: Role of somatic trigger areas in the patterns of hysteria. ***Psychosom Med*** 9:353-363, 1947.
71. Travell J, Rinzler SH: Pain syndromes of the chest muscles: Resemblance to effort angina and myocardial infarction, and relief by local block. ***Can Med Assoc J*** 59:333-338, 1948 (pp. 334, 335, Case 2).
72. Travell J, Rinzler SH: The myofascial genesis of pain. ***Postgrad Med*** 11:425-434, 1952.
73. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. ***JAOA*** 72:61-74, 1973 (p. 8, Figs. 20-23).
74. Weeks VD, Travell J: Postural vertigo due to trigger areas in the sternocleidomastoid muscle. / ***Pediatr*** 47:315-327, 1955.
75. Williams HL: The syndrome of physical or intrinsic allergy of the head: myalgia of the head (sinus headache). ***Proc Staff Meet Mayo Clinic*** 20:177-183, 1945.
76. Williams HL, Elkins, EC: Myalgia of the head. ***Arch Phys Ther*** 23:14-22, 1942.
77. Wyant GM: Chronic pain syndromes and their treatment. II. Trigger points. ***Can Anaesth Soc f 26:216-*** 219, 1979 (Patient 1, and Fig. la).
78. Zohn DA: ***Musculoskeletal Pain: Diagnosis and Physical Treatment,*** Ed. 2. Little, Brown & Company, Boston, 1988 (Figs. 9-2C, 12-1).

CHAPTER 8
Masseter Muscle

with contributions by
Bernadette Jaeger and Mary Maloney

HIGHLIGHTS: The masseter is the muscle most likely to be causing severely restricted jaw opening. Trigger points (TrPs) produce dysfunction, because they increase muscle tension, and they often produce pain. **REFERRED PAIN** from trigger points in the superficial layer of the masseter muscle may be projected to the eyebrow, maxilla, mandible anteriorly, and to the upper or lower molar teeth, which become hypersensitive to pressure and temperature change. In the deep layer of the muscle, TrPs can refer pain deep in the ear and to the region of the temporomandibular joint (TMJ). **ANATOMY:** attachments of the masseter are located, above, on the zygomatic arch and zygomatic process of the maxilla and, below, on the outer surface of the ramus and angle of the mandible. **FUNCTION** of the masseter (superficial fibers) is primarily to elevate the mandible, and for the deep posterior fibers to help retrude it. **SYMPTOMS** of active TrPs in this muscle are chiefly pain and occasionally marked restriction of opening of the jaws. *Unilateral tinnitus* may be a symptom of TrPs high in the *deep* portion of the muscle. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** result from gross trauma, the microtrauma of bruxism or chronic overwork, acute overload, poor posture, significant occlusal imbalance, and holding the mandible in other than a rest position for prolonged periods. **PATIENT EXAMINATION** may reveal restriction of mandibular opening to less than 40 mm, generally accepted to be the minimum normal range of opening for men and women. Normally, the jaws should admit a tier of at least two knuckles between the incisor teeth. **TRIGGER POINT EXAMINATION** is more effective if the jaws are partially open. The superficial anterior fibers are palpated by pincer palpation, but some of the most posterior fibers of the deep layer must be palpated by flat palpation against the mandible. **DIFFERENTIAL DIAGNOSIS** includes tinnitus of neurological origin and painful TMJ disorders. The latter are often seen together with masseter and other masticatory muscle TrPs and may be the precipitating factor. Pain caused by masseter TrPs is often a component of tension-type headache and may be seen with cervicogenic headache. Related TrPs are likely to develop in the ipsilateral temporalis and medial pterygoid muscles, and in the contralateral masseter muscle. Masseter TrP activity is often a satellite manifestation of sternocleidomastoid TrPs. **TRIGGER POINT RELEASE** may be accomplished by spray and stretch, by TrP pressure release, and by reciprocal inhibition to relax the muscle. Forceful stretching maneuvers are avoided, especially if there is any possibility of internal derangement of the TMJ (TM disc dysfunction). **TRIGGER POINT INJECTION** is most accurately performed using a pincer grasp, with one digit localizing the TrP from inside the mouth against the thumb outside the mouth. Extraoral injection of posterior (deep) fibers requires awareness of the location of the facial nerve. **CORRECTIVE ACTIONS** include learning and maintaining good body and head, neck and tongue posture, avoiding abusive oral habits such as exhaustive chewing, clenching and grinding of teeth, chewing gum, and biting ice or finger nails to name a few; the inactivation of related TrPs in muscles that refer pain to the face region, the regular use of self-stretch exercises, and possibly the elimination of premature tooth contacts if significant.

1. REFERRED PAIN
(Fig. 8.1)

Trigger points (TrPs) produce dysfunction (because they increase muscle tension) and they often produce pain. In addition to exhibiting distinctly different referred pain patterns, the superficial and deep layers of the masseter muscle also have a different angulation of fiber direction and therefore somewhat different functions.

Superficial Layer

Myofascial TrPs in the superficial layer of the masseter muscle refer pain mainly to the lower jaw, molar teeth and related gums, and to the maxilla.[45,79,81] When located in the anterior border and *superior* part of this layer, TrPs refer pain to the upper premolar[41] and molar teeth, adjacent gums, and maxilla.[45'79] The maxillary pain is often described by the patient as "sinusitis" (Fig 8.1A). When the TrPs are located just *below* the midbelly of the muscle, they refer pain to the lower molar teeth and mandible (Fig. 8.IB).[79,86] From TrPs along the lower edge of the mandible close to its angle, pain is projected in an arc that extends across the temple and over the eyebrow; it also is referred to the lower jaw (Fig. 8.1C).[40,79,81] These patterns have recently been confirmed by Sola and Bonica.[68] A masseter trigger area at the gonial angle (which is most likely enthesopathy) may refer pain preauricularly in the region of the temporomandibular (TM) joint (also referred to as TMJ).[69] Referred pain and tenderness from TrPs in the masseter (or temporalis) muscle may cause tooth hypersensitivity to any or all stimuli: occlusal pressure, percussion, heat, and cold.

Deep Layer

The TrPs in the underlying deep layer of the masseter muscle over the ramus of the mandible are likely to refer pain diffusely to the midcheek area in the region of the lateral pterygoid muscle and sometimes in the region of the TMJ. When a TrP is found at a specific point close to the posterior zygomatic attachment of the deep portion of the masseter, it is likely to refer pain deep into the ear, as in Figure 8.1D.[8,33,58,79,80] The latter TrP also may cause tinnitus of the ipsilateral ear.[79] The tinnitus may be set off by pressure on the TrP, or may be constant, but the patient may be unaware of its presence until it stops upon inactivation of the TrP. Stretching the jaws wide open may also either activate or interrupt the tinnitus. The tinnitus is usually described as a "low roaring" and is not associated with the deafness and vertigo that is common with a vestibular or central neurological lesion.

Prevalence

Among the masticatory muscles, the masseter very frequently harbors TrPs. In one study of 56 patients with myofascial pain-dysfunction syndrome as defined by Laskin[43] (see Chapter 5), the superficial portion of the masseter was the most commonly involved muscle, and the deep masseter was the fifth most commonly involved.[14] In another study of 277 similar patients, 81% complained of pain. Of these patients with pain, the masseter was the second most commonly involved muscle in regard to tenderness (70% of that group); the lateral pterygoid was tender in 84% of the pain group.[35] Sharav and associates[64] observed that the masseter had the second highest prevalence of active TrPs (69%) of 42 patients with the myofascial pain-dysfunction syndrome. Lateral pterygoid TrPs were found in 83% of the patients. Solberg and coauthors[70] observed tenderness in the superficial masseter with limited mouth opening four times as often in subjects who reported awareness of bruxism as in those who denied awareness of it.

Experimental Studies

Kellgren[40] experimentally induced referred pain from the masseter muscle in a normal subject by injecting 0.1 ml of 6% saline solution into its fibers just above the angle of the mandible. This procedure caused "toothache" of the upper jaw, pain in the region of the TMJ, and pain in the external auditory meatus.[40]

During maximum voluntary tooth clenching, electromyographic changes in the masseter correlated well with the onset of fatigue and the time of muscle exhaustion, but did not relate to the onset of mus-

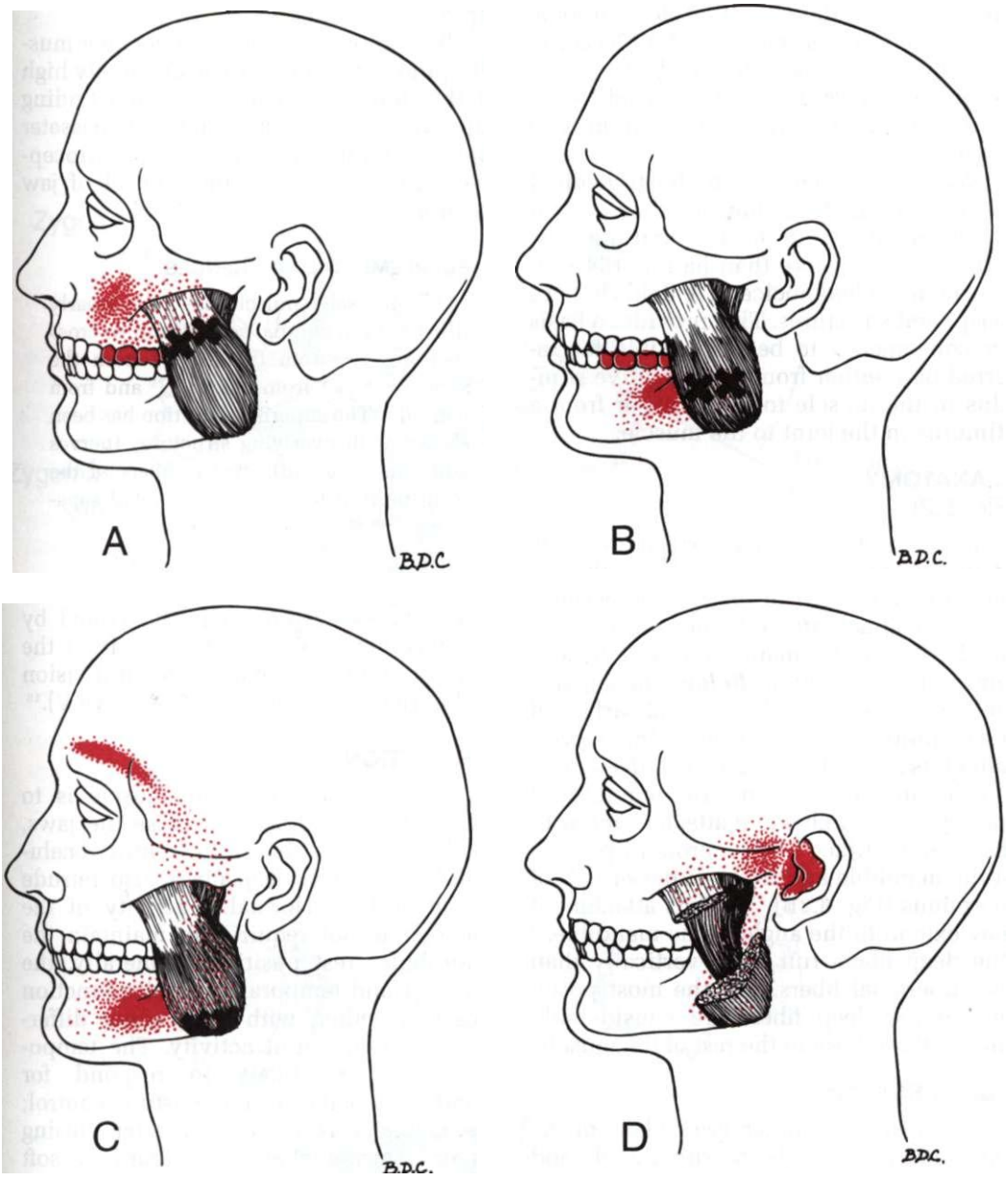


Figure 8.1. The Xs locate trigger areas and trigger points in various parts of the masseter muscle. *Solid red* shows essential referred pain zones, and the *stippled areas* are spillover pain zones. A, attachment trigger points near the musculotendinous junction of the superficial layer, upper portion. **B,** central trigger points in midmuscle of the superficial layer. C, attachment trigger points of the lowest portion of the superficial layer, near its attachment. **D,** trigger point in the upper posterior part of the deep layer below the temporomandibular joint.

cle pain.[15] This finding is in accord with other studies showing that tension-type headache is not correlated with EMG activity;[55] the pain was more likely to be referred from TrPs than to be caused by involuntary motor unit activity of muscle spasm.

Nociceptive neurons in the trigeminal subnucleus caudalis show a strong pattern of convergence from the TMJ and the masseter muscle.[42] More than half of 154 sensory neurons had nociceptive fields in both peripheral structures. This provides a basis for convergence to be responsible for referred pain either from a nociceptive stimulus in the muscle to the joint, or from a stimulus in the joint to the muscle.

2. ANATOMY
(Fig. 8.2)

The superficial and intermediate parts of this muscle are considered together as the *superficial part* of the muscle because they both attach ***above*** to the anterior two-thirds of the zygomatic arch and have a similar fiber direction. ***Below,*** the superficial layer attaches to the external surface of the mandible at its angle and to the inferior half of its ramus (Fig. 8.2A). The *deep layer* attaches ***above*** to the posterior one-third of the zygomatic arch and it attaches ***below*** to the lateral surface of the coronoid process of the mandible and to the superior half of the ramus (Fig. 8.2B).[17,66] This attachment may extend to the angle of the mandible.[29] The deep fibers run more vertically than the superficial fibers, and the most posterior of the deep fibers are considerably shorter than those in the rest of the muscle.

Muscle Structure

In one study,[30] the anterior fibers of the masseter muscle (both superficial and deep) were nearly 87% type I (slow twitch) fibers and nearly 7% type II-B (fast twitch) fibers. The posterior muscle fibers were also predominantly type I fibers (70% superficial and 77% deep) but the posterior part had more type II-B fibers (20% superficial and 15% deep)[30] than the anterior part. Compared to most limb and trunk muscles this is an unusually high proportion of slow twitch fibers, which indicates that the muscle is suited primarily for sustained workloads with only a few brief rapid adjustments.

The number of intrafusal fibers per muscle spindle was found to be unusually high in this muscle (up to 36).[31] This finding supports the understanding that masseter muscle spindles have a strong proprioceptive influence on the fine control of jaw closure.

SUPPLEMENTAL REFERENCES

The masseter muscle has been clearly illustrated in coronal section and in cross section,[2,5,22] from the front,[20] from the side,[4,28,72,76] from below,[25,75] and from behind.[73] The superficial portion has been shown with overlying structures (nerves and parotid gland).[2,48] The fibers of the deep portion have been illustrated separately.[22,29,77]

3. INNERVATION

The masseter muscle is innervated by the masseteric nerve that arises from the anterior branch of the mandibular division of the trigeminal nerve (cranial nerve V).[18]

4. FUNCTION

The chief action of the muscle is to elevate the mandible and close the jaws, as during clenching into centric occlusion.[7,20,54,85] The *deep* fibers also retrude the mandible.[7] Normally, activity of the masseter is not required to maintain the mandibular rest position.[7] Generally, the masseter and temporalis muscles function closely together, with only minor differences in motor unit activity. The temporalis is more likely to respond for mandibular balance and posture control; the masseter is used for greater closing force.[74] During chewing of hard or soft foods, the masseter always responded before the temporalis.[7] Moller, *et al.*[50] found that, in the mandibular rest position, the masseter showed little difference in electrical activity between the sitting and supine postures. In the temporalis muscle, attempts to obtain relaxation with the subject seated upright and the head supported were unsuccessful, but in the supine position it was possible to obtain significant reduction of electromyographic (EMG) activ-

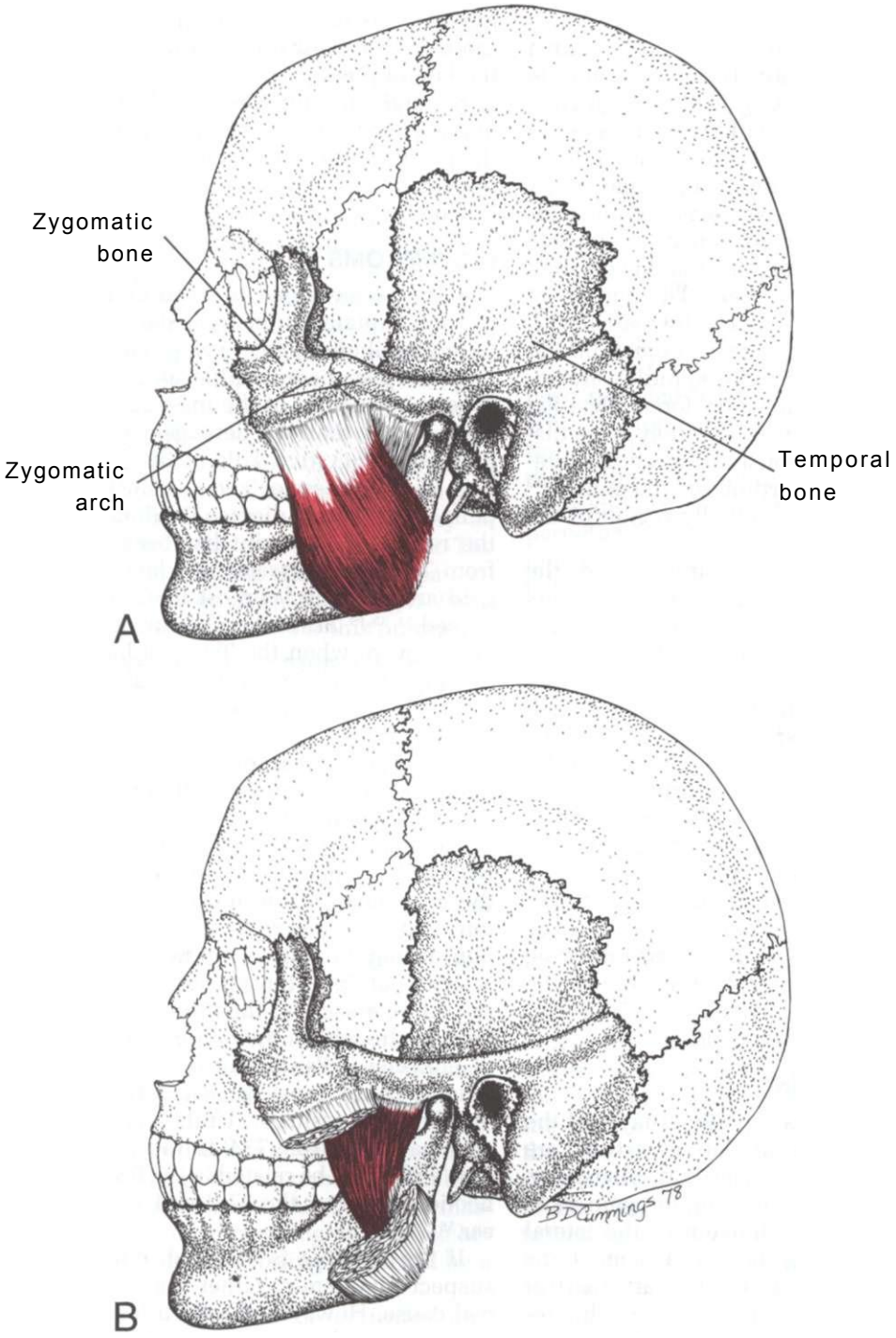


Figure 8.2. Attachments of the masseter muscle. A, superficial layer. B, deep layer, with part of the superficial layer removed.

ity, especially in the anterior part of the muscle.

In this muscle, the deep layers contain a high number of relatively complex muscle spindles corresponding to the remarkable predominance of type I fibers.[30] This could facilitate fine control as the molar teeth are approaching occlusion during chewing. A similar pattern occurs in masseter muscles of the rabbit.[13] A high density of muscle spindles occurs in regions that have a high proportion of type I fibers. The density of spindles and type I fibers increases with distance from the temporomandibular joint, suggesting that the spindles are involved in controlling bite force. Detailed spindle counts showed that they were distributed heterogeneously between the anterior and middle portions of the muscle[13] and not just in the midbelly region of the muscle.

One objective test demonstrated the modulation of reflex activity in patients with symptoms characteristic of active TrPs in the masticatory muscles. A silent period of about 24 msec interrupts masseteric motor unit activity during jaw clench when a jaw-jerk response is produced by a tap on the chin[6,12] or by a tap on a tooth.[7] The silent period results primarily from stimulation of the receptors in the periodontal ligament, which surrounds the teeth.[12] The duration of the silent period was clearly increased among patients with severe symptoms of painful masticatory muscles,[46,67] and was decreased following successful treatment.[64] This is compatible with other data that TrPs affect motor control as well as pain perception.

5. FUNCTIONAL UNIT

Synergists of the superficial layer of the masseter for mandibular elevation are the contralateral masseter and, bilaterally, the temporalis, and medial pterygoid muscles.[7] The superior division of the lateral pterygoid muscle is thought by some to be active during closure or the early part of the mandibular power stroke.[34,44] This remains controversial however, due to the difficulty in ascertaining the actual electrode position during recording and the possibility of EMG noise from adjacent muscles.[84,87] Antagonists to the masseter include the geniohyoid, omohyoid, and hypoglossus muscles, the anterior belly of the digastric, and the inferior division of the lateral pterygoid.

Synergistic with the deep layer of the masseter for retrusion of the mandible is the posterior portion of the temporalis. It is opposed chiefly by the inferior division of the lateral pterygoid muscle.

6. SYMPTOMS

Pain, as described in Section 1, is the major complaint. In many instances, "temporomandibular joint" symptoms are related to poor coordination and increased "spasm" (tension) of the masticatory muscles, rather than to derangement of the joint itself.[35] Active TrPs in the deep portion of the masseter can mimic the TMJ pain of rheumatic disease.[58] When pain in the region of this joint has been referred from TrPs, the masseter and lateral pterygoid are the muscles most likely to be involved.[14,35,64] *Restriction of jaw opening* is more severe when the TrPs are located in the superficial layer of the masseter than when they are in the deep layer of the muscle. Surprisingly, the patient is often unaware of restricted opening if the jaws open wide enough (about 30 mm) to bite a sandwich comfortably.[78]

Unilateral tinnitus may be associated with TrPs in the upper posterior portion of the deep layer of the muscle. This symptom may be a referred sensory phenomenon or may be due to referred motor unit activity of the tensor tympani and/or stapedius muscle of the middle ear. These muscles lie within the pain reference zone of masseter TrPs. Spasm of the stapedius muscle could cause an oscillation of the middle ear ossicles. Unilateral tinnitus also may arise from TMJ intracapsular disease and could be related to the fascial connection between the TMJ and the middle ear.[56]

If the tinnitus is bilateral, one should suspect a systemic, rather than a myofascial cause. However, the deep layer of the masseter can become involved bilaterally, giving rise to bilateral tinnitus. In this case, unilateral fluctuation of its intensity is likely to occur. Bilateral tinnitus may be due to a high serum salicylate level; drug-

induced tinnitus is usually bilateral and dose-dependent,[51] not predominantly unilateral as is typical of deep masseter TrPs. Impairment of hearing is not a feature of active TrPs in the masseter muscle.

Complex symptoms and overlapping patterns of facial pain may be referred from multiple TrPs in the *head and neck muscles. A* good example of this is unilateral or bilateral headache pain, either migraine or tension-type, generated by several different overlapping pain patterns from masticatory and cervical myofascial TrPs (see Fig. 5.2). The practitioner can identify which TrPs are likely to be contributing to the total pattern by sketching the total distribution of pain in detail on a body form *[see* Figs. 3.2-3.4) for each patient, and by comparing the sketch with the characteristic pain patterns of individual muscles that may be contributing to the total pain picture. The muscle guide chapters and the pain-pattern flip chart derived from the *Trigger Point Manual* are very helpful for this purpose.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Postural and Activity Stresses

An excessive forward-head posture *(see* Chapter 5, Section C) places the mandible in a position that puts stress on the masseter muscle and can *activate* or *perpetuate* TrPs in the masseter. Chronic mouth breathing (e.g., through a surgical mask, or due to nasal obstruction) tends to cause excessive forward head positioning and postural changes which indirectly add stress to the masticatory muscles and may activate and perpetuate TrPs in these muscles. Additional postural factors that influence forward-head position are discussed in Chapter 41.

Acute overload situations that can *activate* TrPs in the masseter muscle include sudden forcible contraction of the masseter muscle (as in cracking nuts or ice between the teeth), and biting off thread by a seamstress.

Masseteric TrPs may be *activated and perpetuatedby* sustained or repetitive abusive jaw habits such as clenching or bruxing the teeth, gum chewing, nailbiting, prolonged clamping of the jaws on the mouthpiece of a pipe or cigarette holder,[47] late childhood thumbsucking, and significant occlusal disharmony such as profound loss of vertical dimension due to worn natural teeth, loss of posterior teeth, worn denture teeth, or resorption of alveolar bone.

Psychological Stresses

The masseter muscles are among the first to contract in persons who are in a state of extreme emotional tension, intense determination, or desperation, and they often remain contracted for abnormally long periods of time.[86] These muscles have been reported to be overactive in patients who develop temporomandibular dysfunction.[88] Bell[10] presented case reports that indicate the contribution of life stress situations and bruxism to the development and perpetuation of TrP pain. Schwartz, *et al.*[62] noted the contribution of emotional stress to the development of active TrPs. Unfortunately, the psychological distress component associated with any chronic pain is often overemphasized, to the neglect of the myofascial TrP contribution to internal derangements of the TMJ.[49] It can be grossly unfair to the patient and costly to society to attribute the pain to psychological factors rather than recognizing the psychological factors to be the *result* of the pain, often because the TrP origin of the pain was unrecognized or was inadequately treated.

Other Stresses

Other factors that may *activate* latent TrPs in the masseter muscle include prolonged over-stretching during a dental procedure, immobilization of the mandible in the closed position (by the head halter during continuous neck traction, or by wiring the jaws shut), the direct trauma of an accident, particularly with a blow to the side of the jaw, and overload of the masseter following a motor vehicle accident causing a flexion-extension injury to the suprahyoid or infrahyoid muscles, which in turn produce tension on the jaw and thereby on the masseter muscle. Often overlooked or forgotten is the reflex muscle contraction that occurs with any chronic infection or inflammation. When prolonged, this is believed to contribute to the development of

myofascial TrPs.[32] Chronic pulpal or periodontal inflammation[63] and TMJ arthropathy are frequent causes of masticatory muscle TrP activation which can persist after the inciting infection or inflammation has subsided. Yet lack of recognition of this phenomenon often results in unnecessary endodontic treatment or extraction, or a persistent search for why TMJ treatment has failed to resolve the problem. Similarly, it is important to recognize that masseter muscle TrPs are often satellite TrPs activated and perpetuated by key TrPs in the sternocleidomastoid or upper trapezius muscles.[36] In this situation, appropriate treatment of the key TrPs often obviates the necessity of treating the masseter TrPs directly.

8. PATIENT EXAMINATION
(Fig. 8.3)

The clinician should be aware that TrPs produce dysfunction (because they increase muscle tension) as well as pain. Prior to beginning the physical examination that is addressed in this section, the clinician must take a thorough patient history *[see* Chapter 3). After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram representing the pain described by the patient. The drawing should be in the style of the pain patterns illustrated in this volume, using a copy of an appropriate body form found in Chapter 3, Section 1, Figures 3.2-3.4.

Because the mandible spans the midline and attaches to both sides of the cranium, a unilateral dysfunction, whether due to muscle problems or internal derangement of a TMJ, will also have an effect on the contralateral side. Therefore, assessment should always include bilateral visual and palpatory examination for musculoskeletal dysfunction. The examiner should check specifically for forward-head posture. Forward head posture indirectly induces tension in the supra- and infrahyoid muscles (*see* Chapter 12) which in turn pull downward to create light tensile forces on the mandible. This causes the mandibular elevator muscles to contract to keep the mouth closed. Evaluation of forward-head posture is described in Chapter 5, Section C. Other postural considerations (some of which are factors that can indirectly induce forward-head posture) are discussed in Chapter 41.

Masseter TrPs, whether unilateral or bilateral, may cause significant restriction of mandibular vertical opening which is evident on examination, although the patient may not be aware of it. Unilateral masseter TrPs tend to deviate the mandible toward the affected side, a deviation which is apparent when the patient slowly opens and closes the mouth. This must be differentiated from unilateral TMJ internal derangement, which may also cause the mandible to deviate toward the affected side *[see* Chapter 5). Of course, with a history of painful joint derangement, both factors may be present and ultimately need treatment.

There is one convenient way of obtaining a prompt clinical estimate for the adequacy of intercisal opening that is adjusted to the size of the individual. A patient can use his or her own hand as the measuring instrument. At the very least, the patient should readily pass the "Two-knuckle Test" that is illustrated in Figure 8.3. A tier of the first two knuckles (proximal interphalangeal joints of the second and third digits) should slip readily between the upper and lower incisor teeth. A more critical test is the insertion of a tier of the *distal phalanges* (not knuckles) of the first three fingers placed between the incisor teeth. This was readily accomplished by an asymptomatic population of subjects who were unscreened for masticatory symptoms and/or tender masticatory muscles.[1]

Individuals with active or latent TrPs in the mandibular elevator muscles are very unlikely to pass the more rigorous *"Three-knuckle* Test" which was first reported by Dorrance[27] in 1929. The patient places a tier of the first three knuckles (second, third and fourth digits) of the nondominant hand between the upper and lower incisor teeth. This test is more demanding than the loose two-knuckle test and requires a degree of forcing for many individuals even when they are free of TrPs. This forcing would be unwise for individuals who might have TMJ dysfunction. If the three-knuckle test can be accomplished without forcing, the subject is very unlikely to have

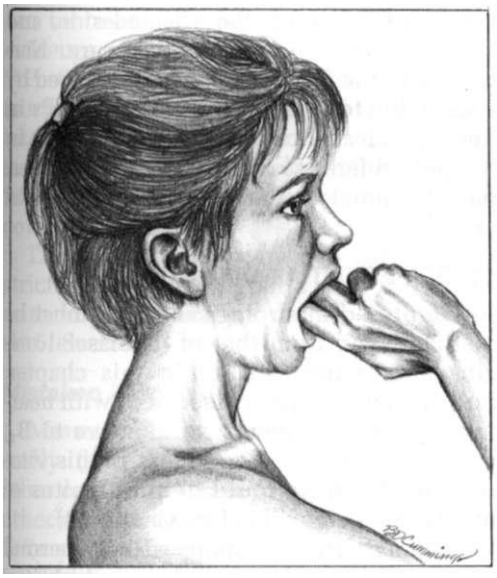


Figure 8.3. Loose Two-knuckle Test. The relaxed, fully opened mouth should **readily** admit the first two knuckles (of either hand) in a tier between the incisor teeth of individuals with normal joint and bone structures and without demonstrable myofascial trigger points.

masseter or temporalis muscle TrPs, or significant TMJ dysfunction, but could have a hypermobile joint.

Measurement of the interincisal opening by forcing three knuckles between the teeth, or wedging another measuring device between the teeth, forces the mouth open slightly and results in a measurement several millimeters larger than that obtained with the usual Boley gauge or a millimeter ruler used without exerting pressure.

If there is doubt about the restriction of mouth opening, the maximal interincisal distance (measured as clearance between the upper and lower incisor teeth) can be measured with a sterilized millimeter ruler and compared to the normal minimum of 40 mm (see Chapter 5, Section C).

It is a remarkable observation that TrP activity in leg muscles due to a Dudley J. Morton foot,[52,53] or TrP activity in certain neck or shoulder-girdle muscles (sternocleidomastoid, trapezius, and scaleni) restricts mouth opening. Inactivation of TrPs in these nonmasticatory muscles can immediately increase the maximal interincisal opening.

Anterior displacement of the TMJ articular disc and postoperative trismus due to activation of TrPs in the medial pterygoid muscle also may restrict jaw opening severely, but temporalis TrPs usually limit it only minimally.

9. TRIGGER POINT EXAMINATION
(Fig. 8.4)

Nearly all of the midmuscle portion of the masseter can be examined for TrPs most effectively by pincer palpation between one digit inside of the cheek and another outside, as illustrated in Figure 8.4 and as illustrated by Ingle and Beveridge.[37] Only a thin layer of mucosa separates the palpating finger and the midportion of the muscle. If the examiner has difficulty localizing the muscle itself, the examiner can ask the patient to bite *gently* on a rubber block or cork. If the muscle has active TrPs,

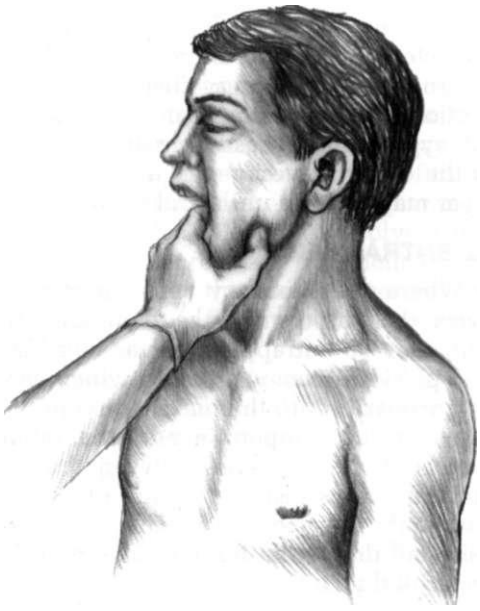


Figure 8.4. Pincer method for locating trigger points in the superficial portion of the masseter muscle. The muscle is lengthened to take up any slack by the patient holding the mouth in a relaxed open position. The examiner's gloved fingers rub across the muscle perpendicular to the direction of the fibers to detect any taut bands.

the taut bands and their exquisite spot tenderness will identify them. With the muscle relaxed, taut bands can be identified by rubbing the muscle fibers between the fingers. The tenderness of the TrP is enhanced if the patient opens the mouth far enough to take up most of the slack in the muscle; usually the width of a tongue depressor placed on its edge between the upper and lower incisors provides this slack. The finger inside the mouth can feel the muscle structure much more clearly than can the finger on the outside, because the parotid gland lies between the skin and much of the midfiber portion of the muscle where many masseter TrPs are located. Tenderness identified from the outside in the region of attachment to the mandible is tenderness of enthesopathy and not primary TrP tenderness. Since enthesopathy results from sustained tension at the attachment of the muscle fibers, it is not surprising that tenderness at the gonial angle disclosed by flat palpation is significantly associated with bruxism.[71]

Sometimes, TrPs in the *deep* layer of the masseter are located more effectively by external flat palpation against the posterior portion of the ramus and along the base of the zygomatic buttress. Pressure on a TrP in the upper posterior portion of the deep layer may activate unilateral tinnitus.

10. ENTRAPMENT

Where the maxillary vein emerges between the masseter and the mandible,[24] the vein may be entrapped by masseter TrPs. The pterygoid venous plexus, which empties primarily into the maxillary vein, lies between the temporalis and the lateral pterygoid muscles and between the two pterygoid muscles; the plexus drains the temporalis muscle *via* the deep temporal vein and drains the infraorbital region *via* the orbital vein.[18]

The resulting engorgement of the deep temporal vein and pterygoid plexus favors bleeding and ecchymosis after injection of TrPs in the temporalis muscle.

The increased firmness of taut bands due to TrPs in the masseter muscle may restrict venous flow from the infraorbital subcutaneous tissues. This engorgement of the orbital vein produces puffiness ("bags") beneath the eye on the affected side, and thus narrows the palpebral fissure. Narrowing of the fissure also may be caused by spasm due to activation of satellite TrPs in the orbicularis oculi muscle, which lies in the pain reference zone of TrPs in the sternal division of the sternocleidomastoid muscle.

11. DIFFERENTIAL DIAGNOSIS

Tinnitus of neurological origin must be distinguished from that of myofascial origin as presented earlier in this chapter. Surprisingly, tinnitus associated with hearing loss was frequently responsive to B_{12} therapy.[65] If the patient is low in this vitamin, B_{12} therapy would help a tinnitus of myofascial origin also (see Chapter 4).

Prolonged pain responses to a thermal stimulus to a tooth may indicate a **pulpitis,** whereas sensitivity to percussion and pressure can result from **apical inflammation of the periodontal ligament.**[11] Referred pain and tenderness from TrPs in the masseter (or temporalis) muscle may cause tooth hypersensitivity to any or all stimuli: occlusal pressure, percussion, heat, and cold. Appropriate treatments for pulpitis, inflammation of the periodontal ligament, and masseter TrPs are quite different.

If patients can open the mouth only 30 mm or less, they may have unilateral or bilateral **anteriorly displaced discs** of the TMTs, especially if they have a history of clicking. These patients should be referred for evaluation by a dentist familiar with the diagnosis and management of temporomandibular disorders.

Trismus is a firm closing of the jaw due to spasm of masticatory muscles that, for example, is characteristic of tetanus. Tetanus also may result from dental sepsis, injury, surgery, needle abscess, and the Morgagni syndrome caused by a malignant tumor. Specifically, trismus can be due to spasm of the masseter muscle from cellulitis in adjacent tissues, spasm of the medial pterygoid muscle from cellulitis in the pterygomandibular space, and spasm of the temporalis muscle from cellulitis in the infratemporal fossa.[9] Attempts to open the jaws are painful because of the spasm. The pain is aggravated if the spastic muscles also have active TrPs. The active TrPs

can be treated by injection if there is no evidence of infection in the region of the TrP. One effective treatment for spasm (such as the spasm of trismus) is to use tetanizing electrical current to fatigue the muscle to the point of release.[57] Following fatigue, muscle release techniques may become effective.

Loss of TMJ play can be a cause of restricted jaw opening and can be determined by mobilization described in Chapter 5, Section C.

Mistaken Diagnoses

Masseter TrPs cause symptoms that are easily (and frequently) misdiagnosed as other conditions. One of the most common other conditions in which masseter muscle TrPs play a frequent contributing role is tension-type headache, especially if TrPs in neck muscles are contributing to the pain.[39] Cervicogenic headache presents a similar situation and frequently involves dysfunctions of the cervical spine that also need correction.[38] Earache of *unexplained origin* is likely to be caused by TrPs in the deep masseter or in the clavicular division of the sternocleidomastoid muscle *(see* Fig. 7.1B). Pain referred to a tooth by TrPs can easily be misinterpreted as being of endodontic origin.[41] This can lead to disastrous results for an innocent tooth.

Related Trigger Points

The main synergists of the masseter (the temporalis and medial pterygoid muscles), tend to develop related TrPs, as does the contralateral masseter.

Masseter TrPs also may originate as satellites due to increased motor unit activity secondary to TrPs in the sternal division of the sternocleidomastoid muscle and also from key TrPs in the upper trapezius.[36]

12. TRIGGER POINT RELEASE
(Figs. 8.5 and 8.6)

Release of myofascial trigger points (TrPs) and subsequent resolution of the associated pain or dysfunction is always dependent on reducing or eliminating as many perpetuating factors as possible first *(see* Section 7). Once these are under control, specific TrP techniques such as those described here will be more effective.

A primary consideration in the treatment of temporomandibular disorders is to correct forward-head posture and poor tongue position, if present *(see* Chapter 5, Section C). Sometimes referred pain from the masseter clears up after only postural correction. This is because masseter muscle tension and/or TrPs (that have been perpetuated by reflex masseter activity counteracting the light tensile forces generated by the supra- and infrahyoid muscles as they pull down on the mandible in head extension) now have been relieved by getting the head back to normal alignment. Anterior head positioning with reflex elevator muscle activity also causes increased intra-articular pressure in the TMJs and can precipitate mild internal derangements in joints with compromised discs. Thus, correction of posture may also resolve early mild TMJ clicks. If posture correction and other treatment strategies directed at the masseter (or other mandibular elevators) alone does not produce the desired result, tension and/or TrPs in the suprahyoid and infrahyoid muscles also may need attention *(see* Chapter 12). Similarly, since masseter TrPs also may originate as satellites to TrPs in the sternocleidomastoid and trapezius muscles, resolution may depend on appropriate management of these primary TrPs first. Masseter and other masticatory muscle TrPs often resolve sufficiently with appropriate treatment of the cervical muscles, that use of specific TrP release techniques for the masticatory muscles is not necessary.

A further potentially complicating factor may be cervical joint dysfunction if present. Most patients will respond to the simple strategies listed above even if they have cervical dysfunction. However, if the dysfunction and TrP pain persist after posture correction and good patient compliance to correction of other perpetuating factors to elevator and cervical muscle TrPs, then referral to a practitioner familiar with evaluation and treatment of both cervical dysfunction and myofascial TrPs may be indicated.

Muscles of mastication, when dysfunctional, are usually involved bilaterally. This is because the mandible is a single bone attached on each side of the cranium,

and movements and functions on one side, whether normal or abnormal, are intimately related and dependent on the other. Although one side may be the primary problem, both sides must be treated. For example, it is impossible to stretch one masseter or temporalis and not the other.

Myofascial TrPs in the masseter muscle can be effectively released by spray and combined manual muscle stretch (Fig. **8.5),** spray and specific masseter myofascial release, TrP pressure release, and a technique known as strumming (described later in this section under Other Release Techniques). Moist heat applied over the muscle prior to the application of TrP release techniques may increase patient comfort and aid relaxation.

Stretch techniques (such as spray and stretch) for release of TrPs should be used with caution whenever there is an intracapsular TMJ disorder. When in doubt about whether or not a TMJ disorder will be aggravated by wide opening, the following noninvasive techniques that do not involve therapeutic stretching can be used. These include TrP pressure release, reciprocal inhibition performed isometrically, and indirect techniques. Refer to Chapter **3,** Section **12** for a general description of these techniques. Emphasis on slow nonforced respiration can augment muscle release with any technique.

Spray and Stretch

Two techniques are presented in detail for stretch release following application of intermittent cold by vapocoolant spray or icing. The first is a combined stretch release effective for inactivating TrPs and releasing tension in the temporalis, masseter, medial pterygoid, and platysma muscles simultaneously (Fig. **8.5** A and B). The other is a spray-and-stretch release technique that is specific for TrPs in the masseter muscle (Fig. **8.6).** In all of these techniques involving the jaw elevator muscles, it is important to remember that both sides of the face must be sprayed or iced in the appropriate pattern *prior* to initiating any jaw opening stretches, since one side cannot be stretched in isolation from the other. In general, the *operator's* hands should passively stretch the *muscle(s)* while the *patient* actively opens the mouth. The operator should avoid assisting the opening or should do so only very gently with little or no force. Any passive assistance to opening is more safely applied on the posterior molars, not on the incisors.

Both of these stretch release techniques start with the patient supine in a comfortable position and with the head supported by a pillow for complete total-body relaxation. Parallel sweeps of vapocoolant (or icing) are directed upward from the upper chest over the muscles, covering the mandible and cheek, and extending over all of the pain reference zones including the temple, forehead, the hairline, and behind the ear. (Fig. **8.5A)** Care is taken to include all areas where the patient experiences referred pain from any of these muscles and to apply the spray bilaterally. To prevent the vapocoolant liquid from trickling into the eye, the patient must be warned to keep the eye closed (it is wise to cover the eye with an absorbent pad). **CAUTION:** Patients with asthma or other pulmonary conditions may not tolerate spray because of the vapors. Ice may be used as an alternate form of intermittent cold (see Chapter **3).** If spray is used, a small cloth or a hand should lightly cover the patient's nose and mouth.

The combined stretch release of the right temporalis, masseter, medial pterygoid, and platysma muscles is applied as described and illustrated in Figure **8.5B.** To accomplish stretch release of specifically the masseter muscle, the clinician first applies vapocoolant or icing bilaterally upward from the mandible primarily over the muscle and cheek, including the forehead and, if indicated, the ear (as in Figure **8.5 A).** When spraying the ear, be sure to warn the patient to be prepared for an alarming sensation for an instant if the vapocoolant enters the ear canal. Immediately following the spray, the clinician proceeds as described and illustrated in Figure **8.6.**

After rewarming the skin with moist heat, stretch release may be repeated if restriction of mouth opening or spot tenderness remains. The patient should open and close the mouth fully (but not forcibly) three times to restore normal muscle coordination.

Warning Note: Yawning has a powerful masseter-relaxing and stretching effect, but

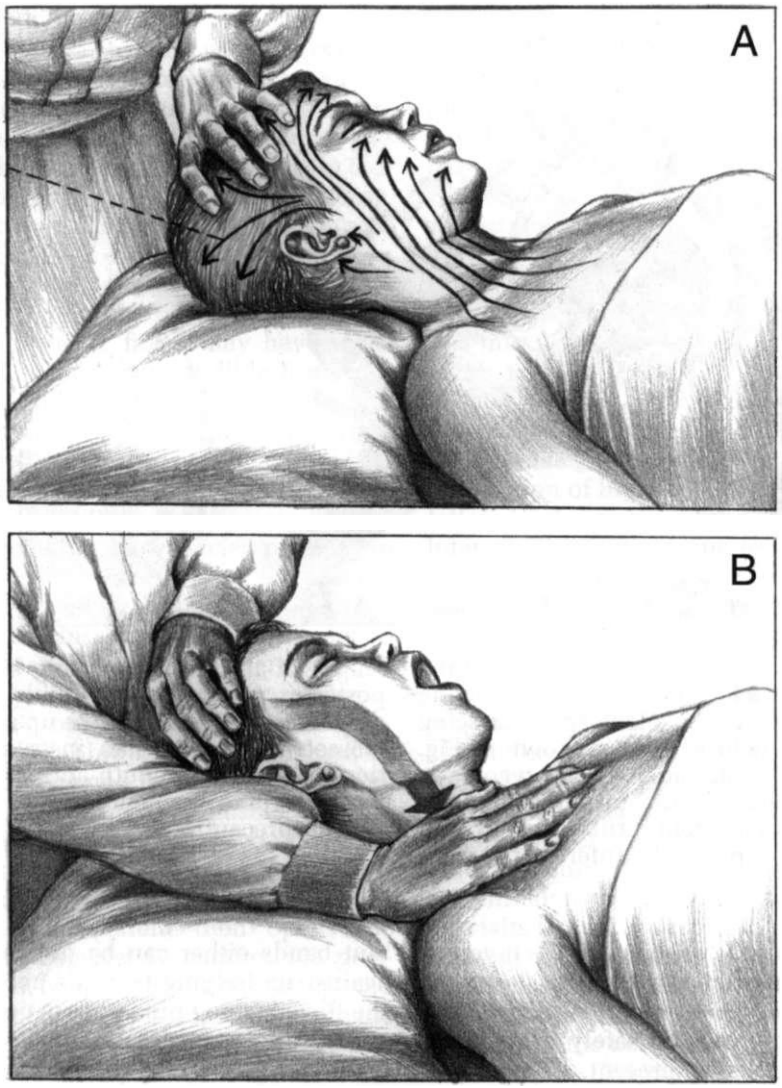


Figure 8.5. Spray and combined stretch for trigger points in the right temporalis, masseter, medial pterygoid, and platysma muscles, patient supine. A, Complete pattern *(thin arrows)* for application of vapocoolant spray (or icing) prior to stretch. The patient's eye should be kept closed and covered with a pad, and the operator should avoid spraying near the eye. The spray should include all areas where the patient is experiencing pain referred from any of these four muscles. For the masseter in particular, the operator applies vapocoolant or icing upward from the mandible over the muscle and cheek, including the forehead and temple. B, Immediately following application of spray, the operator instructs the patient to relax the jaw and then takes up slack in the temporalis muscle by applying upward traction on that muscle with one hand (in this case the left hand). The operator's other hand completes the release procedure with slow firm downward traction *(thick arrow),* starting from the temporalis and moving downward over the masseter and platysma muscles; while the operator maintains the stretch-release, the patient breathes in and opens the mouth, utilizing respiration and reciprocal inhibition to further relax the mandibular elevators. The operator directs the traction pressure inferiorly but *not* medially, to *avoid deviation* to the opposite side, which would place the opposite temporomandibular joint in a loaded, close packed state.

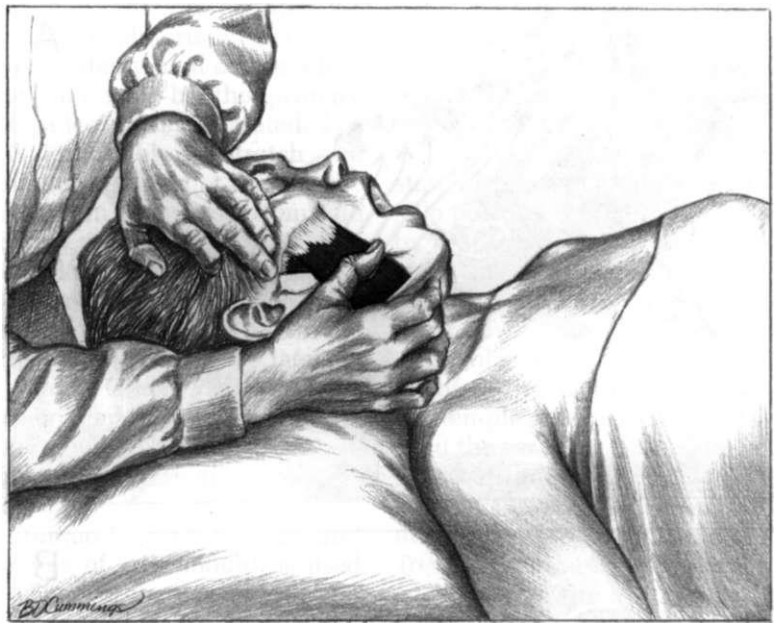


Figure 8.6. Stretch release specifically of the masseter muscle immediately following application of vapocoolant spray or icing (intermittent cold pattern is shown in Fig. 8.5A). The operator anchors the zygomatic arch attachment of the muscle with one hand. The other hand rubs slowly and firmly from superior to inferior over the length of the muscle, taking hold of the posterior part of the mandible with light downward pressure to take up slack in the masseter. The clinician instructs the patient to open the mouth and take in a full deep breath to augment muscle release (see text for precautions).

can cause problems because it involves forceful maximum forward translation of the joint disc. This much mobility of the disc will likely not be safely available if disc dysfunction is present. Therefore it should be employed therapeutically only when there is strong assurance that disc function is completely normal (*see* Chapter 5 for TMJ screening examination). To prevent inadvertent overstretching and excessive opening, especially in hypermobile joints, the patient should be taught to place the tongue tip against the palate just posterior to the incisors, and to open the mouth only as far as that tongue position allows. The mouth should *not* be opened wide when a painful joint click is present. Refer to Chapter 5, Section C.

Other Release Techniques

Trigger point pressure release in the masseter is especially effective because the taut bands either can be pressed directly against underlying bone or held between the fingers using pincer palpation. The basic technique is described in Chapter 3, Section 12; this release should not be excessively painful and consists of light pressure on the TrP until resistance is encountered (until a barrier is engaged), holding that position until release occurs under the palpating finger, and then gently following the movement of the releasing tissues until a new barrier is encountered.

Strumming is a variation of deep massage in which the operator's finger pulls *across* the muscle fibers rather than parallel to the fiber direction. It is particularly effective for at least the anterior half of the masseter because the palpating finger inside the mouth is in direct contact with the muscle fibers, except for a thin layer of mucosa. The patient's mouth should be slightly open in a relaxed position. The

clinician's thumb placed on the skin external to the muscle provides counterpressure to pull against. Strumming consists of pulling the finger across the muscle fibers slowly until the TrP and resistance are encountered, maintaining light contact at that point until the finger senses tissue release, then continuing to pull the finger across the muscle as it releases. Relaxed deep breathing by the patient will facilitate general relaxation during this process. Some of the most posterior fibers may have to be strummed against the mandible from outside the mouth. This technique is most effective when it is performed precisely across the TrPs in the central midmuscle portion of the taut bands. Since it is a dynamic form of TrP pressure release and localized stretching, the clinician must begin gently and progress slowly as the tension in the taut bands gradually releases. Because of the close contact with the sensitive masseter TrPs, excessive pressure is extremely painful and can delay release.

Voluntary opening of the mouth provides **reciprocal inhibition** of the masseter, is readily performed by the patient, and is effective in releasing this muscle. The patient sits with the chin propped on the fist or palm of both hands which are placed symmetrically on the side of each mandible. The patient then opens the mouth to a completely comfortable position that feels as if the mouth is opened fully but not forcibly, taking up slack in the masseter. In that position, the patient performs a gentle *isometric* contraction of the depressors of the mandible by gently pressing the mandible down against the hands for at least 5 seconds. The hands resist any attempt at movement. Then, while inhaling, the patient again gently opens the mouth as before, to take up slack that has developed in the masseter muscles. This can be repeated 3 or more times until no improvement results. Relaxation of the *deep* masseter can be accomplished by having the patient perform a gentle *isometric* contraction that attempts protrusion, but is resisted to prevent movement.

A **technique similar to hold-relax**[82] (briefly described in Chapter 3, Section 12) can be applied to release TrP tension in a tight masseter in *some* patients; the voluntary contraction in this case, however, should be *minimal* rather than maximal. This modification for TrP release involves placing the masseter in a comfortably lengthened position and gently contracting the masseter isometrically against light resistance for about 6 seconds, followed by relaxation, exhalation, and further lengthening to take up the slack created. However, since most patients with masseter TrP involvement evidence excessive tension in this muscle much of the time, other treatment techniques (TrP pressure release, strumming, and reciprocal inhibition) may be more effective than hold-relax.

Masseter TrPs respond well to external application of **high voltage galvanic stimulation** in the hands of trained therapists. Intensity should be increased gradually until the patient is aware of a tingling sensation, but the muscle is not maintained in contraction. (Maloney, Personal Communications, 1996).

Regardless of what technique was used to release the masseter muscle, the patient can maintain the new range of motion and control masseter TrP activity by using the appropriate exercises daily at home (*see* Section 14 of this chapter). These techniques require thorough instructions by the clinician and at least 2 weekly follow up visits to confirm that the patient is performing the exercises correctly.

Normal joint play is necessary for normal joint function; this applies to joints throughout the body, and the TMJ is no exception (see Chapter 5, Section C). One needs to reestablish joint play (if it is lacking) before stretching muscles that require full joint range of motion. Mandibular depression in the long axis is a gentle mobilization that can be performed by placing the thumb over the posterior molar region and gently depressing the mandible 1 to 1.5 mm.

On the other hand, if the TMJ is hypermobile, the patient must learn to limit translation of the mandibular head by avoiding opening the mouth wide, and should perform exercises (isometric exercises in the form of rhythmic stabilization[82]) to improve joint stabilization.[59] Protective tongue position should be used on opening *(see* Chapter 5, Section C). As for

any hypermobile joint, manual inactivation of TrPs should avoid techniques that depend on lengthening the muscle to its full stretch length. Instead, TrP release techniques are applied directly to the muscle, stretching and elongating the muscle fibers without maximal joint movement. Figure 8.5 illustrates this approach.

13. TRIGGER POINT INJECTION (Fig. 8.7)

If the immediate response of masseter trigger points (TrPs) to manual release techniques is not satisfactory, injection of the masseter TrPs usually inactivates them.[10] However, at this point, the clinician must seriously consider the possibility that one or more perpetuating factors have been overlooked *[see* Chapter 4). A detailed description of TrP injection technique is found in Chapter 3, Section 13. Masseter TrPs are identified by pincer palpation as described in Section 9 of this chapter. Masseter TrPs may be injected by a needle held inside the mouth or from the outside, whichever fits the skills of the clinician best. For intraoral injection, a short thin needle (25- or 27-gauge 1-inch) is satisfactory. Intraoral injection has the advantage that it does not require penetration of the parotid gland where the facial nerve also is located as the nerve penetrates the gland.[19] The location of the nerve in relation to the parotid gland and masseter muscle is well illustrated.[3,23]

When the taut band and its TrP are clearly identified against the finger tips by pincer palpation, the needle should be directed specifically into that structure with multiple insertions (peppering) performed without withdrawing the needle. The physician should note carefully any local twitch responses and pain reactions indicating that the needle encountered an active locus in the TrP. A few drops of 0.5% procaine or plain lidocaine is injected whenever an active locus *(see* Chapter 2, Section D) is encountered.

A comparable injection technique is described and illustrated in detail by Cohen and Pertes.[26]

14. CORRECTIVE ACTIONS

Patients suffering from chronic myofascial pain with multiple TrPs should be started on the systematic 6 week program outlined in Chapter 5, Section D. Specific corrective actions for the masseter muscle are outlined below.

Activity Stress

First and foremost, forward head posture must be corrected to reduce masseter muscle activity (refer to Chapter 5 for assessment and correction of forward head posture and refer to Chapter 41 for other factors that may influence head posture). This may require changes to ensure that the patient can breathe through the nose, rather than the mouth. Additionally, the patient should develop awareness of mandibular posture, correct tongue position *[see* Chapter 5, Section C) and reduce daytime clenching, nail biting, exhaustive chewing or other abusive oral habits. Habits of "clenching" the teeth should be revised by the pipe smoker. Those who abuse their mandibular elevators by cracking hard candy or nuts with the teeth and by constantly chewing gum should avoid these activities. Bruxist behavior should be identified and corrected[60] and may require use of an intraoral orthosis (see Chapter 5).

Life stress and tension anxiety that lead to jaw clenching and bruxism should be managed by reducing emotional strain and improving the patient's coping behavior. This may be achieved with simple stress/time management strategies or may require referral to a psychologist or other mental health provider for specific pain and stress management techniques. Wearing a nocturnal occlusal splint reduces bruxism associated with high-stress life situations.[60] Placing the tongue against the roof of the mouth behind the upper incisor teeth while going to sleep (or any time bruxing occurs during waking hours) can be very helpful *[see* Chapter 5).

Myofascial TrPs in the sternocleidomastoid, upper trapezius, and other muscles that refer pain to the head and neck should be inactivated. Muscles that refer pain to the region of the masseter can activate satellite TrPs in that muscle; the key TrPs in those other muscles must be eliminated for sustained relief.[36]

During prolonged neck traction, the patient should wear a dental splint that elim-

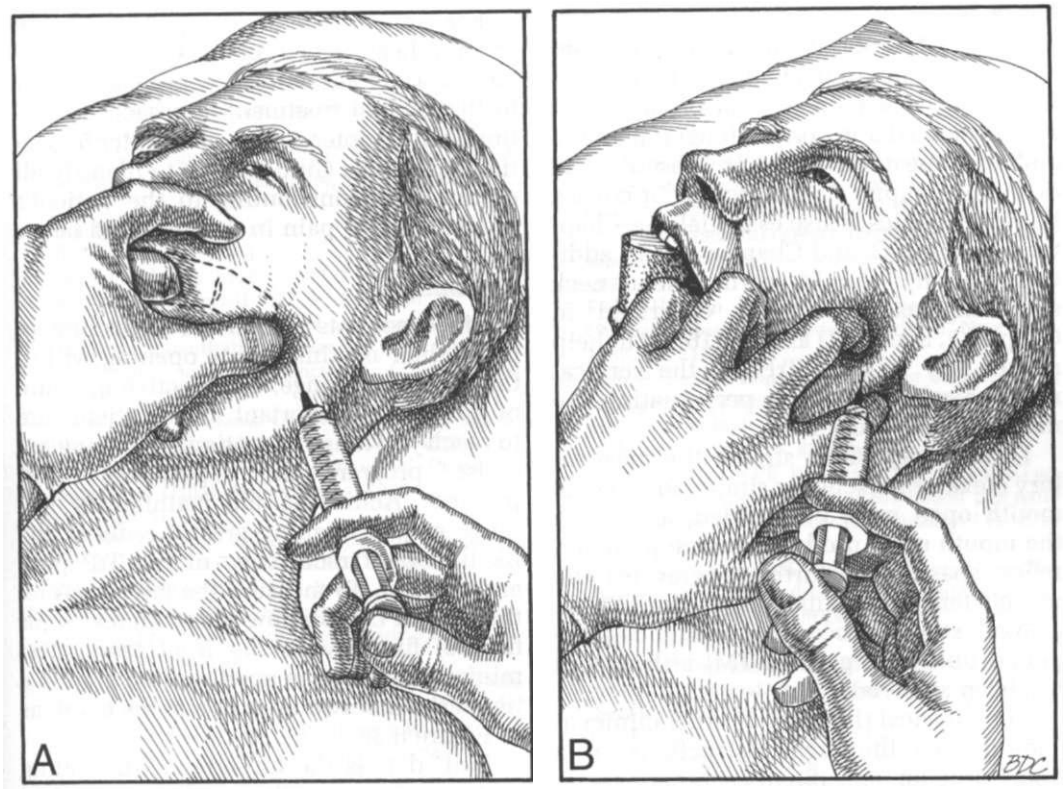


Figure 8.7. Extraoral injection of trigger points in the masseter muscle. A, midbelly of the superficial portion, using pincer palpation to accurately localize trigger points for injection. B, Posteriorly located deep portion of the muscle, using flat palpation to fix the trigger point against the underlying ramus of the mandible, avoiding the facial nerve.

inates premature contacts, provides mandibular stability, and reduces mandibular elevator shortening.

Long dental procedures that require wide opening of the mouth should be preceded by spray and stretch. Periodic relief for the stretched muscles in the form of intermittent breaks allowing closure of the mouth is mandatory. Sedation of the patient may help to prevent severe activation of TrPs. When painful organic intracapsular TMJ disease is present, it must be treated along with treatment of any masticatory muscle TrPs that are also present. A chronic focus of infection may be a contributory factor, especially if the erythrocyte sedimentation rate and white blood cell count are elevated on repeated testing.

The lasting inactivation of myofascial TrPs in the masticatory muscles often requires skillful treatment of the TrPs, dental expertise to deal with primary TMJ dysfunction, and medical expertise to eliminate perpetuating factors of systemic and mechanical origin, including factors in the neck region and, sometimes, in the lower limbs. Often overlooked systemic perpetuating factors are low thyroid function, anemia, vitamin deficiencies, electrolyte disorders, and depression. Mechanical factors include lower limb-length inequality and painful feet due to the destabilizing Dudley J. Morton foot configuration.[53] See Chapter 4 of this volume and Chapter 20 of Volume 2 for details. One vitamin-inadequacy cause of tinnitus may be relieved by supplements of both niacinamide *and* thiamine. Restoration of normal vitamin B_{12} blood serum levels proved helpful in patients with tinnitus associated with hearing loss.[65]

Exercises

The most important and useful exercises a clinician can teach a patient with chronic head and neck pain due all or in part to myofascial TrPs is correct tongue position and body posture. These are described in detail in Chapter 5. Instruction in correct body mechanics is also essential (see Chapter 5, Section C, and Chapter 41). In addition, patients should learn general neck stretching exercises (also described in Chapter 5, Section D and Fig.16.11) to help reduce any primary TrPs in the cervical muscles which may be perpetuating the masticatory muscle TrPs.

Direct attempts to stretch the masticatory elevator muscles by simply forcing the mouth open must be avoided, as forcing the mouth open produces severe pain and reflex spasm that further aggravates the muscle tension and may injure the TMJ. In general, stretch procedures should be postponed until any **painful** TMJ arthropathy has been resolved.

The physical therapist or other clinician should give thorough instructions in a home program specific to each patient. As a part of a home program, the patient should learn TrP pressure release and strumming of the masseter to release tension prior to other exercises. An active opening effort uses reciprocal inhibition to augment relaxation of the masseter. The patient can be taught to release the muscle through lightly resisted opening of the mouth (two fingers below the chin) for a few seconds followed by active opening of the mouth to take up slack in the muscle. The amount of opening can be controlled with the tongue on the palate when needed for protection of the joint. Resisted active opening also has been recommended by others.[83] If any passive stretch is applied to the jaw, it should be done with the fingers on the posterior molars, not on the incisors. To relax the deep masseter, the patient can be taught to perform a brief *isometric* contraction, with the mouth partially open, that attempts protrusion but does not allow movement into protrusion.

When the TMJ is ready for it, yawning can be a useful range of motion exercise but must be done with the precautions identified in Section 12.

For management of patients with chronic head, facial, or neck pain that includes a myofascial TrP component, refer to the general treatment approach in Section D of Chapter 5. Use of Chapter 5, Section A, Muscle Guide, helps to identify all of the TrPs contributing to the patient's myofascial TrP pain in the head and neck.

Conclusion

Since patients are unable to accurately judge their maximum jaw opening within the functional range, an objective measure of progress is important if the patients are to reach *full* range of motion on a home exercise program and appreciate their progress. Achieving and maintaining full range of jaw opening greatly reduces the likelihood of recurrence of the TrP pain and tension. To measure the jaw aperture, the patient checks how many knuckles (or fingers) fit between the front teeth. The minimum goal is an aperture that admits a tier of the first two knuckles of the hand, as in Figure 8.3.

Bell[9] details the importance of dealing with multiple contributory factors. He recommends reduction of life situational stress and development of a positive mental attitude. An intraoral orthosis may help by temporarily offsetting muscle fatigue due to nocturnal bruxism[16,70] until the TrPs are released and the muscles are freed of TrP tension. In addition, correction of nutritional deficiencies and use of stretch exercises help to insure continued normal functioning of the muscles treated for TrPs.

SUPPLEMENTAL REFERENCES, CASE REPORTS.

A detailed case report describes acute restriction of mouth opening following a dental procedure. The patient experienced prompt pain relief and a gradual increase of jaw opening from 15 mm to 51 mm by repeated injections of 0.5% procaine solution into TrPs in the masseter and lateral pterygoid muscles.[78,79]

Another patient experienced tinnitus and "stuffiness" of the ear due to TrPs in the deep division of the masseter muscle. Procaine injection of these TrPs permanently eliminated those symptoms.[79]

REFERENCES

1. Agerberg G, Osterberg T: Maximal mandibular movements and symptoms of mandibular dysfunction in 70 year-old men and women. *Swed Dent J* 67.147-164, 1974.
2. Agur AM. *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 463, Figs. 7.12).
3. *Ibid.* (p. 494, Fig. 7.61).
4. *Ibid.* (p. 495, Fig. 7.62).
5. *Ibid.* (p. 531, Fig. 7.128; p. 532, 7.130).
6. Bailey JO Jr, McCall WD Jr, Ash MM Jr.: Electromyographic silent periods and jaw motion parameters, quantitative measures of temporomandibular joint dysfunction. / *Dent Res* 56:249-253, 1977.
7. Basmajian JV, DeLuca CJ: *Muscles Alive,* Ed. 5. Williams & Wilkins, Baltimore, 1985 (p. 452).
8. Bell WE: *Orofacial Pains-Differential Diagnosis.* Denedco of Dallas, Dallas, Texas, 1973 (p. 94, Fig. 10-1, Case 5).
9. Bell WE: *Orofacial Pains-Classification, Diagnosis, Management.* Year Book Medical Publishers, Inc., Chicago, 1985 (pp. 175, 219, 234).
10. Bell WH: Nonsurgical management of the pain-dysfunction syndrome. / *Am Dent Assoc* 79.161-170, 1969 (Cases 3 and 5).
11. Bellizzi R, Hartwell GR, Ingle JI, *et al.:* Diagnostic procedures. Chapter 9. In: *Endodontics.* Edited by Ingle JI, Bakland LK. Ed. 4. Williams & Wilkins, Baltimore, 1994, pp. 465-523 (see pp. 472-474).
12. Bessette RW, Mohl ND, Bishop B: Contribution of periodontal receptors to the masseteric silent period. *J Dent Res* 53.1196-1203, 1974.
13. Bredman JJ, Weijs WA, Brugman P: Relationships between spindle density, muscle architecture and fibre type composition in different parts of the rabbit masseter. *Eur J Morphol 29(4):297-307,* 1991.
14. Butler JH, Folke IE, Bandt CL: A descriptive survey of signs and symptoms associated with the myofascial pain-dysfunction syndrome. / *Am Dent Assoc* 90:635- 639, 1975.
15. Christensen LV: Some electromyographic parameters of experimental tooth clenching in adult human subjects. / *Oral Rehabil* 7:139-146, 1980.
16. Clark GT, Beemsterboer PL, Solberg WK, *et al.:* Nocturnal electromyographic evaluation of myofascial pain dysfunction in patients undergoing occlusal splint therapy. *J Am Dent Assoc* 99:607-611, 1979.
17. Clemente CD: *Gray's Anatomy,* Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 449).
18. *Ibid.* (p. 1165).
19. *Ibid.* (pp. 1175, 1176).
20. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 603).
21. *Ibid.* (Fig. 606).
22. *Ibid.* (Fig. 608).
23. *Ibid.* (Fig. 622).
24. *Ibid.* (Fig. 624).
25. *Ibid.* (Fig. 647).
26. Cohen HV, Pertes RA: Diagnosis and management of facial pain. Chapter 11. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 361-382).
27. Dorrance GM:. New and useful surgical procedures; the mechanical treatment of trismus. *Pa Med J* 32:545-546, 1929.
28. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (p. 198).
29. *Ibid.* (p. 204).
30. Eriksson PO: Muscle fiber composition system. *Swed Dent J 12(Suppl):8-36,* 1982.
31. Eriksson PO, Butler-Browne GS, Thornell LE: Immunohistochemical characterization of human masseter muscle spindles. *Muscle Nerve 17(1):31-41,* 1994.
32. Fields H: Pain. McGraw-Hill Information Services Company, Health Professions Division, New York, 1987, pp. 213-214.
33. Gelb H: Patient evaluation. Chapter 3. In: *Clinical Management of Head, Neck and TMJ Pain and Dysfunction.* Edited by Gelb H. W.B. Saunders, Philadelphia, 1977 (p. 82, Fib. 3-4).
34. Gibbs CH, Mahan PE, Wilkinson TM, *et al.* EMG activity of the superior belly of the lateral pterygoid muscle in relation to other jaw muscles. / *Prostbet Dent* 5i:691-702, 1983.
35. Greene CS, Lerman MD, Sutcher HD, *et al.* The TMJ pain-dysfunction syndrome, heterogeneity of the patient population. *J Am Dent Assoc* 79.1168-1172, 1969.
:ifi. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J MusculoskePain* 2(3j:29-59, 1994.
37. Ingle JI, Beveridge EE: *Endodontics.* Ed. 2. Lea & Febiger, Philadelphia, 1976 (p. 520).
38. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 9.157-164, 1989.
39. Jaeger B, Reeves JL, Graff-Radford SB: A psychophysiological investigation of myofascial trigger point sensitivity vs. EMG activity and tension headache. *Cephalalgia 5(Suppl* 3J.68-69, 1985.
40. Kellgren JH:. Observations on referred pain arising from muscle. *Clin Sci* 3:175- 190, 1938 (p. 180).
41. Kleier DJ: Referred pain from a myofascial trigger point mimicking pain of endodontic origin. / *Endod* 22f9j:408-411, 1985.
42. Kojima Y: Convergence patterns of afferent information from the temporomandibular joint and masseter muscle in the trigeminal subnucleus caudalis. *Erain Res Rull* 24(4J:609-616, 1990.
43. Laskin DM: Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 79:147- 153, 1969.
44. Lipke DP, Gay T, Gross BD, *et al.:* An electromyographic study of the human lateral pterygoid muscle. / *Dent Res* 565:230, 1977.
45. Marbach JJ: Arthritis of the temporomandibular joints. *Am Fam Phys* 29:131-139, 1979 (Fig. 9F).
46. McCall WD Jr, Goldberg SB, Uthman AA, *et al.:* Symptoms severity and silent periods, preliminary results in TMJ dysfunction patients. *NY State Dent J* 44:58-60, 1978.
47. Mclnnes B: Jaw pain from cigarette holder. *NEngl J Med* 298:1263, 1978.
48. McMinn RM, Hutchings RT, Pegington J, *et al: Color Atlas of Human Anatomy,* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 39).

49. Millstein-Prentky S, Olson RE: Predictability of treatment outcome in patients with myofascial pain-dysfunction (MPD) syndrome. / ***Dent Res*** 58.1341-1346, 1979.
50. Nteller E, Sheik-Ol-Eslam A, Lous I: Deliberate relaxation of the temporal and masseter muscles in subjects with functional disorders of the chewing apparatus. ***Scand } Dent Res*** 79:478-482, 1971.
51. Mongan E, Kelly P, Nies K, ***et al.:*** Tinnitus as an indication of therapeutic serum salicylate levels. ***JAMA*** 226:142-145, 1973.
52. Morton DJ: ***The Human Foot.*** Columbia University Press, New York, 1935.
53. Morton DJ: Foot disorders in women. / ***Am Med Worn Assoc*** 30:41-46, 1955.
54. Moyers RE: An electromyographic analysis of certain muscles involved in temporomandibular movement. ***Am JOrthod*** 36:481-515, 1950
55. Olesen J, Jensen R: Getting away from simple muscle contraction as a mechanism of tension-type headache. ***Pain*** 46:123-124, 1991.
56. Pinto O: A new structure related to the temporomandibular joint and the middle ear. / ***Prosthet Dent*** 12:95, 1962.
57. Rachlin ES: Trigger point management. Chapter 9. In: ***Myofascial Pain and Fibromyalgia.*** Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 173-195).
58. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. ***Arch Phys Med Rehabil*** 62:111-114, 1981.
59. Rocabado M, Iglarsh ZA: ***Musculoskeletal Approach to Maxillofacial Pain.*** J.B. Lippincott Company, Philadelphia, 1991.
60. Rugh JD, Solberg WK: Electromyographic studies of bruxist behavior before and during treatment. ***Calif Dent Assoc J*** 3:56-59, 1975.
61. Schwartz LL: Ethyl chloride treatment of limited, painful mandibular movement. / ***Am Dent Assoc*** 48:497-507, 1954 (Case 4).
62. Schwartz RA, Greene CS, Laskin DM: Personality characteristics of patients with myofascial pain-dysfunction (MPD) syndrome unresponsive to conventional therapy. / ***Dent Res*** 58:1435-1439, 1979.
63. Seltzer S: Dental conditions that cause head and neck pain. Chapter 7. In: ***Pain Control In Dentistry: Diagnosis and Management.*** J.B. Lippincott, Philadelphia, 1978 (pp. 105-136).
64. Sharav Y, Tzukert A, Refaeli B: Muscle pain index in relation to pain, dysfunction, and dizziness associated with the myofascial pain-dysfunction syndrome. ***Oral Surg*** 46:742-747, 1978 (p. 744).
65. Shemesh Z, Attias J, Oman M, ***et al.:*** Vitamin B_{12} deficiency in patients with chronic-tinnitus and noise-induced hearing loss. ***Am f Otolaryngol*** 24f2j:94-99, 1993.
66. Shore NA: ***Temporomandibular Joint Dysfunction and Occlusal Equilibration.*** J.B. Lippincott, Philadelphia, 1976 (pp. 61, 62).
67. Skiba TJ, Laskin DM: Masticatory muscle silent periods in patients with MPD syndrome. / ***Dent Res*** 55.B249 (Abst 748), 1976.
68. Sola AE, Bonica JJ: Myofascial pain syndromes, Chapter 21. In: ***The Management of Pain.*** Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, et al. Lea & Febiger, 1990 (pp. 352-367).
69. Solberg WK: Personal communication, 1981.
70. Solberg WK, Clark GT, Rugh JD: Nocturnal electromyographic evaluation of bruxism patients undergoing short term splint therapy. / ***Oral Rehab*** 2:215-223, 1975.
71. Solberg WK, Woo MW, Houston JB: Prevalence of mandibular dysfunction in young adults. ***J Am Dent Assoc*** 98:25-34, 1979.
72. Spalteholz W: ***Handatlas der Anatomie des Menschen.*** Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 264).
73. ***Ibid.*** (p. 267).
74. Staling LM, Fetchero P, Vorro J: Premature occlusal contact influence on mandibular kinesiology. In: ***Biomechanics V-A.*** Edited by Komi PV. University Park Press, Baltimore, 1976 (pp. 280-288).
75. Toldt C: ***An Atlas of Human Anatomy,*** translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 293).
76. ***Ibid.*** (p. 302).
77. ***Ibid.*** (p. 303).
78. Travell J: Pain mechanisms in connective tissue. In ***Connective Tissues, Transactions of the Second Conference, 1951.*** Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1952 (pp. 114, 115).
79. Travell J: Temporomandibular joint pain referred from muscles of the head and neck. / ***Prosthet Dent 10:745-763,*** 1960 (pp. 748, 750, 752-756).
80. Travell J: Mechanical headache. ***Headache*** 7:23-29, 1967 (p. 27, Fib. 7).
81. Travell J, Rinzler SH: The myofascial genesis of pain. ***Postgrad Med*** 22:425-434, 1952 (p. 427).
82. Voss DE, Ionta MK, Myers BJ: ***Proprioceptive Neuromuscular Facilitation.*** Ed. 3. Harper and Row, Philadelphia, 1985.
83. Wetzler G: Physical therapy. Chapter 24. In: Diseases ***of the Temporomandibular Apparatus.*** Edited by Morgan DH, Hall WP, Vamvas SJ. C.V. Mosby, St. Louis, 1977 (pp. 349-353, Fig. 34-2C).
84. Widman SE, Lillie JH, Ash MM Jr: Anatomical and electromyographical studies of the lateral pterygoid muscle. ***J Oral Rehabil*** 24:429-446, 1987.
85. Woelfel JB, Hickey JC, Stacey RW, er ***al.:*** Electromyographic analysis of jaw movements. / ***Prosthet Dent*** 20:688-697, 1960.
86. Wolff HG: ***Wolff's Headache and Other Head Pain,*** revised by D.J. Dalessio, Ed. 3. Oxford University Press, 1972 (p.550).
87. Wood WW, Takada K, Hannam AG: The electromyographic activity of the inferior part of the human lateral pterygoid muscle during clenching and chewing. ***Arch Oral Biol*** 32:245-253, 1986.
88. Yemm K: Temporomandibular dysfunction and masseter muscle response to experimental stress. ***Br Dent J*** 227:508-510, 1969.

CHAPTER 9
Temporalis Muscle

with contributions by
Bernadette Jaeger and Mary Maloney

HIGHLIGHTS: **REFERRED PAIN** from trigger points (TrPs) in the temporalis muscle can cause *temporal headache and maxillary toothache.* The pain pattern extends mainly over the temporal region, to the eyebrow, the upper teeth, and occasionally to the maxilla and the temporomandibular joint (TMJ). Trigger points also can refer pain, tenderness, and hypersensitivity of the upper teeth to heat and cold. **ANATOMY:** attachments are above to the temporal fossa and to the deep surface of the temporal fascia and below, to the coronoid process of the mandible. **FUNCTION** of this muscle is primarily to close the jaws. In addition, the posterior fibers, and to some extent the middle fibers, bilaterally retrude the mandible; acting unilaterally, they deviate the mandible to the same side. **SYMPTOMS** are pain over the temporal area, often hypersensitivity and aching of the upper teeth, and sometimes patients are annoyed by premature tooth contact. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** may be due to long periods of jaw immobilization (open or closed), bruxism, and clenching of the teeth. Exposure to a cold draft over the fatigued muscle, and direct trauma to the muscle can activate TrPs in it. Temporalis TrPs also may develop secondarily as satellites of key sternocleidomastoid or upper trapezius TrPs, or because of overload from tension in suprahyoid and infrahyoid muscles due to anterior head positioning or secondary to trauma. **PATIENT EXAMINATION** reveals a normal Two-knuckle Test (usually admitting *2 1/2* knuckles), but often reveals incoordinated opening and closing of the jaw, and sometimes altered occlusion of the teeth. **TRIGGER POINT EXAMINATION** of this muscle first requires that the patient allow the mouth to drop open. The central TrPs are usually found in the belly of the muscle about two fingersbreadth above the zygomatic arch. **TRIGGER POINT RELEASE** can be accomplished with several manual techniques. For spray and stretch, the patient is supine, the vapocoolant spray is directed over the muscle and its pain reference zones bilaterally, and then muscle tension is released by manual traction on the muscle with the patient allowing the mouth to relax and open. **TRIGGER POINT INJECTION** is usually more effective for central (midfiber) TrPs than for the attachment trigger points, but injection of two or all three of them may be necessary. The clinician should be careful to avoid the temporal artery. **CORRECTIVE ACTIONS** call for the elimination of mechanical and systemic perpetuating factors, and for a home program which includes correction of forward-head posture and tongue position, the Temporalis Self-stretch Exercise, an active-resistive exercise using reciprocal inhibition, and exaggerated yawning (if there is no contraindicated articular dysfunction of the TM joint).

1. REFERRED PAIN
(Fig. 9.1)

The temporalis muscle is commonly involved in patients with temporomandibular (TM) disorders—either myofascial pain-dysfunction as defined by Laskin[32] *(see* Chapter 5, Section B), or TMJ pain-dysfunction syndrome. Studies have sbown the temporalis to be involved in one-third to nearly two-thirds of the patients.[10,22,31,45]

Headache due to active trigger points (TrPs) in the temporalis muscle is common,[54] and is described as pain felt widely

throughout the temple, along the eyebrow, behind the eye, and can be felt in any or all of the upper teeth.[41,48,49,51] Temporalis TrPs also may refer hypersensitivity to percussion and to moderate temperature change that appears in any or all of the upper teeth on the same side, depending on the TrP location.[48,49] Temporalis TrP_1 (Fig. 9.1A) is an attachment trigger point (ATrP) in the anterior portion of the muscle that refers pain forward along the supraorbital ridge[55] and downward to the upper incisor teeth.[34,48,53] Attachment TrPs 2 and 3 lie in the intermediate portions of the muscle (Fig. 9.IB and C) and refer pain upward in finger-like projections to the mid-temple area and downward to the intermediate maxillary teeth on the same side.[5,7,34,44,48,53,58] fibers of the temporalis deep in the trigger point 3 region, like the deepest masseter fibers, may refer pain and tenderness to the maxilla and the TM joint.[7,48] Central TrP_4 (CTrP) in the posterior portion of the muscle refers pain backward and upward (Fig. 9.ID).[48]

Deep tenderness may be found in each of these pain reference zones even when the corresponding TrPs are latent (clinically silent with respect to pain). Sometimes toothache with hypersensitivity of the upper teeth to ordinary stimuli (biting, heat, cold) is the chief complaint, rather than headache.[48]

The anterior three TrP regions identified in Figure 9.1 are attachment TrPs that are located where one would expect to find musculotendinous junctions. The location of TrP_4 is in the central (midfiber) region of the muscle belly (Fig. 9.1D), which is characteristic of primary TrPs.

EXPERIMENTAL STUDIES

Jensen and Norup[29] compared the pain and tenderness induced by test injections (of 300 mM and 600 mM hypertonic saline or 100 mM potassium chloride) to control injections of isotonic saline into temporalis muscles of healthy volunteers. The test solutions produced significantly more pain than isotonic saline and produced significant transient depression of pain pressure threshold readings (increased tenderness). Forty eight percent of the test injections produced referral of pain, most often to the jaws. Increased pain intensity was significantly associated with the occurrence of referred pain, and with lower pressure pain thresholds at the injection site. The study by Jensen and Norup validates clinically determined referred pain patterns and substantiates the clinical impression that the likelihood of eliciting referred pain from a TrP depends on how hard one presses on it. A subsequent study[25] demonstrated a similar relationship. Hong, *et al.* found a positive correlation between the likelihood of referral of pain from a TrP and its sensitivity to applied pressure.

2. ANATOMY

(Fig. 9.2)

The temporalis muscle arises from the deep surface of the temporal fascia and from the whole of the temporal fossa,[11] superior to the zygomatic arch (Fig. 9.2). The floor of the fossa is formed by the zygomatic, frontal, parietal, sphenoid, and temporal bones.[12]

Inferiorly, the muscle attaches to the medial and lateral surfaces of the coronoid process of the mandible and along the anterior edge of the mandibular ramus, extending almost to the last molar tooth.[11] The temporalis fibers fan out anteroposteriorly from the coronoid process to form three functionally distinct groups. The anterior fibers are nearly vertical, the middle fibers oblique, and the posterior fibers nearly horizontal.[36]

Eriksson[16] reported that different parts of the temporalis muscle showed noteworthy differences in mean percentage of type I (slow twitch) fibers, suggesting differences in function. The superficial anterior and lateral parts of the muscle were 74% type I fibers and the superficial posterior part was only 52%, but the deep part averaged 90% type I fibers.[16]

SUPPLEMENTAL REFERENCES

Anatomy textbooks illustrate this muscle from the lateral view.[2, 11,13,15, 46,47]

3. INNERVATION

The temporalis muscle is supplied by the anterior and posterior deep temporal

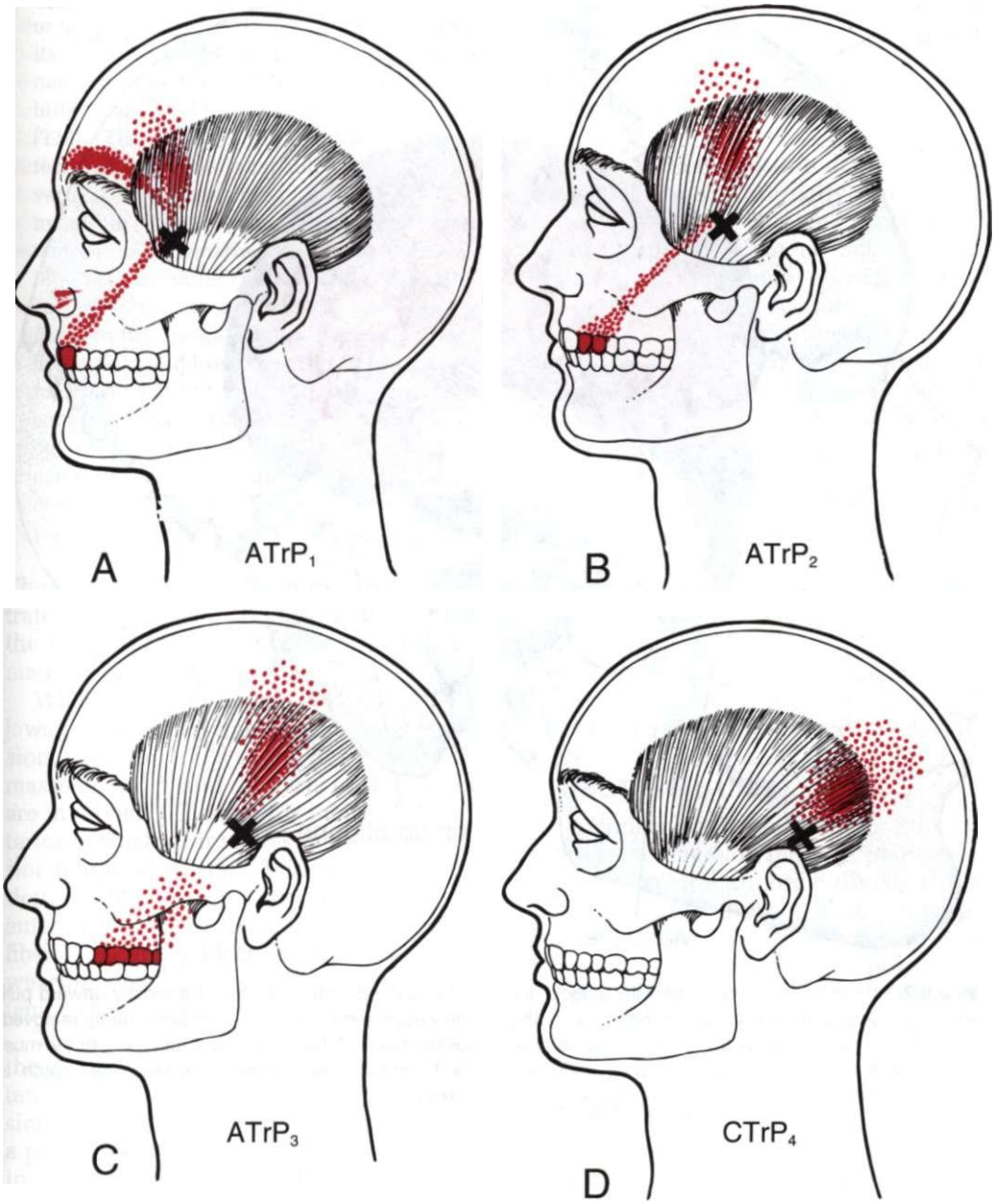


Figure 9.1. Patterns of pain and tenderness referred from trigger points (Xs) in the left temporalis muscle (essential zone *solid red,* spillover zone *stippled).* Three of the trigger points are attachment trigger points *(ATrPs)* which occur at a musculotendinous junction. One is a central trigger point *(CTrP)* which occurs in the midfiber region of the muscle. A, anterior "spokes" represent referred pain arising from ATrP, in the anterior fibers of the muscle. **B** and C, middle "spokes" represent referred pain and tenderness arising from $ATrP_2$ and $ATrP_3$. **D,** posterior supraauricular "spoke" is referred from $CTrP_4$.

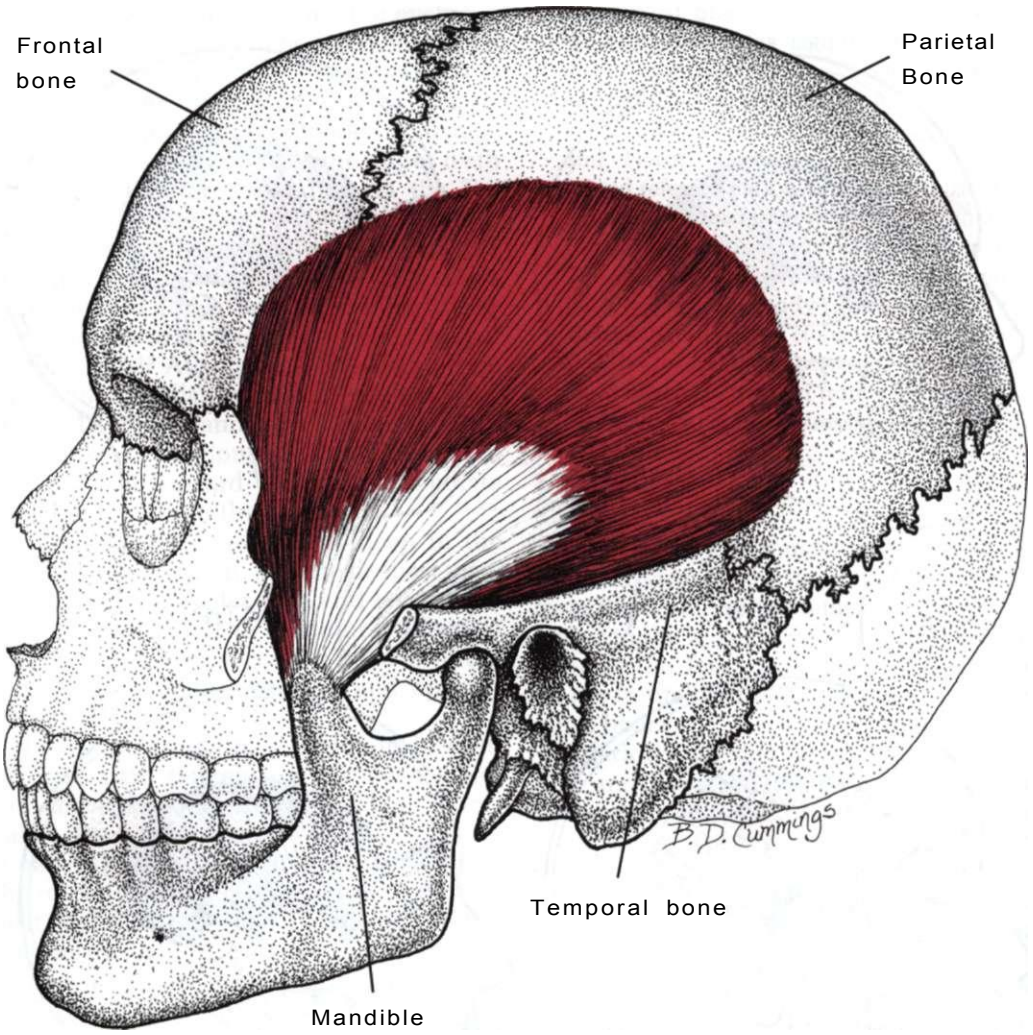


Figure 9.2. Attachments of the temporalis muscle, inferiorly, are chiefly to the coronoid process of the mandible and, superiorly, to the temporal fossa. The anterior fibers of this fan-shaped muscle are nearly vertical, and the posterior fibers are nearly horizontal but change direction and exert a mostly upward pull, The zygomatic arch, which has been partly removed, covers much of the tendinous attachment of the muscle to the coronoid process unless the mouth is opened.

nerves, which branch from the anterior division of the mandibular portion of the trigeminal nerve (cranial nerve V).

4. FUNCTION

All fibers of the temporalis muscle contribute to its primary function of elevation (closure) of the mandible. Sarnat and Laskin[43] state in their text that the posterior fibers

> **. . . should be able to retract the mandible because of their horizontal orientation along the side of the skull; however, these fibers** ***are bent around the posterior root of the zygomatic arch and thus are oriented essentially in a vertical man-***

ner. Therefore, this portion of the temporalis muscle exerts primarily an upward or vertical force on the mandible. Because its fibers pass close to the articular eminence it probably also functions as a stabilizer of the temporomandibular joint (TMJ). The middle oblique portion of the temporalis muscle is capable of exerting a vertical and retracting force on the mandible. That portion of the anterior temporalis originating from the postorbital septum pulls the mandible upward and slightly forward. Finally, the deep fibers of the anterior temporalis that originate along and just above the infratemporal crest pull the mandible upward and somewhat medially. Thus, the morphology of the entire temporalis muscle indicates that its fibers are capable of considerable variability in their direction of pull.[43]

These anatomical features are clearly illustrated by Agur.[2] In some upright postures, the temporalis may function to *keep* the mandible in the rest position.[4]

When the mandible is closed and the jaws are clenched tightly in centric occlusion, the temporalis is activated before the masseter,[37,38,56] and all parts of the muscle are involved.[36] Closure for incisor bite (anterior occlusion) involves mainly the anterior temporal fibers.[37] With normal dentition, gentle closure activates mainly the anterior fibers,[37] or the anterior and middle fibers.[4] If the subject is edentulous and wearing dentures, all three parts of the temporalis contract equally.[4]

The posterior fibers, much more than the middle or anterior fibers, are consistently activated during retraction (retrusion) of the mandible.[4,36,37,56] Bruxism with a posterior thrust of the mandible strongly involves these posterior fibers.[1]

Lateral movements to the same side regularly activate the temporalis,[4] particularly its posterior fibers, more than its anterior fibers.[56] These lateral movements always involve the posterior fibers if the lower jaw is not protruded at the same time. Protrusion conflicts with the retraction function of the posterior temporalis fibers, and therefore inhibits activity in them.[36]

The temporalis muscle was reported to be generally inactive during straight protrusion[4,56] but was active in 5% of efforts,[36] probably to counteract the depressor effect of the primary protruder, the inferior division of the lateral pterygoid muscle.

The important question of whether temporalis motor units normally show activity at rest is clearly resolved only in the supine position, when no activity is observed.[35] The presence of resting motor unit activity in the erect subject is controversial;[4,52] activity is reported as greater in the posterior fibers than in the anterior ones.[4,37] Basmajian and DeLuca[4] state that the temporalis alone is the muscle responsible for keeping the mandible in the rest position during upright posture. Yemm[57] found no activity in repeated recordings of three temporalis muscles in seated subjects at rest with head and trunk erect. These differing conclusions could result from the variation in the rest position, differences in the degree of anxiety-induced muscle tension, variations in electrode technique, head position, and the presence of latent TrPs in the masticatory musculature.

5. FUNCTIONAL UNIT

Synergists of the temporalis for mandibular elevation include, ipsilaterally: the masseter, the superior division of the lateral pterygoid, and the medial pterygoid muscles. Contralaterally, the synergists are the same muscles plus the temporalis.

Antagonists are the inferior division of the lateral pterygoid, digastric, omohyoid and mylohyoid muscles.

6. SYMPTOMS

Patients with temporalis TrPs may complain of head pain, toothache or tooth site pain, as described in Section 1, but are rarely aware of any restriction of jaw opening, which is usually reduced only by 5-10 mm (about 3/8 in). Thus, ordinary mandibular movement does not cause pain. The patients may say, "My teeth don't meet right." If maxillary toothache is a symptom, it may be intermittent, with or without hyperalgesia to percussion, hot

and cold.[48] Because of the potential for attendant hypersensitivity in teeth in the referred site of pain, unaware clinicians may needlessly extirpate pulps or extract perfectly healthy teeth.[49]

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Recent literature indicates no substantial evidence that moderate changes (4-6 mm) in occlusal vertical dimension cause masticatory muscle hyperactivity or the symptoms of temporomandibular disorder.[39]

Trauma and Immobilization

Temporalis TrPs may be activated by bruxism and clenching the teeth, by direct trauma to the muscle, as from a fall on the head, impact from a golf ball or baseball, or impact of the head against the side of the car in a motor vehicle accident, by prolonged jaw immobilization as for an extensive dental procedure, or by cervical traction for neck pain without using an occlusal splint. In the last situation, without a dental splint, the cervical traction immobilizes the mandible in the *fully* closed position, maximally shortening the temporalis and other mandibular elevator muscles. Iatrogenic temporalis TrPs may then add the symptoms of facial pain, toothache, and possibly altered occlusion to the original complaint of neck pain or headache. Sometimes, neck traction has been ordered unnecessarily for neck pain and headache that were caused primarily by TrPs in the upper trapezius, a condition for which traction provides no relief.

Postural and Activity Stress

The mandibular position induced by a **forward-head posture** (see Chapter 5, Section C) produces increased activity in the temporalis muscle and can activate and/or perpetuate TrPs in it. Excessive gum chewing or jaw clenching may activate, and likely perpetuate, masticatory muscle TrPs, including the temporalis.

Bruxism may cause, or result from, temporalis TrPs. In either case, the overuse of the muscle aggravates and perpetuates these TrPs. Restlessness of the masticatory muscles can result from increased neuromuscular irritability due to folic acid deficiency. This restlessness may be expressed as bruxism, and it is comparable to the restlessness of the biceps femoris and calf muscles that is induced by folate deficiency and is known as "restless legs."[9]

An anteriorly displaced TM joint disc may cause the patient to experience a feeling of pressure. In an attempt to do something to relieve the sense of pressure the patient may bite down, which does not correct the disc problem and only perpetuates temporalis (and masseter) TrPs.[33]

Other Factors

Often overlooked or forgotten is the reflex muscle contraction that occurs with any chronic infection or inflammation. When prolonged, this is believed to contribute to the development of myofascial TrPs.[18] Thus, true painful pulpal pathology or an inflamed TM joint, if protracted, may cause temporalis (or other masticatory muscle) TrPs to develop. These TrPs become self-sustaining, and, even after resolution of the pulpal pathology or inflamed joint, may continue to cause intermittent or constant pain, typically referred back to the original site of pain. The unaware clinician, unfortunately, will continue to treat the tooth or the joint instead of the TrPs, with potentially disastrous results.

Excessive tension in suprahyoid and infrahyoid muscles can create light tensile forces which pull down on the mandible. The temporalis and masseter muscles contract to counteract the pull and keep the mouth closed, and TrPs can be activated and/or perpetuated in these muscles. This dysfunctional process can be initiated, for example, when flexion-extension injuries sustained in an automobile accident overload or stress the suprahyoid and infrahyoid muscles; it also can be initiated or perpetuated by an excessive forward-head position.

Especially when the patient is fatigued, temporalis TrPs may be activated by a cold draft over the muscle (e.g., a blast of cold air from a ventilator or air conditioner, or wind through an open car window).[48] Persons with low-normal serum levels of thyroid hormones (T_3 and T_4 by radioim-

munoassay), as well as those clearly hypothyroid, are particularly vulnerable to such muscle cooling.

The temporalis muscle TrPs may be activated as satellites when they lie within the pain reference zone of active TrPs in the upper trapezius and sternocleidomastoid muscles.

Active TrPs in lower limb muscles have been observed to indirectly cause a reduction of maximal interincisal opening, and thus may influence masticatory muscle function; this is an example of dysfunction set up by dynamic and static postural asymmetries, in this instance originating in a weightbearing limb.

8. PATIENT EXAMINATION

Prior to beginning a physical examination, the clinician must take a thorough patient history *(see* Chapter 3 introduction), including a review of habitual body mechanics *(see* Chapter 5, Section C, and Chapter 41).

The examiner should perform a screening examination of the TM joints and should assess the patient's posture, with particular attention to head and neck position *(see* Chapter 5, Section C). Forward-head posture and excessive tension in suprahyoid and infrahyoid muscles should be noted.

The patient performs the Two-knuckle Test *(see* Fig. 8.3) by attempting to place a tier of the proximal interphalangeal joints of the first two fingers of the non-dominant hand between the upper and lower incisor teeth. Usually, about 2 1/2 knuckles of jaw opening can be reached if the temporalis muscle, but not the masseter, is involved. When the posterior fibers of the temporalis harbor active trigger points (TrPs), the mandible is likely to show zigzag deviation during opening and closing of the mouth. Refer to Chapter 5, Section C for details regarding measurement of jaw opening.

9. TRIGGER POINT EXAMINATION
(Fig. 9.3)

Central trigger points (CTrPs) can be found near midfiber in various portions of this muscle; attachment trigger points (ATrPs) can be found at musculotendinous junctions above the zygomatic arch and also at the attachment of the tendon to the coronoid process of the mandible.

The jaws must be *partly* (not fully) open to place the muscle fibers on the degree of stretch required to optimize the palpation of the temporalis TrPs. When the jaws are closed and the muscle is fully shortened and slack, its palpable bands are more difficult to feel; they are less tender, and the local twitch response to snapping palpation may be unobtainable. When the patient allows the jaw to drop in the relaxed open position, it takes up the slack for examination of this muscle (Fig. 9.3). The ATrPs are most likely regions of enthesopathy secondary to the sustained abnormal tension of the taut bands produced by CTrPs. The anterior three regions identified in Figure 9.1 are located in the musculotendinous junction region. If the clinician locates one of these three ATrPs in a taut band and palpates back along the band, it is usually possible to find a corresponding CTrP a few centimeters craniad in the midfiber portion of the same taut band (about 2 fingersbreadth above the zygomatic arch).

The location identified as TrP_4 (Fig. 9.ID) is a TrP in the midfiber region of the posterior portion of the muscle belly and is found above and slightly behind the ear. There is apparently a close relation between the trigger area at a musculotendinous junction and its corresponding central TrP. The presence of one tends to activate the other, and pressure on either frequently can produce much the same referred pain pattern.

Examination of the temporalis muscle for the enthesopathy of the ATrPs is not complete until the insertion region is palpated externally beneath the zygomatic process when the patient's mouth is open and also is palpated internally on the inner surface of the coronoid process from within the mouth. The technique for the internal palpation is similar to that used for examining the inferior division of the lateral pterygoid muscle[30] *(see* Chapter 11, Section 9) except that for the temporalis insertion, pressure is directed outward against the coronoid process, rather than inward toward the pterygoid plate.

Across-the-fiber snapping palpation at TrPs elicits local twitch responses that are

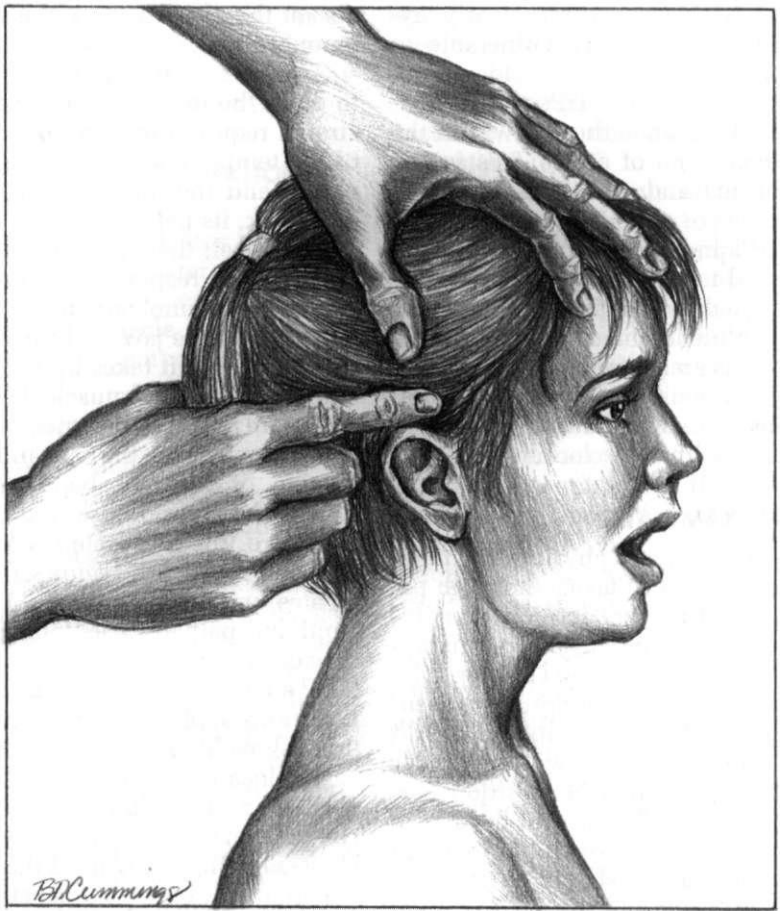


Figure 9.3. Examination of the trigger points in the posterior portion of the temporalis muscle. For examination of all portions, the patient should allow the lower jaw to drop into the relaxed open position to take up slack in the muscle. This accentuates firm bands of muscle fibers, increases the spot tenderness and referred pain of a trigger point to pressure, and increases the sensitivity of the trigger point response to snapping palpation that tests for a local twitch of the taut band fibers.

often *felt* more readily than seen in this muscle.

10. ENTRAPMENT

The temporalis muscle is not known to cause nerve entrapment.

11. DIFFERENTIAL DIAGNOSIS

Other conditions cause symptoms that can appear confusingly similar to those produced by temporalis TrPs or may be present concurrently. Concurrent non-painful disorders may include TMJ internal derangements (*see* Chapter 5, Section C). Painful disorders include diseased teeth, tension-type headaches, cervicogenic headaches, polymyalgia rheumatica, temporal arteritis, and temporal tendinitis.

Grating detected by manual palpation or auscultation with a stethoscope over the TM joints during opening and closing of the jaw or chewing movements, may indicate **internal derangement** of the TMJ *(see* Chapter 5, Section C). Grating sounds alone do not contraindicate treatment of the muscle by stretch, but if joint palpation or movement is painful this calls for expert dental and TMJ examination[21] and may require referral. Clinical evidence indicates that the sustained tension imposed on the

TMJ by TrPs in masticatory muscles may induce TMJ derangement[19] possibly by increasing intra-articular pressure, which in turn may cause a disc, already compromised by thinning posteriorly, to slip forward and become displaced anteromedially *(see* Chapter 5, Sections B and C).

A **diseased tooth** such as one with a nonrestorable carious lesion can produce referred pain over the temporalis muscle that closely emulates the referred pain from a TrP in that part of the muscle.[3]

The common diagnoses of **tension-type headache**[28] and **cervicogenic headache**[26] are very likely to have a significant pain component originating in temporalis TrPs.

The head pain of **polymyalgia rheumatica** is distinguished from that due to temporalis and trapezius TrPs by (1) the more extensive distribution of the *bilateral* polymyalgia pain, which usually includes the shoulders,[8] and often the neck, back, upper arms, and thighs;[23] (2) by the increased erythrocyte sedimentation rate, usually of at least 50 mm/hr and even 100 mm/hr, which is evidence of inflammation with increases in fibrinogen and in the a_2-globulin fraction; and (3) by anemia due to blocked utilization of iron.

The diagnosis of **temporal tendinitis** can be based on tendon-attachment enthesopathy that results from TrPs in the temporalis muscle. The clinician should examine for that possibility before proceeding with palliative care or steroid injections, or worse, a more drastic surgical procedure such as excising the condylar attachment of the muscle.[17] If temporalis TrPs are responsible for the symptoms, inactivating them is much simpler, less invasive, less painful to the patient, and less expensive.

Related Trigger Points

Temporalis muscle TrPs are likely to be associated with TrPs in the ipsilateral masseter (deep division) and in the contralateral temporalis muscle. Less commonly, either or both the medial and lateral pterygoid muscles may be involved, sometimes bilaterally.

Satellite TrPs often develop in the temporalis muscle from key TrPs in the commonly involved upper trapezius[24] and from key TrPs in the sternocleidomastoid muscle.

12. TRIGGER POINT RELEASE
(Figs. 9.4 and 9.5)

Forward-head posture and tongue position should always be corrected first when identified as a problem *(see* Chapter 5, Section C), and the patient should be instructed in *maintaining* good head/neck posture and tongue position. Sometimes, trigger point (TrP)-referred pain will clear up following correction of these two powerful perpetuating factors alone.

When the temporalis has shortened in association with an occlusal abnormality, such as a retrognathic overbite, the muscle should be stretched to its normal resting length *before* fitting dental appliances, so that they can be adjusted and work properly. Correct neutral head position is also critical during adjustment of any appliances. If the head is in extension in the dental chair, the occlusion will be different than when the patient is sitting or standing with correct head and neck alignment.

Because multiple mandibular elevator muscles are likely to develop interacting TrPs, it can be helpful to start with the combined release described in Chapter 8 (Fig. 8.5). If reexamination reveals residual TrPs in individual muscles, those TrPs are more likely to respond to therapy directed specifically to that muscle.

Myofascial TrPs in the temporalis muscle can be released effectively by a number of techniques. These include spray and stretch (Fig. 9.4), self-stretch of the muscle with augmentation techniques (Fig. 9.5), TrP pressure release, and reciprocal inhibition through voluntary opening of the mouth.

Spray and Stretch

To spray and stretch the temporalis muscle, the supine position is preferable (Fig. 9.4). However, the patient may sit in a low-backed armchair (or in a dental chair), reclining the head backward against the operator or headrest to tilt the face upward and reduce postural reflexes.[20, 35] The patient is encouraged to relax.

The vapocoolant spray or icing is applied *bilaterally* from the attachment of the muscle on the coronoid process upward to cover the muscle fibers and all referred

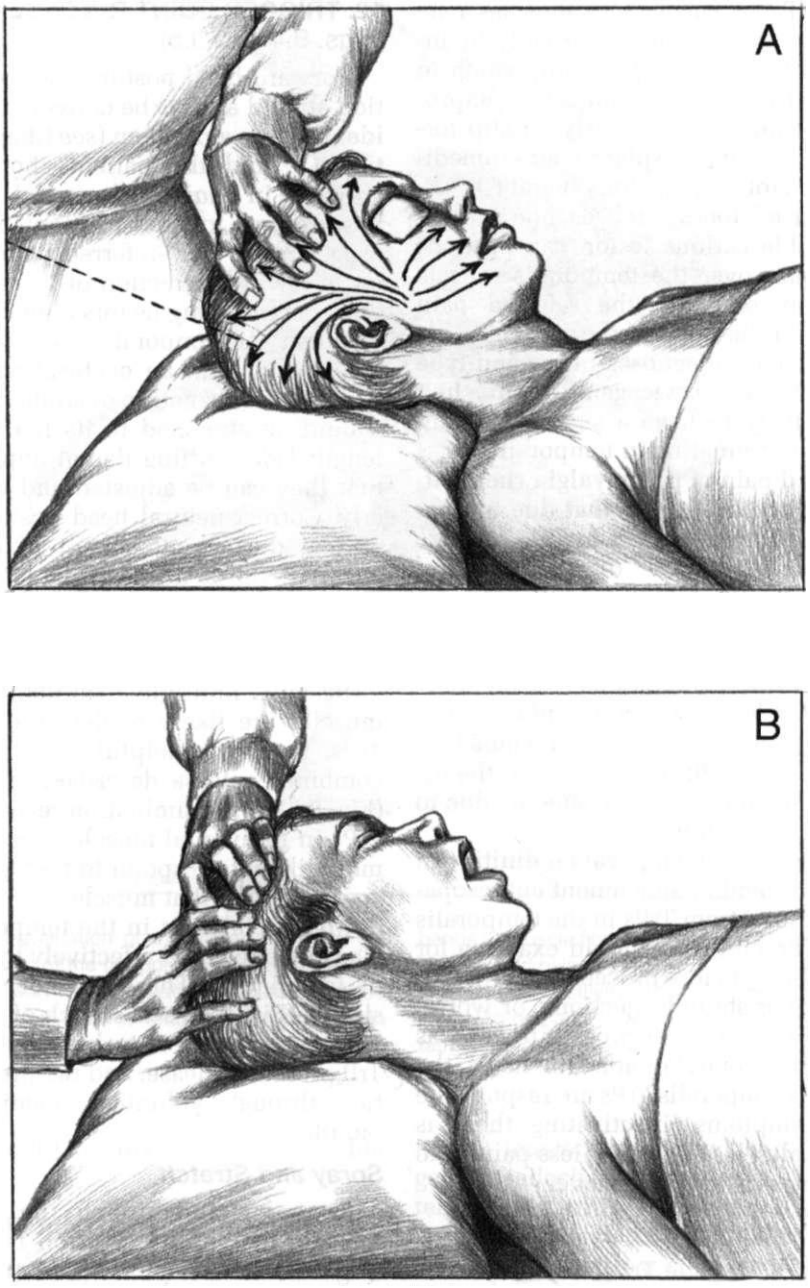


Figure 9.4. Spray and Stretch of the temporalis muscle, patient supine. A, The patient allows the jaws to relax. With the left hand, the operator takes up slack in the temporalis muscle by pulling upward, and with the right hand applies a stream of vapocoolant in the pattern shown, carefully including the entire muscle, its attachments, and its referred pain pattern. Spray should be applied bilaterally, even if only one side is symptomatic. Refer to text for precautions. B, After setting aside the spray dispenser, the operator uses both hands and applies upward traction on the superior and posterior portions of the temporalis muscle to passively elongate the fibers. Then the patient inhales, opening the mouth as far as comfortably possible to further elongate the temporalis muscle. The patient then exhales and allows the mouth to close. The stretching phase is repeated until no further gain in range of motion occurs or until the mouth opens to full normal range of motion. Otherwise, the spray phase shown in A may be repeated. Refer to Figure 8.5 for combined stretch of the temporalis and masseter muscles.

Figure 9.5. Self-stretch of the temporalis muscle. The jaw elevator muscles are elongated by the patient opening the mouth to the comfortable fully opened position. With the fingers spread apart, the patient presses firmly in the upward direction just above the temples and over the ears, stretching the temporalis muscle while taking in a long full breath to augment muscle relaxation. The spray pattern shown in Figure **9.4A** can be used if stretch is to be preceded by application of vapocoolant.

pain areas as shown in Figure 9.4A.[48] The patient's eyes should be protected with dry cotton swabs or pieces of gauze to prevent any of the irritating vapocoolant liquid from splashing into the eyes. Patients with asthma or other respiratory conditions may not tolerate the spray because of the vapors. Ice may be used as an alternate form of intermittent cold *(see* Chapter 3). If spray is used, a small cloth or a hand should lightly cover the patient's nose and mouth. Stretch is applied as described and illustrated in Figure 9.4B. Increase in the jaw opening should be measured and called to the patient's attention.

After a hot pack application to the face, stretch and spray may be repeated. Following stretch and spray, the patient should open and close the mouth fully (but not forcibly) a few times to restore normal muscle function. This whole procedure may be repeated several times at 5-minute intervals (rewarming each time) until no further release occurs. The minimum normal opening for persons of average stature is close to 40 mm in adult men and women. The patient normally should be able to insert a tier of two knuckles between the margins of the incisor teeth *(see* Fig. 8.3).

The patient should practice the technique of self-stretch as illustrated and described in Figure 9.5 to be used at home.

Other Considerations

Therapy of the temporalis muscle for TrPs is not complete until all active TrPs in the upper trapezius and sternocleidomastoid muscles also have been inactivated. The TrPs in the latter two neck muscles can *indirectly* restrict mandibular opening. In addition, Hong[24] found that inactivating the key TrP in the upper trapezius muscle also inactivated a satellite TrP in the temporalis muscle.

When the temporalis and other muscles of mastication are involved with TrPs, they are usually involved bilaterally; because the mandible is connected across the midline, one side cannot be treated without an effect on the other. Therefore, the clinician needs to consider the implications of treatment for the muscles and TM joints on both sides, even if only one side is symptomatic.

When treatment is unsuccessful or the relief lasts only a short time, in addition to considering other muscles of the functional unit, one may look for excessive tension in suprahyoid and infrahyoid muscles, and then release that tension if needed *(see* Chapter 12).

The direct manual techniques that do not require spray and stretch are described in detail in Chapter 3, Section 12. Reciprocal inhibition through voluntary opening of the mouth is described in Chapter 8, Section 12.

Joint play should be restored when it is restricted.

13. TRIGGER POINT INJECTION (Fig. 9.6)

With correction of poor posture, body mechanics, and tongue position, and the elimination of abusive oral habits, many masticatory muscle trigger points (TrPs) will resolve spontaneously. Similarly, attention and appropriate resolution of TrPs in the upper trapezius and sternocleidomastoid muscles will often help to resolve masticatory muscle TrPs, including the temporalis. If, after the above, and after application of temporalis spray and stretch or manual techniques, temporalis TrPs still need specific attention, then TrP injection is an option.

Before injecting temporalis TrPs, the operator first eliminates as many TrPs as possible through spray and stretch and manual techniques described above. In addition, any TrP tension in the masseter muscle should be eliminated to avoid inducing bleeding in the temporal region. Tautness of masseter fibers can entrap venous drainage from the temporalis muscle *(see* Chapter 8, Section 10). If the masseter tension is not released, the patient is more likely to develop a large ecchymosis and a "black eye" following the temporalis TrP injection; the patient should be warned of this possibility.

The lower jaw may be held open to locate the TrP precisely, as for examination (Fig. 9.3). The temporal artery should be identified by its pulsations, and avoided (Fig. 9.6). Using a sterile technique, the needle is directed away from the artery, or angled under it, to avoid puncturing it, as also noted by Bell.[6] After locating the temporalis TrPs by palpation, one finger is placed on the artery to continuously monitor its location, while other fingers localize and fix the TrP for injection. Injecting the midfiber central TrP is usually more effective than injecting the corresponding attachment TrP. It may be necessary to inject both areas for complete prompt relief.

A 2.5-cm (1-in), 23- or 24-gauge needle is used to inject the TrPs with a local anesthetic in an upward direction between the fingers. A 27-gauge needle is too flimsy unless the fast-in, fast-out technique of Hong[24] is used *(see* Chapter 3, Section 13). We recommend 0.5% procaine without epinephrine for intramuscular injection *(see* Chapter 3, Section 13). Alternatively, the 1% lidocaine is much preferred to the 3% mepivacaine and is supplied in the convenient 1.8-ml dental syringes.

Immediately after the injection, maximal passive stretch of the muscle is carried out (Fig. 9.4) while applying vapocoolant spray *bilaterally. A* hot pack follows, then active range of jaw motion. If the range of opening is still restricted, stretch and spray to the temporalis muscle may be repeated bilaterally, after rewarming, to achieve an additional increment of jaw opening.

A similar TrP injection technique is well described and illustrated by dentists.[14]

14. CORRECTIVE ACTIONS

Exercise Program

The clinician should instruct the patient in correct tongue position and body posture. This is described in detail in Chapter 5. Instruction in good body mechanics is also essential *(see* Chapter 5 and Chapter 41). In addition, most patients need to learn general neck stretching exercises (also described in Chapter 5 and illustrated in Fig. 16.11) to help inactivate any key TrPs in the cervical muscles that may be perpetuating the temporalis TrPs.

The patient learns how to passively stretch the temporalis in the supine position by doing the Temporalis Self-stretch Exercise daily (Fig. 9.5). Before this exercise is done, the patient may apply a hot pack over the temporalis muscle, covering the side of the head and face for 10-15 minutes before retiring at night. Alternatively, a wool scarf or sweater over the muscle can provide neutral warmth (keeping body heat in) and comfort.

When the patient is comfortable with this passive exercise, the next step is an active-resistive mouth-opening exercise which helps to overcome restricted motion

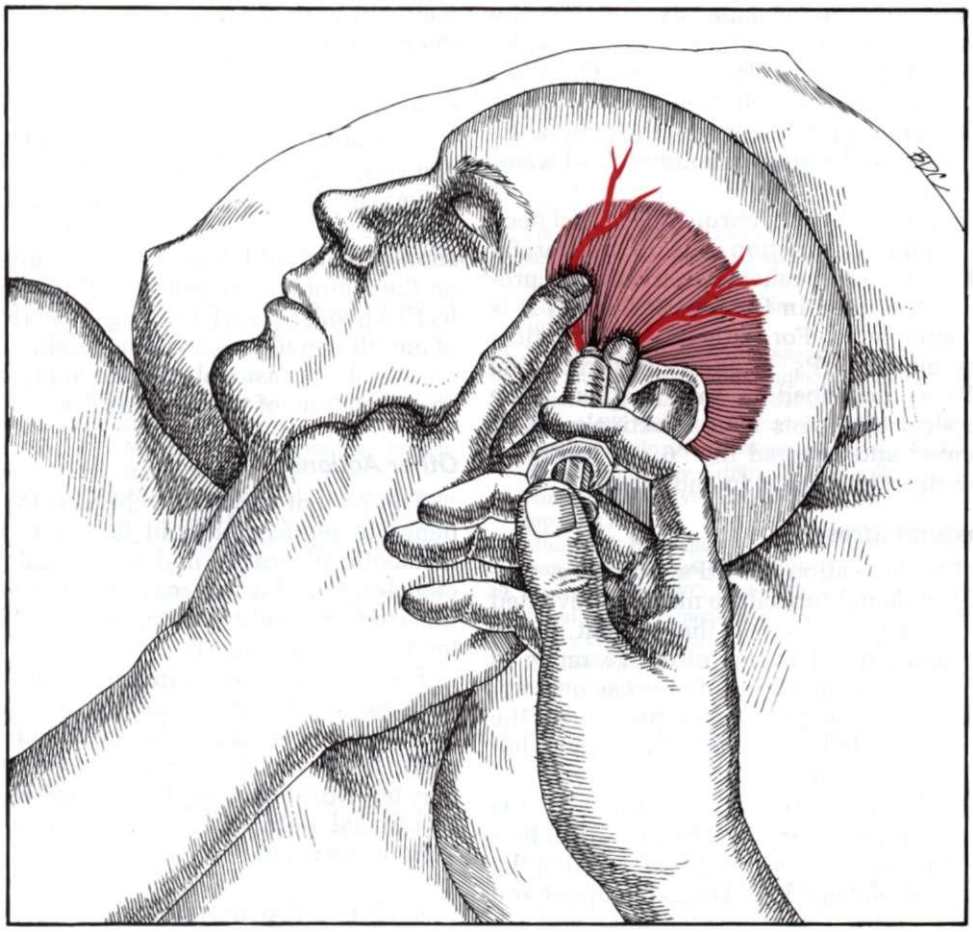


Figure 9.6. Injection of a trigger point in the anterior portion of the temporalis muscle (*light red*). The temporal artery *(dark red)* is avoided. A finger is placed on the pulsating artery to continuously monitor its location, while other fingers localize a trigger point in a taut band and fix it for injection.

through reciprocal inhibition. The patient can release the muscle by lightly resisting opening of the mouth (with two fingers below the chin) for a few seconds, followed by active opening of the mouth to take up slack in the muscle. The amount of opening can be controlled with correct tongue position on the palate *(see* Chapter 5, Section C). This protective maneuver is recommended for patients with TM joint inflammation or painful TM joint derangements (i.e., disc displacement with reduction), so that they will stretch within nonpainful limits or avoid the painful click.[27]

If the posterior fibers of the temporalis muscle are involved, causing the mandible to deviate on opening, the patient must modify this exercise: the patient opens the jaw to stretch while first placing one hand against the opposite maxilla (contralateral to the involved temporalis) and the other hand against the ipsilateral side of the mandible. The lower jaw is pushed away from the side toward which it deviates during opening, while the patient actively assists the motion with the jaw muscles for the most effective stretch. The mandible is gently restored to the starting position before pressure is fully released. When full relief is obtained, the exercises may be reduced to two or three times weekly as a health maintenance measure and be incorporated into a regular post-exercise muscle stretching routine.

If there is no articular dysfunction, the patient is encouraged to induce a wide-open yawn as a regular exercise. The addition of this reflex inhibition helps to obtain full normal stretch length of the temporalis muscle (and other mandibular elevator muscles).

For patients with **chronic** head and neck pain and dysfunction due to myofascial TrPs, the comprehensive treatment program described in Chapter 5, Section D is recommended. For patients with masticatory muscle TrPs, in addition to releasing TrPs as described in this chapter, some physical therapists have effectively incorporated and adapted the "6 X 6" program described by Rocabado and Iglarsh.[40]

Postural Stress

The activation of TrPs during a prolonged dental procedure may be prevented by taking breaks for the patient to go through several cycles of active range of motion with the addition of occasional application of vapocoolant spray over the muscle while the mouth is fully open, but not forced open.

Prolonged maximal shortening of the muscle during sleep may be prevented by a "night guard" or occlusal splint with a flat occlusal plane, which keeps the upper and lower teeth a few millimeters apart and can relieve bruxism. This is especially helpful during periods of high stress.[42] Tongue positioning on the roof of the mouth can help relieve bruxism. A dental splint also should be used during prolonged cervical traction, especially in the patient who has a history of headache.

Body asymmetry and the resultant functional scoliosis should be corrected by appropriate lifts, since this postural stress may activate TrPs in the neck muscles that cause satellite TrPs in the masticatory muscles. If the habit of mouth breathing produces forward-head posture, the mouth breathing should be corrected by eliminating contributory factors, such as nasal obstruction.

Pillow positioning can be of critical importance if the patient likes to sleep on the side and the jaw is allowed to drop laterally for long periods of time. Placement of a corner of the pillow under the jaw and over the shoulder as in Figure 7.7C will maintain normal jaw alignment and avoid the problem.

Activity Stress

The patient should be persuaded to stop chewing gum, eating caramels, biting a pen or pencil, chewing tough meat, and cracking nuts or ice with the teeth. The patient should avoid cold drafts that blow directly on the temple by wearing a night cap, protective hood, or scarf. Prolonged restriction of mouth opening by a face mask should be relieved by occasional removal of the mask and stretching of the jaw muscles.

Other Actions

The patient should be checked for evidence of reduced thyroid function, other metabolic disorders, and nutritional deficiencies, any of which may increase neuromuscular irritability, as described in Chapter 4, Sections C and D.

Because of postural influences through the base of support, elimination of TrP activity in the muscles of the neck, and even of the lumbosacral region and lower limbs, may be critical for *complete* lasting relief of myofascial pain and dysfunction due to TrPs in the masticatory muscles.

SUPPLEMENTAL REFERENCE, CASE REPORT

The diagnosis and management of a patient with temporalis involvement is given in a case report by Travell.[50]

REFERENCES

1. **Adams SH II: Personal communication, 1981.**
2. **Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 496, Fig. 7.64).**
3. **Ailing CC: Personal communication, 1985.**
4. **Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 262, 448-452).**
5. **Bell WE: *Orofacial Pains-Differential Diagnosis.* Denedco of Dallas, 1973 (p. 94, Fig. 10-1).**
6. **Bell WE: Management of masticatory pain. Chapter 12. In: *Facial Pain.* Ed. 2. Edited by Ailing CC III, Mahan PE. Lea & Febiger, Philadelphia, 1977 (pp. 185, 188).**
7. **Bell WH: Nonsurgical management of the pain-dysfunction syndrome. / *Am Dent Assoc* 79.161-170, 1969 (pp. 165, 169, Case 5).**
8. **Bird HA, Esselinckz W, Dixon A, *et al.*: An evaluation of criteria for polymyalgia rheumatica. *Ann Rheum Dis* 38:434-439, 1979.**

9. Botez MI, Fontaine F, Botez T, *et al.:* Folate-responsive neurological and mental disorders: report of 16 cases. *Eur Neurol* 16:230-246, 1977.
10. Butler JH, Folke LE, Bandt CL: A descriptive survey of signs and symptoms associated with the myofascial pain-dysfunction syndrome. / *Am Dent Assoc* 90:635-639, 1975.
11. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 449, Fig. 6-9).
12. *Ibid.* (p. 160).
13. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 608).
14. Cohen HV, Pertes RA: Diagnosis and management of facial pain, Chapter 11. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 361-382 (see p. 378).
15. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (p. 204).
16. Eriksson PO: Muscle fiber composition system. *Swed Dent J 12(suppl)^-3S,* 1982.
17. Ernest EA, Martinez ME, Rydzewski DB, et al.: Photomicrographic evidence for insertion tendinosis: The etiologic factor in pain for temporal tendonitis. *fProsthet Dent* 65:127-131, 1991.
18. Fields H: Pain. McGraw-Hill Information Services Company, Health Professions Division, New York, 1987 (pp.213-214).
19. Freese AS: Myofascial trigger mechanisms and temporomandibular joint disturbances in head and neck pain. *NY State f Med* 59:2554-2558, 1959 (Fig. 1).
20. Funakoshi M, Amano N: Effects of the tonic neck reflex on the jaw muscles of the rat. *J Dent Res* 52:668-673, 1973.
21. Gelb H: Patient evaluation. Chapter 3. In: *Clinical Management of Head, Neck, and TMJ Pain and Dysfunction.* Edited by Gelb H. W.B. Saunders, Philadelphia, 1977 (pp. 73- 116).
22. Greene CS, Lerman MD, Sutcher HD, *et al.:* The TMJ pain-dysfunction syndrome: heterogeneity of the patient population. *J Am Dent Assoc* 79:1168-1172, 1969.
23. Healey LA: Polymyalgia rheumatica. Chapter 50. In: *Arthritis and Allied Conditions.* Ed. 8. Edited by Hollander JL, McCarty DJ Jr. Lea & Febiger, Philadelphia, 1972 (pp. 885-889).
24. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. / *Musculoske Pain* 2(lj:29-59, 1994.
25. Hong CZ, Chen YN, Twehous D, Hong DH: Pressure threshold for referred pain by compression on the trigger point and adjacent areas. / *Musculoske Pain 4(3)m-79,* 1996.
26. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 9.157-164, 1989.
27. Jaeger B: Personal communication, 1997.
28. Jaeger B, Reeves JL, Graff-Radford SB: A psychophysiological investigation of myofascial trigger point sensitivity vs. EMG activity and tension headache. *Cephalalgia 5(Suppl* 3J:68, 1985.
29. Jensen K, Norup M: Experimental pain in human temporal muscle induced by hypertonic saline, potassium, and acidity. *Cephalalgia* 22f2j:101-106, 1992.
30. Johnstone DR, Templeton M: The feasibility of palpating the lateral pterygoid muscle. / *Prosthet Dent* 44:318-323, 1980.
31. Kaye LB, Moran JH, Fritz ME: Statistical analysis of an urban population of 236 patients with head and neck pain. Part II. Patient symptomatology. / *Periodontal* 50:59-65, 1979 (p. 61).
32. Laskin DM: Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 79:147-153, 1969.
33. Maloney M: Personal communication, 1995.
34. Marbach JJ: Arthritis of the temporomandibular joints. *Am Fam Phys* 29:131-139,1979 (p. 137, Fig. 9E).
35. M0ller E, Sheik-Ol-Eslam A, Lous I: Deliberate relaxation of the temporal and masseter muscles in subjects with functional disorders of the chewing apparatus. *Scand J Dent Res* 79:478-482, 1971 (p. 481).
36. Moyers RE: An electromyographic analysis of certain muscles involved in temporomandibular movement. *Am J Orthod* 36:481-515, 1950.
37. Munro RR: Electromyography of the muscles of mastication. In: *The Temporomandibular Joint Syndrome.* Edited by Griffin CJ, Harris R. Vol. 4. of *Monographs in Oral Science.* S. Karger, Basel, 1975 (pp. 87-116).
38. Munro RR, Basmajian JV: The jaw opening reflex in man. *Electromyography* 3 J.191- 206, 1971.
39. Rivera-Morales WC, Mohl ND: Relationship of occlusal vertical dimension to the health of the masticatory system. *J Prosthet Dent* 65:547-553, 1991.
40. Rocabado M, Iglarsh ZA: *Musculoskeletal Approach to Maxillofacial Pain.* J.B. Lippincott Company, Philadelphia, 1991.
41. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Rehabil* 62:107-110, 1981.
42. Rugh JD, Solberg WK: Electromyographic studies of bruxist behavior before and during treatment. *Calif Dent Assoc J* 3:56-57, 1975.
43. Sarnat BG, Laskin DM (eds): *The Temporomandibular Joint: A Riological Rasis for Clinical Practice.* Ed. 4. W.B. Saunders Co., Philadelphia, 1992.
44. Shaber EP: Considerations in the treatment of muscle spasm. Chapter 16. In: *Diseases of the Temporomandibular Apparatus.* Ed 2. Edited by Morgan DH, House LR, Hall WP, Vamvas SJ. C.V. Mosby, St. Louis, 1982 (p. 281, Fig. 16-2B).
45. Sharav Y, Tzukert A, Refaeli B: Muscle pain index in relation to pain, dysfunction, and dizziness associated with the myofascial pain-dysfunction syndrome. *Oral Surg* 46:742- 747, 1978 (Table 1).
46. Spalteholz W: *Handatlas der Anatomica des Menschen.* Ed. 11, Vol. 2, S. Hirzel, Leipzig, 1922 (p. 265).
47. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul, Ed. 2, Vol. 1. MacMillan, New York, 1919 (p. 306).
48. Travell J: Temporomandibular joint pain referred from muscles of the head and neck. / *Prosthet Dent* 10:745-763, 1960 (pp. 748-749, Figs. 3, 13).
49. Travell J: Mechanical headache. *Headache* 7:23-29, 1967 (p. 26).
50. Travell J: Identification of myofascial trigger point syndromes: a case of atypical facial neuralgia. *Arch Phys Med Rehabil* 62:100-106, 1981.
51. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 2 2:425-434, 1952 (p. 247).
52. Vitti M, Basmajian JV: Muscles of mastication in small children: an electromyographic analysis. *Am J Orthod* 68:412-419, 1975.

53. Wetzler G: Physical therapy, Chapter 24. In: *Diseases of the Temporomandibular Apparatus.* Edited by Morgan DH, Hall WP, Vamvas SJ. C.V. Mosby, St. Louis, 1977 (pp. 356, Fig. 24-4).
54. Williams HL: The syndrome of physical or intrinsic allergy of the head: myalgia of the head (sinus headache). *Proc Staff Meet Mayo Clin 20:177-183,* 1945 (p. 281).
55. Williams HL, Elkins EC: Myalgia of the head. *Arch Phys Ther* 23:14-22, 1942 (pp. 18, 19).
56. Woelfel JB, Hickey JC, Stacey RW, *et al:* Electromyographic analysis of jaw movements. / *Pros-thetDent* 20:688-697, 1960.
57. Yemm R: The question of "resting" tonic activity of motor units in the masseter and temporal muscles in man. *Arch Oral Biol* 22:349, 1977.
58. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little Brown & Company, Boston, 1988 (Fig. 12-1).

CHAPTER 10
Medial Pterygoid Muscle

with contributions by
Bernadette Jaeger and Mary Maloney

HIGHLIGHTS: **REFERRED PAIN** from this muscle can appear as a vague ache in the back of the mouth and pharynx, below and behind the temporomandibular joint (TMJ), and deep in the ear. **ANATOMY:** the medial pterygoid muscle spanning between the angle of the mandible and the lateral pterygoid plate on the inside of the jaw forms a sling with the masseter muscle on the outside of the jaw. Together, the two muscles suspend the mandible. **FUNCTION:** Unilateral contraction of the medial pterygoid muscle causes primarily lateral deviation of the mandible to the opposite side. With bilateral contraction it assists in elevation of the mandible and also can assist in protrusion. **SYMPTOMS** caused by active trigger points (TrPs) in this muscle are throat pain, difficulty in swallowing, and painful, moderately restricted jaw opening. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in this muscle can be secondary to lateral pterygoid TrP involvement. The muscle is rarely involved alone. **PATIENT EXAMINATION** usually reveals deviation of the incisal path, generally to the contralateral side, as maximum mouth opening is approached and reveals some restriction of opening. **TRIGGER POINT EXAMINATION** should include palpation for central TrPs with the finger inside the mouth and palpation for attachment TrPs from outside of the mouth. **TRIGGER POINT RELEASE** is usually successful with spray and stretch and other myofascial techniques if active TrPs in other masticatory muscles and in the neck muscles are also inactivated. **TRIGGER POINT INJECTION** may be approached by a needle inside or outside the mouth, but may not be necessary after TrP release techniques have been applied. **CORRECTIVE ACTIONS** include correction of forward-head posture, inactivation of other masticatory TrPs, and self-stretch exercises.

1. REFERRED PAIN
(Fig. 10.1)

The medial pterygoid muscle refers pain in poorly circumscribed regions related to the mouth (tongue, pharynx, and hard palate), below and behind the temporomandibular joint (TMJ), including deep in the ear, but not to the teeth (Fig. 10.1).[8,42,43] Other authors also have found that pain can be referred to the retromandibular and infra-auricular area,[7,22] including the region of the lateral pterygoid muscle, the floor of the nose, and the throat.[37] Patients describe pain from the medial pterygoid as being more diffuse than the pain referred from trigger points (TrPs) in the lateral pterygoid muscle.

Stuffiness of the ear may be a symptom of medial pterygoid TrPs. In order for the tensor veli palatini muscle to dilate the eustachian tube, it must push the adjacent medial pterygoid muscle and interposed fascia aside. In the resting state, the presence of the medial pterygoid helps to keep the eustachian tube closed. Tense myofascial TrP bands in the medial pterygoid muscle may block the opening action of the tensor veli palatini on the eustachian tube producing barohypoacusis (ear stuffiness). Medial pterygoid tenderness was

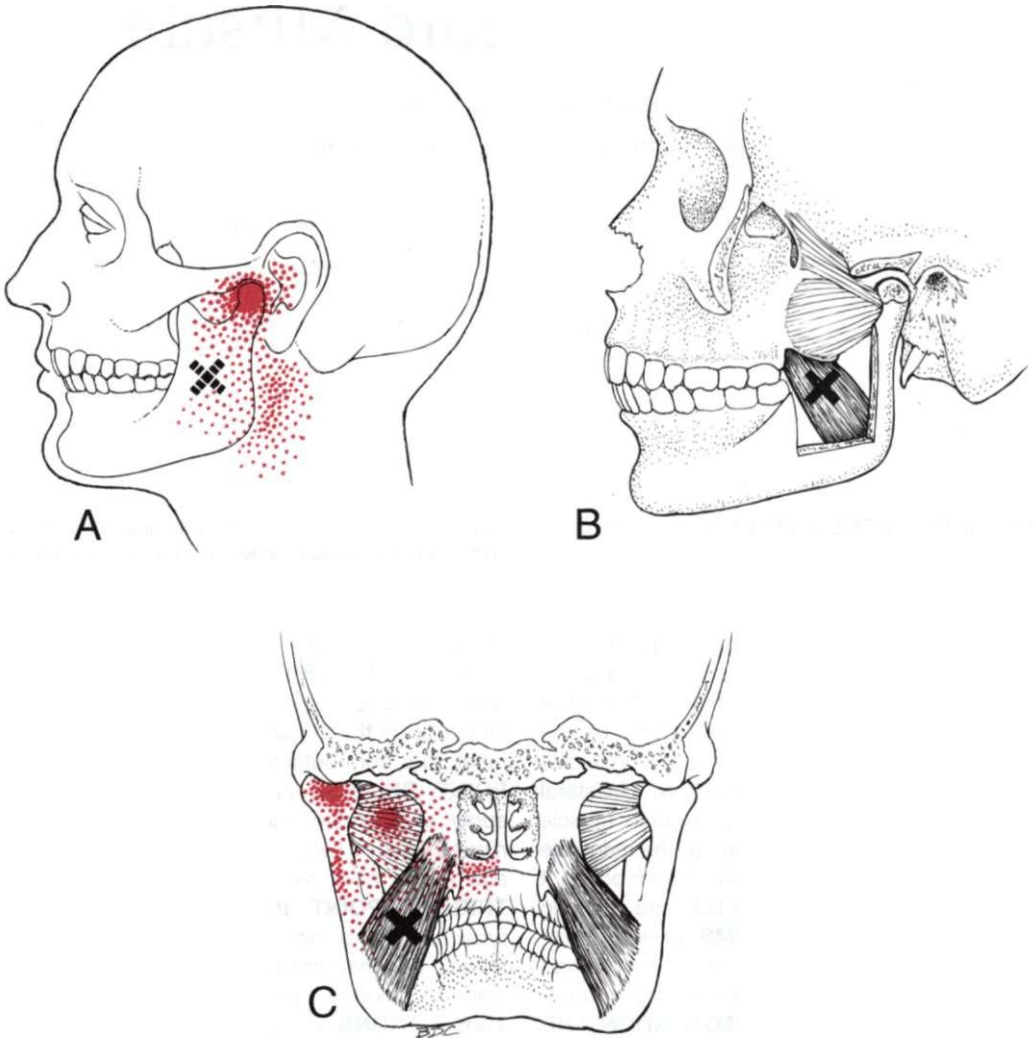


Figure 10.1. Referred pain pattern (red) and location of the responsible trigger point **(X)** in the left medial pterygoid muscle. **A,** external areas of pain to which the patient can point. **B,** anatomical cut-away to show the location of the trigger point area in the muscle, which lies on the inner side of the mandible. **C,** coronal section of the head through the temporomandibular joint, showing an inside view looking forward. Internal areas of pain also appear as stippled red.

confirmed in all 31 patients who were examined and who had this symptom.[1]

2. ANATOMY
(Fig. 10.2)

The medial pterygoid muscle on the inside of the mandible and the masseter muscle on the outside together suspend the angle of the mandible, like a sling. The bulk of the medial pterygoid (Fig. 10.2, *dark red)* attaches *above* to the medial (inner) surface of the lateral pterygoid plate of the sphenoid bone. The inferior division of the lateral pterygoid muscle (Fig. 10.2A, *light red)* attaches to the lateral (outer) surface of the same lateral pterygoid plate of the sphenoid bone.

A small portion of the medial pterygoid muscle often attaches to the lateral surface of the palatine bone, passing over the lateral surface of the lateral pterygoid plate, and thus covers the lower end of the infe-

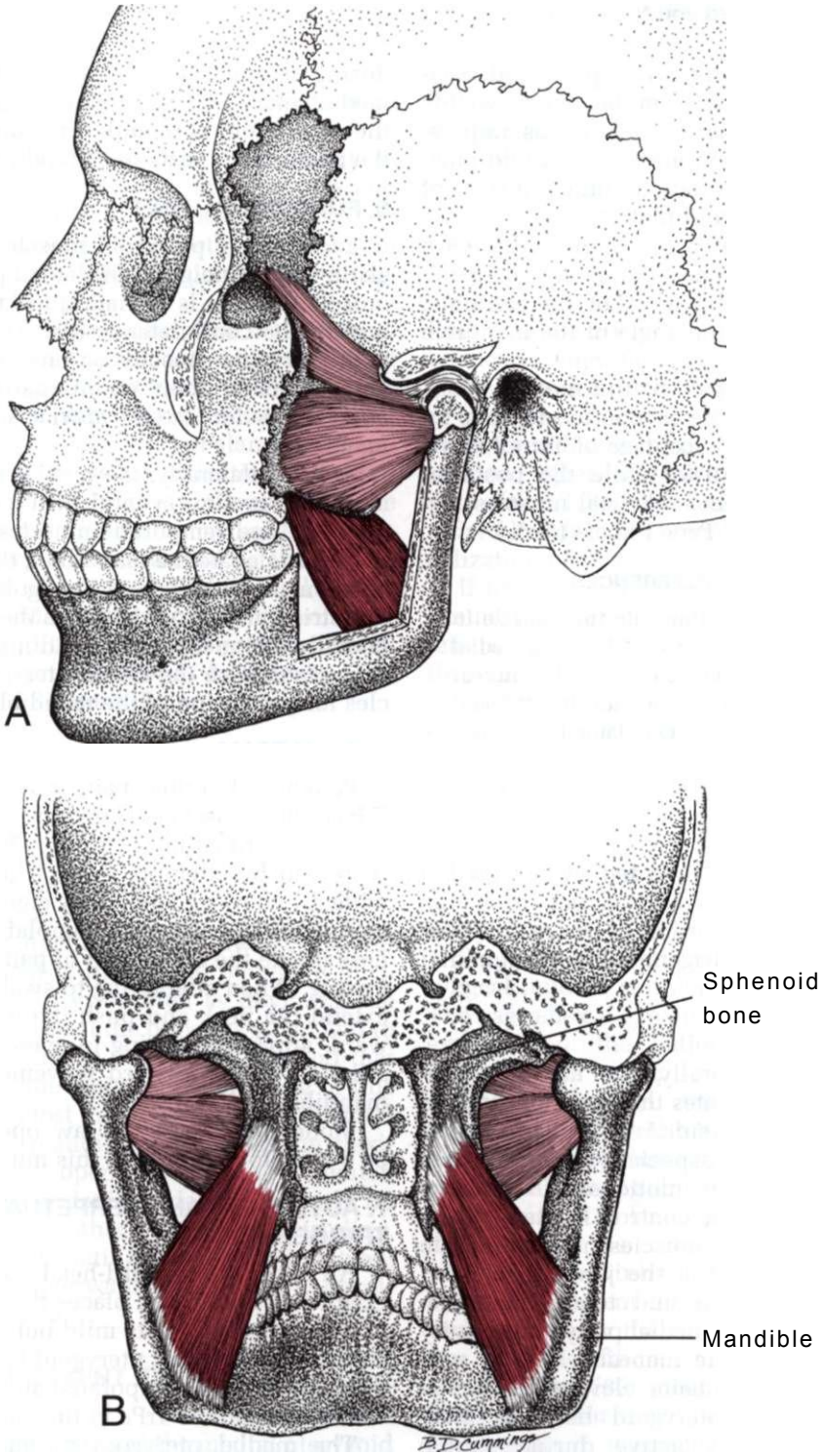


Figure 10.2. Attachments of the medial pterygoid muscle *(dark red)* and its relation to the lateral pterygoid muscle *(light red).* **A,** lateral view showing the medial pterygoid muscle on the inner side of the mandible. Part of the mandible and the zygomatic arch have been removed. **B,** coronal section of the skull just behind the temporomandibular joint, looking forward inside the mouth. The medial pterygoid muscle attaches, above, to the medial *(inner)* surface of the lateral pterygoid plate of the sphenoid bone and, below, to the medial surface of the mandible near its angle.

rior division of the lateral pterygoid muscle. In illustrations from the side view, this variation can give the erroneous impression that the entire medial pterygoid muscle attaches to the lateral (outer) surface of the lateral pterygoid plate.[30]

The medial pterygoid muscle attaches ***inferiorly*** by a short aponeurosis to the lower border of the ramus of the mandible, in the region of the angle of the mandible (Fig. 10.2B).

The anterior part of the medial pterygoid muscle has been reported to have an unusually high percentage of Type I (slow twitch) fibers (79%) while the posterior part was, as in most skeletal muscles, approximately half Type I fibers (52%).[20]

SUPPLEMENTAL REFERENCES

Other authors illustrate this muscle in the lateral (side) view,[13,15,16,30,41] in medial view (from inside the mouth),[31731] in rear view (from inside the mouth),[19,32,39,40] and in cross section.[2,18] One lateral view also shows the overlying pterygoid venous plexus.[16]

3. INNERVATION

The muscle is supplied by the medial pterygoid nerve which arises from the common, fused portion of the mandibular division of the trigeminal nerve (cranial nerve V).

4. FUNCTION

Acting unilaterally, the medial pterygoid muscle deviates the mandible toward the contralateral side.[5,6,25,46] This lateral motion would be especially important during the grinding motions of chewing, which require fine control. Bilaterally, the medial pterygoid muscles help to elevate the mandible (close the jaws) in concert with the masseter and temporalis muscles.[5,6,13,25,46] The medial pterygoid activity is increased if the mandible also is protruded while it is being elevated.[33]

The medial pterygoid becomes electromyographically active during simple protrusion of the mandible, especially if the jaws are only slightly apart,[6] but the activity is less intense if the mandible is voluntarily depressed.[33] Protrusion by the medial pterygoid usually would be inhibited during mandibular depression since the medial pterygoid is a major antagonist to the opening motion and, for protrusion of the mandible, assists the lateral pterygoid.

5. FUNCTIONAL UNIT

Each medial pterygoid muscle is synergistic with its neighboring lateral pterygoid muscle when it is deviating the mandible toward the contralateral side. As a result, both pterygoid muscles on one side act as antagonists to their counterparts on the other side for lateral deviation of the mandible.

Acting bilaterally, the medial pterygoid muscles function synergistically with the masseter and temporalis muscles to close the jaws (elevate the mandible); they act as antagonists to the lateral pterygoid and the digastric muscles, which open the jaws. Bilaterally, the medial pterygoid muscles are synergistic with the lateral pterygoid muscles for protrusion of the mandible.

6. SYMPTOMS

Patients describe pain referred from TrPs in this muscle as shown in Figure 10.1 and as described in Section 1. This pain is increased by attempts to open the mouth wide, by chewing food, or by clenching the teeth. Patients also may complain of soreness inside the throat and of painful swallowing. When attempting to swallow, they extend the neck and push the tongue forward, apparently trying to overcome a restriction in the forward movement of the mandible.

Moderately restricted jaw opening can be a symptom of TrPs in this muscle.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

An excessive forward-head posture (see Chapter 5, Section C) places the mandible in a position that puts mild but persistent stress on the medial pterygoid (along with the masseter and temporalis) and can activate or perpetuate TrPs in this muscle.

The medial pterygoid muscle on one side may develop and retain active TrPs because of the increased stress imposed on it by TrP activity and distorted function of the corresponding muscle on the opposite side. Activation and perpetuation of me-

dial pterygoid TrPs can be secondary to the muscular dysfunction that results from TrPs in the lateral pterygoid muscle.

Sucking of the thumb after infancy or excessive gum chewing may activate and perpetuate TrPs in this muscle. Bruxism (lateral grinding of the teeth), clenching of teeth, anxiety, and emotional tension are common factors.

A less common cause for activation of TrPs is the sustained contraction of medial pterygoid spasm, activated reflexly by cellulitis in the pterygomandibular space.[10]

In the past, occlusal imbalance was considered one cause for activation of medial pterygoid TrPs. It is now thought that the abnormal muscle tension caused by TrPs in masticatory muscles including the medial pterygoid often cause the occlusal abnormalities. Masticatory muscle myofascial TrPs should be inactivated prior to initiating any prosthodontic treatment. *(See* Chapter 5, Section B).

8. PATIENT EXAMINATION

With active medial pterygoid TrPs, the mandibular opening is usually obviously restricted,[8] so that the jaw aperture may not admit two knuckles *(see* Two-knuckle Test, Chapter 8).

During opening of the jaws, unilateral involvement of the medial pterygoid muscle is variously reported as deviating the mandible toward the opposite side,[8] and to the same side, or not at all.[35] We find that deviation due mainly to shortening of this muscle is most marked to the contralateral side as the mandible approaches the maximum mouth opening. The side to which the mandible deviates depends greatly on how severely other protruding, retruding, and lateral-deviating muscles are involved; one medial pterygoid muscle rarely develops TrPs alone.

9. TRIGGER POINT EXAMINATION
(Fig. 10.3)

For examination of the medial pterygoid muscle, the supine patient allows the jaw to drop open as far as is comfortable in order to take up any slack in the muscle. **Palpation for central TrPs in the midmuscle region is performed with gloved fingers intraorally** with the mouth open (Fig. 10.3A). The pad of the palpating index finger faces outward and slides over the molar teeth until it encounters the bony anterior edge of the ramus of the mandible, which lies behind and lateral to the last molar tooth. The belly of the medial pterygoid muscle lies immediately beyond (posterior to) this bony edge. This technique also is illustrated by others.[22,26,36] The muscle can be clearly identified by having the patient alternately clench and relax against a block or cork placed between the teeth while the operator palpates for the changes in tissue tension. When the medial pterygoid harbors active TrPs, digital pressure elicits exquisite tenderness, permitting precise localization of them.

If there is concern for the safety of the examining finger, the block or cork can be left in place between the patient's teeth throughout the TrP examination.

The orientation and texture of this muscle are readily palpable because only a thin layer of mucosa separates the palpating finger from the muscle. Usually one must palpate through thick skin and more subcutaneous tissue including fat. Taut bands are more readily identified and less pressure is required to elicit TrP tenderness than for many muscles.

Palpating this muscle through the pharyngeal mucosa can make the patient gag. The gag reflex is greatly reduced if, during examination, the patient either exhales fully or takes a deep breath,[1] and holds it during examination. Another technique is to tap the ipsilateral temporalis muscle to provide sensory distraction during the examination. Having the patient curl the tip of the tongue as far as possible down the throat behind the molar teeth on the opposite side further inhibits the gag reflex. The harder the patient forces the tongue backward and down the throat, the less sensitive the reflex becomes. Application of a quick acting (30 seconds) topical anesthetic spray for mucous membranes such as Cetacaine® (Cetylite Industries, Inc.), can be used to anesthetize the pharynx if necessary to eliminate the gag reflex in hypersensitive individuals.

To palpate for mandibular attachment TrPs from outside the mouth, the head is

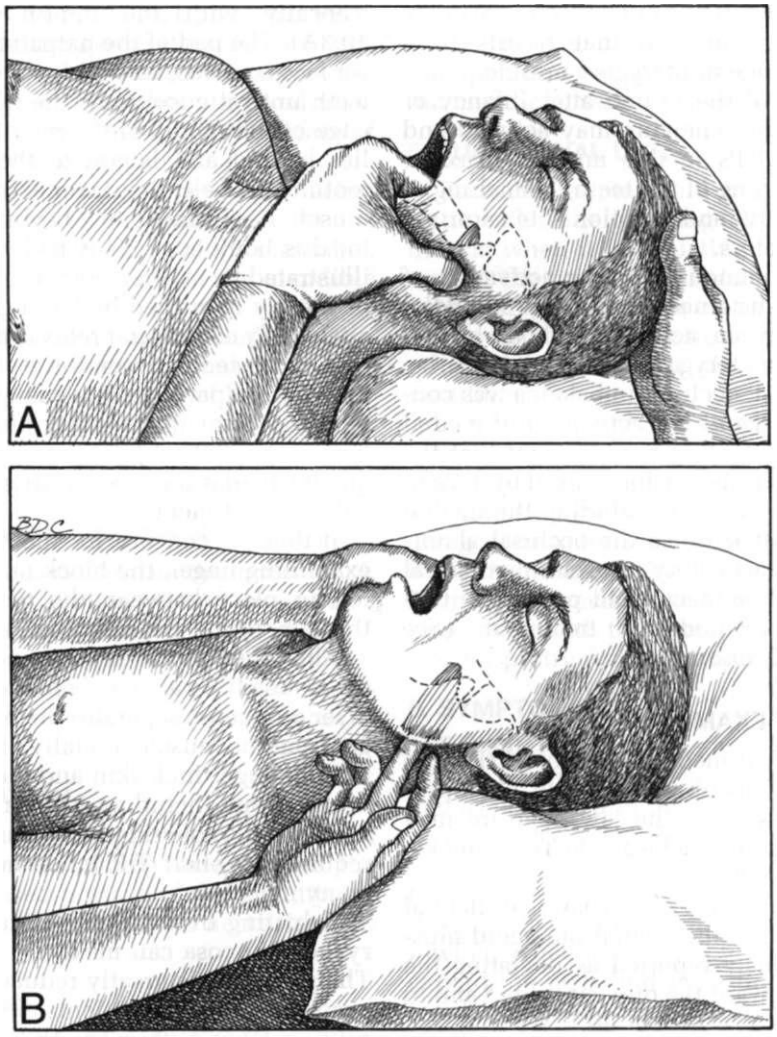


Figure 10.3. Examination of the medial pterygoid muscle for trigger points. **A,** intraoral palpation (with a gloved hand) of trigger points behind the last molar tooth, with the muscle and the ramus of the mandible between the palpating digits. The mouth is opened wide enough for the finger to be placed between the molar teeth. The examiner may wish to prop the mouth open with a cork to protect the finger and help the patient to relax. **B,** extraoral palpation of attachment trigger points in the region of the attachment of the muscle to the inner surface of the mandible, at its angle.

tilted slightly toward the side to he palpated in order to slacken tissues and improve access to the muscle. One finger examines the inner (medial) surface of the mandible by pressing upward at its angle (Fig. 10.3B).[11,47] The firm mass, approximately 1 cm (3/8 in) above the angle of the mandible, just within reach of the finger, is the inferior part of the mandibular attachment of the muscle.

Studies indicate that this muscle is seldom involved alone and is less likely to be tender than are most of the other masticatory muscles.[12,24,38]

10. ENTRAPMENT

A case report by Kahn[28] suggested that the medial pterygoid muscle may have been entrapping the chorda tympani portion of the lingual nerve as it passes be-

tween the medial pterygoid muscle and the mandible,[14] causing an extremely bitter metallic taste that interfered with normal oral functioning. Temporary splints and then fixed bridges that opened the bite approximately 3 mm resolved the problem.

11. DIFFERENTIAL DIAGNOSIS

The medial pterygoid muscle usually develops TrPs in association with functionally related muscles, especially the lateral pterygoid and masseter, as noted in Section 5. Pain in the throat deep behind the angle of the jaw can be caused by a TrP in the posterior lateral part of the tongue on the same side and should be suspected if there is no evidence of TrP activity in the medial pterygoid muscle.

If the patient continues to have difficulty in swallowing following the inactivation of medial pterygoid TrPs, the sternocleidomastoid *(see* Chapter 7), the digastric, and possibly the longus capitis and longus colli muscles (*see* Chapter 12), should be examined for TrPs.

12. TRIGGER POINT RELEASE
(Fig. 10.4)

Important considerations in treatment are to correct forward-head posture when present, encourage correct tongue position, and reduce any abusive jaw habits *(see* Chapters 5 and 41, Section C). Chapter 5 also describes how to identify TMJ disorders that require special consideration.

A number of manual treatment techniques are available for treating trigger points (TrPs) in this muscle. They include spray and stretch, spray and pressure release, and strumming (a form of TrP pressure release), for direct release techniques; postisometric relaxation and resisted jaw opening (using reciprocal inhibition), for increasing vertical range of motion. Physical therapists trained in the use of electrical stimulation have used high-voltage galvanic stimulation to effectively release masticatory muscle TrPs, including those in the medial pterygoid.[20] Ultrasound has also been found by physical therapists to be beneficial for reduction of pain and release of TrP tension, usually applied for 2 minutes at 0.8 watts/cm^2.[29] Each of these techniques is described in Chapter 3, Section 12, and some are described in more detail later in this section. Emphasis on slow, nonforced respiration can augment muscle release with any technique.

Before applying any technique that depends on increased mandibular opening, it is wise to first restore any loss of TM joint play. In the vertical direction, the mandible can be depressed gently 1-2 mm straight downward in long axis distraction. To do this, the clinician places the thumbs on the patient's posterior molar teeth and very lightly presses down, which opens the TM joint (separates the joint surfaces). Any passive movement that is achieved with gentle pressure can be augmented with nonforced exhalation by the patient. If restriction is associated with joint pain or tenderness, mobilization is contraindicated by anyone other than TMJ specialists. Refer to Chapter 5, Section C, under Range of Motion, for precautions and detailed information regarding hypomobility in this joint.

A further potentially complicating factor may be cervical joint dysfunction if present. Most patients will respond to the simple strategies listed in this section even if they have cervical dysfunction. However, if the dysfunction and TrP pain persist after posture correction and after good patient compliance in correction of other perpetuating factors to elevator and cervical muscle TrPs, then referral to a practitioner familiar with evaluation and treatment of both cervical dysfunction and myofascial TrPs should be considered.

Spray and Stretch

The medial pterygoid is stretched along with the masseter and temporalis when the **combined spray-and-stretch release** is applied as shown in Figure 8.5. The specific spray pattern for the medial pterygoid is illustrated here in Figure 10.4A. Be aware that both sides of the face should be sprayed or stroked with ice *prior* to initiating any jaw opening stretches, since one side cannot be stretched in isolation from the other. One should apply caution when using spray near the nose area, especially in patients with asthma and other respiratory conditions. The clinician's hand can cover the patient's nose lightly while spray

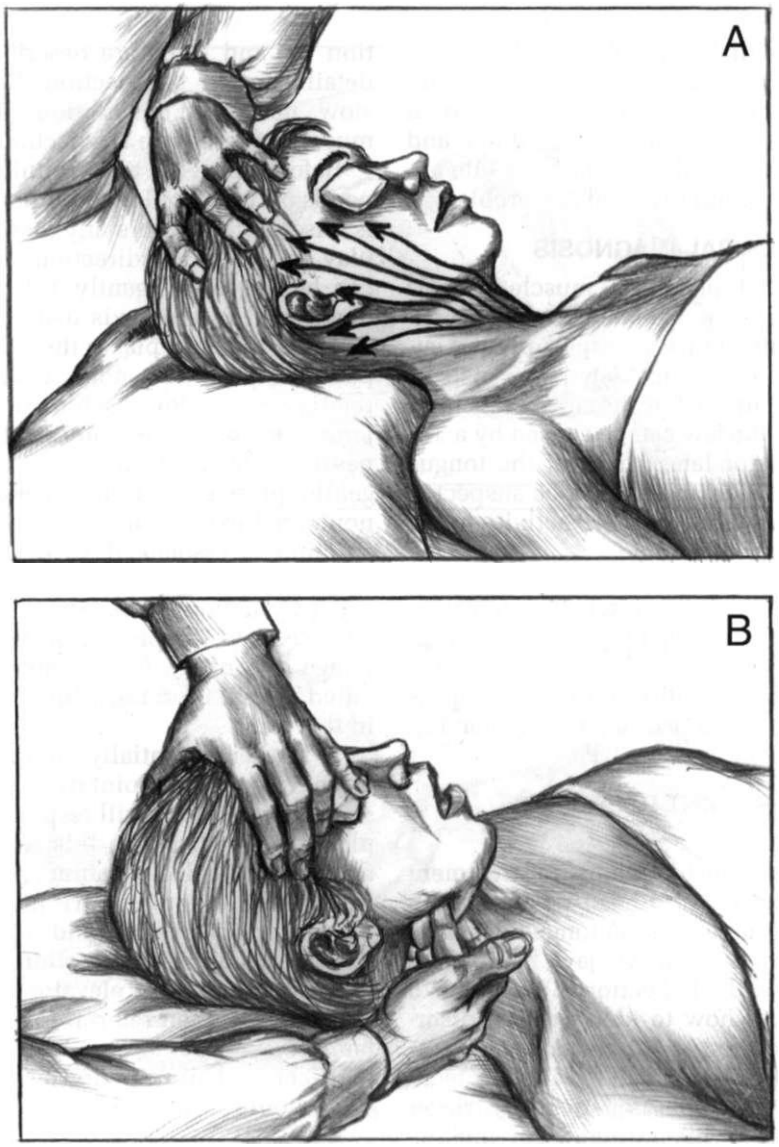


Figure 10.4. Spray pattern (*arrows)* and trigger point pressure release near the mandibular attachment region of the right medial pterygoid muscle. **A, pattern of intermittent cold application** preceding muscle release, patient supine with mouth relaxed and pad protecting eye from spray. The operator applies a stream of vapocoolant or icing in the pattern shown. The patient should exhale while spray is applied and avoid inhaling the spray; icing may be preferable to the spray in patients with respiratory conditions. **B, trigger point pressure release.** With the patient's mouth in the relaxed *open* position, the operator's fingers under the angle of the mandible apply pressure to accessible taut bands near the mandibular attachment and as far upward as possible on the medial pterygoid muscle. When the fingers encounter tissue resistance (the barrier), the operator maintains constant light pressure. When the operator's fingers sense relief of muscle tension (and/or the patient's mouth drops open further), the palpating fingers move to take up the slack until they again encounter tissue resistance (a new barrier). The operator again maintains only light pressure until the muscle releases ("lets go") under the fingers. This process of trigger point pressure release can be repeated for different bands of muscle fibers as needed. The operator's stabilizing hand acts to maintain head position. The operator now asks the patient to open the mouth voluntarily **without forcing,** which will provide reciprocal inhibition and take up slack in the muscle without producing excessive translation of the condyles. See text for additional release techniques for this muscle. See also Figure 8.5 for a combined spray-and-stretch technique that includes the medial pterygoid.

is applied prior to the stretch phase, or inhalation of the vapor can be avoided by applying sweeps of spray only while the patient is exhaling. If assistive stretch is applied to the jaw for opening, the pressure should be applied downward on the posterior molars to provide long axis distraction.

Intermittent cold with pressure release (Fig. 10.4) is not primarily a stretch release across a joint but is a direct TrP pressure release technique. However, it can be compromised in its full effectiveness by restricted range of motion in other masticatory muscles. Either icing or vapocoolant spray can be applied in parallel sweeps as illustrated in Figure 10.4A. The intermittent cold reduces the sensitivity of any enthesopathy. Then digital pressure is applied to the region of musculotendinous junctions along the posterior aspect of the angle of the jaw (Fig. 10.4B) reaching as far up along the medial pterygoid muscle as possible to apply light pressure to muscle fibers as they reach the attachment region. Muscle tension is relieved by applying TrP pressure release (similar to a barrier release approach) as described in the legend to Figure 10.4. The patient allows the jaw to drop in order to take up slack as it develops and to take advantage of newly acquired range of motion if restriction of other jaw elevator muscles does not interfere.

Other Release Techniques

Strumming of central TrPs in this muscle is an intraoral variation of stripping massage and is appropriate when there are multiple taut bands. Unlike the stripping massage movement in the same direction as the muscle fibers (which is more difficult to do because of the location of this muscle), the strumming massage movement is applied midmuscle *across* the fibers, in the region of central TrPs. Where the finger palpates the medial pterygoid muscle inside the mouth, only a thin layer of mucosa and the glove separate the finger from taut bands and TrPs in the muscle. The strumming finger *slowly* slides across from one side of the muscle to the other, releasing one band at a time. As the clinician's finger encounters resistance in the taut band, it applies only minimal pressure at that point, and waits for relaxation of the muscle fibers. This technique involves basically the TrP pressure release method, the effectiveness of which is relatively independent of tension caused by TrPs in the other masticatory muscles.

Postisometric relaxation with respiratory augmentation for opening the mouth is basically a contract-relax technique that can be used to release increased tension and shortening of the muscle due to TrPs (for details, see Chapter 3, Section 12). In this masticatory muscle, muscle-specific relaxation can occur during inhalation (as when yawning) but general body relaxation occurs with exhalation.

Resisted jaw opening is an augmented stretch technique that is based on reciprocal inhibition. Patients are instructed to open the jaw *slowly* against light resistance supplied by the clinician (or later supplied by themselves as a home stretch exercise). The activation of the jaw depressors (digastric, suprahyoid, and infrahyoid muscles) inhibits the elevation function of the medial pterygoid (and all other jaw elevators), providing a useful technique for releasing all of the jaw elevator muscles simultaneously.

Following any of these release techniques, when finished, patients should do three *unforced* cycles of opening and closing the mouth to incorporate the gain as part of their usual active range of motion. As part of the home program the postural considerations and exercise program discussed in the last section of this chapter should be addressed.

Electrical stimulation is a modality used by physical therapists and is a nonstretch technique that can be applied with a sterilized electrode suitable for intraoral use.[27] Clinically, a sinusoidal current of sufficient intensity to maintain a gentle tingle sensation (without muscular contraction) may be effective when applied to the TrPs for 10 minutes or more.[29] This method should be employed only by those specifically trained in its use and is not recommended if the patient finds it unpleasantly painful.

Ultrasound is also used therapeutically by physical therapists[27] and is more effective if directed at the TrPs rather than at a pain reference zone. For this muscle, ultrasound may be effectively applied behind

the gonial angle of the mandible because of its depth of penetration.[34]

13. TRIGGER POINT INJECTION (Fig. 10.5)

The medial pterygoid rarely requires injection of its trigger points (TrPs), since they respond well to the stretch-and-spray technique and to other manual release techniques, provided that active TrPs in other masticatory muscles have been inactivated and are not blocking the jaw opening. On the other hand, Gelb[23] reports that intraoral injection of active TrPs in the medial pterygoid relieves pain arising from TrPs in other muscles on that side of the face. Masticatory muscles tend to induce secondary and satellite TrPs among themselves. When considering injection, it is important to examine both the midmuscle region for central TrPs and the musculotendinous junction region for attachment TrPs.

The central TrPs may be approached for injection with a needle either inside or outside of the mouth. Using the extraoral approach, no major arteries lie in this path of the needle. (The maxillary artery lies posterior to the muscle.[16]) However, the needle must traverse the extensive network of the pterygoid venous plexus,[4] which is a potential source of bleeding. This makes the extraoral approach relatively undesirable. This approach for injection should be avoided in patients who have an abnormal bleeding tendency, as occurs with patients who have an inadequate level of ascorbic acid, are heavy smokers or are taking anticoagulant medication.

If the extraoral approach is selected to inject central TrPs in the muscle through the skin of the supine patient,[9,42] the mouth must be wide open in order to lower the mandibular notch (Fig. 10.5A). However, this wide opening can be contraindicated if there is TMJ derangement, which must be checked carefully (see Chapter 5, Section C). Accurate positioning of the needle at the TrPs is aided by examining the muscle intraorally with the free (gloved) hand, palpating both the needle and the TrP to guide the needle with precision. This technique is similar to the bimanual injection technique described for the piriformis muscle in Volume 2, Chapter 10, Section 13. After disinfection of the skin, the vapocoolant spray is applied for cutaneous local anesthesia to eliminate the pain of the needleprick,[44] as described in Chapter 3, Section 13. The needle is inserted between the condyle and the coronoid process, and is directed caudally along the vertical axis of the ramus of the mandible, as in Figure 10.5B, C, and D. Preliminary examination of a skull is helpful to visualize clearly the path of the needle and the depth of penetration required to enter the belly of the muscle deep to the level of, and posterior to, the lateral pterygoid plate.

Injecting this muscle intraorally is much more satisfactory for those familiar with intraoral injections. To inject the muscle with the needle inside the mouth, the TrP is located by palpation and injected directly through the pharyngeal wall, as illustrated by Gelb.[23] A hyperactive gag reflex must be suppressed to use the intraoral route.

Attachment TrPs may be present inside the angle of the jaw where they are far more accessible to injection than central TrPs. If the attachment TrP tenderness is severe, it may be important to inactivate these prior to injecting central TrPs. If marked TrP tenderness and referred pain remains after injecting central TrPs, also injecting tender attachment TrPs will expedite recovery if these had not been previously injected. Injection of a local anesthetic rather than dry needling is preferable for this TrP tenderness that is caused by enthesopathy.

14. CORRECTIVE ACTIONS

Activity Stress

If the patient has an excessive forward-head posture (see Chapter 5, Section C), the reasons for it should be identified and corrected to reduce activity in this muscle and in other jaw elevator muscles. In addition, the patient should practice correct tongue position (*see* Chapter 5, Section C) and reduce clenching and other abusive jaw habits. Tight pectoralis major and minor muscles are particularly critical and must be released and stretched regularly (*see* Chapters 42 and 43). Good sitting posture should be adopted (*see* Chapter 41, Section C).

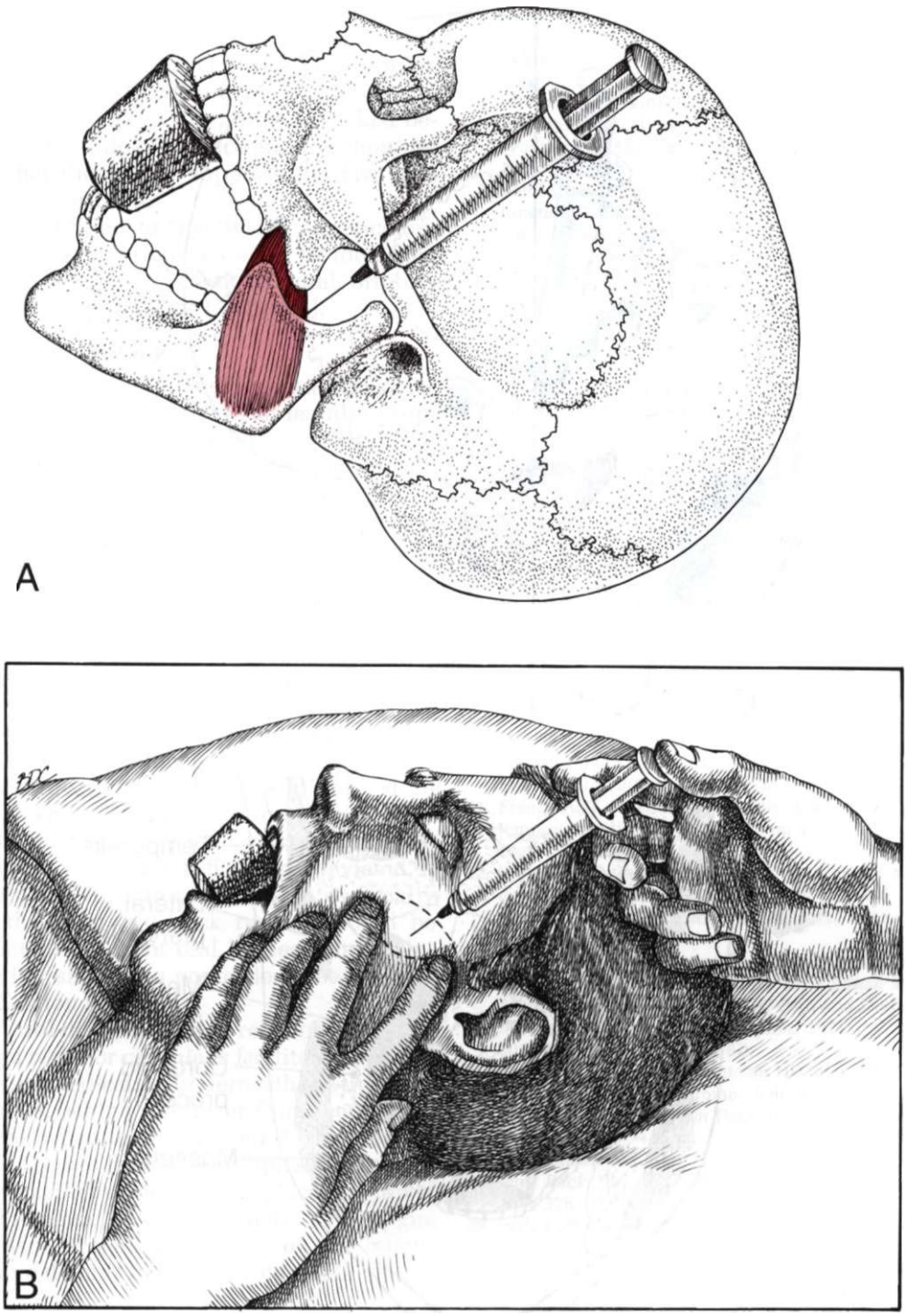
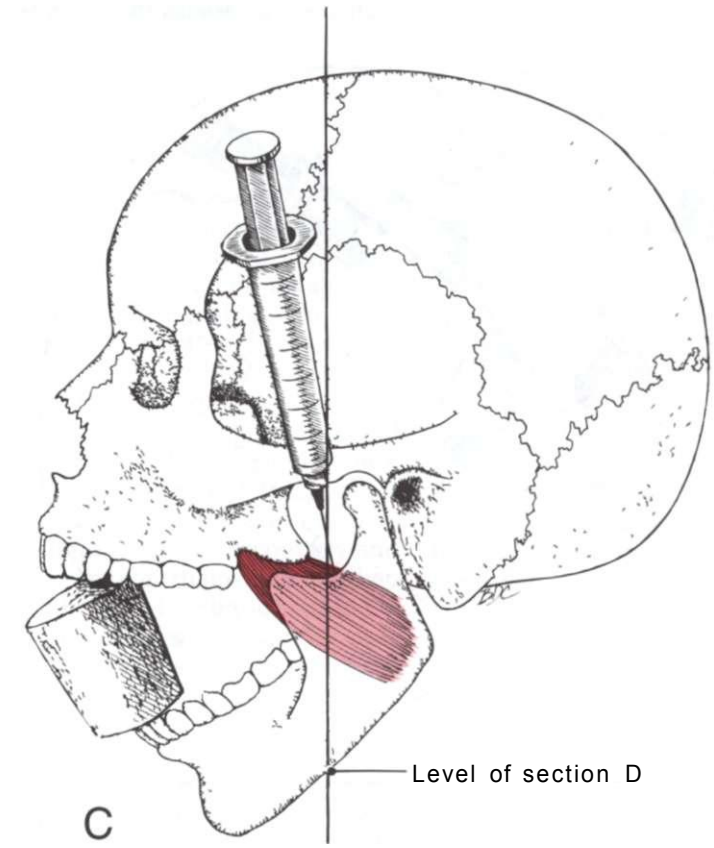


Figure 10.5. Extraoral injection technique for trigger points in the left medial pterygoid muscle. **A,** lateral view showing access to the muscle through the space above the mandibular notch between the coronoid process and the condyle of the mandible. The jaws must be propped wide open to provide access. **B,** injection through the opening above the mandibular notch.



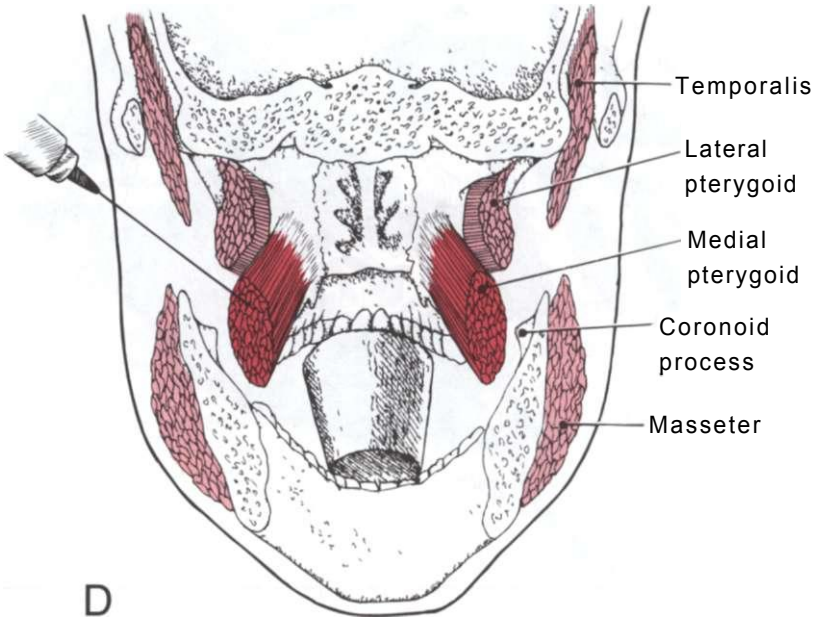


Figure 10.5.—*continued* **C,** lateral view of the injection technique that also indicates the level of the section in Part D. To reach the medial pterygoid muscle using this approach, the needle must penetrate to a depth greater than that of the pterygoid plate. **D,** coronal section of the head, located just behind the needle insertion, looking forward. Note that the medial pterygoid muscle attaches to the medial (deep) surface of the pterygoid plate, and the lateral pterygoid muscle attaches to the outer surface of the plate.

If the patient sleeps on the side, proper pillow positioning can prevent increased muscle activity caused by the jaw dropping down to one side during the night *(see* Fig. 22.6). A corner of the pillow is tucked between the side of the face and the shoulder so that the pillow supports the jaw in a neutral position.

In addition to inactivating masticatory muscle TrPs, bruxism should be identified and treated; use of an intraoral orthosis may be required *(see* Chapter 5).

Exercise Therapy

As the muscle lengthens and becomes less painful, the patient can learn to properly perform active, resistive, and facilitatory exercises for jaw opening and for lateral deviation, using resisted jaw opening (reciprocal inhibition) and lightly resisted lateral deviation as described in Section 12 of this chapter.

Other Measures

Mechanical and reflex perpetuating factors, such as active TrPs in the neck, shoulder-girdle, and sometimes even in the lower-limb muscles, should be eliminated. Common nutritional perpetuating factors are critically important, as discussed in Chapter 4. Factors that increase anxiety and emotional tension, including depression, should be identified and alleviated, if possible. Any chronic infection, especially in the head and neck region, should be treated. Recurrent oral herpes simplex infection should be controlled *(see* Chapter 4, Section F).

Until the dysphagia is relieved, swallowing a tablet or capsule is facilitated by placing the medication underneath the tip of the tongue, behind the lower front teeth; from there, when the head is erect, the medication follows the bolus of liquid being swallowed.[45] When the tablet is placed on *top* of the tongue, as is customary, the tongue presses it against the roof of the mouth where it tends to stick during swallowing.

REFERENCES

1. Adams SH II. Personal communication, 1981.
2. Agur AM. *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:509 (Fig. 7.85).
3. *Ibid.* (p. 467, Fig. 7.20).
4. *Ibid.* (p. 507, Fig. 7.79).
5. Bardeen CR. The musculature. In: *Morris's Human Anatomy.* Ed. 6. edited by Jackson CM. Blakiston's Son & Co, Philadelphia, 1921:377.
6. Basmajian JV, DeLuca CJ. *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985:453-459.
7. Bell WE. Clinical diagnosis of the pain-dysfunction syndrome. / *Am Dent Assoc* 79.154- 160, 1969 (p. 158).
8. Bell WH. Nonsurgical management of the pain-dysfunction syndrome. / *Am Dent Assoc* 79:161-170, 1969 (p. 165).
9. Bell WE. Management of masticatory pain. In: *Facial Pain.* Ed. 2. Edited by Ailing CC HI, Mahan PE. Lea & Febiger, Philadelphia, 1977 (p. 189, Fig. 12-5).
10. Bell WE. *Orofacial Pains-Differential Diagnosis.* Ed. 2. Chicago: Yearbook Medical Publishers, 1979 (pp. 193, 242, 252).
11. Burch JG. Occlusion related to craniofacial pain. In: *Facial Pain.* Ed. 2. Edited by Ailing CC III, Mahan PE. Lea & Febiger, Philadelphia, 1977 (p. 171, Fig. 11-10).
12. Butler JH, Folke LE, Bandt CL. A descriptive survey of signs and symptoms associated with the myofascial pain-dysfunction syndrome. / *Am Dent Assoc* 90:635-639, 1975.
13. Clemente CD. *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 449, 450, Fig. 6-11).
14. *Ibid.* (pp. 1162, 1168).
15. Clemente CD. *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 614).
16. *Ibid.* (Fig. 624).
17. *Ibid.* (Fig. 722).
18. Eisler P. *Die Muskeln des Stammes.* Jena: Gustav Fischer, 1912 (Fig. 25).
19. *Ibid.* (Fig. 26).
20. Eriksson PO. Muscle fiber composition system. *Swed Dent J 12(Suppl):8-38,* 1982.
21. Franks AST. Masticatory muscle hyperactivity and temporomandibular joint dysfunction. / *Prosthet Dent* 25:1122-1131, 1965 (p. 1126).
22. Gelb H, (ed). Patient evaluation. In: *Clinical Management of Head, Neck, and TMJ Pain and Dysfunction.* W.B. Saunders, Philadelphia, 1977 (pp. 85, 96, Fig. 3-14).
23. Gelb H, (ed). Effective management and treatment of the craniomandibular syndrome. In: *Clinical Management of Head, Neck and TMJ Pain and Dysfunction.* W.B. Saunders, Philadelphia, 1977 (pp. 299, 301, 302, 309, 314, Fig. 11-61).
24. Greene CS, Lerman MD, Sutcher HD, *et al.* The TMJ pain-dysfunction syndrome: heterogeneity of the patient population. *J Am Dent Assoc* 79.1168-1172, 1969.
25. Hollinshead WH. *Functional Anatomy of the Limbs and Back.* Ed. 4. W.B. Saunders Philadelphia, 1976:376.
26. Ingle JI, Beveridge EE. *Endodontics.* Ed. 2. Philadelphia: Lea & Febiger, 1976 (Fig. 11- 12B).
27. Kahn J. Electrical modalities in the treatment of myofascial conditions. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin RS. Mosby, St Louis, 1994:197-360.
28. Kahn LJ. Altered taste in a 58-year-old patient. / *Craniomandib Pract* 4(4j:367-368, 1986.
29. Maloney M. Personal Communication, 1993.
30. McMinn RM, Hutchings RT, Pegington J, ef *al. Color Atlas of Human Anatomy.* Ed. 3. Mosby-Yearbook, St Louis, 1993:40.

31. *Ibid.* (p. 49).
32. *Ibid.* (p. 56).
33. Moyers RE. An electromyographic analysis of certain muscles involved in temporomandibular movement. *Am J Orthod* 36.481-515, 1950 (pp. 484, 490, 502).
34. Nel H. Myofascial pain-dysfunction syndrome. / *Prosthet Dent* 40:438-441, 1978 (pp. 440, 441).
35. Schwartz LL, Tausig DP. Temporomandibular joint pain—treatment with intramuscular infiltration of tetracaine hydrochloride: a preliminary report. *NY State Dent J* 20:219-223, 1954 (Cases 3, 4 and 5).
36. Seltzer S. Oral conditions that cause head and neck pain. In: *Pain Control in Dentistry.* J.B. Lippincott, Philadelphia, 1978 (Fig. 8-12).
37. Shaber EP. Considerations in the treatment of muscle spasm. In: *Diseases of the Temporomandibular Apparatus.* Edited by Morgan DH, Hall WP, Vamvas SJ. C.V. Mosby, St Louis, 1977:250.
38. Sharav Y, Tzukert A, Refaeli B. Muscle pain index in relation to pain, dysfunction, and dizziness associated with the myofascial pain-dysfunction syndrome. *Oral Surg 46:742- 747,* 1978.
39. Spalteholz W. *Handatlas der Anatomie des Menschen,* Vol. 2, Ed. 11. Leipzig: Hirzel, 1922:267.
40. Toldt C. *An Atlas of Human Anatomy,* Translated by M.E. Paul. Ed. 2. MacMillan, New York, 1919:295.
41. *Ibid.* (p. 307).
42. Travell J. Temporomandibular joint pain referred from muscles of the head and neck. / *Prosthet Dent* 20:745-763, 1960 (pp. 749, 750, Fig. 5).
43. Travell J. Mechanical headache. *Headache* 7:23-29, 1967 (pp. 26, 27).
44. Travell J. *Office Hours: Day and Night.* World Publishing Co, New York, 1968:296-297.
45. Travell JG. Nonstick trick for pill swallowing. *Patient Care 9:17,* 1975.
46. Vamvas SJ. Differential diagnosis of TMJ disease. In: *Disease of the Temporomandibular Apparatus.* Edited by Morgan DH, Hall WP, Vamvas SJ. C.V. Mosby, St Louis, 1977:190.
47. Whinery JG: Examination of patients with facial pain. In: *Facial Pain.* Ed. 2. Edited by Ailing CC III, Mahan PE. Lea & Febiger, Philadelphia, 1977:159.

CHAPTER 11
Lateral Pterygoid Muscle

with contributions by
Bernadette Jaeger and Mary Maloney

HIGHLIGHTS: The lateral (external) pterygoid muscle is frequently the key to understanding and managing many craniomandibular disorders. Active trigger points (TrPs) in this muscle are tender, and their taut bands are likely to disturb the position of the mandible, its incisal path during opening and closing of the jaws, and the coordination with other muscles. **REFERRED PAIN** from TrPs in this muscle is felt strongly in the maxilla and often includes the *temporomandibular (TM) joint* region. **ANATOMY:** The superior division attaches anteriorly to the sphenoid bone, and posteriorly to the medial surface of the neck of the mandible immediately below the articular disk. The inferior division attaches anteriorly to the lateral pterygoid plate, and posteriorly to the neck of the mandible adjacent to the superior division. **FUNCTION:** Since both divisions of the muscle attach to the neck of the mandible, the traction applied by the superior division during mouth closure affects the condyle and disc complex as a unit. The inferior division protrudes and depresses the mandible with unilateral contraction causing lateral deviation to the opposite side. **SYMPTOMS** include pain in the region of the TM joint and the maxilla, dysfunction of the chewing apparatus, and sometimes tinnitus. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** may result from bruxism, excessive gum chewing, or may develop as satellite TrPs to key TrPs in neck muscles. **PATIENT EXAMINATION** shows some restriction of jaw opening, a distorted incisal path, and often altered occlusion. **TRIGGER POINT EXAMINATION:** The anterior attachment region of the inferior division ordinarily cannot be reached for direct palpation intraorally. Externally, the muscle bellies of both divisions are accessible only indirectly by palpating through the masseter muscle using a specific technique. **TRIGGER POINT RELEASE** of this muscle is limited by the deep location of the muscle and by the bone structure, but may be accomplished by the application of spray and postisometric relaxation. **TRIGGER POINT INJECTION,** therefore, is frequently needed. Injection of TrPs in this muscle is difficult because of their protected position behind the zygomatic arch and coronoid process of the mandible and deep to the masseter muscle. **CORRECTIVE ACTIONS** may initially depend on an occlusal splint and, then, if needed after TrP inactivation, restoration of a normal occlusal pattern and condyle-disc relationship. A home exercise program for improving masticatory muscle function and the elimination of stress factors insure continued relief.

1. REFERRED PAIN
(Fig. 11.1)

The lateral (external) pterygoid muscle refers pain deep into the temporomandibular (TM) joint (TMJ)[5,7,21,57,66,67] and to the region of the maxillary sinus (Fig. 11.1).[9,57,66,67] The pain is strongly associated with functional disorders of that joint.[20,57] In our experience, trigger points (TrPs) in this muscle are the chief myofascial source of referred pain felt in the TMJ area. The myofascial pain syndrome is easily mistaken for the pain of TMJ arthritis.[56]

No distinction has been drawn as to the patterns of pain referred from the two divisions of this muscle; it is sometimes diffi-

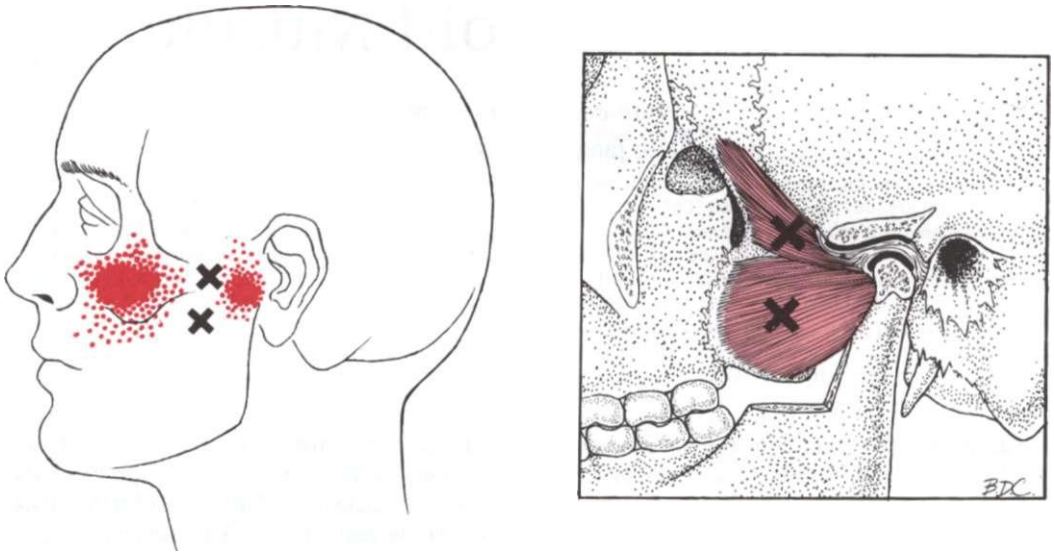


Figure 11.1. The referred pain pattern *(dark red)* of trigger points (Xs) in the left lateral pterygoid muscle *(lighter red)*. See Figure 11.2 legend for anatomical notes.

cult to be sure which division the needle has penetrated. Pain referred to the teeth has not been traced to TrPs in the lateral pterygoid muscle.

2. ANATOMY (Fig. 11.2)

The two divisions of the lateral pterygoid muscle lie deep to, and largely behind, the zygomatic arch and the coronoid process of the mandible. There is full agreement that ***anteriorly*** the *superior* division attaches to the infratemporal crest and to the inferior lateral surface of the great wing of the sphenoid bone, and that the *inferior* division attaches to the lateral surface of the lateral pterygoid plate (Fig. 11 2) [3,13,1,17,62]

The precise attachments of the two divisions ***posteriorly*** at the mandibular end were poorly identified in the past,[13,14,40] except that the fibers attach primarily to the medial half of the neck of the condyle. The fibers of the *inferior* division slant diagonally upward as they proceed posteriorly (Fig. 11.2) and were generally reported to attach to the condylar neck and ramus of the mandible just below the joint.[3,17,26,53,62] Examining 42 joints by the superior approach, Porter[55] found that a few fibers of the inferior division may also attach to the medial portion of the condyle. The fibers of the *superior* division slant diagonally downward and posteriorly toward the TMJ.

When the first edition of this volume was published there was considerable controversy as to the precise location of the **posterior** attachment of the superior division of the lateral pterygoid muscle. A more recent review by Klineberg[35] of studies examining this attachment concluded that there is general agreement that both divisions of the muscle attach into the fovea on the medial half to two-thirds of the neck of the condyle. In some specimens, *few* fibers of the superior pterygoid (superior division of the lateral pterygoid) insert into the foot of the interarticular disc;[35] however, this is contrary to earlier reports that the fibers inserted *primarily* into the capsule and disc. The traction that is applied by the superior pterygoid (superior division) during mouth closure affects the condyle and disk complex as a unit and does not affect the disk selectively.[35]

Rarely, the lateral pterygoid may fuse with the temporalis muscle, but fibers of the two divisions of the lateral pterygoid do not fuse with each other.[3]

It is helpful to remember that the anterior attachments of the *medial* pterygoid muscle and the inferior division of the *lat-*

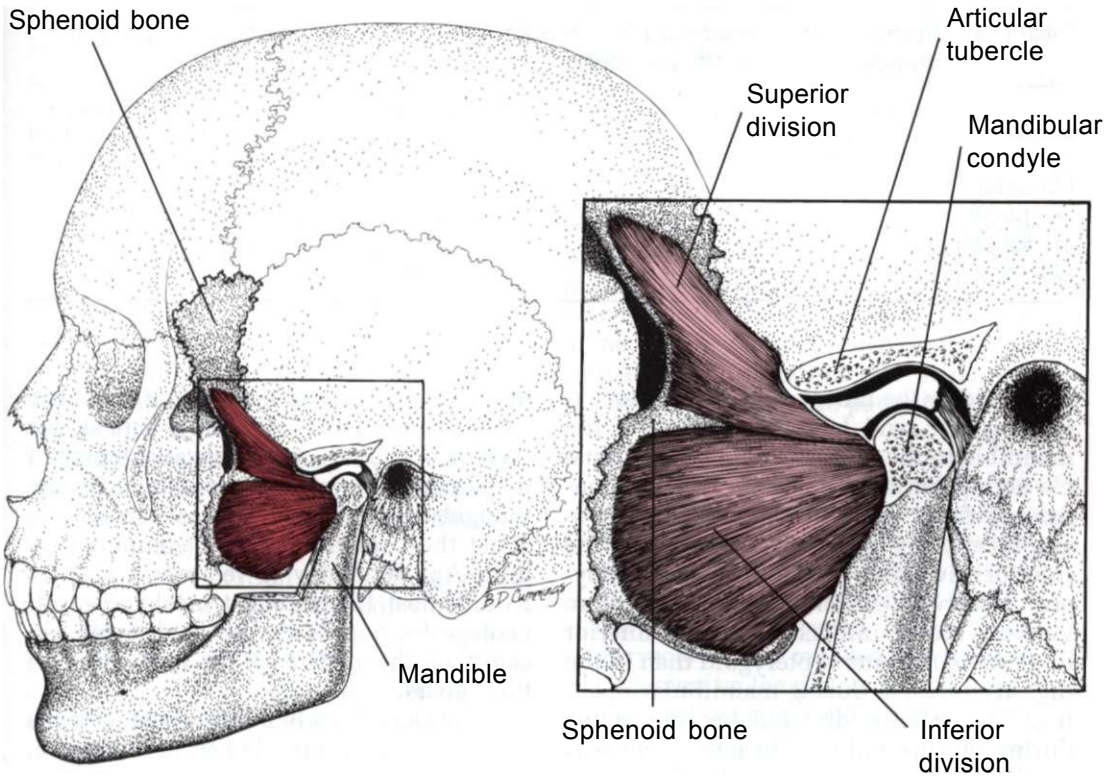


Figure 11.2. Attachments of the lateral pterygoid muscle. The zygomatic arch and superficial portion of the temporomandibular joint have been removed, Both divisions of the muscle attach to the neck of the mandibular condyle. The condyle normally articulates with the posterior surface of the articular tubercle of the temporal bone in this position until the mouth is opened wide as in a yawn.

eral pterygoid muscle are separated by the pterygoid plate of the sphenoid bone *(see* Fig. 11.5C and D). The medial pterygoid fibers attach to the medial (deep) surface of the plate, and the inferior division of the lateral pterygoid attaches to the lateral (superficial) surface of the plate.[40]

The condyle of the mandible must glide forward over the posterior surface of the articular tubercle in harmony with the intermediate articular disc *(see* Fig. 11.2 and Fig. 5.6) to open the jaws fully. This articular disc consists of collagen fibers, not cartilage.[44] The forward glide of the condyle is caused primarily by the inferior division of the lateral pterygoid muscle.

The lateral pterygoid, which acts to open the mouth, has one-tenth or less as many muscle spindles per gram of muscle as the three jaw elevators (Table 11.1). Of all the masticatory muscles, the lateral pterygoid seems to be the one most likely to have TrPs. The lateral pterygoid muscle has short fibers (1.9 cm long), but is relatively thick (4.8 cm^2 cross-sectional area)[69] in proportion to its length.

SUPPLEMENTAL REFERENCES

The lateral pterygoid muscle is clearly illustrated from the side,[1,3,15,17,40,48,61,64] from the rear,[62,65] in cross section,[2,18,19] and in sectional side view.

3. INNERVATION

Both divisions are innervated by the lateral pterygoid nerve from the anterior division of the mandibular branch of the trigeminal nerve (cranial nerve V).[13] The buccal and lingual nerves also may contribute filaments to this muscle.[3]

4. FUNCTION

The functions of the *inferior division* include opening the jaws, protrusion of the

Table 11.1 ***Approximate Density of Muscle Spindles (number of spindles per gram of muscle) in Masticatory Muscles***

Muscle	*Weight[69] (grams)*	*Spindle[37] (number)*	*Spindle Density (spindles per gram)*
Masseter	22	114	5.2
Temporalis	33	342	10.4
Medial pterygoid	8.1	59	7.3
Lateral pterygoid	9.6	6.0	0.6

mandible by the muscles on both sides acting together, and lateral deviation of the mandible to the opposite side by one muscle acting unilaterally.[3,4,13,31,61,68] Confirming these three functions electromyographically by placing a needle in the inferior division *via* the oral route, Moyers[32] observed earlier onset of, and more vigorous motor unit activity in, the inferior division of the lateral pterygoid than in the digastric muscle during mandibular opening. The inferior division became active during closure only if the movement was combined with protrusion. Activation of this inferior division by lateral movement of the mandible to the opposite side increased if the mandible was simultaneously depressed.

The *superior division* has been identified as specifically supporting the apposition of condyle, disc, and eminence during closure of the jaw.[35] Activity at this time would control the rate at which the condyle translates back to its rest position. In his review, Klineberg[35] uses the terms superior pterygoid and lateral pterygoid for the superior division and inferior division, respectively. Klineberg states that traction applied to the superior pterygoid (superior division of the lateral pterygoid) moves the condyle and disc complex as a unit.[35]

Electromyographically, the two divisions have been reported as antagonists in Rhesus monkeys[49] and in man.[23,39] Additional studies in man with fine wire electrodes indicate that the two heads may become active reciprocally during both vertical and horizontal mandibular movements.[43] Anatomically,[12] biomechanically,[23] and electromyographically,[39,49] the *superior division* exerts forward traction at the level of the condylar head during closure of the jaws. Since it is now generally agreed that there is not always a separate attachment of the superior division to the disc, it is now thought that both divisions of the muscle affect the condyle and disc complex as a unit. Any tendency to reciprocal activity would most likely reflect mechanical advantage by one or the other division because of the difference in angulation of their fibers.

Myofascial TrPs in either division of the lateral pterygoid muscle can be the cause, or the result, of premature contacts.[68] Needle-electrode study of the inferior division shows this muscle to be the most active in positioning the mandible during ipsilateral clench as the teeth are forced together by other muscles.[72] The medial pterygoid and both divisions of the lateral pterygoid participate in the lateral and closing movements during grinding of food between the molar teeth.[3,68]

Like the digastric muscle, the lateral pterygoid has a dearth of muscle spindles. The lateral pterygoid has one tenth or less as many muscle spindles per gram of muscle as the other primary muscles of mastication (Table 11.1).[37,47] These jaw depressor muscles, therefore, must need less precise position and movement control than most skeletal muscles and less control than the mandibular elevators. The lateral pterygoid muscle does have an active and coordinated withdrawal reflex. Painful electrical stimulation of the palate[71] consistently resulted in activation of the lateral pterygoid muscle and less frequently the anterior digastric muscle, with inhibition of jaw-closing muscles. The lateral pterygoid responded in all subjects and the an-

terior digastric responded in only 5 of 8 subjects. Widmer[71] concluded that the lateral pterygoid is primary for opening the jaw, and the digastric muscle assists.

5. FUNCTIONAL UNIT

The lateral pterygoid acts bilaterally when the jaws are opened and closed. To depress the mandible, the lower division acts synergistically with the digastric and other suprahyoid muscles.[3,39,49,68] During elevation of the mandible, lateral pterygoid activity controls the return of the condylar head during activity of the masseter and temporalis muscles.[39,49] Mandibular protrusion is assisted slightly by the superficial layer of the masseter and by the medial pterygoid,[68] and by the anterior fibers of the temporalis muscle.[3]

The inferior portion of the lateral pterygoid muscle on one side contributes to mandibular movements to the opposite side and is assisted by the ipsilateral medial pterygoid, contralateral masseter, and anterior fibers of the contralateral temporalis muscle.[60-68]

The paired lateral pterygoid muscles act synergistically for protrusion, but electromyographically are antagonistic to each other for lateral movements of the mandible.[25,52,72]

6. SYMPTOMS

Most patients with temporomandibular joint dysfunction suffer primarily from a muscular disorder, such as that caused by active TrPs in the lateral pterygoid muscle.[29] Severe pain in the TMJ region is commonly referred from TrPs in the lateral pterygoid, the medial pterygoid, or the deep layer of the masseter. This TrP pain referred to the TMJ,[56] as well as altered occlusion due to TrP tension with shortening of the muscles, often has caused treatment to be misdirected to the joint and teeth, with frustrating results. This often happens when the critical role played by TrPs in the lateral pterygoid and other masticatory muscles has been ignored or ineffectively treated.

Severe pain referred by TrPs to the maxilla, with the autonomic concomitant of excessive secretion from the maxillary sinus, may likewise be misdiagnosed as sinusitis, so that the patient thinks of the pain as a "sinus attack."

Patients experiencing tinnitus may have lateral pterygoid TrPs responsible for it.

Myofascial pain on chewing tends to be proportional to the vigor of movement.[7] Clicking sounds in the TMJ area may result from dysfunction of the lateral pterygoid muscles.[46] Although the active range of motion of the jaws may be reduced with active TrPs in the lateral pterygoid muscle alone, the decrease in range (brought about primarily by inhibition due to pain) may not be sufficient for the patient to be aware of it.

> One journal letter-to-the-editor[8] indicates that whatever caused tenderness in the lateral pterygoid muscles was responsible for disabling tinnitus in 39 patients. In 22 of them it was unilateral and in 10 it was bilateral. Palpation of the lateral pterygoid muscles revealed greater muscle tenderness on the symptomatic side with unilateral symptoms and nearly equal tenderness bilaterally in patients with bilateral symptoms. Injection of 1.8 ml of 2% lidocaine into the tender lateral pterygoid muscles resulted in 20% to 100% relief among patients (100% relief in 14 patients) with recurrence of symptoms when the anesthetic effect ended. However, the author noted that among those patients seen subsequent to treatment, soreness of the muscle was greatly reduced, and that in the absence of soreness the patients reported complete relief of tinnitus. Although TrPs were not mentioned, this report is completely compatible with the serendipitous injection of lateral pterygoid TrPs in many of the patients, which could account for the results.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Lateral pterygoid TrPs may develop as satellites in response to TrP activity of the neck muscles, especially the sternocleidomastoid, which, in turn, may be activated by the mechanical stress caused by a lower limb-length inequality, a small hemipelvis, or other lower body postural abnormality.

It is not clear whether degenerative arthritic changes in the TMJ (identified by

their grinding, clicking sounds and crepitus) are a result, or a cause, of TrP activation in the lateral pterygoid muscle. Painful arthritic changes and TrPs seem to intensify each other (see Chapter 5, Section C). The presence of structural changes in the joint may be demonstrated by tomograms, computerized tomography, and arthrograms.

Bruxism may be either the cause or the result of lateral pterygoid TrPs, and contributes strongly to the overuse of this muscle.

The lateral pterygoid muscle can be seriously overloaded by excessive gum chewing, nail biting, persistent thumb-sucking by a child, playing a wind instrument with the mandible fixed in protrusion, and by maintaining mandibular side pressure to hold a violin in playing position.

8. PATIENT EXAMINATION

When the **inferior division** of the lateral pterygoid muscle is affected, there is a slight decrease in jaw aperture that may prevent the entry of a tier of two knuckles between the incisor teeth (see Fig. 8.3, the Two-knuckle Test). Lateral excursion of the mandible is reduced toward the same side as the involved muscle because of the increased muscle tension. When the patient slowly opens and closes the jaws, the midline incisal path of the mandible deviates, wobbling from side to side. The most marked deviation from the midline during movement is usually away from the side of the more affected lateral pterygoid muscle, but this is not a reliable sign because TrP involvement of other masticatory muscles, especially the medial pterygoid, also can produce or alter this finding.

Lateral pterygoid function is practically eliminated by having the patient slide the tip of the tongue backward along the roof of the mouth to the posterior border of the hard palate, which also strongly inhibits translation of the condyles across the articular tubercle. If the incisal path straightens out when the mouth is opened in this way, it is chiefly lateral pterygoid dysfunction that is causing the muscular imbalance. If the incisal path still zigzags, other muscles and/or a TMJ derangement are responsible, and the abnormality may or may not also involve the lateral pterygoid.

Shortening of the inferior division of one lateral pterygoid muscle displaces anteriorly the mandibular condyle to which it attaches, causing premature contact of the anterior teeth on the opposite side and altered occlusion of the posterior teeth on the same side.[7] Little pain is experienced in this displaced resting position, but closing the teeth fully usually induces pain referred to the TM joint on the same side as the involved lateral pterygoid muscle. Vigorous closure increases the pain. Insertion of a tongue blade between the molar teeth on the painful side often eliminates the pain on vigorous clenching. This result strongly implicates the inferior division of the lateral pterygoid muscle on the painful side.[7]

9. TRIGGER POINT EXAMINATION (Fig. 11.3)

Internal (intraoral) palpation of the lateral pterygoid muscle is more direct and reliable than external (extraoral) palpation, but examines only the anterior attachment region of the inferior division of the muscle. The posterior attachment region of both divisions is accessible to external palpation at the neck of the mandibular condyle just below the TMJ. Both muscle bellies can, with proper precautions, be examined externally through the masseter muscle for tenderness and referred pain.

To examine *intraorally* for TrP tenderness in the region of the anterior attachment of the inferior division of the lateral pterygoid muscle, the finger presses posteriorly as far as possible along the vestibule that forms the roof of the cheek pouch. The mouth is opened about 2 cm (3/4 in) and the mandible deviated slightly laterally to the side being examined to improve the clearance, as the finger must squeeze between the maxilla and the coronoid process, along the roots of the upper molar teeth. Several authors have described and illustrated this technique.[10,30,51,54,59] The handle end of a dental mirror or other blunt instrument has been reported by others to substitute for the finger if the space is too tight,[32,41] but it may produce a more concentrated pressure stimulus and may be ineffective for precise identification of structures.

After sliding the finger along the outer side of the cul-de-sac to reach as high as possible along the inner surface of the coronoid process, the examiner presses inward toward the lateral pterygoid plate (see Figs. 11.2 and 11.3B). This pressure reveals *exquisite* tenderness if active TrPs are present in this part of the lateral pterygoid muscle.[21] Trigger point tenderness of temporalis muscle fibers attaching to the medial aspect of the coronoid process, lateral to the palpating finger (or probe), is distinguished from tenderness of lateral pterygoid fibers medial to the finger (or probe) by the patient's response to the direction of pressure.[32]

Some authors question the validity of this intraoral examination,[6,103] largely because one cannot palpate the muscle belly of the inferior division but only the region of attachment. However, since enthesopathy is characteristic of TrPs, this tenderness in the region of the musculotendinous junction could be caused by attachment TrPs in that muscle. A clinician[9] reported this intraoral examination to be very effective diagnostically, as described in detail in Section 15, Case Reports. A simpler but less sensitive test for detecting evidence of TrPs in the inferior division is to evoke pain during contraction of that muscle by trying to protrude the jaw against resistance at the chin.[6]

The finding[63] that 27.6% of a control group of 49 subjects were symptomatic to intraoral digital palpation of the lateral pterygoid muscle indicates that either a considerable percentage of normal individuals have latent TrPs in this muscle, or the examination produces a considerable number of false-positives. None of these subjects found resisted protrusion painful. Among the corresponding group of 59 patients referred for facial pain or temporomandibular disorder,[63] 69.5% found digital palpation painful, but only 27.1% experienced pain during resisted protrusion of the jaw. This functional test is simple and reliable if positive, but could easily miss the diagnosis of lateral pterygoid TrPs in patients whose TrPs were sufficiently active to be sensitive to digital palpation, but not sensitive to increased tension due to voluntary contraction. The pain threshold would depend strongly on the forcefulness of the muscular contraction which was resisted with the examiner' thumb. Although contracting a muscle with active TrPs in the shortened position is likely to be painful, the reliability of this procedure as a diagnostic criterion for TrPs has never been tested.

Using *external* palpation, the lateral pterygoid muscle bellies are inaccessible if the jaws are closed because the superior division lies deep to the zygomatic arch and the inferior division lies deep to the ramus of the mandible. However, **tenderness** of fibers approaching their attachments to the neck of the condyle can be revealed by palpation and can easily be misinterpreted as joint tenderness. With the jaws separated about 3 cm (1 1/8-in), a posterior portion of the inferior division and also of the superior division may be approached externally through masseter fibers and can be reached through the opening between the mandibular notch and the zygomatic arch (Fig. 11.3A).

Because one can palpate the lateral pterygoid muscle externally *only* through the masseter, one must first identify and inactivate any TrP tenderness in the masseter fibers in the area to be examined. When TrP tenderness is present in the masseter, its tense bands are readily palpable, but TrP bands in the underlying lateral pterygoid muscle are too deep to be distinguished by more than their local tenderness and by their referred pain response to pressure. Active TrPs in either the temporalis or the masseter muscle can prevent sufficient mouth opening for satisfactory examination of the lateral pterygoid muscle bellies for tenderness. Unless the temporalis and masseter TrPs are successfully inactivated, only the posterior attachment region can be examined for any enthesopathy.

Although no study is known that specifically identified TrP prevalence among masticatory muscles using taut band criteria, studies that may include other causes of tenderness such as fibromyalgia are useful because the proportion of tender points and TrPs should be relatively constant among muscles in any one study. The lateral pterygoid muscle (inferior division)

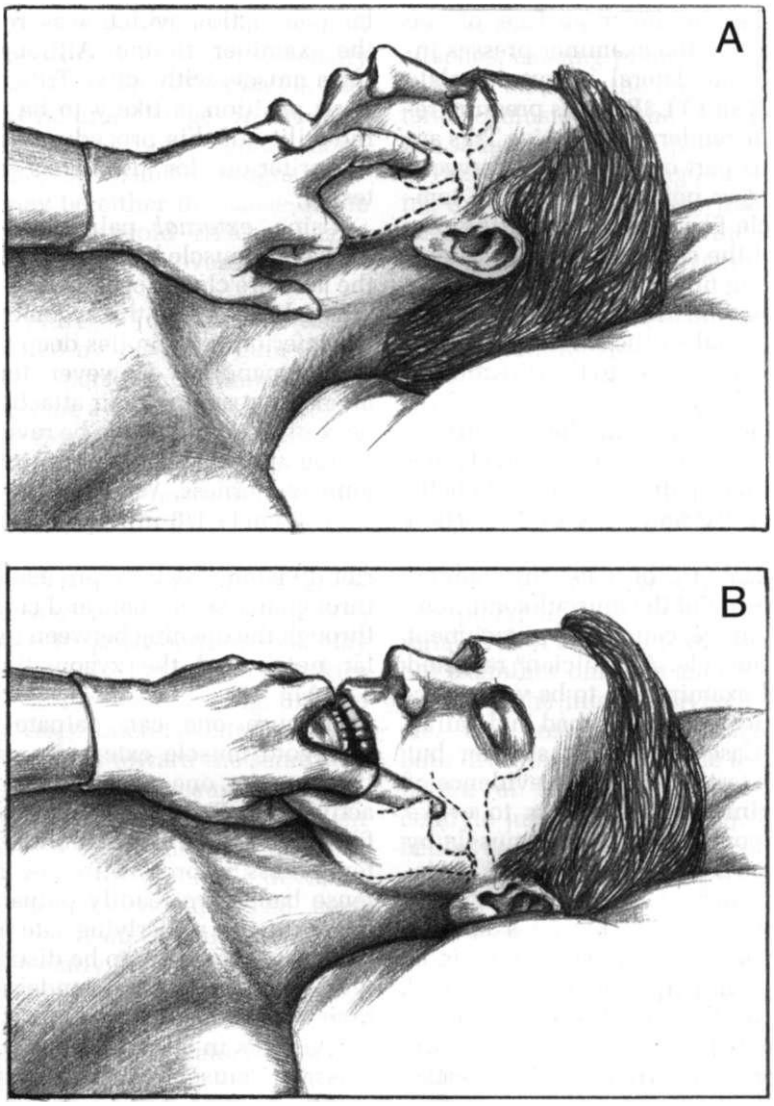


Figure 11.3. External and intraoral examination of the left lateral pterygoid muscle. **A,** External palpation of the posterior part of the muscle bellies of both divisions of the lateral pterygoid through the masseter muscle. The mouth is voluntarily held open by the patient to relax the masseter and permit palpation through that muscle and through the aperture between the mandibular notch and the zygomatic process (dotted lines). External examination permits indirect palpation for tenderness of the posterior parts of both divisions of the muscle as they approach their attachments to the neck of the condyle inferior to the temporomandibular joint. **B,** intraoral palpation permits more direct examination of the region of the anterior attachment of the inferior division. With a gloved hand, the operator slips a finger into the uppermost rear corner of the cheek pouch toward the head of the mandible and then presses medially toward the pterygoid plate. The jaws should be open about 5-8 mm (about 1/4 in) to allow room for the fingertip to squeeze into the space deep to the coronoid process. See text for additional comments regarding examination.

was tender to palpation more frequently than any other masticatory muscle in studies of nearly 300 patients.[20,24,34,51] In these various studies, the lateral pterygoid was tender in from 75% to 100% of patients. A few authors found that other muscles were tender more frequently than the lateral pterygoid, but it was still tender in 31% of 56 patients,[11] and in 20% of 42 patients.[58] These lower values may reflect the difficulty in palpating this muscle, or differences in the patient populations. This suggests that the lateral pterygoid is one of the masticatory muscles most commonly afflicted with TrPs.

10. ENTRAPMENT

The buccal nerve, which arises from the anterior division of the mandibular branch of the trigeminal nerve (cranial nerve V), usually passes between the two divisions of the lateral pterygoid muscle,[13, 65] but sometimes through the superior division.[1] It innervates the buccinator muscle, the skin of the cheek overlying it, the adjacent mucous membrane of the mouth, and part of the gum. Tautness of the lateral pterygoid muscle fibers due to active trigger points theoretically could entrap this nerve to cause buccinator, weakness with numbness and paresthesias in the distribution of the nerve. Mahan,[42] in discussing this, describes such a weird tingling of the cheek area in a number of patients.

11. DIFFERENTIAL DIAGNOSIS

Lateral pterygoid TrPs can produce referred pain that is likely to be interpreted as coming from the TMJ. Refer to Chapter 5, Section C for a description of TMJ problems and screening techniques. The referred tenderness from TrPs does not have the sharp localization nor the intensity of tenderness that is more characteristic of joint inflammation.

The aching facial pain caused by TrPs in the lateral pterygoid should not be mistakenly diagnosed as the paroxysmal electric type pain of tic douloureux. Only the aching pain of lateral pterygoid TrPs can be relieved by inactivating the TrPs.[9] They are separate conditions requiring different treatment.

When the inferior division of the lateral pterygoid muscle harbors active TrPs, its antagonists are likely to develop associated TrPs. Most vulnerable are its chief antagonists for lateral motion of the mandible, the medial and lateral pterygoid muscles on the opposite side. Next are its antagonists for protrusion, the deep masseter and posterior temporalis fibers on the same side.

12. TRIGGER POINT RELEASE (Fig. 11.4)

Of primary importance in treatment is the correction of excessive forward-head posture and correction of poor tongue posture when present *(see* Chapters 5 and 41, Section C).

Because the muscle belly of the lateral pterygoid muscle lies deep to the bulk of the masseter muscle, it is not accessible for direct manual techniques such as trigger point (TrP) pressure release and massage. Retruding the mandible against its restraining ligaments in the condylar fossa elongates (stretches) the muscle only a few millimeters. Spray followed by postisometric relaxation using *gently* resisted protrusion, followed by relaxation can be of some benefit. Spray followed by electrical stimulation can be helpful if applied correctly. Use of electrical modalities is described in principle by Kahn[33] and in Chapter 3, Section 12.

Spray and Postisometric Relaxation

Prespray or icing is applied as illustrated in Figure 11.4 with the patient in the supine position, which inhibits antigravity reflexes and encourages full relaxation of the masticatory muscles. The spray (or ice) is applied bilaterally because one side of the jaw does not function in isolation from the other. Immediately following application of spray or ice, treatment by postisometric relaxation[38] begins.

For postisometric relaxation the patient lies supine, mouth slightly open and relaxed. The clinician stands at the head of the treatment table in position to resist protrusion of the patient' mandible with his thumbs or fingers. The patient is instructed to breathe in and *gently* press his or her chin forward against the clinician's fingers, hold the (isometric) contraction for

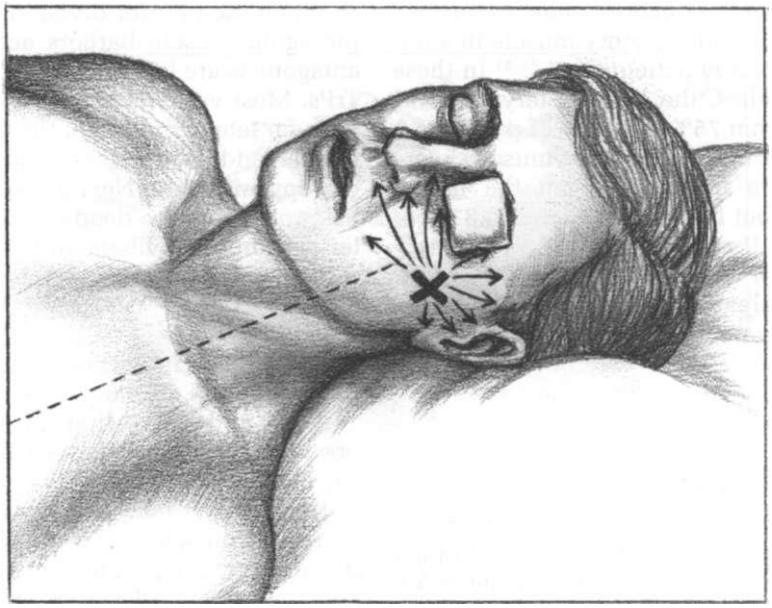


Figure 11.4. Vapocoolant prespray for release of the left lateral pterygoid muscle. Vapocoolant is applied from the trigger point region, covering the muscle and the pain pattern. The gauze protects the eye from misdirected vapocoolant. The clinician should apply the spray only while the patient breathes our in order to avoid inhalation of the vapors. Following the application of vapocoolant, gentle deep inhalation and slow, full exhalation repeated several times aid in relaxation of the muscle. Additional release may be obtained by the application of postisometric relaxation (see text).

a few seconds and then breathe out, relax, and allow the chin to drop back (toward retrusion but without outside assistance). The contraction and relaxation phases (coordinated with respiration) can be repeated 3 to 5 times to release the lateral pterygoid. For self-treatment at home, patients are instructed to use their own fingers for resistance.[38]

Other Methods

Physical therapists trained in the use of electrical stimulation have used high-voltage galvanic stimulation to effectively release the lateral pterygoid.[45] This method requires an intraoral probe small enough to reach directly to the anterior part of the inferior division of the muscle. Stimulation at 120 pulses per second using a pulse pair interval of 230-255 sec has been applied with sufficient intensity that the patient is aware of the stimulation, but does not experience pain. When the patient feels some relaxation of the muscle, the probe is moved to an adjacent stimulation site.

An external modality used by physical therapists has been ultrasound over the area superior to the mandibular notch, with the jaw dropped open. This application must penetrate the masseter (and temporalis insertion). It employs no more than 2 minutes of low intensity ultrasound (around 0.8 watts/cm^2).

13. TRIGGER POINT INJECTION (Fig. 11.5)

General Considerations

Because stretch techniques and direct manual techniques require more skill than usual for this muscle, it may be necessary to inject its trigger points (TrPs). The critical importance of this muscle as a major source of TM joint pain can make it worthwhile to develop the skill necessary to inject it.

The external (extraoral) approach permits injection of the central TrPs in the muscle bellies of both divisions and of the

attachment TrPs at the posterior musculotendinous junctions of both divisions. Only intraoral injection can reach the anterior musculotendinous junction region of the inferior division, and that is probably all it can reach.

In the absence of a history of allergic reactions to procaine, one can use 0.5% procaine in isotonic saline, rather than a long-acting local anesthetic. This reduces the likelihood of adverse reactions. Even if a nerve or blood vessel is penetrated, the dilute procaine is rapidly degraded by procainesterase as the drug enters the blood stream. Lidocaine (Xylocaine) 2%, or mepivacaine (Carbocaine) 3%, have been used successfully by others, but the latter especially requires care to avoid intravascular injection (*see* Chapter 3, Section 13) and provides little or no advantage. Epinephrine-containing solutions are NOT used.

Important signs of effective treatment are the return of the normal range of jaw opening, linearity of the incisal path during opening and closing, nontenderness of the lateral pterygoid muscle to palpation, and cessation of the patient's referred pain.

Extraoral Injection

A sophisticated technique for placing a needle in either division was described by Koole, *et al.*[38] Extraoral injection of either division of this muscle requires detailed knowledge of the anatomy because of the difficulty in palpating the muscle; there are numerous neighboring nerves and vessels including the pterygoid plexus. The needle must be oriented by visualizing the relation of the muscle and its TrPs to surrounding structures. Examination of a skull, in conjunction with the drawings of Figures 11.2 and 11.5, helps to establish a clear three-dimensional image of the lateral pterygoid muscle and its landmarks.

If any of the mandibular elevators (the masseter, temporalis, and/or medial pterygoid muscles) have TrPs with taut bands, they will limit mouth opening. These TrPs should be inactivated in order to provide an adequate mouth opening before trying to inject lateral pterygoid TrPs extraorally.

The volume of the space occupied by the lateral pterygoid muscle is limited by bony structures on all sides. This restricted space makes it a necessity to locate the tenderness of the TrPs precisely by palpation so that only a minimum volume of anesthetic need be injected. To inject the central TrPs in either division of the muscle, the jaws must be opened 22 to 30 mm (about 1 in) or more in order to open the bony window sufficiently. The window is bounded by the zygomatic arch above, the mandibular (semilunar) notch below, the coronoid process in front, and the mandibular condyle behind (Fig. 11.5A). Any taut bands and their tender TrPs in the masseter muscle can make it difficult to be sure that the palpated tenderness is due to TrPs in the underlying lateral pterygoid muscle. Masseter taut bands are more superficial and are oriented at nearly a right angle to the lateral pterygoid fibers, which makes taut bands in the masseter distinguishable (compare Figure 8.2A and 11.2). Masseter TrP tenderness should be eliminated first.

To avoid traversing this region with a dull needle, one disposable needle is used to penetrate the rubber stoppers of the vials, and a fresh needle used for injection. The needle should be replaced immediately if it contacts bone and feels as if the tip has developed a burr which "catches" or "scratches," instead of gliding smoothly through the tissue. A 3.8-cm (1 1/2-in) 22- to 27-gauge needle is adequate. A thinner needle is more likely to miss blood vessels, but may be deflected by connective tissues and by the contraction knots of TrPs unless the thin needle is inserted rapidly with the "fast in, fast out" technique of Hong.[27] When injecting this muscle with a local anesthetic other than 0.5% procaine, it is important not to inject while passing the needle to or from the muscle while the needle is traversing the pterygoid plexus, and in this case, it is important to aspirate for evidence of blood in the syringe before injecting.

To inject central TrPs of the *superior* division, the jaws are opened, the needle is inserted just anterior to the TMJ, and it is directed upward and forward, deep to the zygomatic arch, as illustrated by others.[7,16] The TrPs can be reached only after the full depth of the masseter muscle has been penetrated and the needle tip reaches the

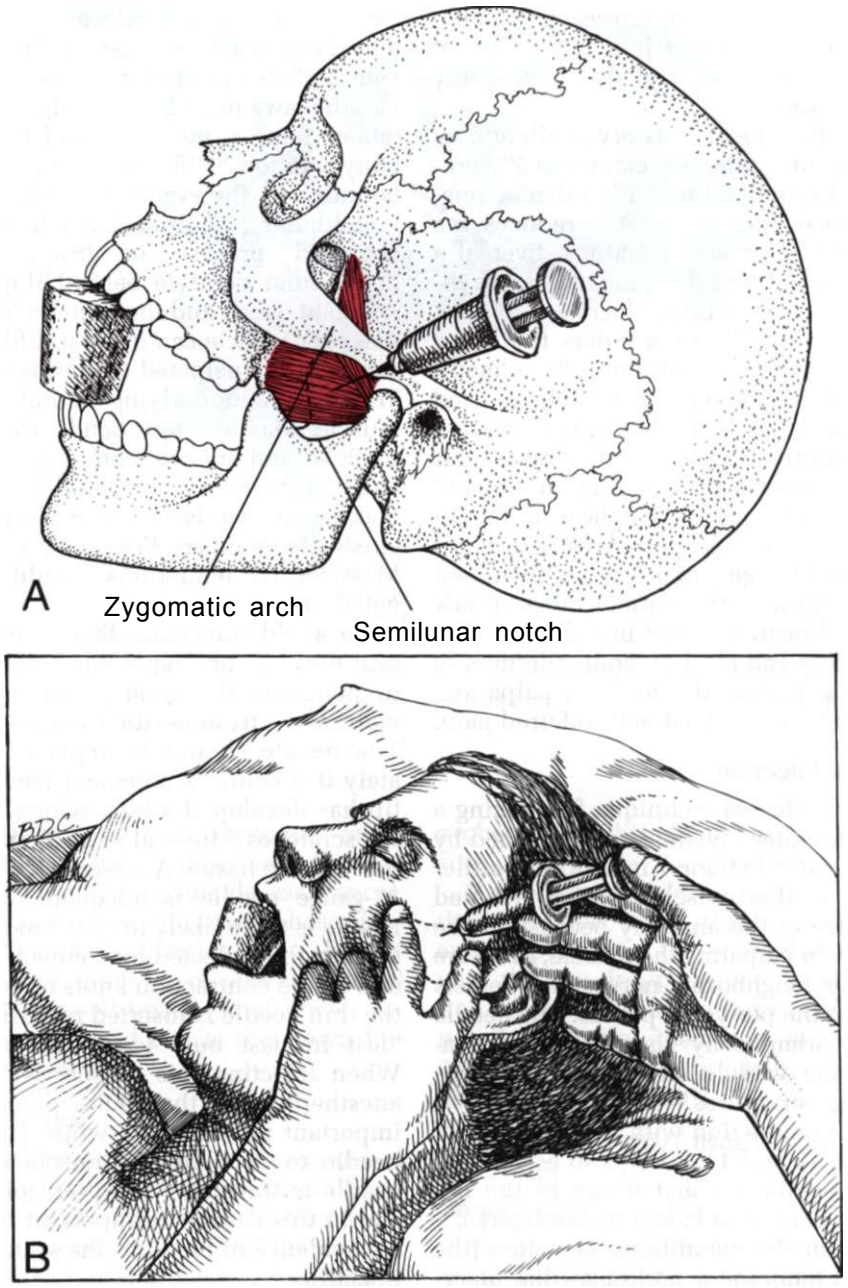


Figure 11.5. Injection technique for central TrPs in the inferior division of the left lateral pterygoid muscle *(dark red)*. **A,** lateral view of its anatomical relationships when the jaw is propped open. The dotted line marks the posterior margin of the pterygoid plate to which the inferior division attaches. The needle reaches the inferior division through the bony aperture bounded by the zygomatic arch above, the semilunar (mandibular) notch below, the coronoid process in front, and the condyle of the mandible behind. **B,** surface markings, same injection as in **A.** *Dotted lines* in **B** outline the palpable bony margins of the aperture.

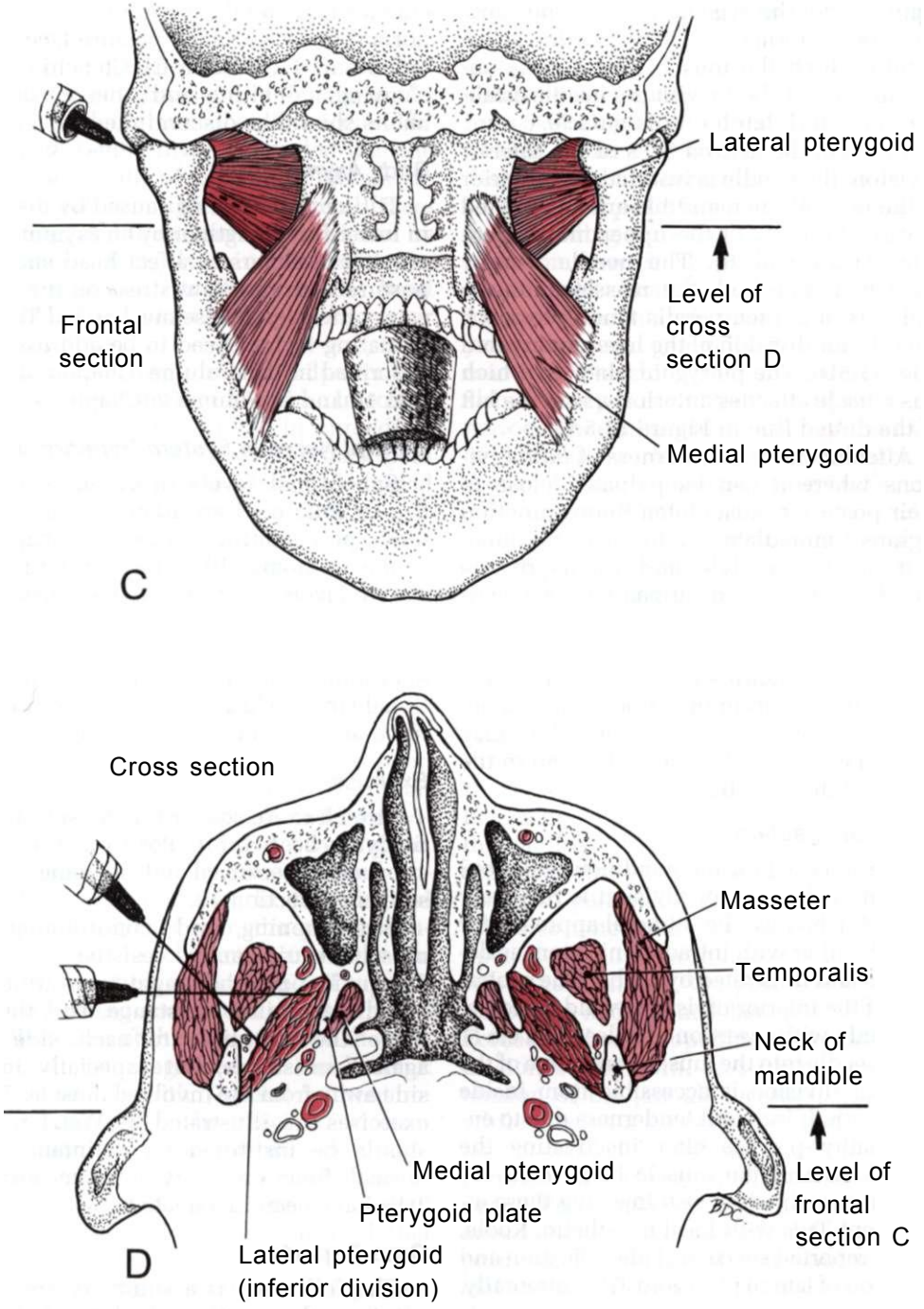


Figure 11.5.—*continued* **C,** frontal section of the head at the level of needle penetration (level of cross section shown in **D).** This view looks forward through the open mouth. The condylar neck of the mandible obscures part of the needle which penetrates the inferior division of the muscle. The medial pterygoid muscle *(light red)* lies in the foreground and attaches to the inner surface of the pterygoid plate. **D,** cross section showing needle penetration of the masseter muscle and then the temporalis *(light red)* as it passes in front of the condylar neck of the mandible above the mandibular notch (level of cross section is shown in **C).** The needles reach the anterior and posterior portions of the inferior division of the lateral pterygoid muscle *(dark red).*

region under the zygomatic arch. The sphenoid bone forms the floor of the space within which the muscle lies. Gently encountering this bone with the needle establishes the full depth of this muscle.

To inject the central TrPs of the *inferior* division, the needle is inserted just anterior to the neck of the mandible and is directed toward the roots of the upper molar teeth (Fig. 11.5A and **B).** The needle usually must penetrate both the masseter muscle and part of the temporalis tendon to reach the inferior division of the lateral pterygoid (Fig. 11.5D). The pterygoid plate, to which this muscle attaches anteriorly, is to the left of the dotted line in Figure 11.5 A.

Attachment TrP tenderness of *both* divisions where it can be palpated close to their posterior musculotendinous junction regions (immediately anterior to the junction of the condyle and ramus of the mandible) usually disappears with inactivation of the central TrPs. If not, the remaining attachment-region tenderness can be carefully identified and injected extraorally. This injection may not require penetration of the masseter muscle but may need to be directed posteriorly deep to the ramus of the mandible.

Intraoral Injection

The anterior (musculotendinous junction) portion of the inferior division is relatively easily reached *via* the intraoral approach for those familiar with intraoral injection, as described and illustrated by Gelb.[22] The central TrPs of the inferior division would be accessible only with insertion of at least 2.5 cm (1 in) of needle into the muscle, and none of the superior division is accessible from inside the mouth. If intraoral tenderness due to enthesopathy persists after inactivating the central TrPs in the muscle belly, recovery will be expedited by also injecting these attachment TrPs with local anesthetic. Koole, *et al.*[36] reported successful identification and injection of lateral pterygoid TrPs intraorally.

14. CORRECTIVE ACTIONS

Activity Stress

Excessive forward-head posture, if present, should be addressed and the patient should be taught correct tongue position *(see* Chapter 5, Section C). The patient also should be instructed in good body mechanics and should learn how to maintain normal head and neck posture *(see* Chapters 5 and 41, Section C). Clenching, gum chewing, nail biting, and other abusive jaw habits should be discontinued.

Body Asymmetry

Tilting of the pelvis caused by disparity in lower-limb length or by an asymmetrical pelvis can adversely affect head and neck posture with resultant stress on the masticatory muscles. These mechanical TrP perpetuating factors need to be addressed, as described in this volume, Chapter 48, Section 14 and in Volume 2, Chapter 4.

Central Nervous System Hyperirritability

Suboptimal levels of vitamins B_1, B_6, B_{12}, or folic acid are likely to act as systemic perpetuating factors *(see* Chapter 4) in the craniomandibular syndromes. Inadequate levels of one or more of these vitamins can aggravate bruxism through increased central nervous system and neuromuscular irritability, as can emotional stress. These factors should be identified and corrected *(see* Chapter 4).

Exercise

Stretch exercises for this muscle are not likely to be helpful. Postisometric relaxation can be carried out at home, as described in section 12.

Strengthening and conditioning the muscle require active resistive exercises. The patient may be taught to protrude the mandible against resistance, and then to move the mandible to each side also against resistance, but especially to the side away from the involved muscle. These exercises are illustrated by Wetzler,[70] but should be instituted for maintenance of normal functions only after masticatory TrPs have been inactivated.

Case Reports

The following is a summary and comments on three patients by Verne L. Brechner, M.D.[9]

The first patient was a 61-year-old female presenting with intense headache involving the right cheek. Six months previously, the patient had lifted a heavy object and had placed it on a shelf above her head. This resulted in pain in the shoulder

and neck radiating into the occipital region of the head. Shortly thereafter, the patient began to experience severe pain in the right zygomatic area while the pain in the shoulders, neck, and occipital region diminished. Dental history revealed that the patient had both of the lower molars removed from the right side several years preceding, and these had been replaced by a prosthesis. Roughly coinciding with the time of muscle strain injury to the head and neck, the patient had ceased to wear this prosthesis.

Examination by intraoral palpation revealed a tender pterygoid muscle. Injection of this tender point resulted in immediate relief of pain in the cheek. The neck pain and the occipital headaches had ceased prior to her examination, and a diagnosis of lateral pterygoid syndrome was made. She was returned to the care of her referring physician with the recommendation that she be refitted for her molar prosthesis.

The second patient was a 68-year-old female complaining of left facial pain of approximately one year's duration. She had been edentulous for many years, and one year previously, because of localized burning pain in the molar area of the gingival margin of the left maxilla, had been advised by her dentist to cease wearing an upper denture. At that time, he had observed a small abrasion in the gingival margin. During this year, the pain gradually changed in character and was no longer recognized as localized. The pain became burning and spread over the entire maxillary area of the face and into the eye. A neurologist diagnosed tic douloureux and treated her with Tegretol without relief. Subsequently, she received electrofaradic neurolysis of the gasserian ganglion. The patient experienced no relief of pain from this procedure. She was placed on Amitriptyline HC1 and was told that nothing else could be done.

Intraoral examination of this patient also revealed an extreme tenderness in the area of the lateral pterygoid muscle. When this area was injected with local anesthesia, the pain was temporarily relieved. She was referred to a dentist, who carefully prepared a set of dentures which fitted well and which improved her occlusion. This reduced the intensity of pain and markedly improved her general appearance and attitude. However, she persisted in chronic benign pain behavior and was admitted to Centinela Inpatient Program for a 5-week period of behavioral reconditioning. Following this program, she was discharged and continued to improve.

The third patient was a 37-year-old female holding an executive position in a large company. She had a headache history of 20 years' duration. Her headaches had increased in intensity during the past 7 years and were generally associated with emotional tension. Her headaches were also severely exacerbated by the menstrual period. Headache was located in the back of the neck radiating up over the back of the head into the forehead, jaw, and eye.

Examination of this patient revealed TrPs in the splenius capitis, supraspinatus, and trapezius muscles. Treatment included local TrP injections, psychological counseling, and biofeedback, with excellent improvement. A contract was made with her that at times of recrudescence of the headache, she could have an emergency appointment for TrP injections. On one of these occasions, injecting the previously successful TrP sites failed to relieve her headache. Further questioning revealed that on this occasion, the headache was not typical of that previously present, but consisted of pain mainly over the maxilla. It was also noted that the patient could not open her mouth more than 1.5 cm, which was a new finding. On previous examinations, she had opened her mouth 3.5 cm. Forced occlusion, opening the mouth, protrusion of the chin, and contralateral extension of the jaw all increased the pain, while placing a separator between the molars decreased it. Palpation of the lateral pterygoid muscle revealed extreme tenderness. The muscle was injected with a local anesthetic, and the patient had immediate pain relief. The lateral pterygoid myofascial pain syndrome has not recurred since then, although she has continued to return on occasion for treatment of her myofascial syndromes involving the supporting muscles of the head and neck.

Comments: The first patient presents an interesting mixture of chronic and acute myofascial pain syndromes. She apparently began with an acute syndrome of neck

and shoulder-girdle muscles caused by a one-time muscle overload that spontaneously resolved over a 6 month period. This indicates that she had no systemic perpetuating factors to prevent their spontaneous resolution. However, the TrPs in these muscles produced satellite TrPs in the right lateral pterygoid muscle that was already subject to muscle overload because of the "altered" occlusion, and the altered occlusion acted as a mechanical perpetuating factor for nearly 6 months. During this period there was no spread or augmentation of the pain symptoms. Inactivating the lateral pterygoid TrPs and refitting the molar prosthesis promptly resolved her pain problem.

The second patient also had chronic face pain but of 1 year duration that began with the onset of altered occlusion. There was gradual spread and augmentation of the pain throughout this period suggesting progressive modification of central pain pathways because of persistent pain. The list of wrong diagnoses and unsuccessful treatments is impressive, and not uncommon in patients with misdiagnosed myofascial TrP who have developed chronic pain. Injections of the lateral pterygoid muscle provided only temporary relief even after resolution of the altered occlusion. A 5-week period of behavior reconditioning improved but did not resolve her pain. We do not yet know how to assure restoration of the normal processing of pain in the central nervous system when pain has been allowed to persist and become chronic. Continuing to relieve the pain as soon as it recurs over a period of months is sometimes successful and is consistently helpful.[28]

The third patient demonstrates a valuable lesson. She had a chronic 20-year TrP pain problem that had developed through neglect, similar to the second patient. However, when she developed an acute lateral pterygoid TrP syndrome that was identified and effectively treated as such, it responded as an acute syndrome. For this area, the nervous system apparently was still processing muscle pain signals normally as acute pain[50] and responded to treatment accordingly.

In two patients, the diagnosis of lateral pterygoid TrPs was made by intraoral palpation, and the treatment was by injection with a local anesthetic.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 504, Fig. 7.76).
2. *Ibid.* (p. 531, Fig. 7.128).
3. Bardeen CR: The musculature. Section. 5. In: *Morris' Human Anatomy.* Edited by Jackson CM. Ed. 6. Blakiston's Son & Co., Philadelphia, 1921 (p. 377, Fig. 377).
4. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 453-453).
5. Bell WE: Clinical diagnosis of the pain-dysfunction syndromes. / *Am Dent Assoc* 79:154-160, 1969 (p. 158).
6. Bell WE: *Orofacial Pains-Differential Diagnosis.* fid. 3. Year Book Medical Publishers, Chicago, 1985 (p. 153).
7. *Ibid.* (p. 351, Fig. 17-11).
8. Bjorne A: Tinnitus aereum as an effect of increased tension in the lateral pterygoid muscle [letter]. *Otolaryngol Head Neck Surg 109(5)^69,* 1993.
9. Brechner VL: Myofascial pain syndrome of the lateral pterygoid muscle. / *Craniomandib Pract* 1(2j:43-45, 1983.
10. Burch JG: Occlusion related to craniofacial pain. Chapter 11. In *Facial Pain.* Ed 2. Edited by Ailing CC III, Mahan PE. Lea & Febiger, Philadelphia, 1977 (pp. 170, 174, Fig. 11-5).
11. Butler JH, Folke LE, Bandt CL: A descriptive survey of signs and symptoms associated with the myofascial pain-dysfunction syndrome. / *Am Dent Assoc* 90:635- 639, 1975.
12. Christensen FG: Some anatomical concepts associated with the temporomandibular joint. *Ann Aust Coll Dent Surg* 2:39-60, 1969.
13. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 451, 1167, Fig. 6-11).
14. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 614).
15. *Ibid.* (Figs. 624, 625).
16. Cohen HV, Pertes RA: Diagnosis and management of facial pain. Chapter 11. In: *Myofascial Pain and Fibromyalgia: Trigger Point Management.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 361-382.
17. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (p. 212, Fig. 24).
18. *Ibid.* (Fig. 25).
19. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 12-14, 20, 21).
20. Franks AS: Masticatory muscle hyperactivity and temporomandibular joint dysfunction. / *Prosthet Dent* 25:1122-1131, 1965 (p. 1126).
21. Gelb H: Patient evaluation. Chapter 3. In: *Clinical Management of Head, Neck and TMJ Pain and Dysfunction.* Edited by Gelb H. W.B. Saunders, Philadelphia, 1977 (pp. 83, 85, 96, Fig. 3-15).
22. Gelb H: Effective management and treatment of the craniomandibular syndrome. Chapter 11. In: *Clinical Management of Head, Neck and TMJ Pain and Dysfunction.* Edited by Gelb H. W.B. Saunders, Philadelphia, 1977 (p. 301, Fig. 11-6G and H).

23. Grant PG: Lateral pterygoid: two muscles? *Am J Anat 138:1-10,* 1973.
24. Greene CS, Lerman MD, Sutcher HD, *et al.:* The TMJ pain-dysfunction syndrome: heterogeneity of the patient population. *J Am Dent Assoc* 79.1168-1172, 1969.
25. Hickey JC, Stacy RW, Rinear LL: Electromyographic studies of mandibular muscles in basic jaw movements. *J Prosthet Dent* 7:565-570, 1975.
26. Honee GL: The anatomy of the lateral pterygoid muscle. *Acta Morphol Neerl Scand* 10:331-340, 1972.
27. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. / *Musculoske Pain* 2(3j:29-59, 1994.
28. Hong CZ, Simons DC: Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. /*Musculoske Pain 1(1*j.89-131, 1993.
29. Ingle JI: "The great imposter." *JAMA* 236:1846, 1976.
30. Ingle JI, Beveridge EE: *Endodontics.* Ed. 2. Lea & Febiger, Philadelphia, 1976 (p. 520, Fig. 11-12).
31. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W.B. Saunders, Philadelphia, 1991 (p. 342).
32. Johnstone DR, Templeton M: The feasibility of palpating the lateral pterygoid muscle. / *Prosthet Dent* 44:318-323, 1980.
33. Kahn J: Electrical modalities in the treatment of myofascial conditions. Chapter 15. In: *Myofascial Pain and Fibromyalgia: Trigger Point Management.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 473-485).
34. Kaye LB, Moran JH, Fritz ME: Statistical analysis of an urban population of 236 patients with head and neck pain. Part II. Patient symptomatology. / *Periodont* 50:59- 65, 1979.
35. Klineberg I: The lateral pterygoid muscle: some anatomical, physiological and clinical considerations. *Ann R Aust Coll Dent Surg* 31:96-108, 1991.
36. Koole P, Beenhakker F, de Jongh HJ, *et al.: A* standardized technique for the placement of electrodes in the two heads of the lateral pterygoid muscle. / *Craniomandib Pract* 8(2j:154-162, 1990.
37. Kubota K, Masegi T: Muscle spindle supply to the human jaw muscle. *J Dent Res* 56:901-909, 1977.
38. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 192, 193, Fig. 6.83).
39. Lipke DP, Gay T, Gross RD, *et al.:* An electromyographic study of the human lateral pterygoid muscle [Abstract]. *J Dent Res Special Issue B* 56.B230,1977.
40. Lockhart RD, Hamilton GF, Fyfe FW: *Anatomy of the Human Body.* Ed. 2. J.B. Lippincott, Philadelphia, 1969 (p. 157, Fig. 266).
41. Mahan PE: Differential diagnosis of craniofacial pain and dysfunction. *Alpha Omegan* 69:42-49, 1976.
42. Mahan PE: The temporomandibular joint in function and pathofunction. Chapter 2. In: *Temporomandibular Joint Problem.* Edited by Solberg WK, Clark GT. Quintessence Publishing, Chicago, 1980 (pp. 33-47).
43. Mahan PE: Personal communication, 1981.
44. Mahan PE, Kreutziger KL: Diagnosis and management of temporomandibular joint pain. Chapter 13. In: *Facial Pain.* Edited by Ailing CC III, Mahan PE, Ed. 2. Lea & Febiger, Philadelphia, 1977 (pp. 201-204).
45. Maloney M: Personal communication, 1993.
46. Marbach JJ: Therapy for mandibular dysfunction in adolescents and adults. *Am J Orthod* 62:601-605, 1972.
47. Matthews B: Mastication. Chapter 10. In: *Applied Physiology of the Mouth,* edited by Lavelle CL. John Wright and Sons, Bristol, 1975 (p. 207).
48. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, St. Louis, 1993 (p. 40).
49. McNamara JA Jr: The independent functions of the two heads of the lateral pterygoid muscle. *Am J Anat* 338:197-206, 1973.
50. Mense S, Simons DG: *Muscle Pain: understanding its nature, diagnosis, and treatment.* Williams & Wilkins, Baltimore. [In Press].
51. Meyerowitz WJ: Myofascial pain in the edentulous patient. *J Dent Assoc S Afr* 30:75- 77, 1975.
52. Moyers RE: An electromyographic analysis of certain muscles involved in temporomandibular movement. *Am J Orthod* 36:481-515, 1950.
53. Perry HT, Marsh EW: Function considerations in early limited orthodontic procedures, Chapter 10. In: *Clinical Management of Head, Neck and TMJ Pain and Dysfunction.* Edited by Gelb H. W.B. Saunders, Philadelphia, 1977 (p. 264).
54. Pinto OF: A new structure related to the temporomandibular joint and middle ear. / *Prosthet Dent* 32:95-103, 1962.
55. Porter MR: The attachment of the lateral pterygoid muscle to the meniscus. *J Prosthet Dent* 24:555-562, 1970.
56. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981.
57. Shaber EP: Consideration in the treatment of muscle spasm. In: *Diseases of the Temporomandibular Apparatus.* Edited by Morgan DH, Hall WP, Vamvas SJ. C.V. Mosby, St. Louis, 1977 (pp. 237, 249, 250).
58. Sharav Y, Tzukert A, Refaeli B: Muscle pain index in relation to pain, dysfunction and dizziness associated with the myofascial pain-dysfunction syndrome. *Oral Surg 46:742-* 747, 1978.
59. Shore NA: Temporomandibular joint dysfunction: medical-dental cooperation. *Int Coll Dent Sci Ed J* 7:15-16, 1974.
60. Silverman SI: Kinesiology of the temporomandibular joint. *Arch Phys Med Rehabil* 43:191-194, 1960.
61. Spalteholz W: *Handatlas der Anatomie des Menschen,* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 266).
62. *Ibid.* (p. 267).
63. Thomas CA, Okeson JP: Evaluation of lateral pterygoid muscle symptoms using a common palpation technique and a method of functional manipulation. / *Craniomandib Pract* 5(2j:125-129, 1987.
64. Toldt C: An *Atlas of Human Anatomy,* translated by M.E. Paul, Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 307).
65. *Ibid.* (p. 295).
66. Travell JG: Temporomandibular joint pain referred from muscles of the head and neck. / *Prosthet Dent* 30:745-763, 1960 (pp. 746, 749, 753).
67. Travell J: Mechanical headache. *Headache* 7:23-29, 1967 (pp. 26-27).

68. Vamvas SJ: Differential diagnosis of TMJ disease. Chapter 13. In: *Diseases of the Temporomandibular Apparatus.* Edited by Morgan DH, Hall WP, Vamvas SJ. C.V. Mosby, St. Louis, 1977 (p. 190).
69. Weber EF: Ueber die Langenverhaltnisse der Fleischfasern der Muskeln in Allgemeinen. Berichte iiber die Verhandlungen der Kbniglich Sachsischen Gesellschaft der Wissenschaften zu Leipzig 3:63-86,1851.
70. Wetzler G: Physical therapy. Chapter 24. In: *Diseases of the Temporomandibular Apparatus.* Edited by Morgan DH, Hall WP, Vamvas SJ, C.V. Mosby, St. Louis, 1977 (pp. 350, 351, Fig. 24-2).
71. Widmer CG: Jaw-opening reflex activity in the inferior head of the lateral pterygoid muscle in man. *Arch Oral Biol* 32:135-142, 1987.
72. Woelfel JB, Hickey JC, Stacey RW, *et al:* Electromyographic analysis of jaw movements. / *Prosthet Dent* 10:688-697, 1960.

CHAPTER 12
Digastric Muscle and Other Anterior Neck Muscles

HIGHLIGHTS: **REFERRED PAIN** and tenderness from trigger points (TrPs) in the posterior belly of the digastric muscle are projected to the upper part of the sternocleidomastoid muscle and therefore deserve to be called "pseudo-sternocleidomastoid" pain. This referred pain will persist after inactivation of sternocleidomastoid TrPs. The anterior belly of the digastric projects pain to the four lower incisor teeth. The other anterior neck muscles can refer pain to the laryngeal region, anterior neck, and to the mouth region. **ANATOMICAL** attachments of the important masticatory digastric muscle are, above, beside the midline symphysis of the mandible for the anterior belly, and to the mastoid notch of the temporal bone for the posterior belly. Below, the two bellies are joined together by a common tendon that is indirectly anchored to the hyoid bone through a fibrous loop. The remaining anterior neck muscles include the suprahyoid group, the infrahyoid group, and the deeper anterior vertebral muscles. **FUNCTION** of both bellies of the digastric muscle in conjunction with the remaining suprahyoid muscles involves opening of the mouth, if the infrahyoid muscles are also activated to stabilize the position of the hyoid bone. The deeply placed anterior vertebral muscles flex the cervical spine or control head position. The **FUNCTIONAL UNIT** includes the inferior division of the lateral pterygoid as a synergist for opening the jaws. The powerful elevators of the mandible are antagonists for closing the jaws. The deep cervical flexors are antagonistic to the posterior cervical muscles. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the digastric commonly occur as a result of TrPs in the antagonistic masseter muscle and other mandibular elevators. Activation can be due to the added stress of habitual mouth-breathing. Activation of TrPs in the suprahyoid muscles, infrahyoid muscles, and in the deeper anterior vertebral muscles (longus colli, in particular) can result from flexion-extension injuries such as those sustained in motor vehicle accidents. **TRIGGER POINT RELEASE** of the posterior belly of the digastric can be accomplished by applying intermittent cold and then postisometric relaxation. The anterior belly of the digastric and other suprahyoid muscles are released as one group and the infrahyoid muscles released as another group following the application of intermittent cold. The deep anterior neck muscles require special consideration. **TRIGGER POINT INJECTION** is performed under direct tactile control of the palpating fingers. **CORRECTIVE ACTIONS** include postural training, TrP pressure release that is self-applied directly to the TrPs, and passive stretch exercises. Measures should be taken to stop the habit of mouth-breathing, to terminate retrusive bruxing, and to correct persistent malocclusion.

1. REFERRED PAIN (Fig. 12.1)

Digastric Muscle

Each belly of the digastric muscle has its own referred pain pattern. Pain arising from trigger points (TrPs) in the *posterior belly* (Fig. **12.1 A)** radiates into the upper part of the sternocleidomastoid muscle,[13] and to a lesser extent to the throat in front of that muscle and under the chin, and sometimes it extends onto the occiput.[14] The pain referred to the sternocleidomastoid muscle is sometimes mistaken as coming from that muscle, but when the sternocleidomastoid is cleared of TrPs the posterior digastric referred pain persists. The occipital component of pain is likely to be associated with referred "soreness" and tenderness, which

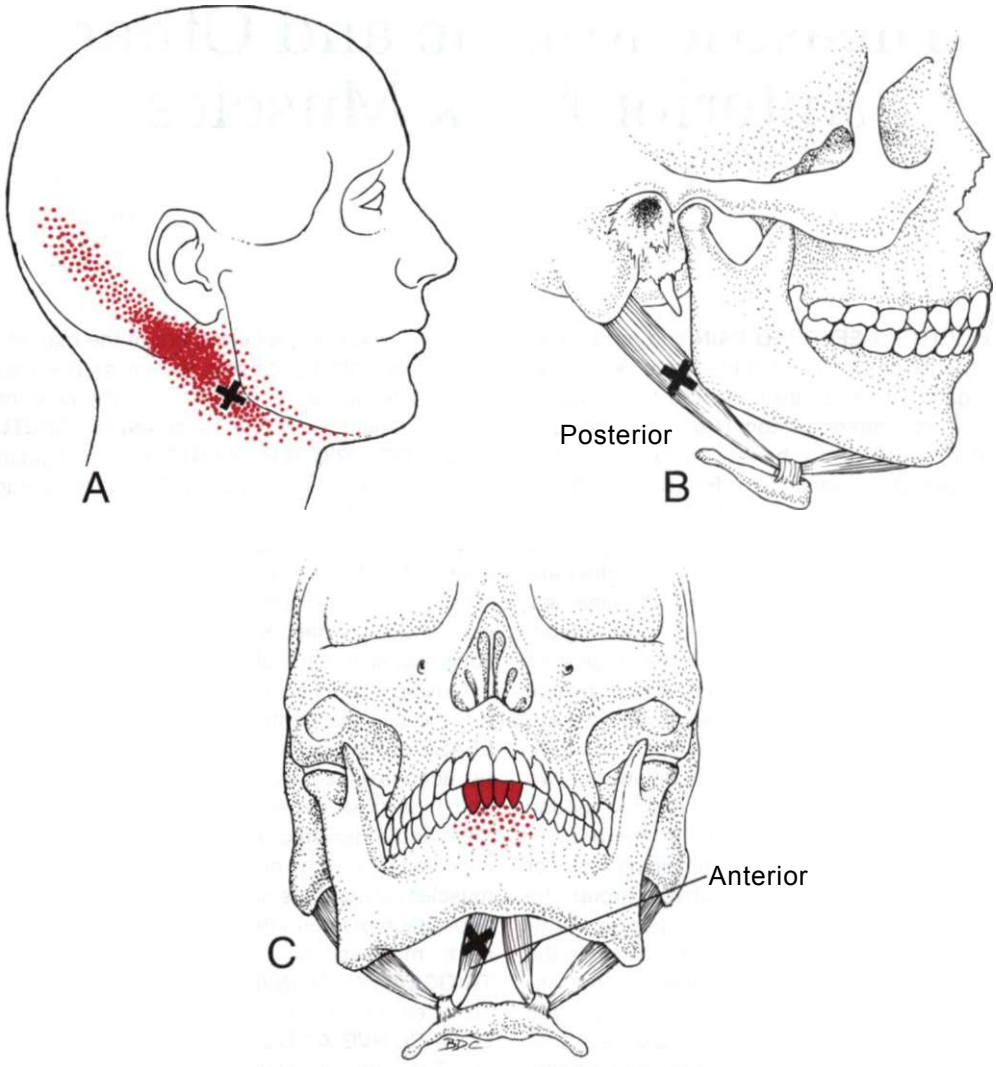


Figure 12.1. Referred pain patterns (essential portion, *solid red;* spillover portion, *stippled red)* of trigger points (Xs) in the right digastric muscle. **A** and **B,** posterior belly, side view. **C,** anterior belly, front view.

may activate satellite TrPs in the occipital portion of the occipitofrontalis muscle. Pain from digastric TrPs may also extend to the ear.[30] See Section 15, Case Report 1 of a patient with Eagle syndrome.

The pain referred from TrPs in the *anterior belly* of the digastric is projected to the four lower incisor teeth and the alveolar ridge below them (Fig. 12.lC) and may be referred to the tongue.[36] The responsible TrP for this bilateral, nearly midline, pain is located just under the tip of the chin and can be in the anterior belly of the digastric muscle on either the left or the right side of the body (Fig. 12.1C).

Other Anterior Neck Muscles

The **mylohyoid** muscle can refer pain to the tongue.[36] See Section 15, Case Report 2 for detailed report of a patient with this condition.

Head and neck pain have been attributed to both the **stylohyoid** muscle and the posterior belly of the digastric.[49] These two

muscles lie close together, have similar functions, are difficult to distinguish by palpation, and are presumed to have similar pain patterns.

The specific pain patterns for most of the deeper anterior neck muscles have not been reported and are not yet clearly established. They can refer to the laryngeal region, anterior neck, and sometimes into the mouth region. Myofascial TrPs in the **longus capitis and longus colli muscles** can cause difficulty in swallowing. Williams[49] reported myalgia (description compatible with TrPs) of the crico-arytenoid posterior muscle with pain on talking and a sore throat for which other physicians could find no cause.

2. ANATOMY (Figs. 12.2, 12.3, 12.4)

Digastric Muscle

The posterior belly of the digastric muscle arises from the mastoid notch on the mastoid process of the temporal bone (Fig. **12.2)** deep to the attachments of the longissimus capitis, splenius capitis and sternocleidomastoid muscles. The anterior belly arises from the inferior border of the mandible, close to its symphysis. The anterior belly passes posteriorly and inferiorly, and the posterior belly passes anteriorly and inferiorly to be united end-to-end by a common tendon that usually attaches indirectly to the hyoid bone through a fibrous loop or sling, the suprahyoid aponeurosis. The common tendon may slide through the fibrous loop.[10]

The tendon common to the two bellies of the digastric muscle perforates the stylohyoid muscle, which lies near the front half of the posterior belly of the digastric.

Suprahyoid Muscles

The **digastric** muscle does not attach directly to the hyoid bone, but only indirectly. The other suprahyoid muscles (Fig. **12.3),** which have their inferior attachment directly to the hyoid bone, include the **stylohyoid,**[8] which attaches above to the styloid process of the temporal bone; the **mylohyoid,** which attaches above to the entire length of the mylohyoid line of the mandible; and the **geniohyoid,** which attaches, above, deep to the mylohyoid muscle on the inner surface of the midportion of the mandible at the symphysis menti.[17] (The hyoglossus muscle is not considered in this suprahyoid group, but it is a muscle that arises from the hyoid bone, passes almost vertically upward, and enters the side of the tongue.[19])

Infrahyoid Muscles

The infrahyoid muscles (Fig. **12.3),** which have their superior attachment to the hyoid bone, include the **sternohyoid,** which attaches below to the sternum, the **thyrohyoid,** which attaches below to the thyroid cartilage, and the **omohyoid** muscle.[17] In addition, the **sternothyroid** attaches above to the thyroid cartilage and below to the sternum. It forms a continuum with the thyrohyoid, and lies deep to the sternohyoid.

The **omohyoid** muscle[18] has a superior belly and an inferior belly separated by a central tendon (Fig. **12.3).** The inferior (caudal) belly attaches below to the cranial border of the scapula near the scapular notch. As the inferior belly passes forward and up to its attachment to the central tendon, it attaches to the clavicle by a fibrous expansion and passes diagonally over the middle and anterior scalene muscles, but deep to the sternocleidomastoid muscle. The central tendon is held in position by a fibrous expansion of the deep cervical fascia that is prolonged caudally to attach to the clavicle and the first rib. From this attachment, the superior belly angles upward to attach to the hyoid bone (Figs **12.3** and **20.7**).[18]

Anterior Vertebral Muscles

The deeply placed anterior vertebral muscles[17] are situated along the anterior (ventral) surface of the vertebral column (Fig. **12.4)** and lie directly deep to the posterior wall of the pharynx. These deep muscles include the **longus colli** (which consists of a superior oblique portion, an inferior oblique portion, and a vertical portion). These groups of muscle fibers ascend from the third thoracic vertebra and attach as far superiorly as the tubercle on the anterior arch of the atlas. Other deep muscles are the **longus capitis,** which is more lateral and more craniad than the longus colli and extends upward from the anterior tu-

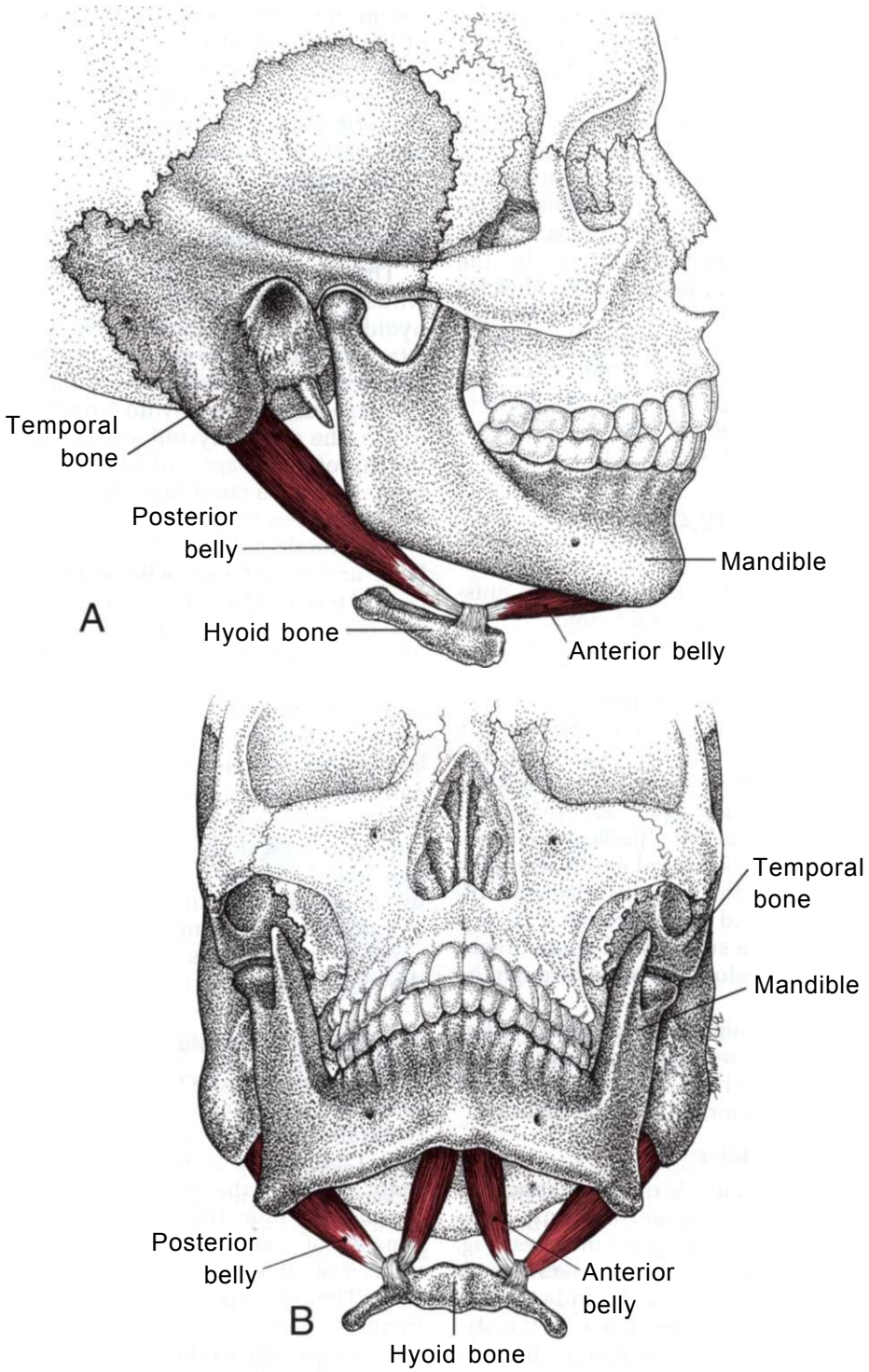


Figure 12.2. Attachments of the digastric muscle. **A,** side view. **B,** front view. The posterior belly attaches superiorly to the mastoid notch and inferiorly, at the muscle's common tendon, by fascial expansion indirectly to the hyoid bone. The anterior belly attaches superiorly to the mandible at the point of the chin and inferiorly, at the common tendon, by fascial expansion indirectly to the hyoid bone.

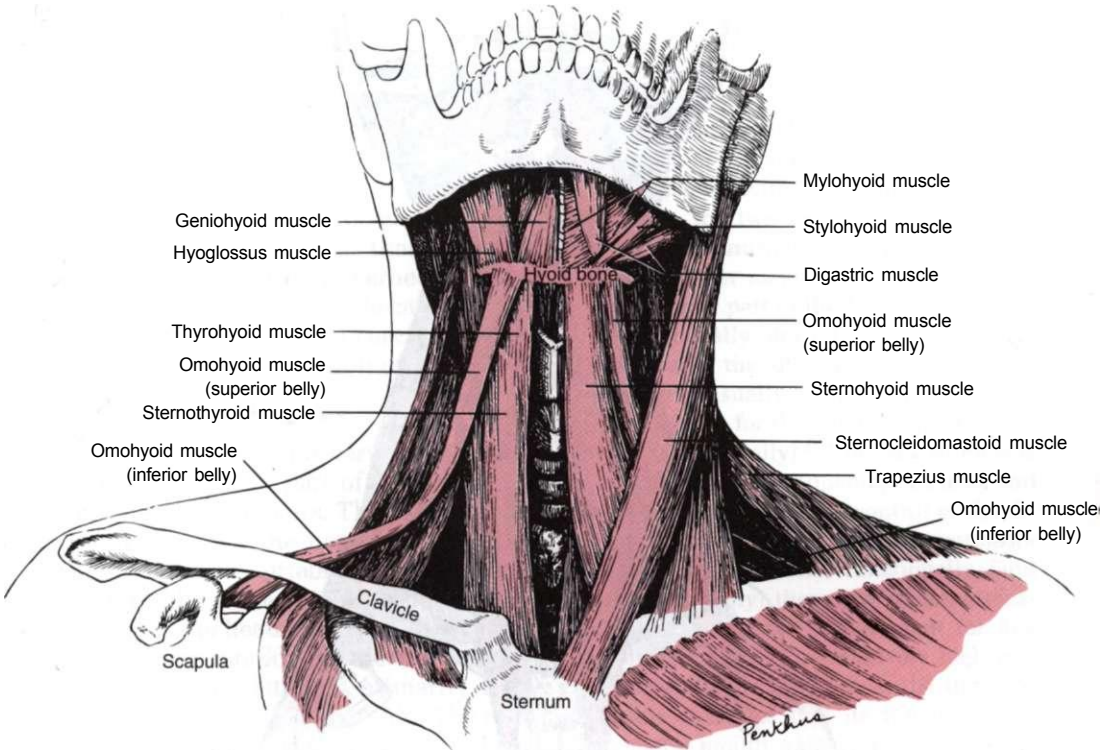


Figure 12.3. Relatively superficial muscles of the anterior neck including the suprahyoid and infrahyoid group. On the right side of the body several of the most superficial muscles (the sternocleidomastoid, sternohyoid, and trapezius) have been removed, (Reprinted with permission from Clemente CD: Gray's *Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985.)

bercles of the transverse processes of C_3- C_6 to the basilar part of the occipital bone, the short **rectus capitis anterior,** which lies deep to the upper longus capitis and passes upward and slightly medially from the lateral mass of the atlas to the basilar part of the occipital bone in front of the foramen magnum, and the short **rectus capitis lateralis,** which arises from the superior surface of the transverse process of the atlas and angles laterally upward to the lateral part of the occipital bone (Fig. 12.4).

Supplemental References

Anatomy textbooks illustrate both bellies of the digastric muscle in level side view[21,39,45,47] and as seen from below in side view,[10,17,25,46] from inside the mouth,[2] and from the front.[20] The relationship between the muscle and underlying neurovascular structures is clearly illustrated in a side view.[5,22] The anterior belly is seen in detail from the side[23] and from below.[3] The posterior belly is seen in detail from the side[4] and from behind.[24]

The attachments of most of the suprahyoid and infrahyoid muscles are shown schematically in side view.[6] The mylohyoid muscle is presented in side view,[9] and the locations of attachments of suprahyoid muscles on the hyoid bone are shown schematically.[8] The infrahyoid muscles are shown in side view.[7]

3. INNERVATION

The geniohyoid of the **suprahyoid** group is innervated by C_1. All of the remaining suprahyoid muscles are innervated by cranial nerves. The mylohyoid and anterior belly of the digastric are supplied by the alveolar branch of the trigeminal (fifth cranial) nerve.[17] The stylohyoid and posterior digastric muscles are innervated by the facial (seventh cranial) nerve, which exits

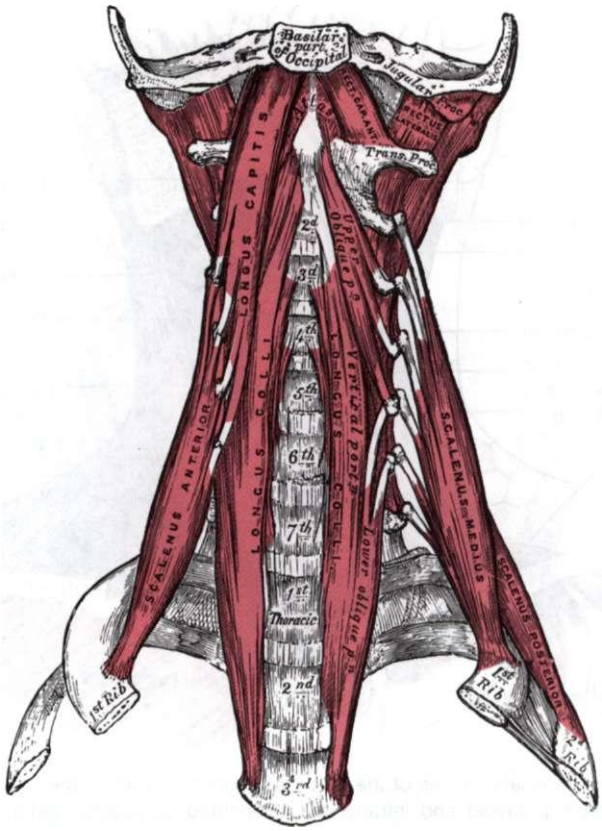


Figure 12.4. The deepest muscles of the anterior neck including the anterior and lateral vertebral muscles. (Reprinted with permission from Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiqer, Philadelphia, 1985.)

the skull through the stylomastoid foramen close to where these muscles attach to the skull.

The ansa cervicalis nerve, which is derived from the first, second, and third cervical nerves, supplies three of the **infrahyoid** muscles: the sternohyoid, sternothyroid, and both bellies of the omohyoid muscle.[17] The thyrohyoid is supplied by fibers from the first cervical nerve, as are the recti capitis muscles of the **deep** group. The **longus capitis** is supplied by branches from the first three cervical nerves, and the **longus colli** is supplied from ventral rami of the second to sixth cervical nerves.

4. FUNCTION

Suprahyoid Muscles

All four of the suprahyoid muscles (Fig. 12.3) characteristically function in pairs and function as a group to open the mouth.[16] The digastric muscles work closely together with the stylohyoid to open the mouth, but can be effective only if the infrahyoid muscles contract and stabilize the position of the hyoid bone. In addition, the mylohyoid muscles also raise the floor of the mouth during swallowing and are active in mastication, sucking and blowing.[6] The geniohyoid together with the digastric unit[10,11] can assist retraction as well as depression of the mandible. These muscles can elevate the hyoid bone.

Infrahyoid Muscles

As a group, all four of these muscles characteristically function in pairs and exert the essential depressive force on the hyoid bone that is required for the suprahyoid muscles to function normally. In

addition, the sternothyroid depresses the larynx after it has been elevated during swallowing, and the thyrohyoid elevates the larynx if the hyoid is fixed. Together these last two muscles form a continuous unit (Fig. 12.3) for depressing the hyoid bone.[6] The greatest intensity of contraction in the cricothyroid muscle occurs in swallowing.[12] Clemente[18] suggests that the omohyoid muscles are also concerned in prolonged inspiratory efforts because they tense the lower part of the cervical fascia and lessen the inward suction of soft parts.

Anterior Vertebral Muscles

These deepest anterior cervical muscles function to flex all or part of the neck and are likely to work in pairs. The longus colli is a weak flexor of the neck and laterally flexes the cervical vertebral column, with rotation to the same side.[35] The longus capitis flexes the head and the upper cervical spine with rotation to the same side. The rectus capitis lateralis primarily tilts the head laterally to the same side. The rectus capitis anterior forward flexes, but does not laterally tilt the head. Both the rectus capitis lateralis and the rectus capitis anterior muscles assist stabilization of the atlantooccipital joint, and their fibers angle in opposite directions.

DIGASTRIC MUSCLE

During mandibular depression, motor unit activity of the anterior belly follows that of the inferior division of the lateral pterygoid. The digastric appears to be less important than the lateral pterygoid for initial opening of the jaws, but is essential for maximum depression, or forced opening.[11] Digastric activity is inhibited during depression of the mandible if the mandible is protruded at the same time. This inhibition would be expected because of the retraction function of the muscle. The digastric is always active during mandibular retrusion.[40] The right and left digastric muscles nearly always contract together, not independently.[11] Coughing, swallowing and retrusion of the mandible strongly recruit the digastric muscles.[11,50]

Together, both bellies of one muscle exert a lateral-deviating force,[10] the effect of which is seen clinically, but only occasionally electromyographically.[40] The digastric muscle was electromyographically active in 85% of records taken during tooth contact which reflexly inhibited the mandibular elevators.[41]

Compared with mandibular elevator muscles, the two bellies of the digastric muscle are unusual. The digastric bellies, like the other major jaw-opening muscle (the inferior part of the lateral pterygoid), are practically devoid of muscle spindles,[26] and the digastric muscle bellies have an unusually low percentage of Type I fibers (24% for the anterior and 38% for the posterior belly).[27] The lack of muscle spindles in the jaw-opening muscles and the lack of evidence for control of the digastric muscle by the jaw-closing proprioceptors[48] suggest that functionally (and understandably) these jaw opening muscles do not have a requirement for fine position control. The relatively high percentage of type II fibers in the digastric muscle indicates that its function is to open the mouth quickly without having to maintain sustained tension. On the other hand, the definitely larger percentage of Type I fibers in the lateral pterygoid could relate to its need to maintain forward traction to keep the mandibular condyle in a forward-translated position as long as the mouth is held in a wide open position.

5. FUNCTIONAL UNIT

Muscles that are synergistic with the digastric muscle for opening the jaws (depressing the mandible) include the inferior division of the lateral pterygoid, and the stylohyoid (and other suprahyoid muscles), with the infrahyoid strap muscles stabilizing the hyoid bone. The previous section 4 includes detailed descriptions of the interactions of these anterior neck muscles as functional units. For retrusion of the mandible, synergists of the digastric are the posterior fibers of the temporalis and the deep portion of the masseter.

Antagonists to the jaw-opening action are the mandibular elevators: the masseter, the temporalis, the medial pterygoid, and the superior division of the lateral pterygoid. The deep longus colli and capitis and

the rectus capitis anterior are antagonists to the posterior cervical muscles.

6. SYMPTOMS

If the patient has posterior digastric TrPs, the primary complaint may not be pain but may be of difficulty swallowing and a sensation of a lump in the throat, or that something is stuck and won't go down. The patient is likely to palpate or point to the sternocleidomastoid muscle on the involved side. Although head rotation range of motion may not be reduced, the patient is likely to avoid turning the head to the involved side because the movement is likely to elicit referred pain or aggravate the swallowing problem. The posterior digastric referred pain pattern, as shown in Figure 12.1 A, concentrates in the region of the superior part of the sternocleidomastoid muscle. However, the patient may not become aware of the digastric referred pain component until after concurrent sternocleidomastoid TrPs on the same side have been inactivated. Then, pain and soreness persist in the upper part of the sternocleidomastoid muscle, which remains diffusely and moderately tender to palpation but free of taut bands and local twitch responses. This development can be very perplexing to the clinician unless the possibility of posterior digastric TrPs is investigated.

The chief symptom from TrPs in the anterior belly of the digastric is pain in the region of the lower incisor teeth. The source of this tooth pain can also be perplexing if the clinician considers only the teeth as the source of pain and overlooks examination of the anterior digastric muscle. Glossodynia can be caused by TrPs in the anterior belly,[36] as described in Section 15, Case Report 2.

Taut band tension from TrPs in the omohyoid muscle can (through its pull on the fibrous expansion of cervical fascia that attaches to the first rib) contribute to the dysfunction associated with an elevated first rib.

Patients with TrPs in the longus capitis and/or longus colli muscles are likely to complain of difficulty swallowing and of a lump in the throat. When these symptoms occur in a person who has sustained a cervical flexion-extension injury ("whiplash") in a rear-end automobile collision, longus colli TrPs may be a source of the problem. Rocabado and Iglarsh[43] reported that patients with a "spasm" in the longus colli (one source of which is trigger points) may complain of dry mouth, a sore throat without infection, a persistent tickle in the throat, or a lump in the throat upon swallowing.

Active TrPs in laryngeal muscles may produce a hoarse voice.

One should be aware that unresolved *posterior* neck pain may result from sustained TrP tension of these anterior muscles and tightening of their fasciae.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Activation of TrPs in the digastric muscle may be secondary to myofascial dysfunction of muscles in its functional unit; masseter TrPs have been specifically identified.[14] Hong found that key TrPs in the sternocleidomastoid muscle could induce satellite TrPs in the digastric muscle.[31] Lewit (personal communication, 1993) found the reverse also to be true. Inactivation of TrPs in the digastric also inactivated its satellite sternocleidomastoid TrPs.

Overload due to bruxing, by retruding the mandible, and due to mouth-breathing (one sign of which is inward, rather than outward, flaring of the nostrils during inhalation) predisposes to activation of TrPs in the digastric muscle. Mouth-breathing may result from mechanical blockage (as by nasal polyps), structural distortion (deviated septum) of the nasal passages, sinusitis, or recurrent allergic rhinitis.

The activation of TrPs in "Myalgia" of the posterior belly of the digastric and of the medial pterygoid muscle has been attributed to mechanical irritation caused by an elongated styloid process, the "Eagle syndrome."[34] The patient with this syndrome complains of pain in the angle of the jaw on the side of involvement, and also may have symptoms of dizziness and visual blurring with "decreased" vision on the same side. This pain can be caused by TrPs in the posterior digastric and stylohyoid muscles. Active TrPs in these muscles can result in sustained elevation of the hyoid. The tenderness at the styloid process

and calcification of the stylohyoid ligament can represent enthesitis and subsequent calcification due to the sustained tension caused by TrP taut bands. The dizziness and blurred vision can be caused by associated TrPs in the adjacent sternocleidomastoid muscle. The presence of abnormal elongation of the styloid process by calcification of the stylohyoid ligament is palpable from inside the mouth.[34] It may be necessary to remove the excess calcium surgically to provide relief. Pressure of the calcified process against the carotid artery during extreme rotation of the head may cause pain and dizziness.

Flexion-extension injuries, such as those sustained in motor vehicle accidents, can activate TrPs in the suprahyoid muscles, infrahyoid muscles, and in the deeper longus colli and longus capitis muscles; forward-head posture can perpetuate them.

8. PATIENT EXAMINATION

Rocabado and Iglarsh[43] state that "the hyoid bone influences movements of the mandible, swallowing, and sound formation in speech." The examiner should assess the hyoid for free movement laterally in both directions, and should be able to palpate muscle tension in suprahyoid and/or infrahyoid muscles when movement is restricted.

Sustained TrP tension of the posterior digastric and stylohyoid muscles can overload, and help to activate TrPs in, the antagonistic fibers of the contralateral posterior temporalis and of the contralateral masseter's deep division; tautness of these antagonists may nearly balance the mandibular deviation induced by the digastric. If the contralateral muscles are cleared of active TrPs, the mandible is then free to deviate to the side of the affected posterior belly of the digastric muscle. If deviation is due solely to posterior digastric TrPs, the mandible is pulled over as the jaws start to separate, but with further opening, it returns to the midline.

An indicator that TrPs in the posterior digastric muscle are contributing to a patient's difficulty in swallowing is improvement of the symptom by clenching the teeth while swallowing. Clenching may reciprocally inhibit the digastric TrP activity responsible for the difficulty. Difficulty in swallowing can also be caused by TrPs in the longus colli.

If the patient has an ossified extension of the styloid process (Eagle syndrome) with involvement of the posterior digastric and stylohyoid muscles, the patient should be checked for TrPs in the mylohyoid and longus colli muscles.

One test of **anterior digastric** TrP involvement as a source of lower incisor tooth pain is to ask the patient to pull the corners of the mouth down vigorously enough to tense the anterior neck muscles. When positive, this **Anterior Digastric Test** activates the toothache and indicates the likelihood of TrPs in the anterior belly of at least one digastric muscle.

Myofascial TrPs that restrict side bending of the neck include TrPs in the upper trapezius and in both the clavicular and sternal divisions of the sternocleidomastoid muscle. Less frequently, a tense omohyoid muscle stands out under the skin like a rope as it stretches over other neck structures and attaches to the scapula. Adson[1] relieved pain and dysesthesia resulting from pressure on the brachial plexus due to abnormal tension in the omohyoid by surgically sectioning the muscle.

When the omohyoid muscle develops TrPs and becomes tense, it can act as a constricting band across the brachial plexus.[44] Because the tense muscle stands out prominently when the head is tilted to the contralateral side, the omohyoid is easily mistaken for the upper trapezius or a scalene muscle. When the omohyoid harbors TrPs, it can prevent full stretch of the trapezius and scalene muscles, and therefore also must be released. Rask[42] reported the diagnosis and treatment of four patients whose primary cause of pain was myofascial TrPs in this muscle.

Recognition of muscle balance is always important, and between the suprahyoid and infrahyoid muscles it is particularly critical because (except for the stylohyoid ligament) the hyoid bone is "floating" between them. The concept of inhibited and excitable muscles contributing to imbalance[32] is becoming increasingly accepted.[29] The digastric muscle has been identified as being prone to weakness and inhibition;[37]

however, no experimental data substantiating this impression is known to have been published. Since TrPs can so profoundly disturb normal muscle responsiveness, and since the masseter-digastric relationship is unusually dependent upon reflex interaction because of the dearth of digastric muscle spindles, electromyographic (EMG) studies of the functional behavior of these muscles before and after release of the TrPs in one muscle or the other should be very informative. Such a study could readily be extended to include the infrahyoid muscles.

In cases of unresolved *posterior* cervical, thoracic, or lumbosacral pain, it is wise to assess anterior structures for TrP tension and to assess the patient for a forward-head posture.

9. TRIGGER POINT EXAMINATION (Fig. 12.5)

Tension in either belly of the digastric muscle can be assessed by finding abnormal resistance when trying to shift the hyoid bone from side to side. The **posterior** belly of the digastric muscle is examined with the patient supine and the head extended, in order to enlarge the space for palpation between the neck and the angle of the mandible. This posterior belly of the digastric muscle (and the stylohyoid muscle) are palpated (Fig. **12.5**A) by rubbing across (perpendicular to the direction of) the fibers behind the angle of the mandible,[15] and by sliding the finger upward toward the ear lobe along the anterior border of the sternocleidomastoid muscle, while pressing inward against the underlying neck muscles. The initial pressure on active TrPs in the posterior belly elicits exquisite local tenderness; sustained pressure may reproduce the patient's more distant neck and head pain.

The **anterior** belly of the digastric muscle is examined with the patient supine, the head tilted back and the neck extended (Fig. **12.5B).** With the patient relaxed, the examiner palpates the soft tissues just beneath the point of the chin on both sides of the midline. A tender nodule may be felt in the muscle belly at the point of central TrP tenderness. Tenderness at the base of the greater horns of the hyoid bone is likely to be attachment TrP tenderness due to enthesopathy for which Ernest and Salter[28] presented strong histopathological evidence.

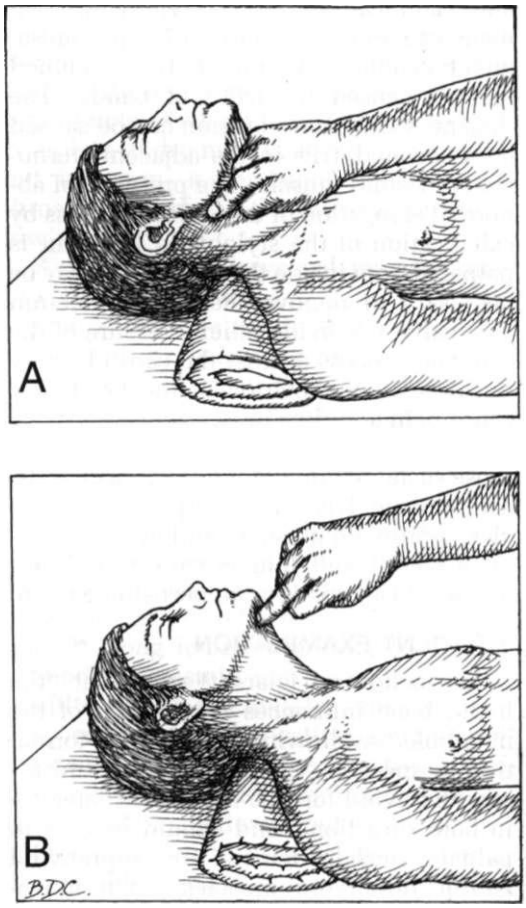


Figure 12.5. Examination of the digastric muscle. **A,** posterior belly: palpated between the angle of the jaw and the mastoid process, against the underlying neck structures. **B,** anterior belly: the head is tilted back and the neck extended, with the jaws closed, to stretch the muscle as it is palpated against the underlying soft tissues, as described in text.

If the inferior belly of the omohyoid muscle has a tender TrP and taut band it can be mistaken for the anterior scalene muscle, although the two muscles have different fiber directions. The omohyoid muscle is more superficial than the scalene muscles, comes out from beneath the sternocleidomastoid muscle, and crosses diagonally over the anterior scalene (see Fig. 20.7). It can cross at about the same level as the location where scalene TrPs can be

found, depending on which scalene digitation is involved and depending on head position.

Myofascial TrPs in the longus capitis muscle can be palpated behind the posterior pharyngeal wall through the open mouth. Those in the longus colli can be palpated with difficulty by placing the examining finger along a lateral border of the trachea between the sternocleidomastoid muscle and the thyroid cartilage,[43] and slowly advancing it by separating the musculature from the adjacent trachea by gentle rocking and wiggling motions of the finger. When the finger encounters the vertebral column, that region is explored for TrP tenderness.

10. ENTRAPMENT

No neurological entrapments are attributed to TrP activity in the digastric muscle; however, Loch *et al.*[38] reported that among 85 anatomical specimens they found 7 cases of compression of the external carotid artery (in some cases including the posterior auricular artery) solely by the stylohyoid muscle without ossification of the styloid process.

11. DIFFERENTIAL DIAGNOSIS

Active TrPs in the **posterior belly** of the digastric are a common problem when severe restriction of mouth opening due to masseter and/or temporalis TrPs has been present for a long time. Digastric TrPs rarely occur when only the neck muscles are involved with TrPs; they usually occur only if some of the mandibular elevator muscles also are involved. With posterior digastric involvement, TrPs also may occur in the retrusion synergists: the posterior fibers of the temporalis and the deep fibers of the masseter, often on the contralateral side. In painful Eagle syndrome, the posterior digastric and stylohyoid are likely to harbor active TrPs; the longus colli also may become involved.

With **anterior digastric** involvement, other TrPs are likely to develop in the antagonistic masseter on the same side.

12. TRIGGER POINT RELEASE (Figs. 12.6, 12.7, and 12.8)

Cervical muscles frequently work in pairs and are involved bilaterally; therefore, they should be treated bilaterally. Excessive forward-head posture should be corrected, and the patient should be given instruction for home follow-through.

Spray and Release

Digastric Muscle. To apply intermittent cold and release (stretch) for the right digastric muscle, the clinician sprays the vapocoolant (or applies icing) in the pattern illustrated in Figure 12.6A which includes the anterior and posterior bellies of the muscle. The intermittent cold is applied *prior* to release of the muscle. If vapocoolant spray is used, it is applied while the patient exhales in order to avoid inhalation of spray vapor. Extra precaution must be taken with patients who suffer from respiratory problems; icing is an acceptable substitute. The sweeps of spray (or icing strokes) are applied over the entire muscle and over the referred pain zone, including the area under the chin, the upper part of the sternocleidomastoid muscle, the mastoid area, and extending to the occiput if involved. The vapocoolant application also includes the skin covering the painful lower teeth in the case of anterior digastric involvement.

Figure 12.6B illustrates stretch release of the right digastric muscle using postisometric relaxation with the patient supine. The clinician's left hand resists the patient's attempt to open the mouth (isometric contraction phase) while the right thumb exerts minimal pressure on the hyoid bone on the side of trigger point (TrP) involvement. The clinician instructs the patient to open the mouth gently and breathe in, to hold the breath momentarily, and then to breathe out slowly and relax fully. As tension releases under the light pressure applied by the clinician's thumb, the hyoid bone moves toward the left (contralateral) side. The respiratory-augmented isometric contraction and relaxation phases can be repeated to gain complete release of trigger point tension. This procedure was fully described and illustrated by Lewit.[37]

Suprahyoid and Infrahyoid Muscles. To apply spray and release to the **suprahyoid** muscles, the clinician has the patient extend the head and neck sufficiently to

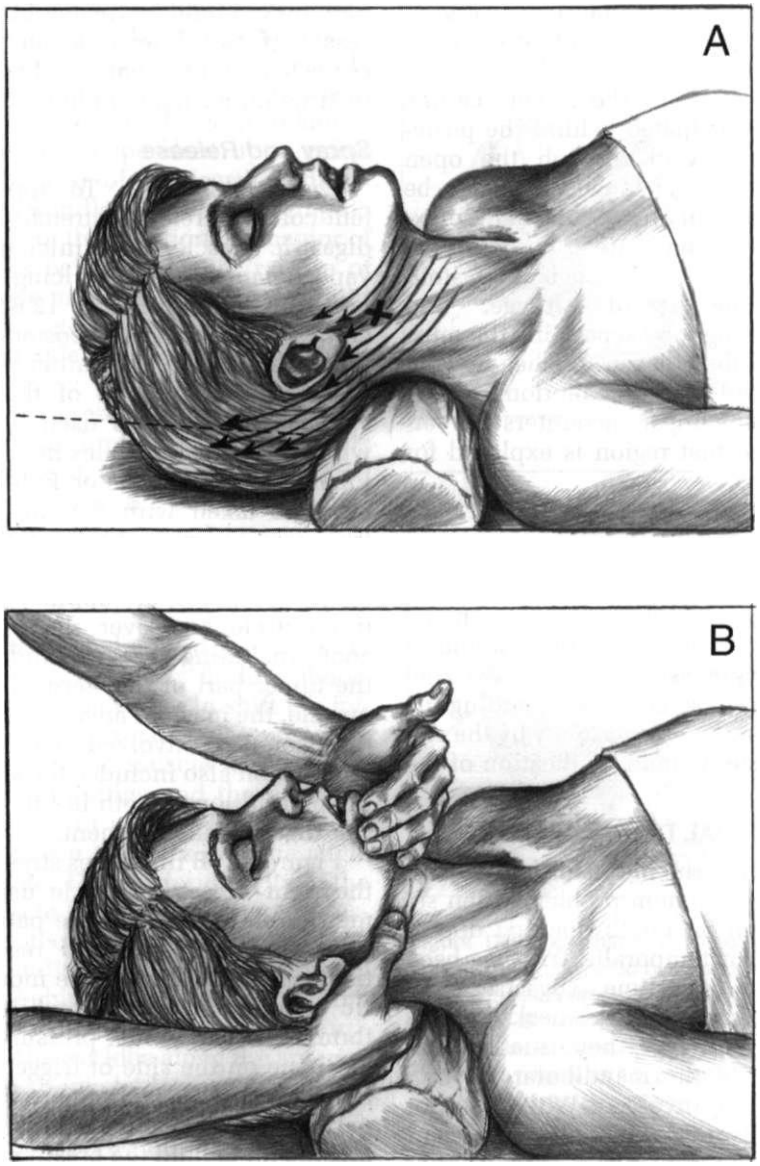


Figure 12.6. Application of intermittent cold and release (stretch) to the right digastric muscle. See text for description of techniques. **A,** Vapocoolant (or icing) pattern *(arrows)* for anterior and posterior bellies of the muscle. **B,** Stretch release of the right digastric muscle utilizing postisometric relaxation, patient supine. The relation of the thumb to the hyoid bone is illustrated. (Adapted from Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991.)

take up the slack in the anterior neck muscles, but no farther. Parallel sweeps of spray are applied as illustrated in Figure 12.7A.

The clinician then lengthens and releases the suprahyoid group of muscles (Fig. 12.7B) by stabilizing (anchoring) the hyoid bone using thumb and index fingers of one hand (right hand, in this case) and by taking up soft-tissue slack in an upward direction toward the mandible with the other (left) hand. This stretches and releases the **anterior digastric** along with other suprahyoid muscles.

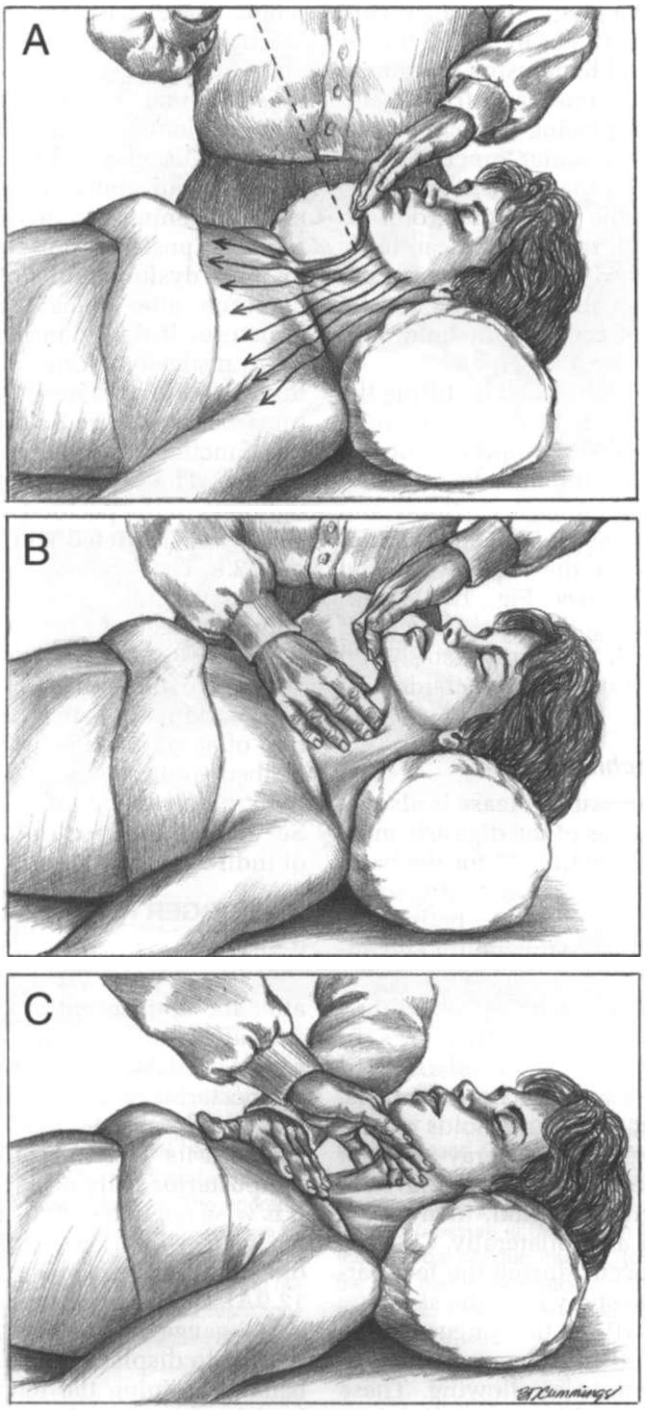


Figure 12.7. Spray and release of the suprahyoid and infrahyoid muscles. **A,** left half of spray pattern (arrows). **B,** manual release of the suprahyoid group of muscles. **C,** manual release of infrahyoid muscle group. See text for details.

The clinician lengthens the **infrahyoid** muscle group (Fig. 12.7C) by stabilizing or anchoring the hyoid bone using the thumb and index fingers of one hand (right hand in Fig. 12.7C) and placing the other hand across the sternoclavicular junctions, applying pressure with that hand caudalward to take up slack in the infrahyoid group. To facilitate the stretch release, one can use a contract-relax procedure by having the patient actively press the tip of the tongue against the roof of the mouth, hold, and then relax.

The **omohyoid** is stretched by tilting the head to the contralateral side and depressing the scapula while applying downsweeps of spray over the muscle.

Anterior Vertebral Muscles. Spray and stretch can be applied in a manner similar to that described for the suprahyoid and infrahyoid muscles (see Fig. 12.7), with slightly more head and neck extension if not contraindicated. The spray also should include both heads of the sternocleidomastoid bilaterally.

Other Release Techniques

Trigger point pressure release is also effective for both bellies of the digastric muscle *[see* Chapter 3, Section 12 for the basic technique). Hong[30] used stretching massage for the anterior digastric belly with one finger placed inside the mouth and another outside.

To relieve patients who speak with a hoarse voice because of active TrPs in the laryngeal muscles, the head is tilted back to stretch the anterior neck muscles. While the patient sings and holds a note ("Ahhh-"), the vapocoolant spray is swept upward from the sternum and clavicles covering the laryngeal region, then to the chin and mastoid area bilaterally. Clearing of the tone may occur during the few parallel sweeps of the spray over the skin.

Patients with TrPs in the longus capitis and/or longus colli muscles are likely to complain of difficulty swallowing. These TrPs have been treated by some clinicians by the application of 1.0 watt/cm^2 of **ultrasound** directed along the lateral border of the spinal column.

Additional Anterior Releases. Patients experiencing flexion/extension injuries in motor vehicle accidents usually develop TrP tightness of the posterior cervical muscles and of the suprahyoid and infrahyoid muscles that requires release. Usually, these patients also have developed tightness of muscular and fascial tissues in the pectoral and abdominal regions. Tension and shortening in anterior structures can overload posterior muscles and contribute to joint dysfunction. Posterior pain may continue after release of the posterior structures if tight anterior structures have been neglected. Release of tightness in these additional anterior myofascial structures below the neck also is essential for full functional recovery of these accident victims. The techniques for releasing this tightness in the pectoral and abdominal regions are illustrated and described in Figure 12.8. These releases may be done either before or after spray and stretch to involved muscles.

Another way of approaching treatment is through **indirect techniques,** for example, working in a direction to find a position of ease that effects release. Jones[33] described indirect techniques for dealing with involvement of anterior structures. See Chapter 3, Section 12 for a discussion of indirect techniques.

13. TRIGGER POINT INJECTION (Fig. 12.9)

If trigger point (TrP) sensitivity persists **after** the application of spray and release, and **after** TrP pressure release, injection can be tried. With the patient supine, either the posterior or anterior belly of the digastric muscle may be fixed between the fingers and its TrPs injected. When injecting the **posterior** belly of the digastric muscle, it is wise not to penetrate the external jugular vein which is readily identified by blocking the vein lower in the neck (Fig. 12.9A). During injection with a 3.8-cm (1 1/2 in) 22-gauge needle (Fig. 12.9B), one finger is used to displace the vein, while the taut band containing the tender TrPs is localized between two fingers for tactile guidance of the needle. The internal carotid neurovascular bundle lies deep to the muscle.[522] It is avoided by determining the size of the muscle by palpation to begin with, and then by injecting within the confines of

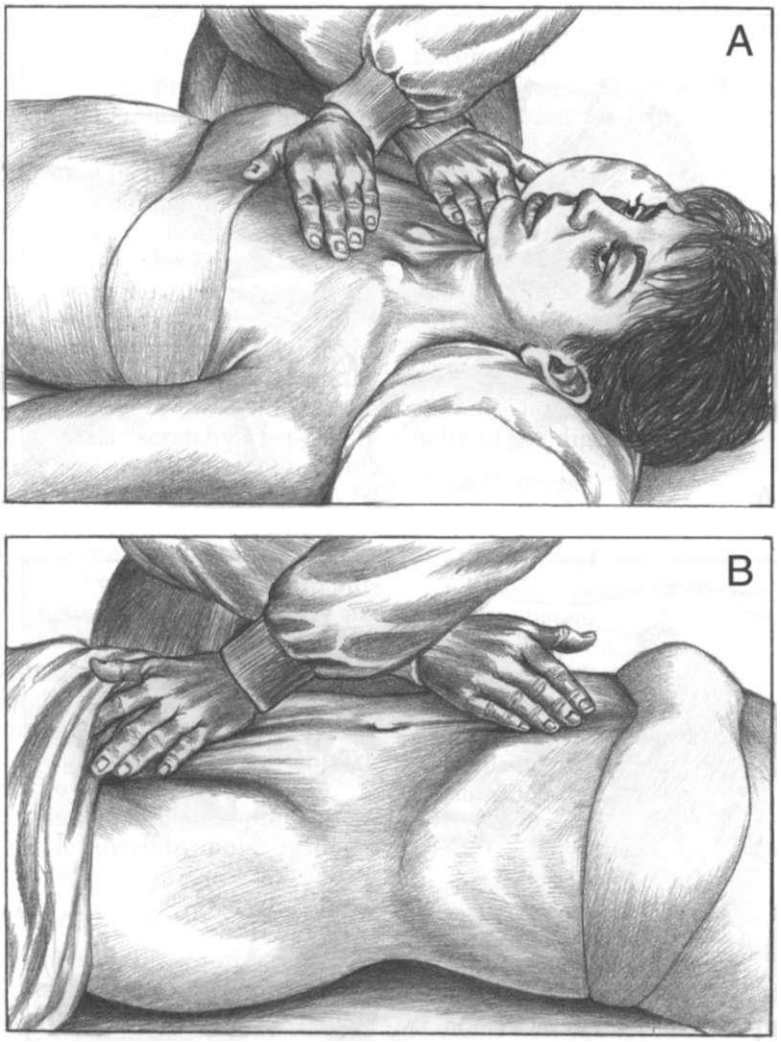


Figure 12.8. Additional anterior releases. **A, pectoral region** myofascial release. One hand applies gentle pressure in a diagonal superior and lateral direction at the shoulder joint region, and the other hand applies pressure in an opposite direction at the sternum, releasing the pectoral myofascial tissues. The operator does not force, but rather encourages the release by applying gentle pressure just to the barrier (resistance of the tissues). The hands then follow the releasing tissues (taking up slack) to the point of next resistance (barrier); the clinician waits for release again, repeating until release is complete and movement of the tissues is not restricted. This release should be repeated on the other side of the body. **B, abdominal region** myofascial release. One hand applies pressure in a caudal direction at the pubic area, and the other hand applies counter-pressure in a diagonal upward direction. The upward pressure is directed in line with the fibers of the external oblique muscle, toward the shoulder of the side that the operator is releasing (the right in this case). This release also should be performed on the contralateral side.

the muscle; the needle is directed posteriorly, as illustrated (Fig. 12.9B). A 27-gauge needle can be used, but only with the Hong technique *(see* Chapter 3, Section 13).

A local twitch response is an important indicator of a successful injection. When injecting these posterior digastric TrPs, no effort is made to distinguish the posterior belly of the digastric from the stylohyoid muscle. Needle penetration of these TrPs may cause a flash of pain over the occipital region, especially if that spillover pat-

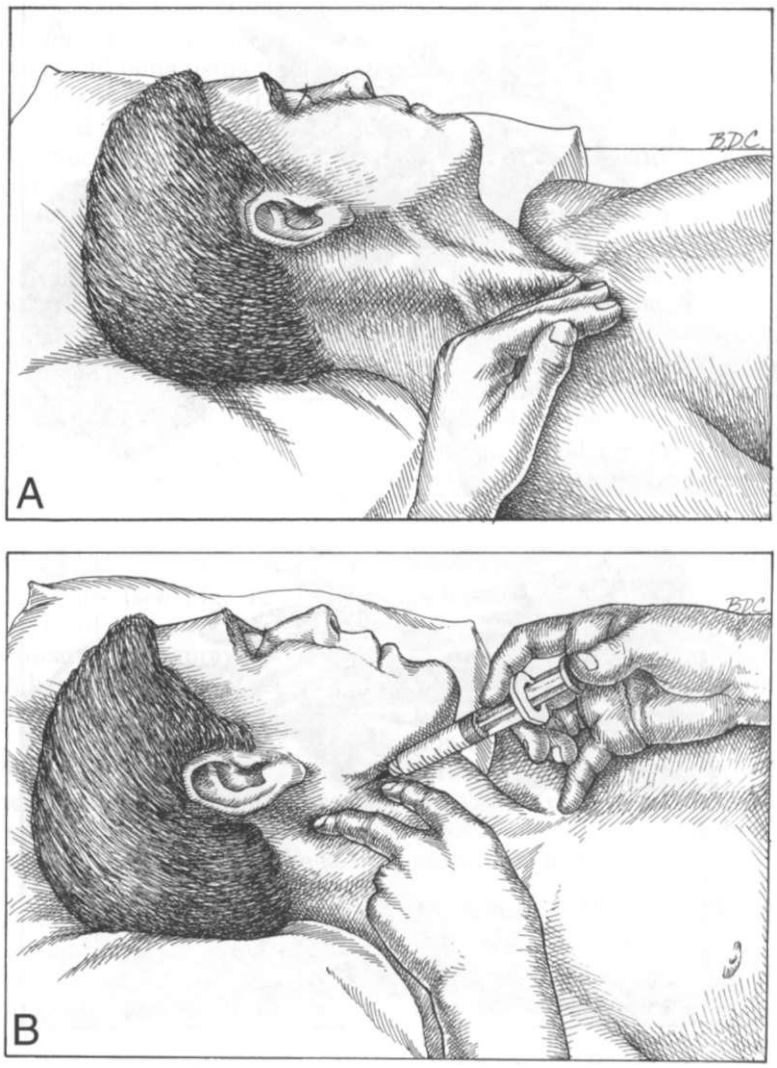


Figure 12.9. Injection of the posterior belly of the digastric muscle. **A,** manual occlusion of the external jugular vein to demonstrate its path near the angle of the jaw. **B,** injection of the muscle belly using the index finger to displace the external jugular vein to one side. The middle finger presses against the sternocleidomastoid muscle on the posterior aspect of the digastric, and the posterior belly is fixed between the two fingers at the TrP.

tern is part of the patient's current pain complaint.

To inject TrPs in the digastric **anterior** belly, the head and neck of the patient are extended, and the TrP spot tenderness in the taut subcutaneous muscle fibers is localized between two fingers of the palpating hand for injection.

If one finds it necessary to inject the other suprahyoid or the infrahyoid muscles, a shorter and a small (1 inch, 27-gauge) needle is recommended with due consideration given to the local anatomy.

Injection of the **longus colli muscle** is difficult and requires an advanced level of practitioner experience and technique. The guide fingers are placed along a lateral border of the trachea and slowly advanced by separating the musculature from the adjacent trachea by gentle rocking and wiggling motions of the fingers. This palpatory advance stops when the fingertips reach the

anterior portion of a vertebra, and the depth beneath the skin is carefully noted. Changes in direction of pressure help to locate the areas of maximum tenderness.

The longus colli can be a very thin muscle. Here, the Hong technique *[see* Chapter 3, Section 13) is recommended for holding the syringe. The needle is advanced along the path identified by the fingers. It is advanced very slowly and gently as it approaches the depth of the vertebral structures to minimize hard contact with the bony vertebra. Even gentle contact with the bone can bend the tip of the needle into a "fishhook" that feels "scratchy," especially whenever the needle is retracted. When this happens, the needle should immediately be withdrawn and replaced. No "fishhook" should develop if the needle is moved sufficiently slowly and gently. The anterior surface of the longus colli is very gently explored with the needle tip in the regions where palpation against the anterior surface of the vertebral column elicited the greatest deep tenderness. The operator's palpating finger should remain in contact with the TrP during the entire course of TrP injection.

After injection, stretch and spray are repeated at once, followed by moist heat over the anterior neck.

14. CORRECTIVE ACTIONS

Postural analysis and training are covered in Chapters 5 and 41, Section C.

Self-application of TrP pressure release on the superficial TrPs can be quite effective for the superficial muscles. The patient must understand the concept of referred pain and learn exactly where to press on the posterior belly of the digastric muscle deep to the angle of the mandible, rather than on the sternocleidomastoid muscle where the "soreness" of the referred tenderness is usually felt by the patient.

The patient can be instructed in self-treatment of the digastric muscle utilizing a technique similar to that described previously in Section 12 and as described by Lewit.[37] The patient can perform postisometric relaxation while sitting at a table, chin supported by one hand, and using the other hand at the hyoid bone.

Steps should be taken to have the patient stop retrusive bruxism and to restore breathing through the nose, rather than through the mouth. The latter favors depression and retrusion of the mandible causing activation and shortening of the digastric muscles. Malocclusion may require permanent correction when symptoms persist after the masticatory TrPs have been inactivated.

The patient can do an active jaw-protrusion exercise, lying supine. If the mandible deviates to one side during active opening and closure, the patient should rhythmically resist deviation, pushing the mandible to the opposite side with the fingers while the jaws are less than half open. This exercise helps to stretch a tight posterior belly of the digastric muscle.

Case *Reports*

Case Number 1 (see reference 30)

A 42-year-old white male complained of progressive pain in the floor of the mouth with radiation to both ears for 10 years before the final diagnosis was made of Eagle syndrome. Surgical amputation of the elongated styloid process was performed, but the patient still had persistent pain in the floor of the mouth and in the ears. Examination revealed several trigger points (TrPs) in bilateral digastric muscles (both heads) with marked local twitch responses and typical referred pain patterns to the anterior neck and the ear. Other muscles involved included the longus colli, suprahyoid, and infrahyoid muscles. The patient was treated with injection of TrPs in muscles of the floor of the mouth, and stretching massage of the anterior belly of the digastric muscle (with one finger placed inside the mouth and another finger outside). After 2 months of treatment, he had made significant improvement. Subjectively, the severity of pain had reduced to only 50% of the previous level and the frequency of severe pain attack was also reduced by half. Six months later, he reported that he had further improvement to only 20% of original pain level.

Case Number 2 (see reference 86)

The patient is a 59-year-old female who was referred by her physician for evaluation of painful, burning tongue and a lesion of the anterior dorsum. The burning sensation had been present for 9 years. The lesion was first noticed by the patient at the time the pain began. It presented as

a slightly raised, whitish area with interspersed erythema.

An incisional biopsy had been performed on the lesion, and the histology was reported as mild nonspecific chronic inflammation. Wide local excision and lingual frenectomy completed later failed to give the patient relief, and the lesion soon recurred. A fourth surgical procedure was equally unrewarding. A variety of medications had been prescribed, including corticosteroid, antibiotics, analgesics, and vitamins. None effected relief.

The patient reported that she periodically had brief, intermittent, spontaneous relief, and there had been changes in the pattern and intensity of the lesion. The local tenderness was increased by cold, heat, spices, salt, and the near presence of volatile liquids, such as cleaning fluid.

Examination and Diagnosis. On physical examination, Dr. Konzelman found the patient to be an apparently healthy, cooperative, and alert person. Her medical history was unremarkable except for thyroid surgery of a benign nodule in **1946,** hysterectomy in **1957,** and surgery of the tongue in **1975, 1976, 1980,** and **1981.** She had no apparent neurologic deficits. A review of her systems showed them to be essentially normal. Screening laboratory studies included a hematology profile and SMAC**-22.** All values were within normal limits.

All of the patient's oral tissues appeared normal except for surgical scars of the right ventrolateral aspect of the tongue and the ill-defined lesion of the right anterior dorsum and lateral border. The lesion was serpiginous over a **1** cm area and consisted of a depapillated center with slight peripheral hyperkeratinization, but no induration.

Palpation of the stomatognathic muscles revealed tenderness and palpable muscle TrPs in the right mylohyoid and in the anterior belly of the digastric, which referred pain to the tongue when palpated.

The examinations led Dr. Konzelman to diagnose the patient's problems as the following:

1. Benign migratory glossitis, clinically pathognomonic.
2. Glossodynia secondary to referred myofascial TrPs.

Treatment. Diagnostic therapy consisted of **1** hour of transcutaneous electric neural stimulation (TENS) through transcranial leads in the area of the coronoid notches, with a cervical spine ground. (Please note that most conventional TENS units cannot be used transcranially. Two units that *can* safely be used in this way are the Myomonitor, when used with a cervical spine ground, and the Pain Suppressor, a high-frequency TENS). The purpose of this treatment was to relax the muscles innervated by cranial nerves V and VII, which are accessible through the coronoid notch. The patient experienced complete pain relief which lasted for about **48** hours. Dr. Konzelman explained the nature of the patient's tongue lesion and advised her that no treatment was indicated.

She returned in **1** week, and her muscles were again pulsed with TENS for **1** hour. At this visit, she was given a flat intraoral orthotic to disengage dental intercuspation and to permit a more relaxed mandibular muscle movement. Her glossodynia has not returned.

NOTE: Dr. Konzelman commented that glossodynia secondary to referred myofascial pain has been known and treated for many years (L. A. Funt, Personal communication).

REFERENCES

1. **Adson AW: Cervical ribs: symptoms, differential diagnosis and indications for section of the insertion of the scalenus anticus muscle. / *Int College Surg* 26:546-559, 1951 (p. 548).**
2. **Agur AM: *Grant's Atlas of Anatomy,* Ed. 9. Williams & Wilkins, Baltimore, 1991:507 (Fig. 7.79).**
3. ***Ibid.* (p. 569, Fig. 8.33).**
4. ***Ibid.* (p. 496, Fig. 7.64).**
5. ***Ibid.* (p. 562, Fig. 8.20).**
6. ***Ibid.* (p. 561, Fig. 8.19).**
7. ***Ibid.* (p. 563, Fig. 8.24).**
8. ***Ibid.* (p. 565, Fig. 8.26).**
9. ***Ibid.* (p. 625, Fig. 9.17).**
10. **Bardeen CR: The musculature. Section 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (pp. 378, Fig. 379).**
11. **Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 431, 453-456, 467).**
12. ***Ibid.* (p. 469).**
13. **Bell WH: Nonsurgical management of the pain-dysfunction syndrome. / *Am Dent Assoc* 79:161-170, 1969.**
14. **Bonica JJ, Sola AE: Neck pain. Chapter 47. In: *The Management of Pain,* Ed. 2. Edited by Bonica JJ,**

Loeser JD, Chapman CR, et al. Lea & Febiger, Philadelphia, 1990 (pp. 848-867).
15. Burch JG: *Occlusion related to craniofacial pain.* Chapter 11. In: *Facial Pain.* Ed. 2. Edited by Ailing III CC, Mahan PE. Lea & Febiger, Philadelphia, 1977 (p. 171, Fig. 11-11).
16. Carlsbo S: An electromyographic study of the activity of certain suprahyoid muscles (mainly the anterior belly of digastric muscle) and of reciprocal innervation of the elevator and depressor musculature of the mandible. *Acta Anat* 26:81-93, 1956.
17. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 457-463, Figs. 6-15, 6-16, 6-17).
18. *Ibid.* (pp. 460, 461, Fig. 6-15).
19. *Ibid.* (pp. 1428, 1429).
20. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 598- 600).
21. *Ibid.* (Fig. 608).
22. *Ibid.* (Figs. 580-582).
23. *Ibid.* (Figs. 623, 625).
24. *Ibid.* (Fig. 754).
25. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (p. 275, Fig. 34).
26. Eriksson PO: Muscle fiber composition system. *Swed Dent J 12(Suppl):6-3S,* 1982.
27. Eriksson PO, Eriksson A, Ringvist M, et al.: Histochemical fibre composition of the human digastric muscle. *Arch Oral Biol* 27(3J:207-215, 1982.
28. Ernest EA III, Salter EG: Hyoid bone syndrome: a degenerative injury of the middle pharyngeal constrictor muscle with photomicroscopic evidence of insertion tendinosis. / *Prosthet Dent 66(l):78-83,* 1991.
29. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 146, 147).
30. Hong CZ: Eagle syndrome manifested with chronic myofascial trigger points in digastric muscle. *Arch Phys Med Rehabil* 70.A-19, 1989.
31. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. / *Musculoske Pain* 2(2j:29-59, 1994.
32. Janda V: Evaluation of muscular imbalance. Chapter 6. In: *Rehabilitation of the Spine: A Practitioner's Guide.* Edited by Liebenson C. Williams & Wilkins, Baltimore, 1996 (pp. 97-112).
33. Jones LH: *Strain and Counterstrain.* The American Academy of Osteopathy, Colorado Springs, 1981 (pp. 57-59, 66-69).
34. Kelly RJ, Jackson FE, DeLave DP, *et al:* The Eagle syndrome: hemicrania secondary to elongated styloid process. *US Navy Med* 65:11-16, 1975.
35. Kendall FP, McCreary EK, Provance PG: *Muscles, Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 320, 321).
36. Konzelman JL Jr: Glossodynia: a case report. / *Craniomandib Pract* 3(1J:82-85, 1984.
37. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 24, 192, 193, Fig. 6.84a).
38. Loch C, Fehrman P, Dockhorn HU: [Studies on the compression of the external carotid artery in the region of the styloid process of the temporal bone]. *Laryngorhinootologie* 69(5j:260-266, 1990.
39. McMinn RM, Hutchings RT, Pegington J, et al.: *Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, St Louis, 1993 (pp. 44, 46).
40. Moyers RE: An electromyographic analysis of certain muscles involved in temporomandibular movement. *Am JOrthod* 36:481-515, 1950.
41. Munro RR, Basmajian JV: The jaw opening reflex in man. *Electromyography* 11:191- 206, 1971 (p. 205).
42. Rask MR: The omohyoideus myofascial pain syndrome: report of four patients. / *Cranio Prac* 2:256-262, 1984.
43. Rocabado M, Iglarsh ZA: *Musculoskeletal Approach to Maxillofacial Pain.* J.B. Lippincott Company, Philadelphia, 1991 (pp. 119, 120, 152, Fig. 13.4).
44. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am JPhys Med* 34:585-590, 1955.
45. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2, S. Hirzel, Leipzig, 1922 (p. 271).
46. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul, Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 292).
47. *Ibid.* (p. 297).
48. van Willigen JD, Morimoto T, Broekhuijsen ML, et al.: An electromyographic study of whether the digastric muscles are controlled by jaw-closing proprioceptors in man. *Arch Oral Biol 38(6):497-505,*1993.
49. Williams HL: The syndrome of physical or intrinsic allergy of the head: myalgia of the head (sinus headache). *Proc Staff Meet Mayo Clin* 20:177-183, 1945 (p. 181).
50. Woelfel JB, Hickey JC, Stacey RW, *et al.:* Electromyographic analysis of jaw movements. / *Prosthet Dent* 10:688-697, 1960.

CHAPTER 13
Cutaneous I: Facial Muscles (Orbicularis Oculi, Zygomaticus Major, Platysma and Buccinator)

HIGHLIGHTS: The orbicularis oculi, zygomaticus major, platysma, and the buccinator muscles serve as examples for trigger point (TrP) involvement, which may be found in any of the muscles of "facial expression." **REFERRED PAIN** to the nose is rarely caused by TrPs in any muscle except the orbicularis oculi. The zygomaticus major refers pain in an arc close to the side of the nose and up to the forehead. The platysma refers a prickling sensation over the lower jaw. **ANATOMICAL** attachments of these skin muscles are usually to subcutaneous fascia; only rarely do they attach to bony structures. **FUNCTION** of the orbicularis oculi is to close the eye tightly, and of the zygomaticus major is to draw the corner of the mouth upward and laterally, as in smiling. Functions of the platysma are to tense the skin of the anterior neck and to pull the corner of the mouth downward. The buccinator assists the tongue in moving food about the mouth during chewing. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in these skin muscles may occur because the muscles often lie in the pain reference zones of TrPs in the sternocleidomastoid and masticatory muscles. **TRIGGER POINT EXAMINATION** requires careful exploration of the subcutaneous tissue, using pincer (simultaneous intraoral and extraoral) palpation when possible, and flat palpation when necessary. **TRIGGER POINT RELEASE** by spray and stretch is usually more effective for the platysma than for the other muscles. **TRIGGER POINT INJECTION** effectiveness requires injection precisely into each TrP of these muscles. **CORRECTIVE ACTION** for these skin muscles involves chiefly the inactivation of key TrPs in other muscles responsible for these satellite foci of hyperirritability.

1. REFERRED PAIN (Figs. 13.1 and 13.2)

Orbicularis Oculi (Fig. 13.1 A)

This is one of the few muscles from which trigger points (TrPs) refer pain to the nose (Fig. 13.1 A). No muscle is known to refer pain to the tip of the nose. Less intense pain may be felt in the cheek close to the nose and over the upper lip, homolaterally.[25]

Zygomaticus Major (Fig. 13.1B)

The TrPs in this muscle refer pain in an arc that extends along the side of the nose and then upward over the bridge of the nose to the mid-forehead (Fig. 13.IB).[25]

Platysma (Fig. 13.1C)

Active TrPs in the platysma usually overlie the sternocleidomastoid muscle, and refer a strange prickling pain to the skin over the lateral surface of, and just below, the mandible on the same side (Fig. 13.1C). A platysma TrP just above the clavicle may refer hot prickling pain across the front of the chest.

Buccinator (Fig. 13.2A)

From TrPs in the buccinator muscle, the patient experiences pain locally in the cheek (Fig. 13.2A) and pain referred deep to the cheek as a subzygomatic ache in the jaw.[16]

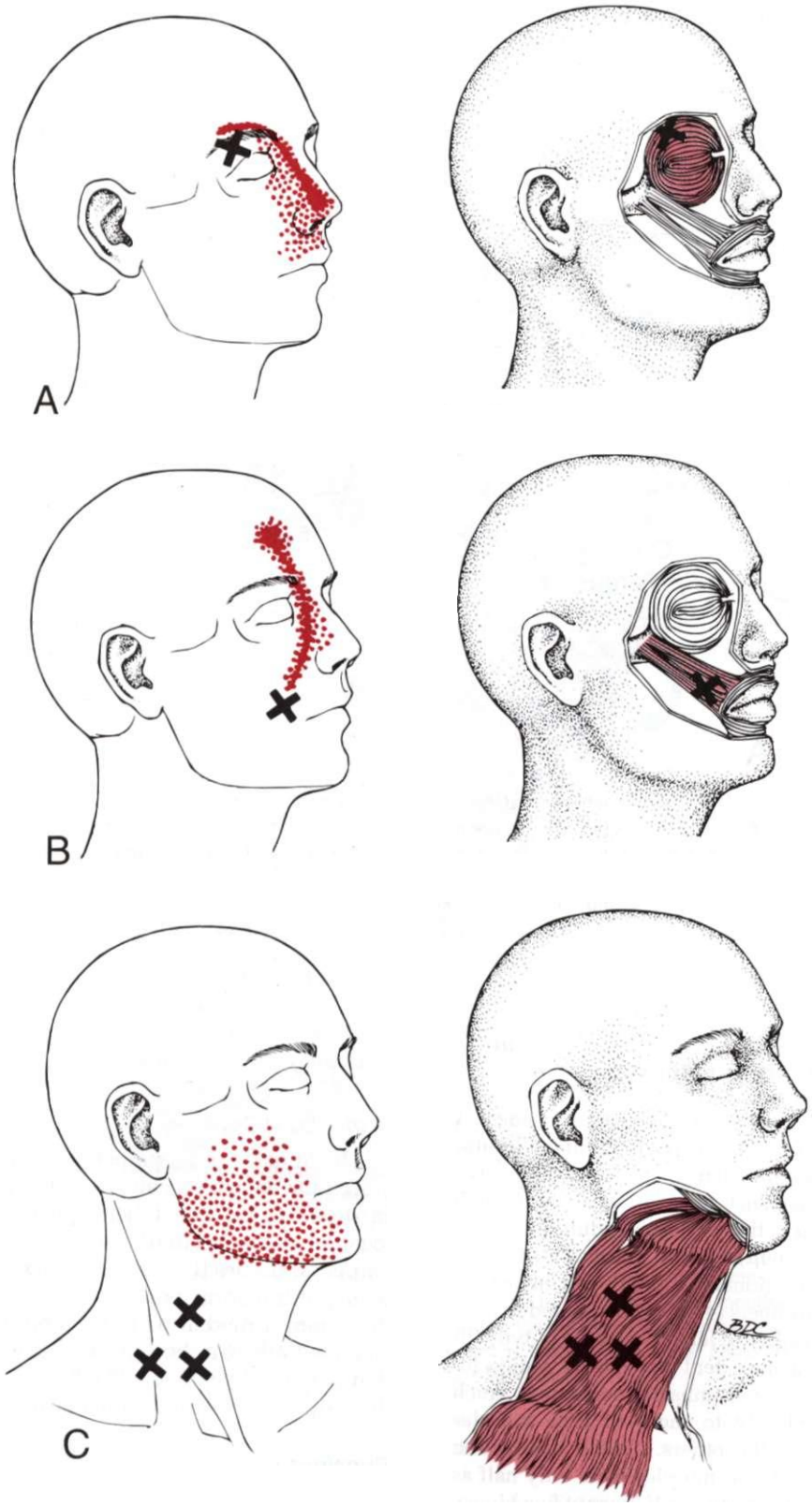


Figure 13.1. Pain patterns (dark *red)* and the trigger points (Xs) from which the pain is referred. **A,** orbital portion of the right orbicularis oculi muscle. **B,** right zygomaticus major muscle. C, right platysma muscle.

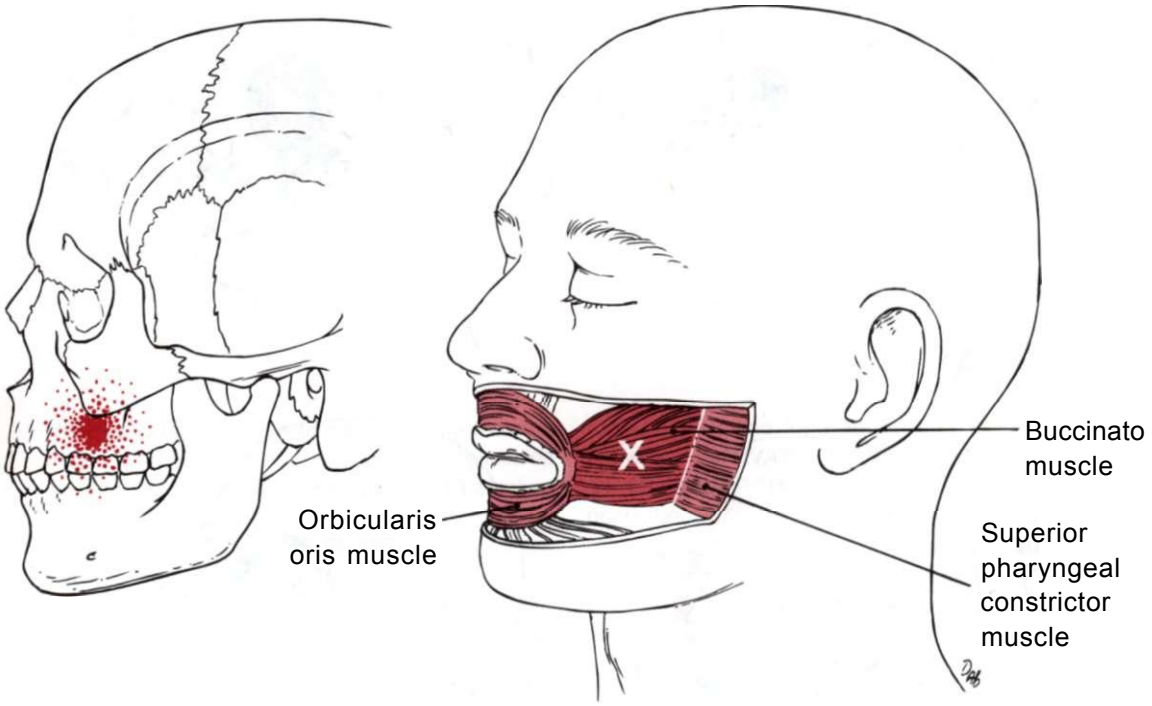


Figure 13.2. Pain pattern and attachments of the buccinator muscle. **A,** pain pattern (*dark red)* showing location of pain in the cheek and deep to it in the subzygomatic portion of the jaw. **B,** trigger point (X) in the central portion of the buccinator muscle *(medium red).* The buccinator blends anteromedially with the fibers of the orbicularis oris muscle (*light red).* Posterolaterally, it attaches chiefly to the tendinous inscription that also anchors the superior pharyngeal constrictor muscle *(light red).*

2. ANATOMY (Figs. 13.2 and 13.3)

The fibers of these cutaneous muscles lie within the superficial fascia.

The mean diameter of types I and IIA fibers in surgically excised human levator labii, zygomaticus major, orbicularis oris, and platysma muscles was nearly half (32-40u,)[23] that of normal adult limb muscles (57-69u).[6] These muscles also had a disproportionately high percentage of type IIA fibers (48-68%) compared to limb muscles (29%) largely in place of type IIB and to some extent type I fibers. Values for the platysma muscle compared much more closely to those of limb muscles than did the others. Twitch contraction times of facial muscles were only half as long as limb muscle.[17] Twenty five biopsy samples[23] of these facial muscles were taken only from the central part of each muscle, distant from the myotendinous junction. Motor endplates and fine motor nerves were seen in every specimen, frequently in large numbers.

Orbicularis Oculi

This muscle has a palpebral portion contained in the eyelids, and an orbital portion surrounding the lids. Fibers of both portions together form a circular path around the palpebral fissure (Fig. 13.3). Fibers of the orbital portion form bony attachments along the superior medial part of the orbit and attach medially to a short fibrous band, the medial palpebral ligament. The fibers surround the palpebral fissure in concentric curves.[8]

Buccinator

The buccinator (Fig. 13.2B) is the principal muscle of the cheek forming the lateral wall of the oral cavity.[10] Anteromedi-

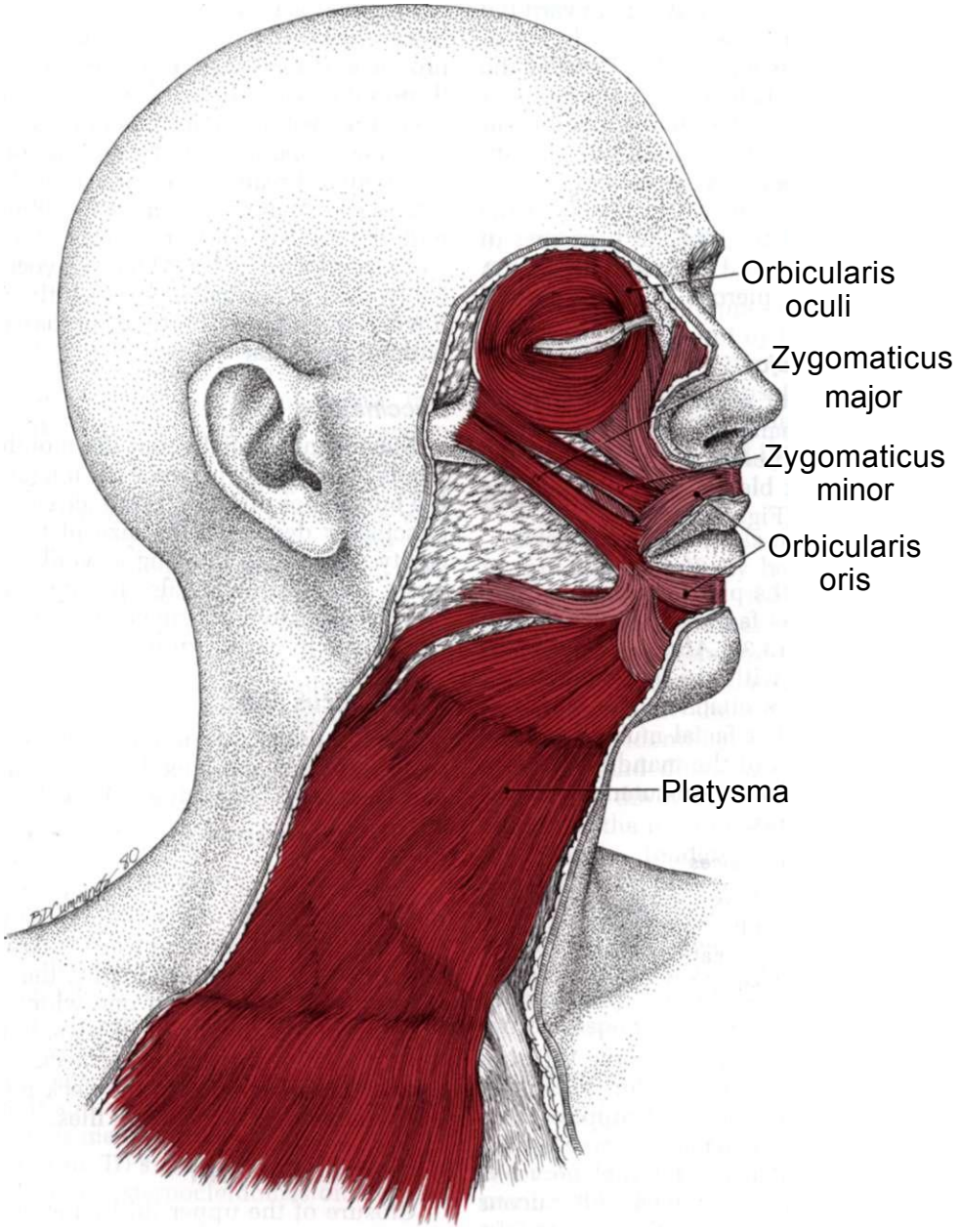


Figure 13.3. Attachments of selected facial muscles and face-related cutaneous muscles. The orbicularis oculi, the zygomaticus major, and the platysma are *dark red.* The palpebral portion of the orbicularis oculi covers only the eyelids; the remaining fibers are the orbital portion. The zygomaticus major reaches from the zygoma to the corner of the mouth. The platysma connects the skin muscles near the mouth to the subcutaneous fascia of the upper chest. The orbicularis oris is *light red.*

ally, buccinator fibers converge toward the angle of the mouth where they divide to become continuous with the fibers of the orbicularis oris. Laterally, the buccinator attaches chiefly to the pterygomandibular raphe, a tendinous inscription that also anchors the superior pharyngeal constrictor. Posterolaterally, some fibers attach to the outer surfaces of the alveolar processes of the maxilla above and the mandible below. The muscle is pierced by the parotid duct.[13]

Zygomaticus Major

This muscle of mouth control attaches ***above*** to the malar surface of the zygomatic bone and ***below*** to the angle of the mouth, where it blends with fibers of the orbicularis oris (Fig. 13.3).[9]

Platysma

The fibers of the platysma muscle lie in the subcutaneous fascia of the lower face and neck (Fig. 13.3). ***Above,*** many of its fibers interlace with the orbicularis oris while other fibers attach to the corner of the mouth, to other facial muscles, and to the lower margin of the mandible; ***below,*** the fibers attach to the subcutaneous fascia of the upper thorax.[11]

Supplemental References

Other authors have clearly illustrated the orbicularis oculi,[1,8,14,19,22] the buccinator,[3,10,20] the zygomaticus major,[1,9,14,18,22] and the platysma.[2,8,15]

3. INNERVATION

The facial nerve (cranial nerve VII) supplies the motor nerve fibers for these muscles of facial expression and supplies deep facial sensation.[26] The buccal nerve branch of the trigeminal nerve (cranial nerve V) supplies the skin of the cheek and mucous membrane of the mouth in the region of the buccinator muscle.[12]

4. FUNCTION

With concentric electrodes, the motor unit potentials of facial muscles were approximately half the duration and half the amplitude of limb muscles.[7]

Orbicularis Oculi

Activation of only the palpebral portion of the orbicularis oculi produces gentle, but rapid closure of the eye, as in blinking. Additional activation of the orbital portion produces strong closure of the eye which throws the skin into folds at the lateral angle of the eyelid.[4,8] Paralysis of the orbicularis oculi abolishes tight closure of the eye, which threatens the cornea with devastating dehydration and may interfere with the drainage of tears, causing them to spill over the lower lid.[18] Electromyographically, the eye normally closes gently by allowing the upper lid to drop passively without muscular contraction.

Buccinator

Movement of food about the mouth depends on interplay between the tongue and the buccinator muscles. Contraction of the buccinator decreases the size of the oral cavity. Whistling, blowing a wind instrument, and swallowing also use these muscles.[20] The paired buccinator muscles also participate in facial expression.

Zygomaticus Major

This muscle draws the angle of the mouth upward and laterally, as in smiling and laughing,[4,9] or saying, "Whee."

Platysma

Contraction of the platysma muscle pulls the angle of the mouth downward and the thoracic skin upward.[11] Also, as confirmed by electromyography, the muscle becomes active when one widens the aperture of the already open jaws, but not during swallowing or during neck movements.[5] It corresponds to the neck muscle that a horse uses to shake off flies.

5. FUNCTIONAL UNIT

Closure of the upper lid by the orbicularis oculi is antagonized by the levator palpebrae muscle. The tongue works with the buccinator muscles to control the food during chewing. The muscles of exhalation work in close cooperation with the buccinator muscles when one is blowing a wind instrument. The orbicularis oris frequently works in concert with the buccinators.

The zygomaticus major muscle is assisted by the parallel zygomaticus minor,

which also is known as the zygomatic head of the quadratus labii superioris. The platysma TrPs apparently develop in relation to involvement of the sternocleidomastoid muscle, which it overlies in parallel.

6. SYMPTOMS

Patients report pain as described in Section 1. Individuals with myofascial dysfunction of the orbicularis oculi muscle may complain of "jumpy print." When reading type with strong black and white contrast, the letters seem to jump, making it difficult to focus on them.

Prickling pain due to platysma TrPs feels like multiple pinpricks. The sensation is not like the tingling caused by an electric current, a feature which usually denotes a neurologic origin. Patients who experience this prickling pain in the face in combination with headaches from TrPs in the sternocleidomastoid muscle are often greatly concerned and baffled, as are their physicians.

When the buccinator is involved, subzygomatic jaw pain may be aggravated by chewing. The patient may have a perception of difficulty in swallowing, although the swallowing movement appears normal.[16]

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Habitual frowning, squinting (due to photophobia or astigmatism), or TrPs in the sternal division of the sternocleidomastoid muscle (which refer pain to the orbit) may activate TrPs in the orbicularis oculi muscle.[25] Myofascial dysfunction of the masticatory muscles that is severe enough to cause trismus may activate TrPs in the zygomaticus major muscle.

Platysma TrPs are activated secondarily by TrPs in the sternocleidomastoid-scalene family of muscles.

Buccinator TrPs may be activated by ill-fitting dental appliances.

8. PATIENT EXAMINATION

Activation of TrPs in the orbicularis oculi muscle may produce a unilateral narrowing of the palpebral fissure that resembles the ptosis of Horner's syndrome, but without the change in pupillary size. When upward gaze is tested, these patients tilt the head backward, because they cannot raise the upper eyelid sufficiently to look up.

Trigger-point tightness of the zygomaticus major muscle may cause restriction of the normal jaw opening by 10 or 20 mm; the opening can be improved by inactivating the TrPs in this muscle.

9. TRIGGER POINT EXAMINATION

Orbicularis Oculi

The TrPs in the upper orbital portion of this muscle are found by flat palpation, by running the tip of the examining finger crosswise over the muscle fibers that lie above the eyelid, just beneath the eyebrow and against the bone of the orbit.

Buccinator

Trigger points in this muscle are found in mid-cheek, halfway between the angle of the mouth and the ramus of the mandible. The examiner uses pincer palpation between fingers inside and outside of the mouth to find a taut band running in the direction of the muscle fibers. The band can be identified by sliding the inside finger up and down against the counterpressure of the outside finger, across the direction of the muscle fibers, while squeezing gently. Tenderness of the TrP is augmented by pressing the cheek outward, which places the buccinator muscle on increased tension. Snapping palpation of the band at the tender active TrP produces a painful, palpable, and usually visible, local twitch response in this superficial muscle.

Zygomaticus Major

To examine the zygomaticus major, the patient relaxes, either sitting or supine and the jaws are propped open as wide as is comfortable. Most of the length of the muscle can be palpated for spot tenderness by pincer grasp, placing one digit inside the cheek and one outside (see Fig. 13.6A). The palpable band is appreciated chiefly by the outside finger. See Figure 13.IB for a location of TrPs in this muscle.

Platysma
(Fig. 13.4)

Local twitch responses are not observed in the orbicularis oculi or the zygomaticus

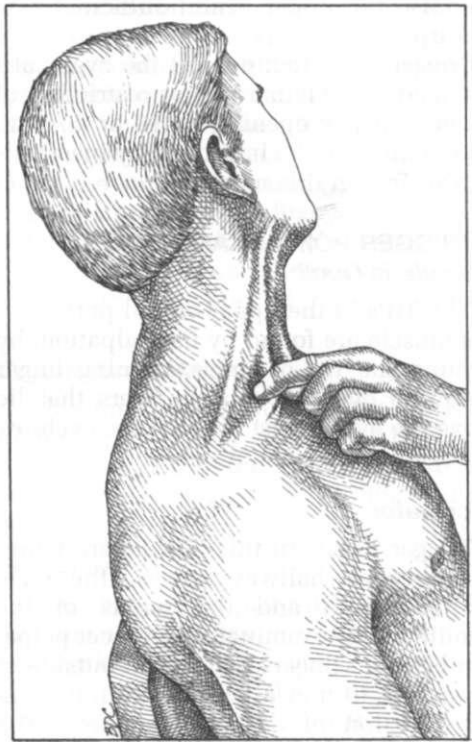


Figure 13.4. Rolling the skin of the neck between the thumb and fingers tests for the presence of active trigger points in the platysma muscle.

major, probably because it is difficult to put them on sufficient stretch. However, the twitch response of the band is likely to be seen and felt during examination of the platysma. The patient tips the head back far enough to tighten the muscle, and then the examiner pinches successive lines of skin across the muscle fibers (Fig. 13.4) approximately 2 cm (1 in) above the clavicle. Rolling the skin and platysma between the digits usually sets off the referred prickling sensation in the face (Fig. 13.1C).

10. ENTRAPMENT

No nerve entrapments have been observed due to active TrPs in these muscles.

11. DIFFERENTIAL DIAGNOSIS

Pain caused by TrPs in the orbicularis oculi, buccinator, and/or zygomaticus muscles is easily attributed erroneously to a form of tension headache. Patients with pain from buccinator TrPs are very likely to receive a misdiagnosis of temporomandibular joint (TMJ) syndrome or dysfunction, especially since they have trouble chewing and swallowing. TM joint dysfunction should be ruled out.

The sternocleidomastoid, scalene and masticatory muscles on the same side often harbor active TrPs, and platysma TrPs are rarely, if ever, seen in the absence of TrPs in one of these other muscles.

12. TRIGGER POINT RELEASE

Orbicularis Oculi

The vapocoolant spray-and-stretch technique is unsatisfactory for this muscle because of difficulty in obtaining adequate stretch and of keeping the liquid out of the eye. Pressure release of orbicularis oculi trigger points (TrPs), by rolling and progressively squeezing the TrP between the fingers as the tension releases, can be effective.

Buccinator

Spray and stretch of the buccinator is more effective than for the orbicularis oculi because the operator can stretch the muscle by pressing the cheek outward. Using this approach, postisometric relaxation and trigger point pressure release can also be effective. Electrotherapy has also been reported to be useful[16] as described in the case report at the end of this chapter.

Zygomaticus Major (Fig. 13.5A)

Either sitting or supine, the patient relaxes with the mouth opened as wide as is comfortable. The fibers of the zygomaticus major muscle are lengthened by pulling the cheek outward with one finger, as shown in Figure 13.5A (with glove applied). While the operator maintains tension on the muscle fibers, and while the patient exhales, the spray is applied upward over the muscle and then over the distribution of the referred pain. However, it is difficult to obtain an adequate stretch of this long slack muscle, so that stretch and spray may be ineffective. If the patient has asthma or another respiratory condition, ice stroking may be substituted for the spray. If spray is used, the operator's hand or a cloth can block the spray to protect the nose area.

Zygomaticus TrPs can be effectively inactivated by using trigger point pressure release and by stroking massage of the nodule and the taut band.

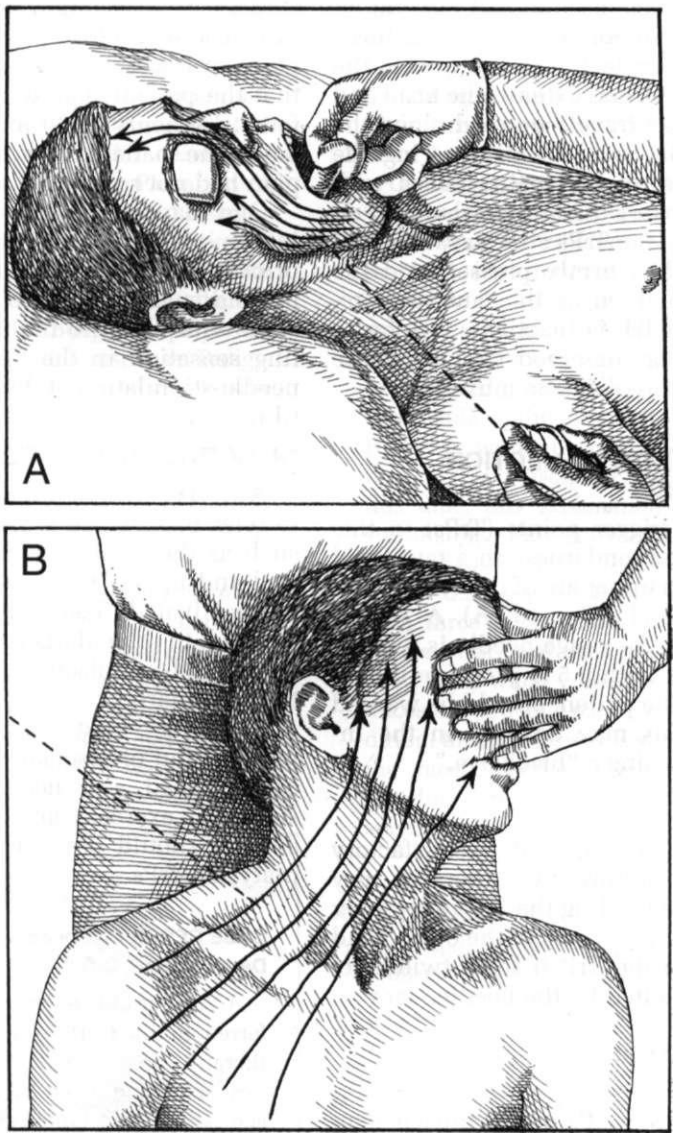


Figure 13.5. Stretch position and spray patterns *(arrows)* for two skin muscles. A, the zygomaticus major. A glove should be worn on the operator's examining hand. The fingers pull the corner of the mouth down and forward, away from the zygoma. The patient is instructed to breathe out (exhale). As the patient exhales, the operator protects the eye and applies vapocoolant to the cheek and face area as shown. Ice stroking followed by deep massage is an alternative treatment. **B,** platysma muscle. The head and neck are extended and the face turned to the opposite side, as the spray is applied upward (see also Figure 8.5 for combined stretch release including the platysma muscle). **CAUTION:** Spray only while patient is exhaling. Patients with asthma or other respiratory conditions may not tolerate the spray. Ice stroking may be used instead.

Platysma
(Fig. 13.5B)

With the patient seated, and the arm on the same side anchored, the operator turns the patient's face to the side opposite the involved muscle, and extends the head and neck. The spray travels upward along the line of the platysma fibers, covering the muscle and its referred pain pattern with parallel sweeps of the vapocoolant. If, on reexamination, the TrPs still show signs of activity, a TrP is firmly pressed and the pressure maintained as the taut band releases and the TrP is inactivated. Refer to Figure 8.5 for a combined stretch release that includes the platysma muscle.

13. TRIGGER POINT INJECTION

Orbicularis Oculi

Locate the trigger points (TrPs) in this muscle by focal tenderness in a taut band palpated in the upper arc of the orbital portion of the muscle (Fig. 13.1A). A 16-mm (5/8-in), 25- or 26- gauge needle is used to inject the TrPs with 0.5% procaine in isotonic saline. The patient should be warned that ecchymosis may develop in the injected area, causing a "black eye."

Buccinator

If necessary because of unsatisfactory response to noninvasive therapy, dry needling while holding the TrP in a pincer grasp between the fingers of the other hand will inactivate the TrP if local twitch responses are elicited by the needle.

Zygomaticus Major
(Fig. 13.6)

Injection of the TrPs in this muscle usually is more effective than treatment by stretch and spray. A pincer grasp holds the TrP between the digits (as during examination) for injection of the taut band at its most tender point under tactile guidance (Fig. 13.6).

Platysma

Injection is rarely required to clear this muscle of active TrPs. When used, it should be followed by several active contractions followed by relaxation in the lengthened position.

Lapeer[21] reported relieving neck pain of eight months duration following neck surgery by inserting a 34-gauge acupuncture needle into the platysma muscle at the point of lowest skin resistance to a depth that the patient reported as painful. After four treatments of 20 minutes each in 10 days, the patient was pain-free and the pain had not returned 6 months later.

One may accidentally encounter a TrP in the platysma and evoke the referred prickle when injecting the underlying sternocleidomastoid muscle. The patient may react with alarm to the unexpected prickling sensation in the face caused by the needle-stimulation of the platysma TrP, until its cause is explained.

14. CORRECTIVE ACTIONS

Any TrPs in other muscles that are likely to refer pain to the same side of the face, such as the masticatory, sternocleidomastoid and upper trapezius muscles, should be inactivated. The "jumpy print" symptom due to orbicularis oculi TrPs has been eliminated by injecting the active TrPs in the orbicularis oculi.

Following treatment of the platysma muscle, and of associated TrPs in muscles of the head and neck, regular passive stretching exercises and resumption of full activity should prevent recurrence of the platysma TrPs.

CASE REPORT (of buccinator TrP seen by Darryl D. Curl, D.D.S.[4])

A 23-year-old Caucasian female was referred to Dr. Curl 8 months into splint therapy for "TMJ syndrome." This headache pain dated back to over 2 years ago while receiving orthodontic care. Seven months into splint therapy the patient reported a new onset of left face pain to her dentist. This pain failed to respond to additional modifications of her full-occlusion mandibular splint and the dentist could find no explanation for it.

The patient explained: "My left cheek hurts, maybe it's my teeth (pointing to the left buccal area) and I can't swallow very well." She described a sudden onset of deep, almost continuous, aching subzygomatic pain in the left face. It had been present for nearly 2 months. The pain was ag-

gravated by chewing and swallowing her food had become difficult. She was unaware of any trauma or other factor associated with the onset of this pain.

A thorough routine medical history and physical examination of the head and neck including the temporomandibular joint apparatus and cranial nerves revealed nothing remarkable. Palpation of muscles for TrPs revealed a slight discomfort to pressure in the posterior portion of the left temporalis muscle and a palpable taut band with focal tenderness in the left buccinator muscle. Snapping palpation produced a local twitch response. Bidigital compression elicited local cheek pain and reproduced deep subzygomatic ache in the left jaw. Infrared thermography of the left and right sides of the head and neck showed a region over the left buccinator muscle $\geq$ 1°C above the surrounding temperature.

Electrotherapy was applied with electrodes placed on each side of the buccinator TrP, one inside and the other outside of the mouth. The current applied was 500 UA of 800 Hz direct current pulses switched between negative and positive pulses every 2 seconds. It was administered for approximately 7 minutes. After three treatments, each two days apart, the patient reported complete resolution of symptoms: no facial pain and normal swallowing. Examination revealed no remaining TrP tenderness or referred pain, and a symmetrical thermal pattern. Two months later, the patient remained symptom-free.

Comment: Activation of this buccinator TrP near the end of her orthodontic care would have contributed to the symptoms that led to the erroneous diagnosis of "TMJ syndrome." Shifting of this TrP between degrees of latent and mild activity could account for the various degrees of comfort experienced with the full occlusal mandibular splint for nearly eight months. However, without a competent examination of this muscle for TrPs throughout this time, this explanation can only be speculation. Full activation of this TrP then led to her referral to Dr. Curl.

The diagnosis of this TrP was made with three of the most reliable criteria: (1) a spot of focal tenderness (2) in a taut band that, when compressed, (3) reproduces the patient's pain complaint. In addition, the diagnosis was substantiated by the most discriminating and skill-demanding criterion, a local twitch response.[24]

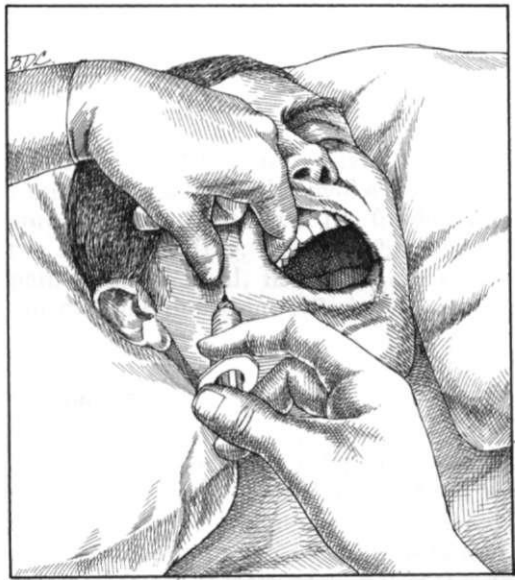


Figure 13.6. Injection of the right zygomaticus major muscle, using pincer grasp to localize the trigger points between the digits.

REFERENCES

1. **Agur AM: Grant's Atlas of Anatomy. Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 462, Fig 7.10).**
2. ***Ibid.* (p. 550, Fig. 8.1).**
3. ***Ibid.* (p. 504, Figs. 7.76; p. 532, Fig. 7.130; p. 584, Fig. 8.54; p. 593, Fig. 8.65)**
4. **Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 462, 478).**
5. ***Ibid.* (p. 466).**
6. **Brooke MH: The pathologic interpretation of muscle histochemistry. Chapter 7. In: *The Striated Muscle.* Edited by Pearson CM, Mostofl FK. Williams & Wilkins, Baltimore, 1973 (pp. 86-122).**
7. **Buchthal F, Rosenfalck P: Action potential parameters in different human muscles. *Acta Psych Et Neurol Scand* 30f3/2j:125-131, 1955.**
8. **Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 440-443).**
9. ***Ibid.* (p. 444).**
10. ***Ibid.* (pp. 446-447).**
11. ***Ibid.* (pp. 456-457).**
12. ***Ibid.* (p. 1167).**
13. ***Ibid.* (p. 1434).**

14. Clemente CD: *Anatomy,* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 576, 603, 604).
15. *Ibid.* (Fig. 575).
16. Curl DD: Discovery of a myofascial trigger point in the buccinator muscle: a case report. / *Craniomandib Pract* 7(4j:339-345, 1989.
17. Hawrylyshyn T, McComas AJ, Heddle SB: Limited plasticity of human muscle. *Muscle Nerve* 29:103-105, 1996.
18. Hollinshead WH: *Anatomy for Surgeons.* Ed. 3, Vol. 1, *The Head and Neck.* Harper & Row, Hagerstown, 1982 (p. 293)
19. *Ibid.* (pp. 95, 297).
20. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W.B. Saunders, Philadelphia, 1991 (pp. 339-341).
21. Lapeer GL: Postsurgical myofascial pain resolved with dry-needling. Treatment protocol and case report. / *Craniomandib Pract* 7(3j:243-244, 1989.
22. McMinn RM, Hutchings RT, Pegington J, *et ah: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, St. Louis, 1993 (pp. 38, 39).
23. Schwarting S, Schroder M, Stennert E, *et al.:* Enzyme histochemical and histographic data on normal human facial muscles. *ORL* 44:51-59, 1982.
24. Simons DG: Clinical and etiological update of myofascial pain from trigger points. / *Musculoske Pain 4(1/2):* 97-125, 1996.
25. Travell J: Identification of myofascial trigger point syndromes: a case of atypical facial neuralgia. *Arch Phys Med Rehabil* 62:100-106, 1981 (Fig. 5).
26. Willis WD, Grossman RG: *Medical Neurobiology.* C.V. Mosby, Saint Louis, 1973 (p. 366).

CHAPTER 14
Cutaneous II: Occipitofrontalis

HIGHLIGHTS: **REFERRED PAIN** from trigger points (TrPs) in the frontalis belly of the occipitofrontalis muscle ("scalp tensor") projects locally over the forehead. Pain from TrPs in the occipitalis belly is projected to the back of the head and through the cranium to the back of the orbit ("behind the eye"). **ANATOMICAL** attachments of these epicranial muscle bellies are, above, to the galea aponeurotica. Anteriorly, the frontalis attaches to the skin of the forehead, and posteriorly, the occipitalis attaches to the occipital bone. **FUNCTION** of these muscles is to wrinkle the forehead; the occipitalis assists the frontalis. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the frontalis belly may arise from direct trauma, or secondarily as satellites from TrPs in the clavicular division of the sternocleidomastoid muscle, or from the overload stress of habitually wrinkling the forehead. **TRIGGER POINT EXAMINATION** is easily accomplished by flat palpation of the muscle against the underlying skull for taut bands, TrP tenderness, and local twitch responses. **ENTRAPMENT** of the supraorbital nerve can be caused by TrPs in the frontalis muscle. **TRIGGER POINT RELEASE** using spray and stretch is usually unsatisfactory for these muscles, but trigger point pressure release is remarkably effective. **TRIGGER POINT INJECTION** in these scalp muscles requires a finer needle than for most muscles. **CORRECTIVE ACTIONS** include training the patient to avoid prolonged, intense frowning or wrinkling of the forehead and the inactivation of key TrPs, particularly in the clavicular division of the sternocleidomastoid muscle.

1. REFERRED PAIN (Fig. 14.1)

Frontalis (Fig. 14.1A)

The trigger points (TrPs) of the frontalis muscle belly evoke pain that spreads upward and over the forehead on the same side (Fig. 14.1A). The referred pain remains local, in the region of the muscle, like that from TrPs in the deltoid muscle.

Occipitalis (Fig. 14.1B)

"Fibrositic nodules" or "myalgia" (used in the sense of myofascial TrPs) of the occipitalis muscle belly are a recognized source of headache.[14,17] Occipitalis tenderness was found in 42% of 42 patients with ipsilateral face and head pain associated with the myofascial pain-dysfunction syndrome.[15]

Active TrPs in the occipitalis muscle belly (Fig. 14.IB) refer pain laterally and anteriorly, diffusely over the back of the head and through the cranium, causing intense pain deep in the orbit. Kellgren[14] reported that the injection of hypertonic saline into normal occipitalis muscle gave rise to "earache." Cyriax[9] similarly injected muscles and fascia of the head and neck to map referred pain patterns. He found that injection into the galea aponeurotica between the frontalis and occipitalis muscle bellies referred pain homolaterally behind the eye, in the eyeball, and in the eyelids. These referred pain patterns were later confirmed clinically by Williams.[17]

2. ANATOMY (Fig. 14.2)

The major cutaneous muscle of the scalp (the epicranial muscle) is the occipitofrontalis, which has two muscle bellies: the frontalis anteriorly and the occipitalis

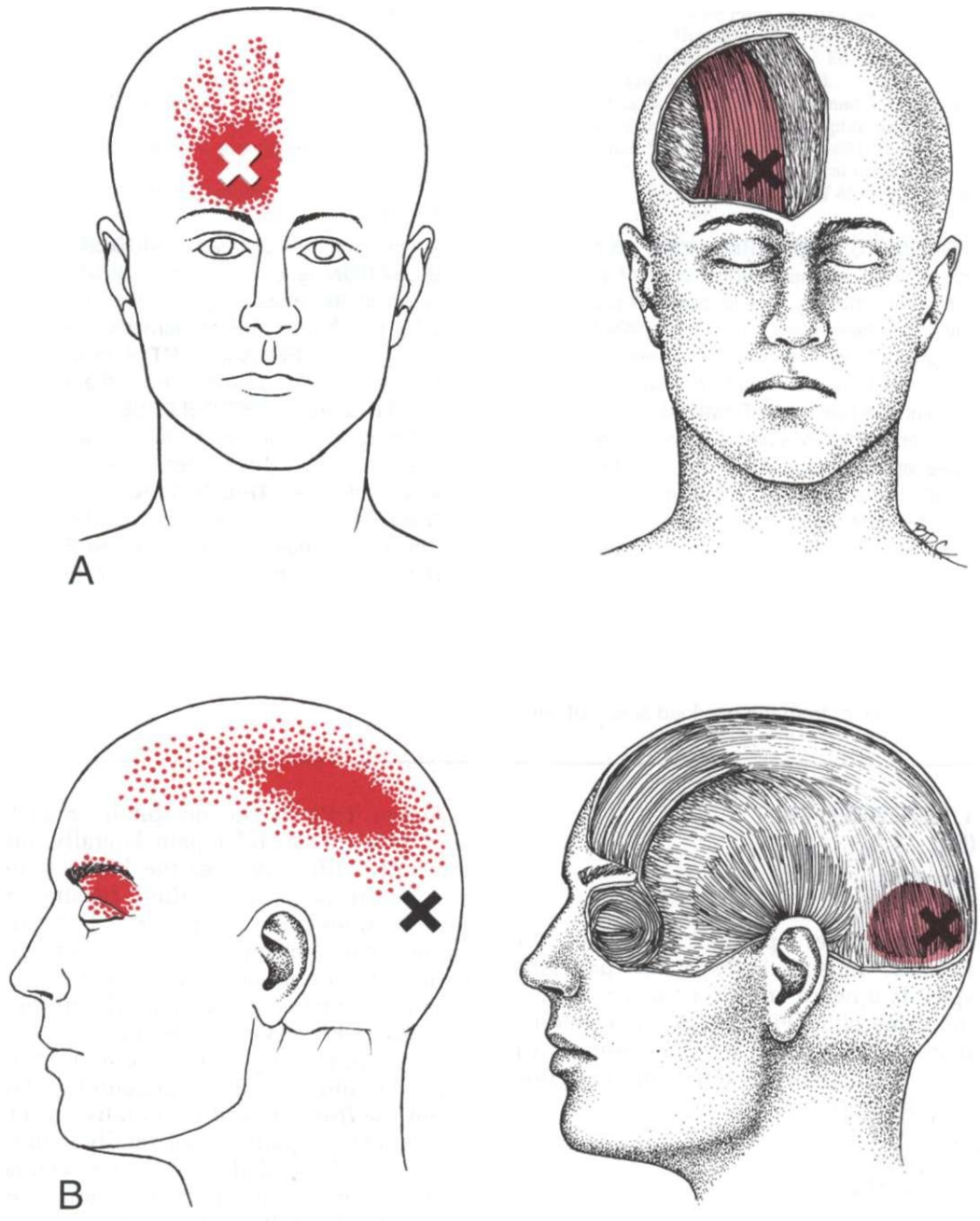


Figure 14.1. Pain patterns (*dark red)* referred from trigger points (Xs) in the occipitofrontalis muscle *(medium red).* **A,** right frontalis muscle belly. **B,** left occipitalis muscle belly.

posteriorly. These bellies attach ***above*** to one large, flat tendinous sheet, the galea aponeurotica, which covers the vertex. The galea is firmly connected to the skin, but slides over the periosteum[16] (Fig. 14.2).

The frontalis muscle belly attaches ***below and in front*** to the skin over the eyebrow, where it interdigitates with the orbicularis oculi muscle. The occipitalis muscle belly attaches ***below and behind*** to the superior nuchal line of the occipital bone.[3,5]

Supplemental References

The frontalis has been illustrated by other authors in side view,[2, 3, 5, 7] from above,[11] from in front,[6, 16] in cross section,[12] and from the side with associated vessels and nerves.[8]

The occipitalis has been illustrated in side view,[3,5,7, 10] from behind,[1] and from the side with associated vessels and nerves.[8]

3. INNERVATION

The epicranial muscle is supplied by the facial nerve (cranial nerve VII).

4. FUNCTION

The frontalis muscle belly raises the eyebrow and wrinkles the forehead;[13] act-

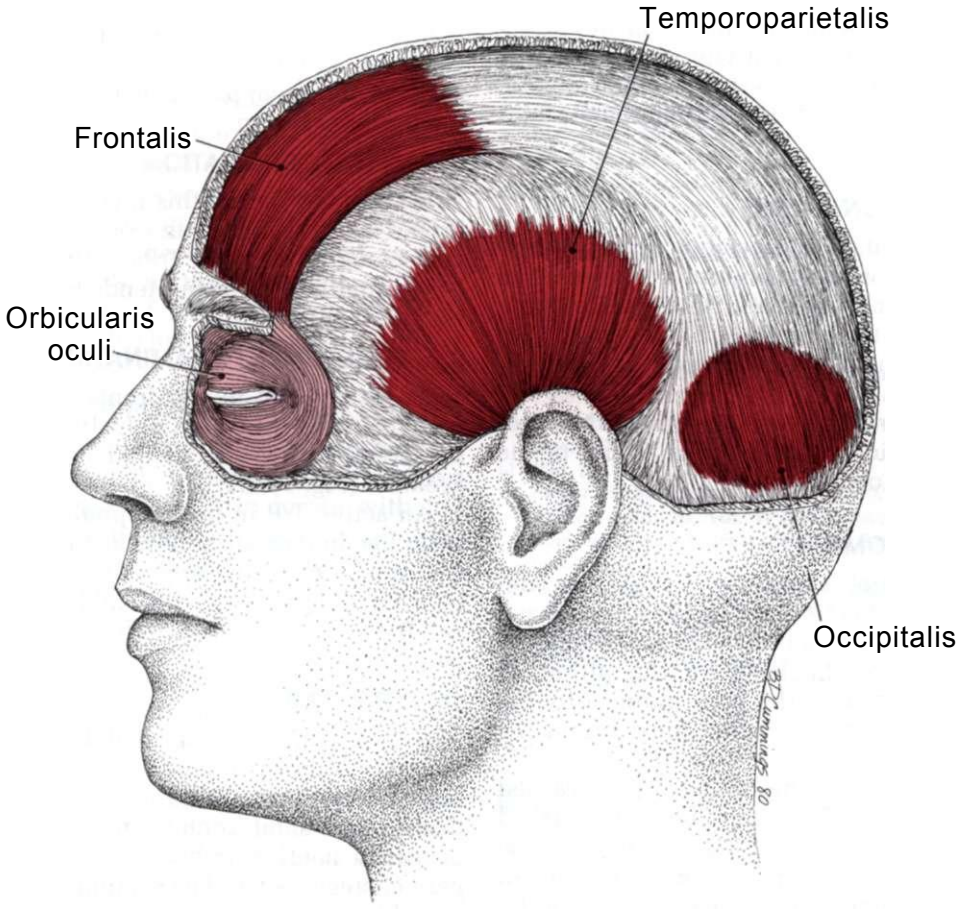


Figure 14.2. Attachments of the left epicranial muscles *(dark red);* the *frontalis* and *occipitalis* bellies of the occipitofrontalis muscle, and also the *temporoparietalis* muscle. Each connects above to the tendinous galea aponeurotica. Below and anteriorly, the frontalis attaches to the skin near the eyebrow; the occipitalis anchors to bone along the superior nuchal line, and the temporoparietalis to the skin above the ear. The cutaneous *orbicularis oculi* muscle is shown in light red.

ing bilaterally, this produces an expression of surprise or attention.[5] The occipitalis and frontalis, acting together, further retract the skin of the forehead, opening the eyes widely in an expression of horror. This action shifts the scalp toward the occiput, which makes the hair stand up, because the hair bulbs in the frontal region slant backward.[3] The occipitalis anchors and retracts the galea posteriorly, so that the frontalis can more effectively pull against it. This action gives rise to the combination name of the occipitofrontalis muscle.

Because the frontalis is associated with the increased muscle tension of anxiety, it is commonly monitored for biofeedback. Contrary to some statements in the literature, all electrical activity in the frontalis ceases at complete rest in normal subjects (in the absence of specific emotional states or expressions).[4]

5. FUNCTIONAL UNIT

The frontalis and occipitalis muscle bellies function as synergists in tandem. The frontalis may contract with, or independently of, the perpendicularly placed corrugator muscle, which shortens the eyebrows in a frown.

The frontalis is an antagonist to the procerus, which pulls the medial end of the eyebrow down.[4]

6. SYMPTOMS

The chief symptom is pain, as described in Section 1. The patient with occipitalis TrPs cannot bear the weight of the back of the head on the pillow at night because of the pain induced by the pressure on the TrP, and must lie on the side.

The deep aching occipital pain caused by occipitalis TrPs must be distinguished from the more superficial scalp tingling and hot prickling pain due to entrapment of the greater occipital nerve by the posterior cervical muscles. The patient with pain referred from myofascial TrPs finds, as a rule, that moist heat provides relief. The patient with head pain due to nerve entrapment cannot tolerate heat, but prefers the cold of an ice pack.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

In the frontalis, TrPs are likely to develop as satellites of TrPs in the clavicular division of the sternocleidomastoid muscle, which refer head pain to the frontal region. Frontalis TrPs also may be activated by work overload, especially in anxious or tense people with great mobility of facial expression, and in people who persistently use the frontalis in an expression of attention with raised eyebrows and wrinkled forehead.

Occipitalis TrPs are likely to occur in patients with decreased visual acuity and/or with glaucoma, due to persistent, strong contraction of forehead and scalp muscles. These TrPs likewise may be activated as satellites of posterior cervical TrPs, which refer pain and tenderness to the occipital region.

8. PATIENT EXAMINATION

No specific sign of this myofascial syndrome has been noted on examination. Be suspicious of frontalis TrPs if the patient has frontal headache and tends to frown a lot.

9. TRIGGER POINT EXAMINATION

An active TrP in the frontalis muscle belly is identified by flat palpation as spot tenderness above the medial end of the eyebrow (Fig. 14.1 A).

An active TrP in the occipitalis muscle belly lies in a small hollow just above the superior nuchal line approximately 4 cm (1.5 inch) lateral to the midline (Fig. 14.1B). Spot tenderness is located by flat palpation.

10. ENTRAPMENT

Active TrPs in the medial half of the frontalis belly apparently can entrap the supraorbital nerve. This entrapment produces a unilateral frontal "headache" with primarily neuritic rather than myofascial pain characteristics. The symptoms are relieved by inactivating (by digital pressure, massage, or injection) the frontalis TrPs that are responsible.

11. DIFFERENTIAL DIAGNOSIS

Pain caused by TrPs in these scalp muscles is likely to be diagnosed as tension-

type headache without recognition of the treatable source.

Active TrPs in the frontalis are often found as satellites in association with long-standing TrPs in the clavicular division of the sternocleidomastoid muscle on the same side. Lasting relief also may depend on inactivating related TrPs in neck muscles.

In patients with occipital aching pain, muscles that refer pain to the occiput, especially the posterior digastric and semispinalis cervicis, should be checked for TrP tenderness and for referral of occipital pain that the patient recognizes. In addition, the possibility of an occipital neuralgia of neurological or myofascial origin should be considered. See Chapter 16, Sections 6, 10, and 11.

12. TRIGGER POINT RELEASE

The frontalis responds poorly to stretch and spray because the muscle is so difficult to stretch. It does respond well to massage and/or to pressure release of its trigger points (TrPs). The same treatment may be used for TrPs in the occipitalis muscle. Deep massage of TrPs in this muscle is effective and also has been recommended by others.[9,17]

13. TRIGGER POINT INJECTION

The frontalis muscle fibers are thin and very superficial, which makes its trigger points (TrPs) difficult to locate with the needle tip. To inject it, a 2.5 cm (1 inch), 24- or 25-gauge needle is directed across the muscle fibers (parallel to the eyebrow), nearly tangent to the skin. Dry needling with an acupuncture needle is also effective if it produces a local twitch response.

The occipitalis muscle belly is thicker than the frontalis and may require a longer, 3.7 cm (1.5 inch) needle. Injection of these posterior TrPs is technically more satisfactory since they seem to lie in a small hollow which holds sufficient muscle mass to receive the needle. However, considerable probing of the area may be necessary to locate them.

14. CORRECTIVE ACTION

When there is occipitofrontalis involvement, the patient should avoid persistent frowning and vigorous wrinkling of the forehead. In addition, for prevention of recurrences of TrP activity, the patient should learn to use digital pressure release of the TrPs.

Any related key TrPs in the clavicular division of the sternocleidomastoid and posterior neck muscles should be inactivated.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 240, Fig. 4.56).
2. *Ibid.* (p. 462, Fig. 7.10).
3. Bardeen CR: The musculature. Section 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (pp. 364, 371, Fig. 372).
4. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (p. 463).
5. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 438-441).
6. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 603).
7. *Ibid.* (Figs. 604, 608).
8. *Ibid.* (Fig. 623).
9. Cyriax J: Rheumatic headache. *Br Med J* 2:1367-1368, 1938.
10. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (p. 170, Fig. 18).
11. *Ibid.* (p. 184, Fig. 20).
12. Ferner H, Staubesand J: *Sobotta Atlas of Human Anatomy.* Ed. 10, Vol. 1, *Head, Neck, Upper Extremities.* Urban & Schwarzenberg, Baltimore, 1983 (p. 67).
13. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W.B. Saunders, Philadelphia, 1991 (pp. 340, 341).
14. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3:175-190, 1938 (p. 181).
15. Sharav Y, Tzukert A, Refaeli B: Muscle pain index in relation to pain, dysfunction, and dizziness associated with the myofascial pain-dysfunction syndrome. *Oral Surg* 46:742- 747, 1978.
16. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 260).
17. Williams HL: The syndrome of physical or intrinsic allergy of the head: myalgia of the head (sinus headache). *Proc Staff Meet Mayo Clin* 20:177-183, 1945 (p. 181).

CHAPTER 15
Splenius Capitis and Splenius Cervicis Muscles

HIGHLIGHTS: **REFERRED PAIN** from trigger points (TrPs) in the splenius capitis appears in the vertex of the head. Pain from the splenius cervicis is projected upward to the occiput, diffusely through the cranium, and intensely to the back of the orbit—an *"ache inside the skull."* Sometimes, splenius cervicis pain is referred downward to the shoulder girdle and to the angle of the neck. **ANATOMICAL** attachments of the splenii are below to the spinous processes of the lower cervical and upper thoracic vertebrae. Above, the splenius cervicis attaches to the transverse processes of the upper cervical vertebrae, and the splenius capitis attaches to the mastoid process of the skull. The splenius cervicis and capitis lie superficial to the semispinalis capitis and other paraspinal muscles, deep to the trapezius, and posterior and medial to the levator scapulae. **FUNCTIONS** of the splenii include working together to extend the head and neck and individually to rotate the head and neck, turning the face toward the same side. **SYMPTOMS** of headache and/or neck pain with homolateral blurring of vision can be due to active TrPs in the splenius cervicis and splenius capitis muscles. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in these muscles are often due to sudden overload, such as whiplash, or caused by holding the head and neck in a forward, crooked position for a prolonged period. These neck muscles are especially vulnerable when they are tired and the overlying skin is exposed to a cold draft. **PATIENT EXAMINATION** reveals moderate restriction of passive head and neck flexion and rotation to the opposite side, and painful restriction of active head and neck rotation to the same side. **TRIGGER POINT EXAMINATION** requires that the relation of the splenii to adjacent muscles be kept clearly in mind. Most of their course lies between and deep to other muscles. The **DIFFERENTIAL DIAGNOSIS** distinguishes TrP sources from other causes of pain in patients complaining of headache and those who have suffered whiplash. With TrPs in the splenii, one frequently finds multiple and varied cervical articular dysfunctions. The myofascial problem of stiff neck should not be confused with the neurological disease, spasmodic torticollis. Trigger points in at least 7 other head and neck muscles have similar or overlapping pain patterns. **TRIGGER POINT RELEASE** of the splenius capitis and splenius cervicis using spray and stretch is performed with an up-stroke pattern of the vapocoolant. These TrPs also respond to pressure release and deep massage. **TRIGGER POINT INJECTION** of the splenius capitis should be done only with extreme caution, with the needle aimed caudad, *below* the junction of the and C_2 vertebrae, to avoid the vertebral artery. When accurately located, the more caudal splenius cervicis trigger points usually respond well to injection therapy. **CORRECTIVE ACTIONS** include eliminating perpetuating sources of muscle strain, and performing daily passive self-stretch of the splenii.

1. REFERRED PAIN (Fig. 15.1)

A trigger point (TrP) in the splenius capitis muscle usually refers pain to the vertex of the head on the same side (Fig. 15.1A).[36,47,49,50] The splenius capitis is one of many neck and masticatory muscles that commonly cause headache.[25,28,30,43]

A TrP in the upper end of the splenius cervicis (Fig. 15.1B, pattern on the *left* figure) usually refers a diffuse pain through the inside of the head that focuses strongly

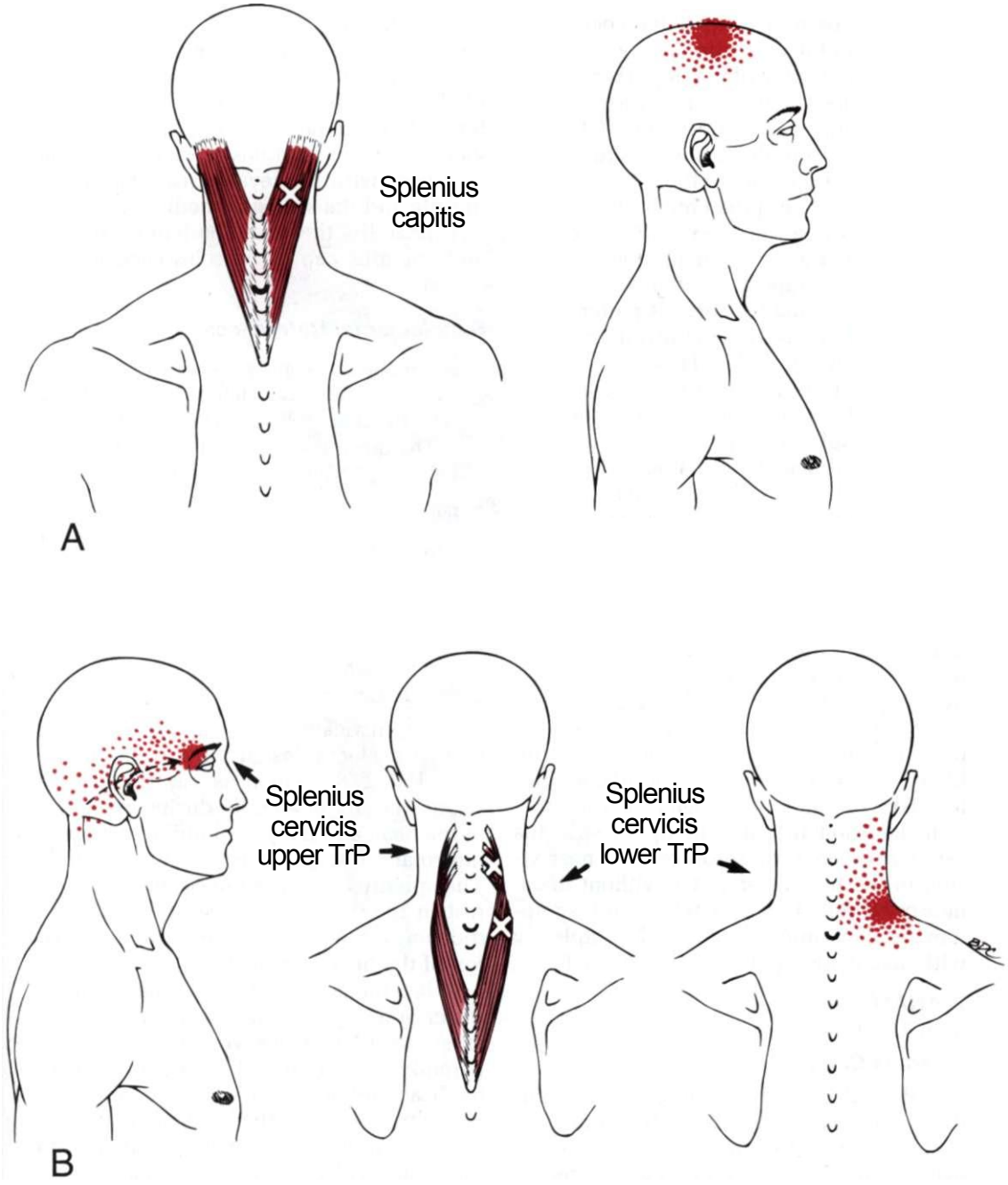


Figure 15.1. Trigger points (Xs) and referred pain patterns *(dark red)* for the right splenius capitis and splenius cervicis muscles *(medium red).* **A,** an unusually craniad location of a splenius *capitis* trigger point, which, near the level of C_2, is just caudad to the exposed vertebral artery. **B,** pressure applied to the tender region of the craniad musculotendinous junctions of the splenius *cervicis* muscle refers pain to the orbit (pain figure on the *left).* The black dash *line* and *arrow* indicate that the pain seems to shoot through the inside of the head to the back of the eye. Splenius cervicis central trigger points (located mid-muscle) refer pain to the angle of the neck (figure on the *right).* The lower X in the *middle* figure locates this splenius cervicis trigger point region,

behind the eye on the same side, and sometimes refers pain to the ipsilateral occiput.[45] A TrP in the lower portion of the splenius cervicis at the angle of the neck (Fig. 15.IB, *middle* figure) refers pain upward and to the base of the neck (pattern in *right* figure). This pattern generally lies within the upper part of the pain pattern of the levator scapulae but with some spread medially.

One of three case reports[25] described pain in the side of the neck that radiated into the head (trapezius pattern) and above the right eye (splenius cervicis pattern) in a patient with active TrPs in the trapezius and splenius capitis muscles. This pain had previously been diagnosed as occipital neuralgia. The other two patients had active TrPs in both the splenius capitis and the splenius cervicis muscles. One patient described continuous pressure-like pain in the left occipital region. The other described continuous pressure-like pain that radiated to the forehead and described *numbness* in the occipital region. These cases illustrate the degree of variability of pain patterns seen in individual patients. The report of numbness instead of pain in the occipital region by one patient is a reminder that TrPs can refer numbness and anesthesia instead of pain and hyperesthesia. Patients are more likely to describe pain than numbness, unless asked about any *change* in sensation.

In addition to pain, an upper splenius cervicis TrP may cause blurring of near vision in the homolateral eye, without dizziness or conjunctivitis. Sometimes this symptom resolves immediately and completely with inactivation of the responsible TrP.

2. ANATOMY (Fig. 15.2)

Splenius Capitis

Below, this muscle attaches in the midline to the fascia over the spinous processes of the lower half of the cervical spine and over the first three or four thoracic vertebrae (Fig. 15.2). ***Above and laterally,*** its fibers attach to the mastoid process and to the adjacent occipital bone underneath the attachment of the sternocleidomastoid muscle.[3,11]

Splenius Cervicis

This muscle lies to the lateral side and caudal to the splenius capitis. The splenius cervicis, like the capitis, attaches ***below*** in the midline to the spinous processes; the cervicis fastens from the T_3 to T_6 vertebrae. The muscle connects ***above*** to the posterior tubercles on the transverse processes of the upper two or three cervical vertebrae. On these posterior tubercles, the cervicis forms the most posterior of a triple attachment with the levator scapulae in the middle and the scalenus medius in front.

Bilaterally, the paired splenius cervicis and splenius capitis muscles each form a "V" shape.[12]

Supplemental References

The splenius capitis has been illustrated by other authors as seen from behind,[2,12,18] from the side,[3,14,19] and in cross-section.[22]

The splenius cervicis is presented from behind[12,19,31] and in cross-section.[21]

3. INNERVATION

Both muscles are innervated by lateral branches of the dorsal primary divisions of spinal nerves C_2-C_4, frequently also C_1, sometimes C_5, and rarely C_6.[20]

4. FUNCTION

Splenius Capitis

A sophisticated study using implanted fine-wire electrodes in 15 subjects determined that the splenius capitis showed strong activity bilaterally during extension of the head and neck, and unilaterally during rotation of the face to the same side.[8] The splenius capitis showed no activity at rest in the upright balanced position, and did *not* become active during lateral flexion of the head and neck.[8,44]

When the face is rotated to one side with the chin tilted upward, the splenius capitis muscles on *both* sides work vigorously. Apparently, the muscle on the same side rotates the head and neck, while the opposite muscle helps to extends the head and neck.[44]

Early stimulation experiments on an unspecific splenius muscle described lateral inclination and extension with rotation of the head to the stimulated side.[17] Subsequent authors attributed extension and lateral flexion of the head and neck to activity of one splenius capitis muscle,[11] and attributed extension of the head and neck to its bilateral contraction.[11,31] A significant lateral flexion function is highly questionable.

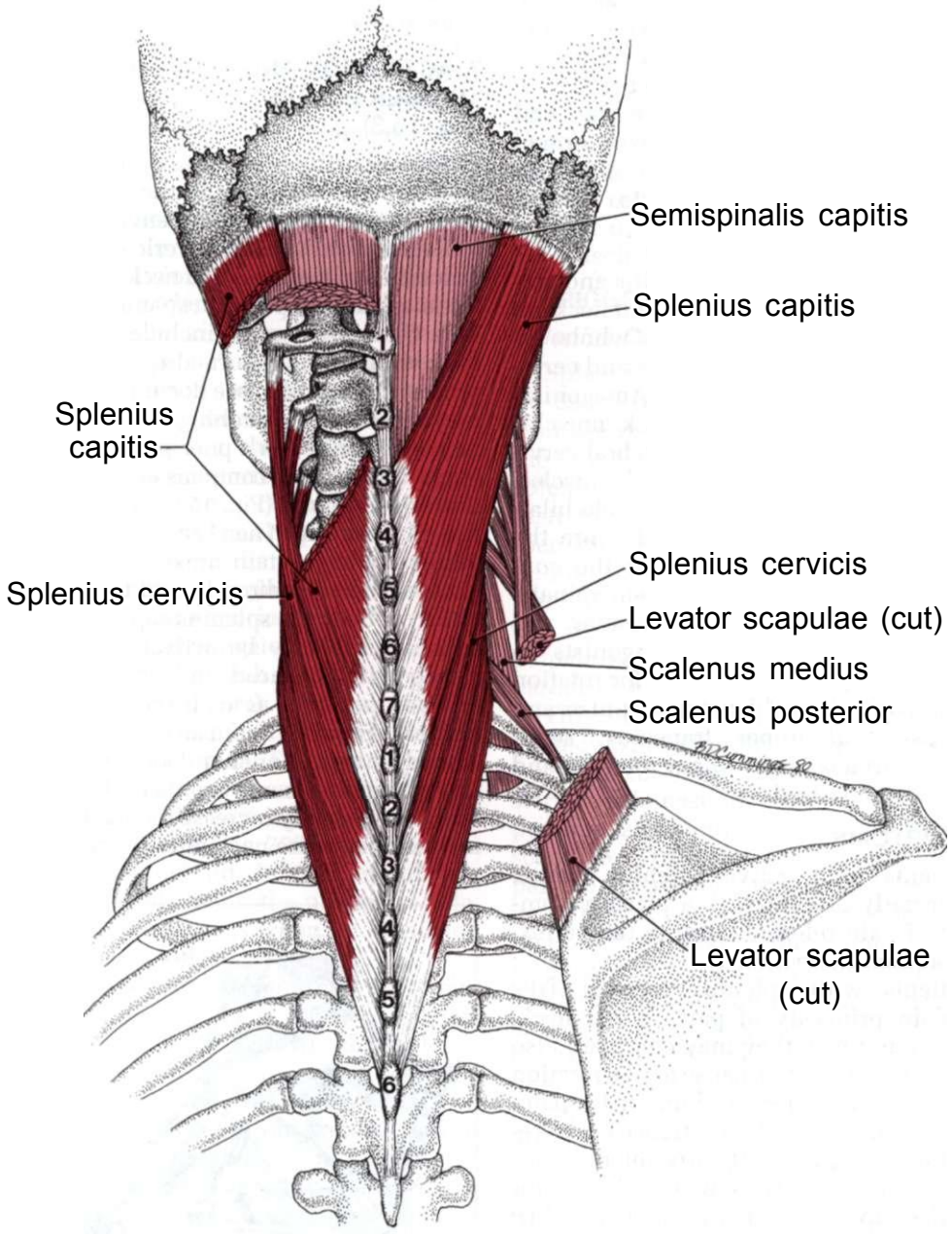


Figure 15.2. Attachments of the right splenius capitis muscle (*upper dark red),* and of the right splenius cervicis muscle *(lower dark red).* Adjacent muscles are shown in *light red.* The levator scapulae *(right side,* cut) crosses over the upper part of the splenius cervicis, with which it has common attachments to the transverse processes of the upper cervical vertebrae. The trapezius muscle (not shown) covers much of both splenii.

Splenius Cervicis

No electromyographic data specific to the splenius cervicis muscle were found. As one would expect, contraction of one muscle causes rotation and sidebending of the cervical spine to the same side and both muscles bilaterally cause extension of the neck.[11] Others [5,31] attribute only rotation and extension to this muscle.

5. FUNCTIONAL UNIT

Synergists of the splenii capitis and cervicis for extension of the head and neck are the posterior cervical group as a whole, especially the semispinalis capitis and cervicis muscles acting bilaterally. Antagonists for extension are anterior neck muscles which include the anterior vertebral cervical muscles, the hyoid-attached muscles, and the sternocleidomastoid muscle bilaterally. The synergists for rotation are the *ipsilateral* levator scapulae and the *contralateral* upper trapezius, semispinalis cervicis, deep spinal rotator muscles, and sternocleidomastoid. The antagonists to the splenii capitis and cervicis for rotation are the *contralateral* levator scapulae and the *ipsilateral* upper trapezius, semispinalis cervicis, deep spinal rotators, and sternocleidomastoid.

6. SYMPTOMS

Patients with active splenius capitis TrPs usually present with a primary complaint of pain referred close to the vertex, as described in Section 1.

Patients with splenius cervicis TrPs complain primarily of pain in the neck, cranium and eye; they may complain also of a "stiff neck,"[38,45] because active rotation of the head and neck is limited by pain. However, the patient experiences less restriction of rotation with only splenius cervicis involvement than with only levator scapulae involvement. Simultaneous TrP activity in both the levator and splenius muscles may almost completely block *active* head rotation to that side. Involvement of the splenius cervicis may become apparent because of residual pain and stiffness following elimination of TrP activity in the levator scapulae.

Pain in the orbit and blurring of vision are disturbing symptoms that occasionally are referred homolaterally to the eye from TrPs in the upper part of the splenius cervicis muscle.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS (Fig. 15.3)

Several kinds of stress can activate and/or perpetuate TrPs in the splenii: postural, impact, activity, and environmental.

Postural stresses that overload extension or rotation of the head and neck are likely to initiate and perpetuate splenius cervicis TrPs. Clinical examples include working at a desk with the head turned to one side and projected forward to see documents or a display screen, bird-watching through binoculars while seated in a poor position that extends the neck to compensate for a strong thoracic kyphosis (Fig. 15.3), and assuming a similar posture of head and neck extension while playing certain musical instruments such as the accordion. In addition, TrPs in either, or both, the splenius capitis and splenius cervicis may be activated by falling asleep with the head and neck bent in a crooked position, as with the head on the armrest of a sofa without an adequate pillow. A cold air conditioner or cool draft blowing on the exposed neck, together with muscular fatigue, greatly increase the likelihood of activation of these neck-muscle TrPs.

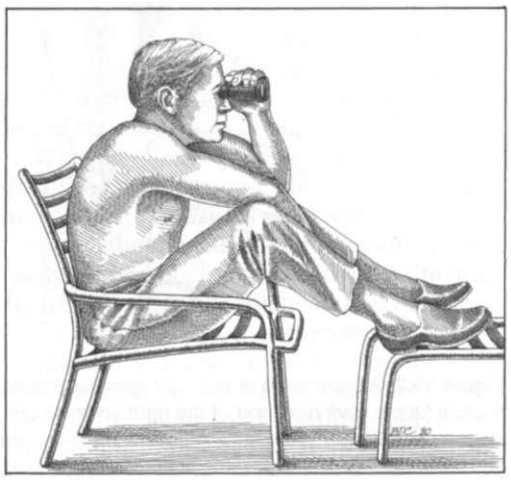


Figure 15.3. Poor "bird-watching" posture that places the splenius cervicis muscles in sustained contraction. This posture should not be held for a prolonged period.

One patient developed a splenius capitis syndrome after acquiring contact lenses.[46] He held his head in a cocked position at his desk to avoid reflections on the lenses from overhead lights. Adjustments in neck posture to see through the middle section of trifocal lenses may have the same result.

A common cause of splenius capitis (and other axial muscle) TrPs is the **impact stress** of motor vehicle accidents. These muscles are susceptible to the trauma of a rear-end collision in an automobile followed by a sudden stop,[41] especially if the head and neck are somewhat rotated at the time of impact. A significant number of patients suffering acceleration-deceleration injury of the neck[37]—which is sometimes identified by the outmoded term "whiplash"—are notorious for developing refractory head and neck pain symptoms. These patients are rarely properly examined and treated for the part of their pain that is of muscular origin. Baker,[4] who was well-trained and experienced in the diagnosis of TrPs, investigated 34 muscles for myofascial TrPs in each of 100 occupants (drivers or passengers) who sustained a single motor vehicle impact and identified the direction of impact. The splenius capitis was the second most frequently involved muscle: in 94% of subjects in impacts from the front, in 77% of subjects in impacts from behind, in 75% of subjects when hit broadside on the passenger side, and in 69% of subjects when hit broadside on the driver's side.

The **activity stress** of pulling on a rope while rotating or projecting the head forward may activate TrPs in the splenii. These muscles, and the levator scapulae muscle as well, are vulnerable when one pulls excessive weight on exercise equipment pulleys or when one lifts excessive weight; the stress is accentuated when the subject rotates the head and neck and/or projects the head forward.

Environmental stress that can activate both splenius cervicis and levator scapulae TrPs may occur with marked skin cooling, especially when the muscles are tired. An example is exposure to a breeze when a person relaxes in a wet bathing suit in the shade (even on a warm day) after the fatigue of swimming.

8. PATIENT EXAMINATION

The patient shows painful restriction of *active* head rotation to the same side, while passive rotation is restricted to the opposite side. Flexion of the chin onto the chest may lack a distance of one or two finger widths. One is likely to uncover the splenius cervicis TrP involvement when the pain and restricted rotation improve but the pain fails to clear up after TrPs in the levator scapulae muscle have been inactivated.

9. TRIGGER POINT EXAMINATION

Splenius Capitis

Splenius capitis TrPs can be identified by flat palpation, and usually are found near the region where the upper border of the upper trapezius muscle crosses the splenius capitis (*see* Fig. 20.7). As also illustrated by others, this relationship can be seen clearly.[13, 32] These TrPs are located mid-muscle, as also illustrated by Rachlin.[40] The examiner must know the direction of fibers (*see* Figs. 16.2 and 20.7) and palpate across the fibers for a taut band harboring a TrP.

This muscle can be palpable subcutaneously within the small muscular triangle *[see* Fig. 20.7) bounded anteriorly by the sternocleidomastoid, posteriorly by the upper trapezius, and caudad by the levator scapulae.[14] To locate the splenius capitis, palpate the mastoid process and the prominent sternocleidomastoid muscle (easily identified by asking the patient to look *away* from the side being examined, and sidebend the head *toward* the side being examined). Then the operator places one finger posterior and medial to the sternocleidomastoid, below the occiput, and palpates contraction of the diagonal **splenius capitis** fibers by asking the patient to turn the face *toward* the side being examined and extend the head against *light* resistance supplied by the operator. Once the splenius capitis has been identified in this muscular triangle, it can be palpated for taut bands and TrPs. In some patients, the splenius may be taut enough to be clearly palpable without active assistance from the patient.

If in doubt, one can also identify the upper border of the trapezius muscle (see Figs. 6.6 and 20.7) by having the patient

well supported in a reclining position with full muscular relaxation and then feel for muscle contraction as the patient suddenly makes a brief forceful arm abduction movement against light resistance. One palpates the splenius capitis for taut bands and tenderness along and/or deep to the border of the upper trapezius muscle at approximately the level of the C_2 spinous process.

Levator scapulae contraction can be distinguished if needed when the patient elevates that scapula against resistance, since the splenius capitis remains slack.

Williams[49] ascribed tenderness at the insertion of the splenius capitis muscle on the mastoid process, and in the portion of the muscle just distal to this attachment, to a splenius capitis TrP. Tenderness in this location is more likely to be caused by enthesopathy secondary to tension caused by a TrP that is mid-muscle.

Splenius Cervicis (Fig. 15.4)

The splenius cervicis is not readily palpable. From behind, all of it is covered by the upper or middle trapezius muscle. Only a small patch of it is not covered by the splenius capitis and/or the rhomboid minor muscle posteriorly, or by the levator scapulae laterally.[12]

The best chance of eliciting the tenderness of mid-muscle splenius cervicis TrPs is from the side, through or around the levator scapulae. If the skin and subcutaneous tissues are sufficiently mobile, the operator slides the palpating finger anterior to the free border of the upper trapezius at approximately the level of the C_7 spinous process, to and beyond the levator scapulae muscle. If the levator scapulae is not tender, but additional pressure directed medially toward the spine is painful, this is likely a splenius cervicis TrP that can be tested for reproduction of the patient's pain complaint. In patients with mobile connective tissue the taut bands may be palpable running caudad diagonally from lateral to medial. The levator scapulae can be felt to contract with shoulder elevation, but the splenius cervicis contracts with neck extension.

Posteriorly, digital pressure to splenius cervicis TrPs is applied mid-muscle approximately 2 cm lateral to the spine at approximately the level of the C_7 spinous process (Figs. 15.2 and 15.4, lower position of finger), which is just above the angle of the neck.[13] Tenderness may also be from trapezius TrPs, which are associated with taut bands angled laterally, not medially, in the caudad direction. If the tenderness is deep to the trapezius, it may be from either splenius cervicis or levator scapulae TrPs. If straight flexion of just the neck (increasing tension chiefly on the splenius cervicis fibers) increases the sensitivity of the tenderness, it is more likely from splenius cervicis TrPs. Both the splenius capitis and splenius cervicis muscles are elongated by neck flexion, but only the splenius capitis is further elongated by flexion of the head on the cervical spine.

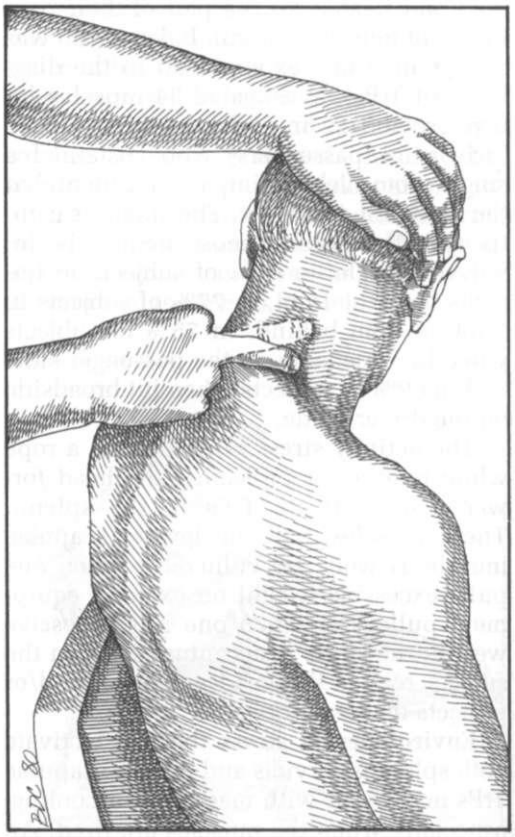


Figure 15.4. Examination of the splenius cervicis muscle. The lower finger *(solid lines)* palpates the mid-muscle trigger point. The *dash-line* finger *(above)* palpates tenderness near the region of a musculotendinous junction.

In some patients, pressure applied from the lateral aspect of the neck directly toward the spine cephalad to the C_7 level elicits tenderness in the region of the cephalad attachments of the splenius cervicis. This tenderness is likely to be enthesopathy from taut band tension and is cephalad to the TrP tenderness.

10. ENTRAPMENT

The authors are not aware of any nerve entrapment due to involvement of these muscles.

11. DIFFERENTIAL DIAGNOSIS

With TrPs in the splenii, one frequently finds multiple and varied cervical articular dysfunctions. The most common seems to be a C_2 dysfunction, particularly when the splenius capitis is involved. Another common articular dysfunction relating to that TrP is an occipitoatlantal dysfunction. Dysfunctions at C_4 and C_5 are likely with splenius cervicis TrPs.

Patients with head and neck pain following a motor vehicle accident frequently receive one of a number of descriptive diagnoses including whiplash syndrome or injury, hyperextension strain or injury, hyperextension-flexion injury, and acceleration-deceleration injury; all terms that do not address the tissue-source of the patient's symptoms. The frequently predominant TrP component of the pain is overlooked and inadequately treated. Clinicians familiar with TrPs are prone to concentrate exclusively on posterior muscle involvement. Baker[4] found that the upper body muscles that most commonly develop active TrPs following a motor vehicle accident are the splenius capitis and semispinalis capitis. However, recovery of patients with TrPs in these posterior muscles often requires inactivation of TrPs in anterior neck and chest muscles, such as the sternocleidomastoid and the pectoral muscles, especially the pectoralis minor.

Pain patterns referred by other TrPs that may appear similar to or overlap the patterns of the splenius capitis and splenius cervicis include those from the: semispinalis cervicis, suboccipital, levator scapulae, sternocleidomastoid, upper trapezius, temporalis, and deep masseter muscles.[48]

Acceleration-deceleration injuries[37] (whip-lash) are increasingly a common problem in medical practice which has generated widespread concern and frustration as to its pathophysiology and treatment.[10,35,39] It was recently extensively reviewed by Barnsley, *et al*.[6] Whiplash is recognized in France as *coup du lapin* (rabbit's blow), in Germany and Austria as *Schleudertrauma* (slinging trauma), in Italy as *colpo di frusta* (whiplash), in Norway as *nakkesleng* (rapid neck hyperextension), and in Sweden as *pisksnart skada* (whiplash injury).[23]

The dissatisfaction with diagnosis and treatment of pain following acceleration-deceleration injury stems largely from the fact that there are multiple sources of the pain, which are of variable importance in individual patients.[34] In this Mailis, *et al.* study, only 9% of 32 patients with thoracic outlet syndrome following a motor vehicle accident were referred with consideration of that diagnosis. Sixteen of the 32 patients were treated surgically and all received conservative treatment including "TrP injection." Only 20% of the non-surgical group and 47% of the group receiving surgery experienced good pain relief. This study makes no mention of a specific muscular examination, particularly examination of the pectoralis minor for TrPs, which are a critical source of pain in many whiplash patients.[27] This observation is supported by the 67% prevalence of "thoracic outlet syndrome" on the side of the driver's shoulder strap.[34] An adequate evaluation for pain following whiplash injury includes competent and thorough examination for active TrPs, which are much more responsive to treatment when identified and treated promptly.[27]

Demonstration of kinking and fanning in cervical spine radiographs that include flexion and extension views in clinically proven "whiplash" patients compared to normal controls showed a diagnostic sensitivity of 81% and an accuracy of 80%.[26] One cause for this finding could be muscular distortion of cervical mechanics associated with increased tension of muscles caused by taut bands of TrPs.

A carefully controlled study confirmed that painful zygapophysial joints were

present in 54% of 50 consecutive patients with chronic neck pain following whiplash.[7] These patients were not examined specifically for TrPs, and myofascial TrPs frequently coexist with painful articular dysfunctions. The TrPs in the neck muscles and cervical zygapophysial joints at corresponding levels can have remarkably similar pain patterns.[9]

In a study of 120 young patients referred for whiplash syndrome,[42] 76% had abnormal upper extremity nerve conduction studies that persisted for 6 months in 70% of them. Abnormal brain auditory-evoked potentials appeared in 64% of the subjects, which also persisted. This is evidence that there are neurological abnormalities associated with whiplash which could increase motor dysfunction, aggravate TrPs, and act as perpetuating factors of TrPs.

Active TrPs rarely appear in the splenii muscles alone; usually, either or both the levator scapulae and other posterior cervical muscles also are involved.

Myofascial TrP involvement of the splenii, levator scapulae, upper trapezius and sternocleidomastoid muscles must be distinguished from spasmodic torticollis (wry neck),[1,24] which is a neurological condition characterized by paroxysmal or clonic contractions of the involved muscles, especially the sternocleidomastoid. The latter also may exhibit tonic spasm. In spasmodic torticollis, hypertrophy of the muscles develops, associated with fibrotic change and permanent contracture. In contrast, the apparent shortening and tautness of a muscle due to myofascial TrPs in that muscle does not cause hypertrophy. Also, in the case of TrPs, there is a steady resistance to stretch without paroxysmal or clonic contractions. Spasmodic torticollis, like the dystonias, appears to have a central nervous system origin,[24] and the irritable focus in the brain may be treated surgically.[1, 15, 16] The differential diagnosis of "stiff neck" of myofascial origin[45] is discussed further in Chapter 7, Section 11 and in Chapter 19.

12. TRIGGER POINT RELEASE (Fig. 15.5)

The splenii generally are released together with their synergists as part of one treatment. Tightness of one muscle may prevent full stretch of one or the other of the synergistic parallel units.

The patient is seated and the axis of the shoulder girdle is checked to be sure that it is horizontal. If not, the pelvis is leveled and the spine straightened by adding an ischial lift under the ischial tuberosity on the side of the smaller hemipelvis *[see* Fig. 48.10).

The patient sits in a comfortable armchair with good elbow support, so that he or she can relax the shoulder-girdle muscles while leaning against the chair back. The patient's head is rotated 20% or 30% away from the involved splenii, while the head is gently flexed toward the opposite side (Fig. 15.5A). At the same time, the vapocoolant spray is swept upward over the muscles and occiput to the vertex. The spray should also cover the angle of the shoulder where pain is referred from the splenius cervicis, as well as the lateral aspect of the head as far forward as the eye (protect the eye from spray). Another form of intermittent cold, such as ice stroking, may be used instead of spray.

Immediately, the operator grasps the head between both hands and applies upward traction while gently further flexing and rotating the head toward the opposite side to take up any slack in the muscles (Fig. 15.5B). The patient should simultaneously look down and slowly exhale. The effect is potentiated by reciprocal inhibition if the patient also tries to actively tilt the head in the direction of the stretch. The patient thus learns exactly how to stretch these muscles at home.

A hot pack over the treated muscles promptly follows the application of intermittent cold and stretch.

13. TRIGGER POINT INJECTION (Fig. 15.6)

Splenius Capitis

The splenius capitis muscle can be injected safely in the region of its mid-muscle trigger points (TrPs) with appropriate precautions. The semispinalis capitis muscle lies deep to the splenius capitis muscle (Fig. 15.2), and provides a buffer between it and the unprotected portion of the vertebral artery (*see* Fig. 16.8). Also, the exposed artery lies cephalad to the C_1 spinous process (*see* Fig. 16.5). Therefore, the sple-

nius capitis can be safely injected if needle insertion is angled caudad, aims below the junction of the C_1 and C_2 vertebrae (Fig 15.6), and remains close to a frontal plane to control the depth of penetration.

The patient lies on the side opposite the involved muscle, with the head supported on a pillow between the cheek and shoulder, without bending or rotating the head and neck. The TrP is located by palpation, as described in Section 9. When injecting a TrP in the splenius capitis muscle, the needle is inserted below and lateral to the posterior occipital triangle through which the vertebral artery passes (see Fig. 16.5).

Splenius Cervicis

The patient lies on the side opposite the involved muscle, with the head supported on a pillow between the cheek and shoulder, without bending or rotating the head and neck. Splenius cervicis TrPs are located mid-muscle[40] and are found by palpation at approximately the level of the C_7 spinous process, as described in Section 9. At this level, the splenius cervicis muscle lies medial and deep to the levator scapulae and continues caudad deep to the rhomboid and serratus posterior superior muscles. Its mid-muscle TrP is located between the lower end of the splenius capitis and the levator scapulae muscles, and is best injected with the needle directed from lateral to medial (Fig. 15.6), while the needle point is kept superficial to the rib" posterior to the plane of the transverse processes. In this approach, the needle enters the splenius cervicis

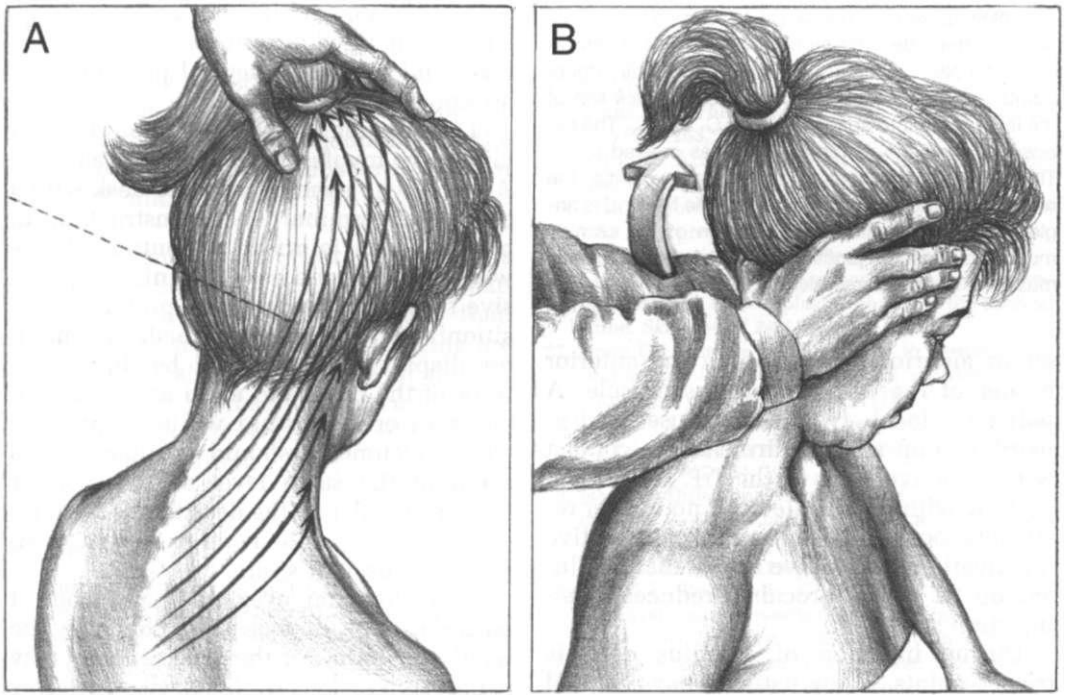


Figure 15.5. Stretch position and vapocoolant spray or icing pattern for trigger points in the right splenius capitis and splenius cervicis muscles. **A,** the head and cervical spine are rotated toward the opposite side and slightly flexed while a stream of vapocoolant *(thin arrows)* is applied in an up-sweep pattern to the vertex. In addition, for the splenius cervicis, the spray should cover the angle of the shoulder and also across the lateral aspect of the head (not shown) as far forward as the eye (protect the eye from spray). **B,** immediately, the clinician takes up slack that has developed in the splenii by applying upward traction while further flexing and rotating the head and neck *(thick arrow)* toward the opposite side. Release of the splenii is augmented if the patient slowly exhales, looks down, and then gently attempts to actively assist the direction of movement into the lengthened position.

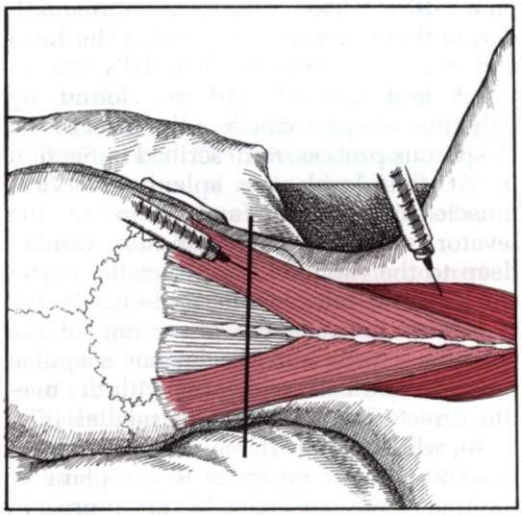


Figure 15.6. Injection of the trigger point area in the midportion of the splenius capitis muscle *(light red)* and in the mid-portion (lower trigger point region) of the splenius cervicis muscle *(dark red)* with the patient lying on the side. The semispinalis capitis is shown without color. Injections of splenius capitis trigger points are **avoided** craniad to the *thick black line* at the level of the interspace between C_1 and C_2. The exposed part of the vertebral artery lies craniad to C_1. Therefore, for injections of the splenius capitis, the needle is inserted below the level of the line and is angled caudad. Injection to the region of the craniad musculocutaneous junction of the splenius capitis muscle is not recommended.

either anterior to or through the anterior border of the upper trapezius muscle. A palpated local twitch response and/or painful jump sign confirm needle contact with an active locus in the TrP. Additional probing with the needle until no further responses occur helps to confirm effective inactivation of all active loci in that TrP. Injection of 0.5% procaine reduces post-injection soreness.

During injection of splenius cervicis trigger points, a few patients have fainted as a result of the strong autonomic stimulus associated with release of this trigger point. This fainting usually followed multiple large twitch responses with visible deviation of the head in the direction of the twitch. The fact that the syncope consistently followed rapid jerking motion of the head suggests that this marked response relates to altered vestibular input. When the head moves, it is likely that fibers of the splenius capitis and splenius cervicis contract together. (Personal communication: R. Shapiro, 1996).

If the patient is being treated for a "stiff neck," any TrPs in the levator scapulae should be injected at the same time as those in the splenius cervicis.

14. CORRECTIVE ACTIONS

Postural Stress

As patients become aware that certain activities initiate and perpetuate their splenius TrPs, they learn to avoid the postural strain by improving posture, keeping the head and neck erect and the thoracic spine extended, and minimizing excessive twisting and turning of the head and neck.

Body asymmetry due to a lower limb-length inequality or small hemipelvis should be corrected. An excessively long walking cane should be avoided. Neck strain is avoided also by sleeping with the head and neck in a neutral position with appropriate pillow support.

The ergonomic approach should be included in myofascial pain management.[33] Anyone who spends time at a desk with a computer terminal needs instruction in how to keep joints in a neutral posture when possible and how to minimize excessive twisting movements or prolonged positioning with the head turned. The monitor display screen should be directly in front of the body and at an angle that encourages erect posture while minimizing glare. Documents should be placed on a stand at the same level as the monitor (rather than flat on the desk to one side) for optimum viewing to avoid excessive twisting and muscular strain.

Reflections on eyeglasses and contact lenses can be managed by changing the relative position of the light source or by using tinted lenses. Trifocal eyeglasses should not be worn by patients susceptible to splenius cervicis TrPs.

Activity Stress

Care should be taken when pulling weights while using exercise equipment. Excessive weight should be avoided, and the subject should learn to pull the weight without rotating the head and neck or projecting the head forward.

Environmental Stress

Chilling the skin of the neck, especially when the muscles are fatigued, often activates TrPs in posterior neck muscles. The patient learns to keep the neck warm by sleeping in a high- necked sleeping garment, by wearing a turtle-neck sweater or scarf during the waking hours, and by avoiding cold drafts.

Exercise Therapy

The patient releases tightness in these muscles by reaching up as in Figure 16.11A and gently flexing and turning the head to the position shown in that figure and in Figure 15.5B of this chapter. This is best done sitting on a stool or standing in a warm shower as the patient uses postisometric relaxation, with or without augmentations, to release the muscle. After release, the patient moves the head and neck *slowly and gently* through the full range of flexion, extension, and rotation three times. If adjacent muscle fibers remain sore and taut, the exercise is repeated, slightly changing the head position and thereby the angle of pull to engage muscle fibers that still feel uncomfortable and tight when stretched.

The patient should stretch the muscles in one direction at a time, release the tension, turn the head slightly, and then stretch in the next direction. Swinging the head around at the full range of motion ("head rolling") can seriously *overload* adjacent lines of taut muscle fibers, and worsen the condition.

REFERENCES

1. Adson AW, Young HH, Ghormley RK Spasmodic torticollis. *J Bone Joint Surg 28:299-* 308, 1946.
2. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 235, Fig. 4-49).
3. *Ibid.* (pp. 552-555, Fig. 8-4).
4. Baker BA: The muscle trigger: evidence of overload injury. /*Neurol Orthop Med Surg* 7:35-44, 1986.
5. Bardeen CR: The musculature. Section. 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (p. 447).
6. Barnsley L, Lord S, Bogduk N: Whiplash injury. *Pain* 58:283-307, 1994.
7. Barnsley L, Lord SM, Wallis BJ, *et al.:* The prevalence of chronic cervical zygapophysial joint pain after whiplash. *Spine 20(l):20-25,* 1995.
8. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 468-469).
9. Bogduk N, Simons DG: Neck pain: joint pain or trigger points? Chapter 20. In: *Progress in Fibromyalgia and Myofascial Pain,* Vol. 6 of *Pain Research and Clinical Management.* Edited by Vaer0y H, Mersky H. Elsevier, Amsterdam, 1993, pp. 267-273.
10. Cisler TA: Whiplash as a total-body injury. *J Am Osteopath Assoc 94(2):145-148,* 1994.
11. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 466, 467).
12. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 523, 524).
13. *Ibid.* (Fig. 527).
14. *Ibid.* (Figs. 576, 608).
15. Cooper IS: *Parkinsonism. Its Medical and Surgical Therapy.* Charles C Thomas, Springfield, 111., 1961 (pp. 224-228).
16. Cooper IS: Cryogenic surgery of the basal ganglia. *JAMA* 281:600-604, 1962.
17. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 513).
18. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer. Jena, 1912 (Fig. 55).
19. *Ibid.* (Fig. 52).
20. *Ibid.* (p. 396).
21. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (pp. 60-66).
22. *Ibid.* (pp. 64-70)
23. Evans RW: Whiplash around the world. *Headache* 35(5j:262-263, 1995.
24. Foltz EL, Knopp LM, Ward AA Jr: Experimental spasmodic torticollis. *JNeurosurg* 26:55-67, 1959.
25. Graff-Radford S, Jaeger B, Reeves JL: Myofascial pain may present clinically as occipital neuralgia. *Neurosurgery* 29(4j:610-613, 1986.
26. Griffiths HJ, Olson PN, Everson LI, *et al.:* Hyperextension strain or "whiplash" injuries to the cervical spine. *Skel Radiol* 24(4j:263-266, 1995.
27. Hong CZ, Simons DG: Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. *JMuscuioske Pain* 2(lj:89-131, 1992.
28. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia 9(3):157-164,* 1989.
29. Jaeger B: Differential diagnosis and management of craniofacial pain. Chapter 11. In: *Endodontics.* Ed. 4. Edited by Ingle JI, Bakland LK. Williams & Wilkins, Baltimore, 1994, pp 550-607.
30. Jaeger B, Reeves JL, Graff-Radford SB: A psychophysiological investigation of myofascial trigger point sensitivity vs. EMG activity and tension headache. *Cephalalgia 5(Suppl* 3J:68, 1985.
31. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 198, 199).
32. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 301, 319).
33. Khalil T, Abdel-Moty E, Steele-Rosomoff R, *et al.:* The role of ergonomics in the prevention of myofascial pain. Chapter 16. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 487-523.
34. Mailis A, Papagapiou M, Vanderlinden RG, *et al.:* Thoracic outlet syndrome after motor vehicle acci-

dents in a Canadian pain clinic population. *Clin] Pain 11:*-316-324, 1995.
35. Malleson A: Chronic whiplash syndrome. Psychosocial epidemic. *Can Fam Physician* 40:1906-1909, 1994.
36. Marbach JJ: Arthritis of the temporomandibular joints. *Am Fam Phys* 29:131-139, 1979 (Fig. 9C).
37. Merskey H, Bogduk N: *Classification of Pain.* Ed. 2. International Association for the Study of Pain, Seattle, 1994 (p. 107).
38. Modell W, Travell JT, Kraus H, ef *al:* Contributions to *Cornell Conferences on Therapy.* Relief of pain by ethyl chloride spray. *NY State J Med* 52:1550-1558, 1952 (p. 1551).
39. Munker H, Langwieder K, Chen E, *et al:* Injuries to the cervical spine in automobile accidents. *Ver-sicherungsmedizin 47(l):26-32,* 1995.
40. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360.
41. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Re-habil* 62.107-110, 1981.
42. Serra LL, Gallicchio B, Serra FP, *et al:* BAEP and EMG changes from whiplash injuries. *Acta Neuro-logica 16(5-6]:262-270,* 1994.
43. Sola AE: Trigger point therapy. Chapter 47. In: *Clinical Procedures in Emergency Medicine.* Edited by Roberts JR, Hedges JR. W.B. Saunders, Philadelphia, 1985.
44. Takebe K, Vitti M, Basmajian JV: The functions of semispinalis capitis and splenius capitis muscles: an electromyographic study. *Anat Rec* 279:477-480, 1974.
45. Travell J: Rapid relief of acute "stiff neck" by ethyl chloride spray, *f Am Med Worn Assoc* 4:89-95, 1949 (p. 91, Fig. 3; p. 93 Case 3).
46. Travell J: *Office Hours: Day and Night.* The World Publishing Company, New York, 1968 (p. 271).
47. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 2 2:425-434, 1952 (p. 427).
48. Travell JG, Simons DG: *Trigger Point Pain Patterns,* parts 1 and 2. Williams & Wilkins, Baltimore, 1993 (Wall Charts).
49. Williams HL: The syndrome of physical or intrinsic allergy of the head: myalgia of the head (sinus headache). *Proc Staff Meet Mayo Clin* 20:177-183, 1945.
50. Wyant GM: Chronic pain syndromes and their treatment. II. Trigger points. *Can Anaesth Soc J* 26:216-219, 1979 (Case 2, Table 1).

CHAPTER 16
Posterior Cervical Muscles: Semispinalis Capitis, Longissimus Capitis, Semispinalis Cervicis, Multifidi and Rotatores

with contributions by
M.L. Kuchera, I.J. Russell, and R. Shapiro

HIGHLIGHTS: Combining knowledge of the close association of trigger points (TrPs) with motor endplates and the close association of TrPs with articular dysfunction, the complex and often enigmatic posterior cervical musculoskeletal pain symptoms become easier to understand. **REFERRED PAIN** from the upper portion of the semispinalis capitis is felt in a band-like pattern projected above the orbit, and from the middle portion of the muscle it is felt over the posterior occiput. Referred pain from the longissimus capitis concentrates on the region of the ear or just behind and below it. Multifidi TrPs refer pain strongly upward to the suboccipital region, and downward over the neck and upper part of the shoulder girdle. **FUNCTION** of the posterior cervical muscles is primarily extension of the head and neck by the longer more superficial fibers and rotation by the deeper more diagonal fibers. **SYMPTOMS** due to active TrPs in these muscles are pain, marked restriction of head and neck flexion, and restriction of neck rotation. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are usually caused by sustained partial neck flexion when reading, writing, operating a computer terminal, or sewing; by holding a stooped posture; or by gross trauma. **TRIGGER POINT EXAMINATION** reveals tenderness to palpation, and sustained pressure on an active TrP elicits pain that is recognized as the clinical pain complaint. A taut band is sometimes palpable in the more superficial muscles. **ENTRAPMENT** of the greater occipital nerve is commonly caused by tension due to TrPs in the upper portion of the semispinalis capitis and/or the upper trapezius muscles. **TRIGGER POINT RELEASE** using spray and stretch requires that the direction of stretch, the direction of the muscle fibers, and the direction of application of intermittent cold be coordinated to correspond with either the chiefly longitudinal fibers or the more diagonal fibers. The types of articular dysfunction commonly associated with TrPs in the deep diagonal semispinalis cervicis, multifidi, and rotatores muscles are often corrected by suboccipital decompression, or other manipulative medicine techniques. **TRIGGER POINT INJECTION** is simplified by noting at which segmental levels the TrPs typically occur for each of the posterior cervical muscles. Injection of TrPs in the upper portion of the semispinalis capitis muscle above the level of the second spinous process should be avoided because of the proximity of the unprotected vertebral artery; however, this muscle can be injected below that level if appropriate precautions are taken. **CORRECTIVE ACTIONS** include improved posture, adoption of ergonomic work practices, adjustment of eyeglasses, use of a cervical pillow, and performance of the combined self-stretch exercise in the shower.

1. REFERRED PAIN
(Fig. 16.1)

Semispinalis Capitis

The referred pain pattern elicited from the region of attachment of the semispinalis capitis to the skull, which is at location 1 in Figure 16.1A, is shown in red in Figure 16.1**B.** Pressure on the area represented by the upper **X** in Figure 16.1**B** produces a pain pattern that travels forward like a band and encircles the head halfway, reaches maximum intensity in the temporal region, and continues on forward over the eye. The tenderness at this location is likely an area of enthesopathy induced by the sustained tension of the taut band fibers of a trigger point (TrP) in the upper third of the semispinalis capitis (location 2, lower **X** in Fig. 16.1**B),** which has a similar pain distribution.

The pain pattern characteristic of a TrP at location 3 (Fig. 16.1A) that lies in the middle third of the semispinalis capitis muscle (which is more superficial than the multifidi and rotatores) is similar to the pain pattern shown in Figure 16.lC.

Referred pain patterns of the middle and lower parts of the semispinalis capitis muscle, and the referred pain pattern of the semispinalis cervicis muscle, overlap part of the pain distribution of the C_2-C_3 zygapophysial joint.[13]

Longissimus Capitis

The pain pattern of the longissimus capitis (not illustrated) concentrates in the region of the ear or just behind and below it. The pain may extend a short distance down the neck and also may include a periorbital region behind the eye.[38,65]

Semispinalis Cervicis

The location of TrPs and the pain pattern are not illustrated separately for this muscle. It is likely to refer pain into the occipital region in a pattern similar to that shown in Figure 16.1C for the middle semispinalis capitis.

Cervical Multifidi

A multifidus TrP in the cervical region refers pain and tenderness cephalad to the suboccipital region and sometimes down the neck to the upper vertebral border of the scapula (Fig. 16.1**D)** in adults[44,66,78] and in children.[10] Bonica and Sola[14] illustrated a similar referred pain pattern. Similar referred pain patterns also were reproduced by injection of hypertonic salt solution into the posterior cervical muscles.[29,81] The pain arising from the cervical multifidi is analogous to the pattern of pain arising from the corresponding deep layer of muscles found in the lumbar spine (*see* Fig. 48.2B), in that both refer pain locally adjacent to the spinous process and may refer additional patterns several segments away from the TrP.

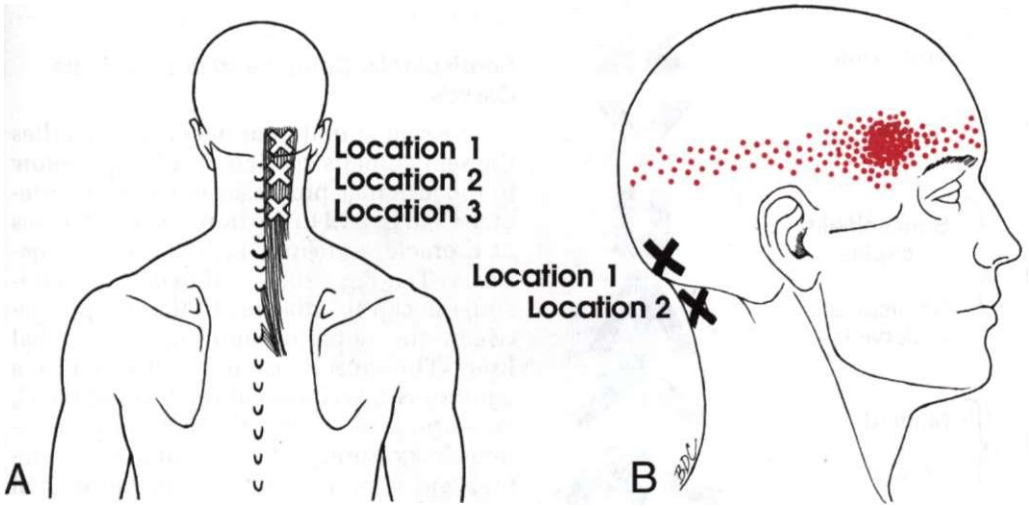
Rotatores

When present, TrPs of cervical rotatores produce midline pain and tenderness at the segmental level of the TrP, analogous to that described for thoracolumbar rotatores under **Deep Paraspinal Muscles** in Chapter 48. Pain is elicited by application of pressure or tapping on the spinous process(es) of the vertebra(ae) to which the muscle attaches. This tenderness testing is used also to identify dysfunctional spinal articulations.

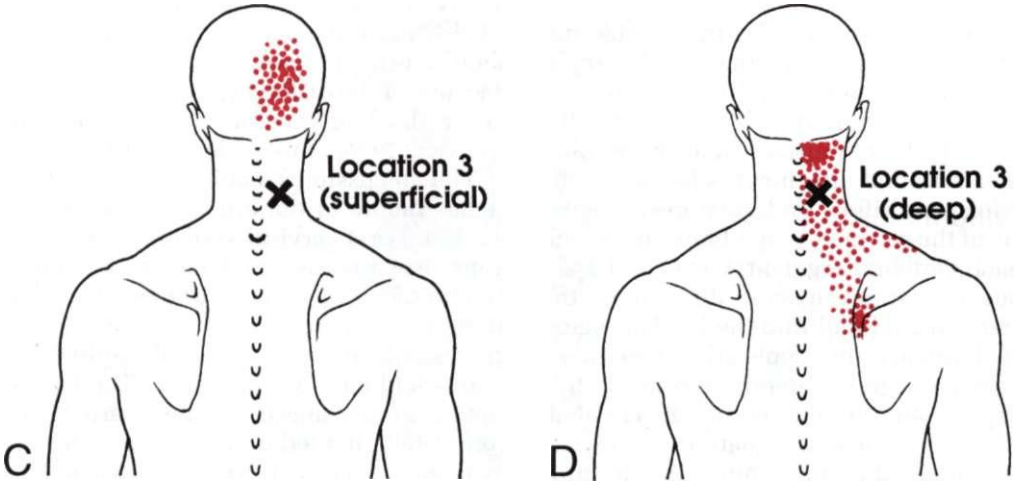
2. ANATOMY
(Figs. 16.2 and 16.3)

The posterior neck muscles have been divided anatomically into four layers[71] with fibers running in different directions at some levels, suggesting the plies of a tire (Fig. 16.2). The most superficial, the bilateral upper trapezius fibers, converge above, tending to form a "A," or roof-top shape. The next deeper, the bilateral splenius fibers, converge below to form a "V" shape. The semispinalis capitis fibers of the third layer lie nearly vertical, parallel with the vertebral column. All of the remaining, deepest fibers return to the "A" configuration. These include the more deeply placed semispinalis cervicis of the third layer and the multifidi and rotatores fibers, which constitute the fourth layer. Knowledge of this fiber arrangement is helpful in order to stretch and release these muscles effectively. The erector spinae muscles of the cervical spine include the longissimus capitis and cervicis, iliocostalis cervicis, and the variable spinalis capitis and cervicis.[25]

However, in terms of functional anatomy, these muscles divide into two groups:



Upper Semispinalis Capitis



Middle Semispinalis Capitis

Multifidi

Figure 16.1. Referred pain patterns *(red)* and their trigger points (Xs) in posterior cervical muscles. **A,** three common trigger point locations. Trigger points of the upper semispinalis capitis are expected at locations 1 and 2. One may find trigger points of the middle semispinalis capitis more superficially at location 3 and trigger points of the multifidi, rotatores, and possibly the semispinalis cervicis at a deeper level of location 3. **B,** pain pattern characteristic of trigger point locations 1 and 2 of the semispinalis capitis. Location 1 likely identifies an area of enthesopathy. Trigger points at location 2 in the upper third can contribute to entrapment of the greater occipital nerve. **C,** trigger point and pain pattern of middle semispinalis capitis. The semispinalis cervicis also can refer pain in a similar pattern. **D,** characteristic location and pain pattern of the deeply-placed cervical multifidi trigger points.

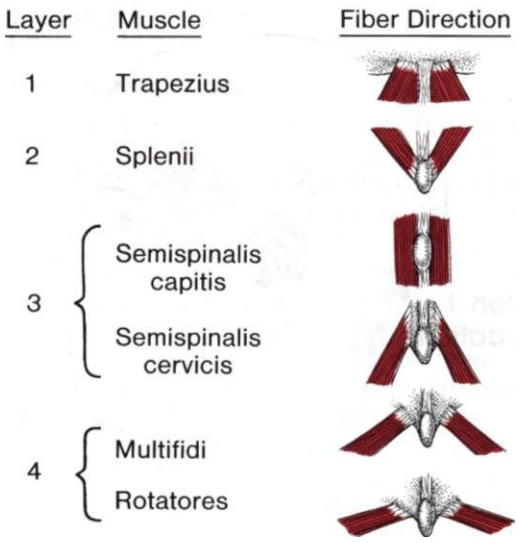


Figure 16.2. The changes in direction of successively deeper fibers in the four layers of the posterior cervical muscles, with layer 1 representing the most superficial muscle fibers and layer 4 the deepest fibers.

the group of four muscles that attach and control movement of the head (upper trapezius, splenius capitis, semispinalis capitis, and longissimus capitis), and the group of three muscles that have only spinal vertebral attachments (semispinalis cervicis, multifidi, and rotatores). Digitations of the second group of muscles attach at each vertebral segmental level and analogous digitations extend throughout the thoracic region and into the lumbar region with basically the same arrangement. At successively greater depth, muscles of this group become shorter and more angulated.

The anatomical designation of the second functional group of muscles into three names, semispinalis, multifidi, and rotatores, is quite arbitrary. In fact, there is a full and continuous transition of lengths at each spinal level. Digitations attaching at every vertebra span (bridge across) 0 to 5 vertebral segments.[23,67-69]

Each of these multiple digitations has its own endplate zone. Thus, with so many muscular digitations present, there are *many* endplate zones in the cervical paraspinal musculature on each side. Since TrPs are specifically associated with the endplate zone, knowing the location of the endplate zones identifies where one may find TrPs among these muscles.

Semispinalis Capitis and Longissimus Capitis

The **semispinalis capitis** muscle overlies the semispinalis cervicis. It attaches ***below*** to the articular processes of cervical vertebrae C_4 to C_6 and to the transverse processes of thoracic vertebrae T_1 to T_6, and sometimes T_7 (Fig. 16.3). ***Above,*** the semispinalis capitis attaches to the occiput between the superior and inferior nuchal lines. The muscle usually is divided by a tendinous inscription at the level of the C_6 vertebra. Less frequently there is a tendinous inscription at the level of the C_2 vertebra, and it is most marked in the medial fibers that arise from the thoracic vertebrae.[7,23] These inscriptions can divide the muscle so there can be three endplate zones, one in the middle of each third of the muscle. The endplate zone of the upper third of the semispinalis capitis should be a nearly transverse line at the suboccipital level. The endplate zone of the middle third should lie at approximately the C_3-C_4 level. Because of the differing fiber lengths in the lower third of the muscle, the endplate zone would be more widely distributed.

The **longissimus capitis** (Fig. 16.3) attaches ***below*** to the articular processes of the last 3 or 4 cervical vertebrae and to the transverse processes of the upper 4 or 5 thoracic vertebrae. It attaches ***above*** to the skull along the posterior margin of the mastoid process, deep to the splenius capitis and sternocleidomastoid muscles.[25] The longissimus capitis muscle is often partially or completely divided into two muscle bellies by a tendinous inscription.[7,25,33] Such a division would produce two endplate zones.

Semispinalis Cervicis

The semispinalis cervicis (not illustrated here) lies deep to the semispinalis capitis and attaches ***below*** to the transverse processes of the first to the fifth or sixth thoracic vertebrae. ***Above,*** it attaches to spinous processes of the second to fifth cervical vertebrae. Toward its cephalic end, it becomes thicker and more muscular. The fibers of the semispinalis cervicis usually span 5 vertebrae.[7,23,67] The diago-

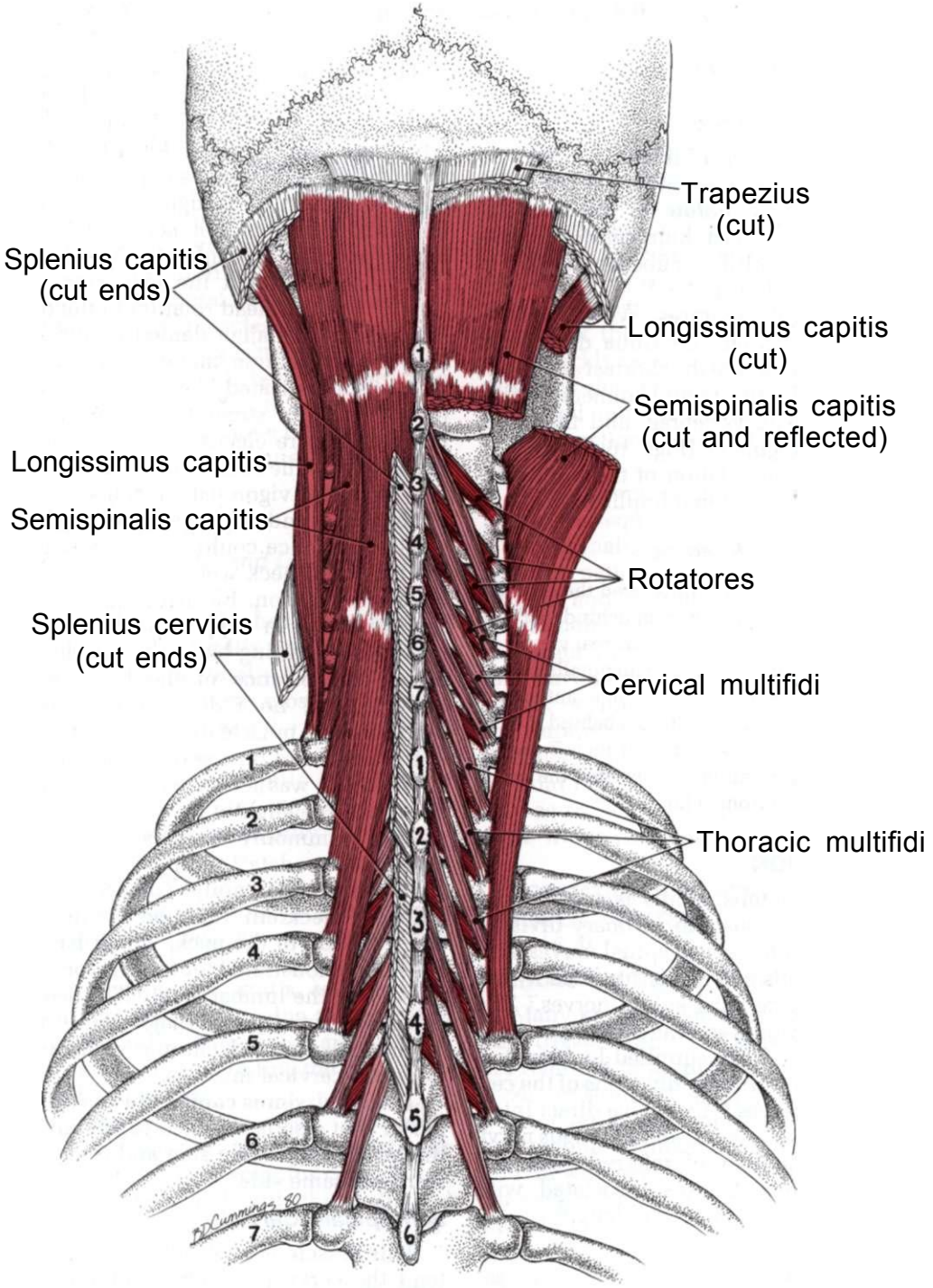


Figure 16.3. Attachments of the posterior cervical muscles. **Left side,** the fibers of the longissimus capitis and semispinalis capitis muscles *(medium red)* lie almost vertically, between the skull and the thoracic vertebrae. The semispinalis cervicis is not shown here *(see* Fig. 48.4). It is intermediate between the semispinalis capitis and multifidi in depth, fiber length, and angulation of fibers. **Right side,** the deepest layer, comprised of the multifidi *(light red)* and rotatores *(dark red)*. They travel diagonally to form, bilaterally, the roof-top "A" shape.

nal orientation of the digitations of this muscle can be seen in Figure 16.2 and in Figure 48.4 of this volume.

Multifidi and Rotatores

The cervical **multifidi** attach *above* to the spinous processes of vertebrae C_2 to C_5.[68] They attach *below* to the articular processes of the last four cervical vertebrae, C_4 to C_7; multifidus fibers cross two to four vertebrae (Fig. 16.3).[23]

The cervical **rotatores,** when present, also begin at C_2 and continue downward segmentally. They are the shortest and deepest paraspinal muscles and connect to adjacent or alternate vertebrae, and therefore, are the most angulated (Figs. 16.2 and 16.3). The degree of angulation of these muscles has important functional implications.

Supplemental References

Other authors have illustrated the semispinalis capitis as seen from behind,[1,26,33,80,67] and from the three-quarters rear view.[34] They have portrayed the semispinalis cervicis from behind.[27,33,61] Some authors have shown the multifidi from behind,[27,35,61,68] from a three-quarters rear view,[73] and from the side,[3] and have presented the rotatores as seen from behind.[2,35,69]

3. INNERVATION

The semispinalis capitis is supplied by branches of the posterior primary division of the first 4 or 5 cervical spinal nerves, and the semispinalis cervicis is supplied by the third to sixth cervical spinal nerves.[7] The longissimus capitis and the deeper posterior cervical muscles are supplied by branches of the posterior primary divisions of the cervical spinal nerves.[23] Extensive direct interconnections between C_2 and the vagus nerve result in a number of referred pain and parasympathetic reflexes associated with semispinalis capitis hypertonicity.[50]

4. FUNCTION

Functions of the semispinalis capitis primarily relate to head movement whereas the deeper intervertebral muscles are primarily concerned with spinal stabilization and spinal movement.

Semispinalis Capitis and Longissimus Capitis

The **semispinalis capitis** has one main action, extension of the head, and it functions in antigravity control of the head when one leans forward. Electrical stimulation of the semispinalis capitis produced head extension and slight inclination to the same side, but not neck extension.[31] Based on other considerations, other authors also identified the extension function[7,23,51,59] and head rotation to the opposite side.[23] Basmajian denied rotation and also did not include lateral flexion.[9]

A sophisticated electromyographic (EMG) study of strengthening exercises,[59] using fine- wire electrodes in 15 subjects, reported that the semispinalis capitis muscle responded vigorously during extension of the head and neck hut, with training, electrical silence could be achieved while the head and neck were held in the erect, balanced position. Electrical activation of these muscles in support of the head appeared only during body activity that disturbed the balance of the head on the body.[59] Also, no EMG activity was observed in this muscle during lateral flexion of the head, and during head rotation.

No study was found which specifically examined the slightly forward-flexed head posture commonly assumed for reading. The exercise data[59] strongly suggest that the semispinalis capitis consistently provides a checkrein function during even slight flexion of the neck, which has been so well demonstrated for the erector spinae muscles at the lumbar level.[8] Abuse of this checkrein activity is a major cause of the frequently observed chronic strain of the posterior cervical muscles.

The **longissimus capitis** muscle is an extensor that also is reported to laterally flex the head to the same side and rotate it toward the same side.[25,51]

Semispinalis Cervicis

This muscle is reported to primarily extend the cervical vertebral column,[23,47,51] and to rotate it to the opposite side.[23,51] The caudal attachments of this muscle to the relatively immobile thoracic vertebrae serve primarily as anchors for movement of the cervical spine. A study by Pauly[59] suggests that the semispinalis cervicis at times

provides a checkrein function during even slight flexion of the neck.

Multifidi and Rotatores

No description of the functions of this group of muscles specifically for the cervical area was found, but generally, when acting bilaterally, these deep muscles extend the vertebral column. Acting unilaterally, they rotate the vertebrae to the opposite side.[23,47,51] The multifidi were identified as contributing to lateral flexion of the spine.[23]

These deeper muscles seem to be designed for control and are said to control positional adjustments between vertebrae, rather than movements of the spine as a whole.[48] The more oblique rotatores are the most effective of these muscles for spinal rotation.

5. FUNCTIONAL UNIT

Semispinalis Capitis and Longissimus Capitis

For extension of the head, *synergists* of the semispinalis capitis and longissimus capitis include, bilaterally, the deep suboccipital muscles that lie mostly vertically, the upper trapezius, and the splenius capitis. *Antagonists* include the head flexors, especially the rectus capitis anterior and the anterior fibers of the sternocleidomastoid muscles acting bilaterally.

Semispinalis Cervicis

For extension of the neck, *synergists* of the semispinalis cervicis are the splenius cervicis bilaterally, the longissimus cervicis, the semispinalis capitis, and the levator scapulae bilaterally, plus the multifidi acting bilaterally. *Antagonists* are the anterior neck muscles, including the strap muscles and longus colli.

For rotation of the neck, the semispinalis cervicis functions *synergistically* with the contralateral splenius cervicis and levator scapulae, and with the ipsilateral multifidi and rotatores.

Multifidi and Rotatores

For extension and rotation of the neck, a synergist of the multifidi and rotatores is the semispinalis cervicis. For each separate movement, additional synergists and antagonists are the same as those listed above for the semispinalis cervicis.

6. SYMPTOMS

Patients complain of headache with pain as described and illustrated in Section 1 and are likely to be referred with the diagnosis of tension-type headache[46] or of cervicogenic headache.[45] With chronic headache sufferers, the pain pattern is likely to be a composite of referred pain from several neck and masticatory muscles (*see* Fig. 3.5).

Patients are likely to be bothered also by tenderness over the back of the head and neck, so that pressure there from the weight of the head on a pillow at night may quickly become intolerable. They usually experience some degree of painfully restricted motion of the neck in one or more directions, especially head and neck flexion.

With entrapment of the greater occipital nerve as a sequel to prolonged activation of a semispinalis capitis or upper trapezius muscle, patients complain of numbness, tingling and burning pain in the scalp over the homolateral occipital region ("occipital neuralgia") in addition to headache. They may have received anesthetic blocks of the greater occipital nerve, with relief only for the duration of the local anesthetic effect. Patients with nerve entrapment usually prefer cold rather than heat. They look for an ice-bag to relieve the burning neuropathic pain, which can now obscure the TrP pain.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS
(Fig. 16.4)

Activation of Trigger Points

Many kinds of one-time events can activate trigger points (TrPs) but other factors are required to perpetuate them.

Acute Trauma. Falling on the head, experiencing forceful head movement in an automobile accident, or diving head-first and hitting the head can produce forceful neck flexion and muscle strain even in the absence of fracture. The strain activates TrPs in head and neck muscles. Baker[6] examined 34 muscles bilaterally of 100 occupants (drivers or passengers) who sustained a single motor vehicle impact. All of these patients complained of symptoms typical of acceleration-deceleration ("whiplash") injury. All had active myofascial TrPs. The semispinalis capitis was the

third most frequently involved muscle in 73% of subjects suffering impact from the front, in 69% of subjects when hit on the passenger side, in 63% of subjects when hit on the driver's side, and in 62% of subjects when hit from behind. Automobile impact from any direction is likely to activate semispinalis capitis TrPs.

Acute Overload. Hubbell and Thomas[43] reported their examination of four postpartum patients who complained of head and neck pain, who had received epidural analgesia, and who had a long second stage of labor associated with prolonged pushing. These authors concluded that these patients had no evidence of the usual diagnosis of postdural-puncture headache as the cause of the pain. They diagnosed the patients as suffering from cervical myofascial pain because the posterior cervical muscles and/or their attachments exhibited point tenderness. No additional specific examination for myofascial TrPs was reported. Myofascial TrPs deserve serious consideration in this group of patients.

Perpetuation of Trigger Points

Chronic stress that eventually activates TrPs, if continued, will also perpetuate them.

Postural Stress. Reading or working at a desk while sitting with a forward-head posture or with the neck in sustained flexion commonly activates and perpetuates posterior cervical TrPs. This undesirable position (Fig. 16.4C) maybe assumed because: (1) the lenses of the eyeglasses have too short a focal length, (2) the frames of the eyeglasses are adjusted improperly (Fig. 16.4A), (3) the chair has inadequate lumbar support or no lumbar support, (4) there is ergonomically incorrect location of work equipment, such as a keyboard,[57,72] (5) the tension caused by TrPs in the pectoralis major muscles produces round-shouldered posture and increases thoracic kyphosis (see Fig. 41.4D),[76] or (6) the patient is emotionally depressed.[16]

Excessive cervical extension at night tends to activate and perpetuate TrPs in the posterior cervical muscles by placing these muscles in the shortened position for a prolonged period. This posture occurs when a person lies supine without a pillow on a mattress that is too hard, or when a too-hard, poorly-fitted pillow is placed under the shoulders and neck. Sometimes young people (in particular) lie prone on the floor, propped up on elbows to support the head, while watching television. This position places the posterior cervical muscles in a shortened position for a prolonged period of time.

Since the more longitudinal posterior cervical muscles commonly function bilaterally, TrP involvement of one side soon leads to at least some functional disturbance of the contralateral muscles, which can affect TrPs in them also.

A patient with a long supple neck is more prone to develop active TrPs in the posterior cervical muscles than one with a short stocky neck because of the greater leverage and demand placed on the muscles for muscular support.

Key Trigger Points. Hong[42] pointed out that the semispinalis capitis may develop satellite TrPs in response to key TrPs in either an upper trapezius muscle or a splenius capitis muscle. Elimination of key TrPs in either of these two muscles usually inactivates the TrPs of the semispinalis capitis without specific treatment of the semispinalis capitis muscle itself. Conversely, inactivating only the satellite TrP results in its reactivation and perpetuation by the key TrP.

Neuropathy. Increased nerve irritability due to entrapment, as in spinal radiculopathy, can be a significant factor in the activation and perpetuation of these posterior cervical TrPs. A comparable response has been well documented for lumbar paraspinal muscles.[22]

Facet Joint Arthritis. Halla and Hardin, Jr.[41] identified a distinctive clinical syndrome in 27 patients with C_1-C_2 facet joint osteoarthritis. Occipital TrPs were one of the major features of the syndrome. This strong association between cervical osteoarthritis and myofascial TrPs is compatible with the observation of Jaeger,[45] who found the semispinalis capitis muscle to be one of the most frequently involved. This indicates that cervical osteoarthritis is likely to activate and/or perpetuate cervical myofascial TrPs. It is possible that other arthritic conditions, such as rheumatoid arthritis and seronegative spondyloarthropathies, may have a similar influence on TrPs.

Neck Constriction. A bathing cap that is too tight or a heavy overcoat with a tight

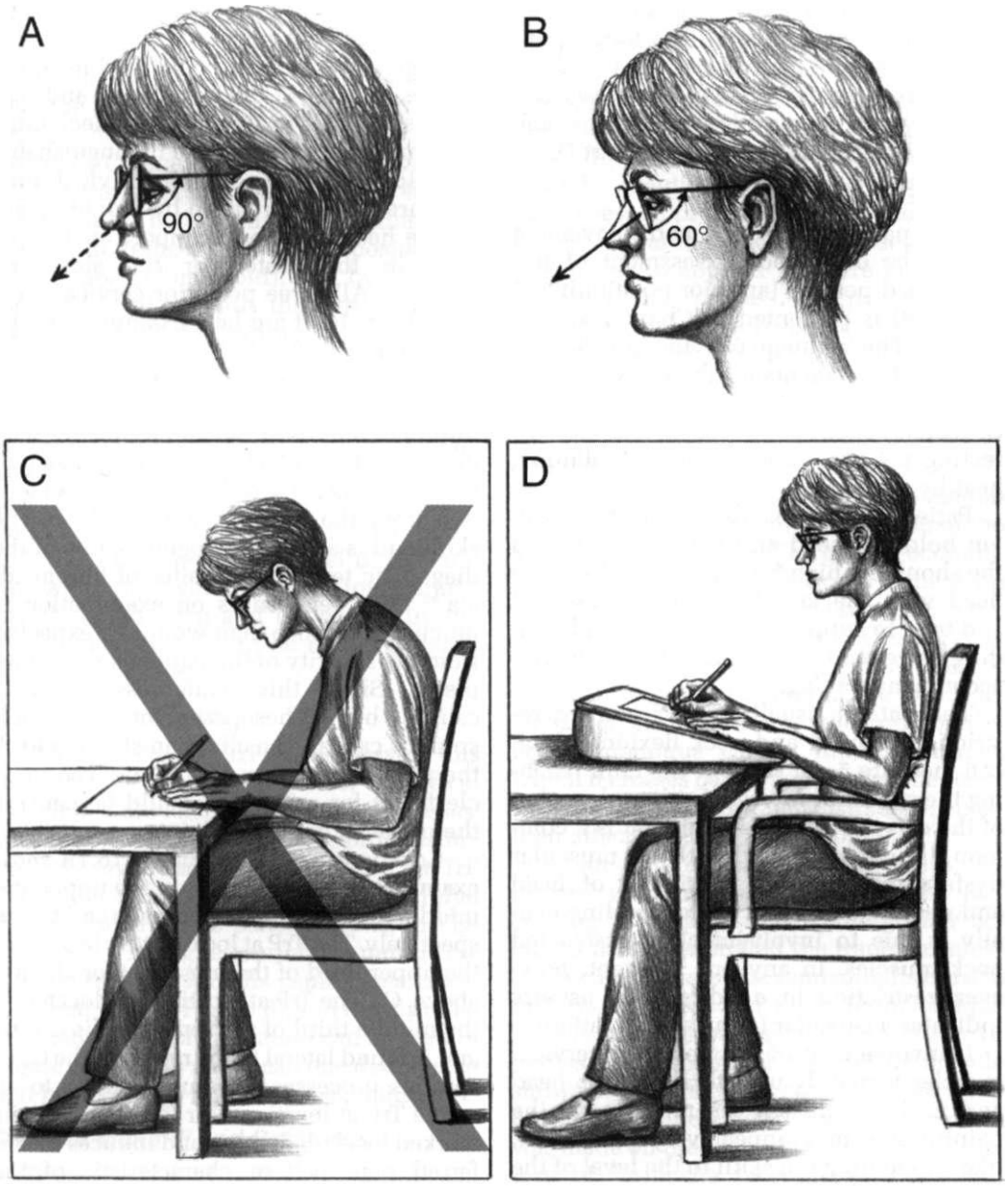


Figure 16.4. Causes and corrections of unnecessary load on the posterior cervical muscles. A, view obstructed by the lower rim of the eyeglasses, which must be compensated by a forward tilt of the head in order to read. **B,** unobstructed view for reading with the head in an erect, balanced position, after the axis of the lens has been tilted 30° or more, to bring the lower rim against the cheek. **C,** the red **X** indicates undesirable posture. The sustained spinal flexion with work placed flat on a low desk causes checkrein overload of the posterior cervical muscles. The poor posture is aggravated by having lenses with too short a focal length and rims that obstruct the line of vision. Additionally, the lack of an armrest for adequate elbow support creates a drag on the upper trapezius muscle. Lack of lumbar support in the backrest favors reversal of the normal lordotic curve and the low table top increases flexion of the spine. **D,** good posture of subject writing at a higher table with a tilted work surface, and sitting in a chair with armrests and added thoracolumbar junction support to lift the sternum. The higher table provides more adequate knee room, and the tilted board can be pulled even closer to the body for forearm support in the absence of adequate armrest support. All of these contribute to a stress-free, balanced head position.

collar that compresses the posterior cervical muscles and impairs their blood flow, may activate and perpetuate TrPs (by aggravating their energy crisis), as is described for the trapezius muscle in Chapter 6 and for TrPs in general in Chapter 2, Part D.

8. PATIENT EXAMINATION

The patient's posture and movement should be evaluated. Assessment of forward-head posture (anterior positioning of the head) is presented in Chapter 5, Section C. The influence of the position of other body segments on the positioning of the head is covered in Chapter 41, Section C, which also include suggestions for correcting poor posture and for maintaining healthy posture.

Patients with posterior cervical TrPs often hold the head and neck upright with the shoulders high;[81] they may position the head with the face tilted up somewhat[81] and tend to suppress the bobbing and nodding movements of the head that ordinarily accompany talking.

The patient usually shows marked restriction of head and neck flexion, which can measure 5 cm short of the chin reaching the sternum. Altered segmental motion of the cervical spine to palpation is a common finding associated with the muscular dysfunction. Marked restriction of head and neck rotation and of sidebending usually is due to involvement of associated neck muscles. In any one segment, however, restriction in *all* directions usually indicates a capsular (or arthritic) pattern.

If involvement of the posterior cervical muscles is mainly unilateral and the head and neck are flexed, the muscles on the painful side may appear very prominent, like a rope from the skull to the level of the shoulder girdle.

9. TRIGGER POINT EXAMINATION

Semispinalis Capitis

With the new understanding that TrPs occur in the endplate zone and the well established fact that endplate zones occur in the middle portion of muscle fibers,[28] as described in Chapter 2, Section C of this manual, it is now possible to identify where TrPs are likely to occur in the posterior cervical muscles, based on a knowledge of anatomy and the expected locations of endplate zones.

Slight flexion of the head and neck enhances tension of the taut bands and tenderness of TrPs in the posterior neck muscles and makes them more distinguishable by palpation if the posterior cervical musculature has been relaxed by providing adequate head and body support for the patient in the seated or the side-lying position. All three posterior cervical locations (Fig. 16.1) are best examined by flat palpation.

Location 1 (Fig. 16.1A) at the musculotendinous junction region is likely to feel indurated and often must be pressed very firmly to elicit referred pain. This region of tenderness is usually found a centimeter or two from the midline at the base of the skull and is also in the region of one of the diagnostic tender point sites of fibromyalgia.[80] Deep tenderness on examination is much less intense than would be expected from the severity of the patient's pain complaint. Since this tenderness is likely caused by enthesopathy of the semispinalis capitis, the clinician should check the midbelly portion of the involved muscle fibers for a TrP that could be causing the patient's pain complaint.

Locations 2 and 3 in Figure 16.1A show examples of TrP locations in the upper and middle thirds of the semispinalis capitis, respectively. The TrP at location 2 is located in the upper third of the muscle at, or slightly above, C_1. The TrP at location 3 is located in the middle third of the semispinalis capitis and is found lateral to the region of the C_3-C_4 spinous processes. Pressure applied to an active TrP at location 2 or location 3 elicits marked local tenderness and induces the referred pain pattern characteristic of the muscle containing the TrP. It is difficult to elicit a detectable local twitch response by manual palpation of this muscle in many patients. However, if the upper trapezius is relaxed, one may be able to palpate a taut band in the semispinalis capitis that is distinguished by its vertical fiber direction.

Sola[66] identified two TrP locations for the lower portion of the semispinalis capitis muscle and illustrated that they also referred pain to the suboccipital region and, in addition, to the vertex.

Longissimus Capitis

The longissimus capitis muscle lies deep to the lateral part of the splenius capitis near the level of the C_3 vertebra.[17] From the level of the C_2 spinous process[18] to the junction of C_3-C_4, one can attempt to palpate TrP tenderness and taut bands of the **longissimus capitis** by locating the splenius capitis (lateral to the trapezius and posterior to the sternocleidomastoid), and by pressing anteriorly and medially through the lateral part of the splenius capitis. If the splenius capitis has TrPs and taut bands they must first be released, or the deeper tenderness of the longissimus capitis may not be distinguishable. If the longissimus capitis has severe TrPs it should be prominent and firm, and its nearly vertical fibers help to distinguish it from the more diagonal fibers of the splenius. Superior to the level of C_2[19] and inferior to the level of C_4,[20] the longissimus capitis is too deep and is covered by too many other muscles to be reliably identified, even indirectly.

Semispinalis Cervicis

One palpates for TrP tenderness of this intermediate-to-deep posterior cervical muscle 1-2 cm lateral to spinous processes. A common TrP location is at approximately the C_4-C_5 level, and deep pressure on the TrP may elicit referred pain over the occipital region similar to the pattern shown in Figure 16.1C. The digitations of this muscle are deep to the semispinalis capitis (see cross-sectional view in Fig. 16.8), and the diagonal orientation of each digitation can be seen in Figure 48.4. Only rarely can one distinguish taut bands in this relatively deep muscle.

Cervical Multifidi and Rotatores

Trigger points of cervical **multifidi** can be located approximately halfway between a spinous process and a lower transverse process, exemplified at location 3 in Figure 16.1A and D.

Since there are digitations of the cervical multifidi for every segmental level from C_2 inferiorly, and since some digitations span more than one vertebra, TrPs in the multifidi could be found at any level between these processes starting at about the interface between spinous processes C_3 and C_4, and continuing inferiorly as thoracic multifidi.

The deepest muscles in the fourth layer, the **rotatores,** are often not as fully developed in the cervical region as they are in the thoracic region. These muscles lie too deep for the fiber direction of their taut bands to be identified by palpation. They must be identified by characteristic deep tenderness to pressure applied deep in the groove lateral to spinous processes, and by tenderness to applied pressure or tapping on the spinous process. The pain distribution of the rotatores is essentially midline pain at the segmental level.

10. ENTRAPMENT
(Fig. 16.5)

The greater occipital nerve is the medial branch of the dorsal primary division of the second cervical nerve. The greater occipital nerve supplies sensory branches to the scalp over the vertex and motor branches to the semispinalis capitis muscle.[24] This cervical nerve emerges below the posterior arch of the atlas above the lamina of the axis (Fig. 16.5). It then curves around the lower border of the obliquus capitis inferior muscle, which it crosses before penetrating the semispinalis capitis and trapezius muscles near their attachments to the occipital bone.[4]

In an autopsy study of 20 cases (40 nerves) without history of headache (according to hospital files),[15] the greater occipital nerve penetrated the trapezius muscle in 45% of cases, the semispinalis muscle in 90% of cases, and the inferior oblique muscle in 7.5% of cases. Eleven of the 18 nerves that penetrated a trapezius muscle showed evidence of compression. This finding was unexpected because selection was made on the basis of no established history of headache (according to hospital charts). Apparently, some degree of nerve compression at the point of a trapezius muscular penetration is not uncommon.[15]

After penetrating the trapezius muscle, the nerve remains subcutaneous[24,56] and has no muscular branches. Entrapment symptoms apparently develop when TrP activity in one of the muscles that it penetrates (the semispinalis capitis or the upper trapezius) produces taut bands of muscle fibers that compress the nerve as it penetrates the muscle.

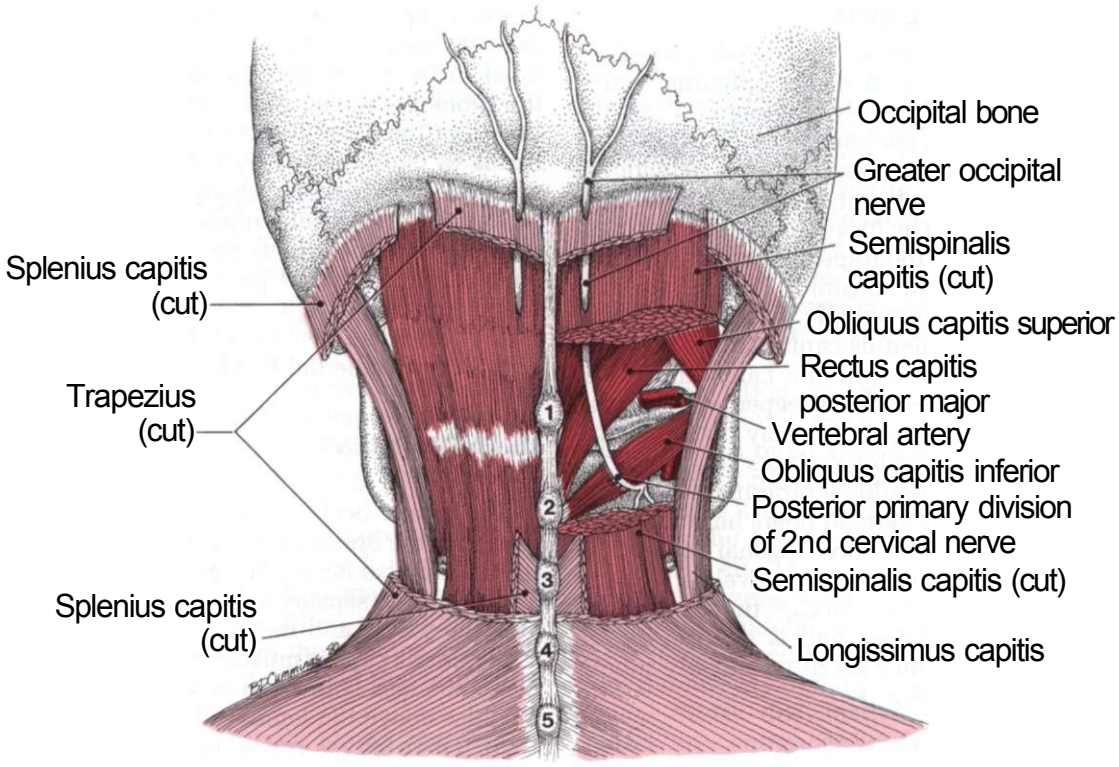


Figure 16.5. Course of the second cervical nerve, which becomes the greater occipital nerve and then penetrates the semispinalis capitis *(light medium red)* and trapezius muscles *(light red)* to continue beneath the scalp. Entrapment can occur where the nerve passes through the semispinalis muscle. Note the vertebral artery *(darkest red)* in the suboccipital triangle, which is bounded by the rectus capitis posterior major and the obliquus capitis superior and inferior muscles *(dark medium red)*.

The symptoms associated with entrapment of the greater occipital nerve are described in Section 6. They are often relieved by inactivation of TrPs in the semispinalis capitis and/or upper trapezius muscles, which usually respond well to local procaine injection or dry needling.

11. DIFFERENTIAL DIAGNOSIS

For patients with widespread pain lasting at least 3 months, **fibromyalgia** must be considered. A brief examination of the designated tender points of fibromyalgia[80] will allow the diagnosis to be established or excluded clinically with confidence. Fibromyalgia patients commonly also have myofascial TrPs that contribute to their pain.[37,40] Finding a positive occipital tender point should alert the examiner to the possibility that this is an enthesopathy secondary to a semispinalis capitis TrP.

Halla and Hardin, Jr.[41] indicated that atlantoaxial (C_1-C_2) **facet joint osteoarthritis** produces a distinctive clinical syndrome different from those associated with only subaxial degenerative joint disease of the cervical spine, and different from other articular dysfunctions of the cervical spine. This syndrome was seen mainly in elderly women who also have osteoarthritis at other sites, and who experienced occipital and postauricular pain. Physical signs were limited head rotation, tender points or TrPs confined to the occipital area, palpable cervical crepitus, and abnormal head position to one side.[41] The crepitus of the C_1-C_2 arthritis and the taut bands and recognition of pain on palpation of TrPs would be the two most clearly distinguishing characteristics.

Bogduk and Simons[13] have reported **overlapping pain patterns** of cervical zygapophysial joints and posterior cervical

muscles. The C_2-C_3 zygapophysial joints in particular need to be considered in diagnosis when dealing with TrPs in the semispinalis capitis and semispinalis cervicis muscles. The C_3-C_4 and C_4-C_5 zygapophysial joints refer pain in patterns that overlap partly with the pain distribution of cervical multifidi TrPs.

Beal[11,12] reports palpatory spasm [or TrP contracture] tissue texture changes, and cervical restricted motion at assumed to be secondary to viscerosomatic reflexes from cardiac, upper gastrointestinal and pulmonary disorders. A series of chiefly left-sided cervical somatic dysfunctions were attributed to similar visceral sources by DAlonzo and Krachman,[30] and the cervical dysfunctions are associated with pain patterns that overlap partly with the pain distribution referred from posterior cervical muscles.

Articular Disorders

The differential diagnosis of neck pain must include a wide variety of articular disorders which can cause symptoms in the cervical area but are usually diagnosed on the basis of patterns of involvement at other sites in the body. There is a limited number of arthritic conditions which typically involve the cervical spine. In addition to osteoarthritis, which is relatively noninflammatory, they can include rheumatoid arthritis and the seronegative spondyloarthropathies.

Segmental Dysfunction (Articular Dysfunction or Somatic Dysfunction). Satisfactory management of head and neck pain of musculoskeletal origin often requires careful evaluation of posterior cervical muscles for TrPs and cervical joints for restricted mobility. Often both of these findings are present, and frequently both must be treated.

Jaeger[45] examined each of 11 patients with symptoms of **cervicogenic headache** for TrPs in 7 head and neck muscles and for cervical spine dysfunction. All patients had at least 3 active myofascial TrPs. In 8 patients, TrP palpation clearly reproduced the headache. Ten of the 11 patients (91%) had a specific segmental dysfunction of the occipitoatlantal (OA) joint or of the atlantoaxial (AA) joint. The temporalis muscle was the one most likely to have TrPs (7 patients), and the semispinalis capitis was the next most likely (6 patients). Trigger points were predominantly on the most symptomatic side. Trigger points were found in other posterior cervical muscles in only two patients. Among the posterior cervical muscles, suboccipital articular dysfunction was most likely to be associated with TrPs in the semispinalis capitis.

SEMISPINALIS CAPITIS. One frequently finds a combination of OA, C_1, and C_2 dysfunctions in relation to semispinalis capitis TrPs.

LONGISSIMUS CAPITIS. With TrP tension in this muscle, one will frequently see apparent elevation of the first rib concurrent with T_1 articular dysfunction. Part of this muscle spans the region from the mastoid process to the transverse process of T_1, which allows it to indirectly affect the first rib through its pull on the costotransverse junction. Resultant rotation of the vertebra produces the *apparent* rib elevation.

SEMISPINALIS CERVICIS, MULTIFIDI, AND ROTATORES MUSCLE GROUPS. The semispinalis cervicis, multifidi, and rotatores muscle groups can form articular dysfunctions at various levels of the cervical and upper thoracic spine depending on the specific attachments.

BILATERAL POSTERIOR CERVICAL INVOLVEMENT. A simple extension dysfunction of the T_1, T_2, T_3, and T_4 segments is another important articular dysfunction associated with TrP involvement of bilateral posterior cervical muscles that attach to or span the upper thoracic vertebrae. This is particularly true of the semispinalis cervicis, multifidi, and rotatores with attachments in the upper thoracic region, as well as the semispinalis thoracis digitations that extend to and cross these upper thoracic vertebral segments. The upper thoracic segments are particularly difficult to isolate. One should, however, treat these extension dysfunctions from T_1 to T_4 by using a manual stretch technique that also incorporates contract-relax and forward flexion progressing down the spine segment by segment.

Arthritic Disorders. The **inflammatory disorders** have the potential to cause erosions at the atlantoaxial articulation which can progress to lysis of the transverse ligament and subluxation of the

odontoid process of C_2.[36] Posterior motion of the odontoid process with flexion of the head can cause compression of the cervical spinal cord with life-threatening consequences.

In addition to a careful history and examination for systemic disease, a person suspected of having symptomatic arthritic involvement of the neck should have imaging confirmation. A pair of lateral neck X-rays in voluntary flexion and extension can help to identify inappropriate motion (> 4 mm) of the odontoid process away from the internal margin of the ring of C_1. Imaging of subaxial disease will require computed tomography with contrast, magnetic resonance imaging, or even myelography. Prevention of these lesions is the objective of immunosuppressive and anti-inflammatory drug therapy, but once deformity or instability are established, surgical stabilization may be necessary.

In **rheumatoid arthritis,** subaxial involvement of the cervical spine can cause subluxation (forward slipping) of one vertebra on another (e.g., C_5 on C_6) and compress the cord. These lesions tend to be less painful than those at C_1 or C_2 but are more likely to cause loss of distal motor function.

Osteoarthritis causes osteophytic spur formation on the upper and lower borders of the cervical vertebral bodies which can limit neck motion, cause tendon popping over irregular surfaces, or even narrow the neural foramen sufficiently to cause radiculopathy.

The **seronegative** (meaning negative blood rheumatoid factor test) **spondyloarthropathy** disorders can include ankylosing spondylitis, Reiter's syndrome, reactive arthritis due to inflammatory bowel disease, or reactive arthritis associated with psoriasis.[5] A typical pathologic process in these patients is painful enthesopathy (inflammation at the site of attachment of ligament or tendon to bone) which tends to heal with diastrophic calcification.

In ankylosing spondylitis, the spinal ligaments tend to calcify *symmetrically* from the sacroiliac joints upward until the entire spine is fused into what looks on X-rays like a vertical stick of bamboo (the so-called bamboo spine). In the other disorders like Reiter's syndrome, the inflammatory involvement of the axial skeleton tends to be more *asymmetric* (skipping vertebral levels and involving only one side of some vertebrae). In any of these conditions, neck pain can be a prominent symptom, and involvement of the atlantoaxial joint can place the cervical cord at risk of serious injury. The presence of systemic symptoms, such as conjunctivitis and urethritis in Reiter's syndrome, can be helpful in establishing the correct diagnosis.

Related Trigger Points

In addition to the bilateral posterior cervical muscles, the upper semispinalis thoracis and the erector spinae muscles that extend into the thorax also are likely to become involved. The segmental level of TrP involvement often can be identified by a *flattened spot* in the normally smooth curvature of the thoracic region; when tested by forward flexion, at least one spinous process fails to stand out prominently as expected. Multiple bilateral deep short rotatores can look like the longer but less angulated multifidi in this respect; however, multifidi involvement would not cause as much restricted rotation as the rotatores do, and the multifidi are less likely to cause a contiguous series of pressure sensitive vertebrae with restricted joint mobility. Restriction of motion may respond well to appropriate bilateral stretch and spray of the deep paraspinal muscles that span the level of the flattening. Alternatively, manual techniques designed to affect both joint and muscle function may be employed.

When the posterior cervical muscles have been treated and patients continue to complain of suboccipital pain and soreness, especially in the neighborhood of the mastoid process, the examiner should check for active TrPs in the trapezius muscle (see Fig. 6.2), in the posterior belly of the digastric muscle (*see* Fig. 12.1), and in the upper medial portion of the infraspinatus muscle (see Fig. 22.1) on the same side as the pain. Trigger points in the latter two muscles cause little restriction of head motion and are easily overlooked.

Counterstrain Tender Points. Jones[49] mapped a series of tender tissue texture changes typically located near bony attach-

merits of tendons, ligaments, or in the belly of some muscles. Those located in the upper posterior cervical region, at the tips of the C_1 transverse processes, and along the mandibular rami are associated with impaired or altered function of the upper cervical segment. Jones's system of treatment, which approximates origin and insertion of muscles for 90 seconds in a comfortable position followed by a slow return to a more neutral resting position, has been used to treat muscle "spasm," headache, and tender points, as well as to restore cervical function. While there are no adequate studies of a correlation between these Jones points and latent trigger points, clinicians using both systems comment on significant overlap.[39,55]

Neuropathy

Clinically, cervical radiculopathy can activate TrPs in the posterior cervical muscles that, following surgery, are then perpetuated by other factors. This is a common cause of cervical postlaminectomy pain syndromes.[63] Since the radiculopathy and the TrPs can occur separately or concurrently, each condition must be diagnosed on its own criteria. Cervical radiculopathy from C_4-C_8 rarely fails to cause limb signs or symptoms. *Posterior* cervical TrPs alone do not produce limb symptoms. Cervical radiculopathy is much more likely to show a positive **Sperling test,** pain elicited by spinal compression applied as downward pressure on the head with the upright cervical spine slightly extended. Positive electrodiagnostic findings are helpful in identifying cervical radiculopathy. The strong relation between lumbar radiculopathy and TrPs in lumbar paraspinal muscles was recently demonstrated by Chu.[22]

One should distinguish between the local neurologically projected pain of a Tinel's sign (produced by tapping on the point of entrapment) and referred pain from a TrP. The shock-like tingling or "pins and needles" of the Tinel's sign is produced by pressure on a point of constriction, for example, where the greater occipital nerve passes through a semispinalis capitis or upper trapezius muscle (Fig. 16.5). Neural pain is usually projected along the distribution of the nerve. In comparison, TrP referred pain usually is a deep aching pain that is less well localized and has a nonneural distribution, since its location is determined by intraspinal pathways. Trigger points respond to snapping palpation with a local twitch response of the taut band. One must *avoid* injection at the point of neural entrapment, whereas injection of the TrP in the muscle that is contributing to the entrapment is appropriate therapy.

Another potential source of confusion is peripheral compression neuropathy, such as carpal tunnel syndrome at the wrist[70] and ulnar tunnel neuropathy at the elbow. These conditions can produce the perception of neck or shoulder pain. The examiner may be able to support the clinical impression of compression neuropathy by tapping on the point of entrapment. However, this Tinel's test does not have a very high specificity.

12. TRIGGER POINT RELEASE (Figs. 16.6 and 16.7)

Treatment with full-range stretching is *contraindicated* across joints that exhibit primary hypermobility. When there are trigger points (TrPs) in muscles that cross hypermobile joints, the TrPs should be inactivated using techniques that do not extend the muscles to maximum length. Such alternative therapies include TrP pressure release, hold-relax (but with a *mild* contraction, not maximum), counterstrain, indirect myofascial release, TrP injection, deep stroking or stripping massage, high voltage galvanic stimulation, and ultrasound. The muscles of these patients who exhibit primary hypermobility require strengthening with stabilizing exercises, not overall lengthening. It should be noted that secondary hypermobility of one joint may be compensatory to restricted motion in adjacent areas[54] due to articular dysfunction or TrPs. Treatment of the primary restriction in mobility is often effective in resolving compensatory hypermobility.

An important implication of recognizing the presence of atlantoaxial subluxation is that manipulation of the neck in such a patient, especially at surgery when the patient's own protective musculature is not in control, can result in cord compression. Even the neck manipulations associated

with spray and stretch can place the cervical cord at risk or injury. It should be recognized, however, that the voluntary and involuntary muscular effort involved in stabilizing the unstable arthritic neck can cause soft-tissue pain, such as myofascial pain, which may require specific treatment in addition to standard management of arthritis.

Patients who complain of neck "stiffness" generally have restricted head and neck movements in several directions due to a combination of involved muscles.[74,75] Range of motion is tested for flexion, extension, rotation, and sidebending grossly and at segmental levels. Restriction in *all* directions may indicate a capsular (or arthritic) pattern rather than a dysfunctional one. As a rule, stretch and spray are applied first to the muscles that are causing the greatest restriction of movement. When movement is severely restricted in all directions, it is usually best to start by applying gentle manual traction to the neck, and next start to restore flexion, sidebending, rotation, and extension. Upward traction can release compressive forces on the upper cervical articulations and can be applied as described for suboccipital decompression in Chapter **17,** Section **12.** The degree of involvement of individual muscle groups must be assessed for each patient, and the overlapping functions of these muscles should be considered.

It helps to visualize clearly the location and direction of the muscle fibers being passively stretched (Fig. **16.2).** Several neck muscles, with fibers running in varying directions, may contribute to a specific neck movement; therefore, stretch and release of fibers in only one direction with unidirectional parallel sweeps[58] usually release that movement only partially. Adjacent tight restricting muscle fibers also must be released. After one application of stretch and spray to release all directions of restricted movement, and after rewarming, it may be necessary to repeat the application to achieve complete restoration of normal movement.

When stretching and spraying the neck muscles to improve motion, first treat the suboccipital muscles (Chapter **17)** and the upper cervical muscles. Next, treat the long-fibered lower cervical and upper thoracic muscles, and then the long-fibered low thoracic and lumbar muscles (*see* Fig. **48.6)** are stretched and sprayed. The diagonal neck muscles that combine extension with rotation also must be released. This requires a flexion and rotation stretch. Descriptions of these stretches follow.

Longitudinal Posterior Neck Muscles

To stretch and spray the more **longitudinal posterior cervical muscles,** the patient sits in an armchair (Fig. **16.6A).** The hips are moved forward slightly to better recline the trunk against the backrest. The patient lets the head and neck hang forward and relaxed, as the clinician's hand monitors and encourages this movement to take up the slack in the extensors while the vapocoolant is applied upward over the back of the neck and head (Fig. **16.6A).** Next the patient is asked to slump forward (Fig. **16.6B)** as the operator continues to take up slack (but does *NOT* use force) and applies a downspray pattern bilaterally to cover the long paraspinal muscles from the occiput to the lower thorax. This stretch is facilitated if the patient tries to "hump the back" which adds reciprocal inhibition and voluntary stretch. This procedure can be continued down the lower thoracic and lumbar spine as illustrated in Figure **48.6,** letting the arms hang down between the knees.

This technique can be effectively combined with postisometric relaxation described in detail in Section **12** of Chapter **3. Caution:** The operator should not apply forceful pressure to the head in the positions shown in Figure **16.6 A** and **B** because the force could stress the cervical spine enough to cause complications in spines that are medically compromised. Figure **16.6C** shows a safer technique (refer to the figure legend for details).

Figure **16.6C** illustrates and describes a manual release technique for longitudinal lower posterior cervical and upper thoracic muscles that gives the clinician more direct control of the release process and provides a much better "feel" for what is happening to the muscles. This method is specifically indicated in patients who have degenerative joint disease or other compromise of the joints spanned by the muscles being released. Manual cervical traction with the patient in a supine position is an

alternate release procedure, aided by gentle contractions of the posterior cervical muscles followed by relaxation.

In addition, a specific treatment for the commonly involved **longissimus capitis** muscle employs the combination of a myofascial release technique with a contract-relax technique. The patient is in the supine position, and the clinician cradles the patient's head and, with the other hand, the clinician applies pressure along the distal attachments of the muscle. This positioning

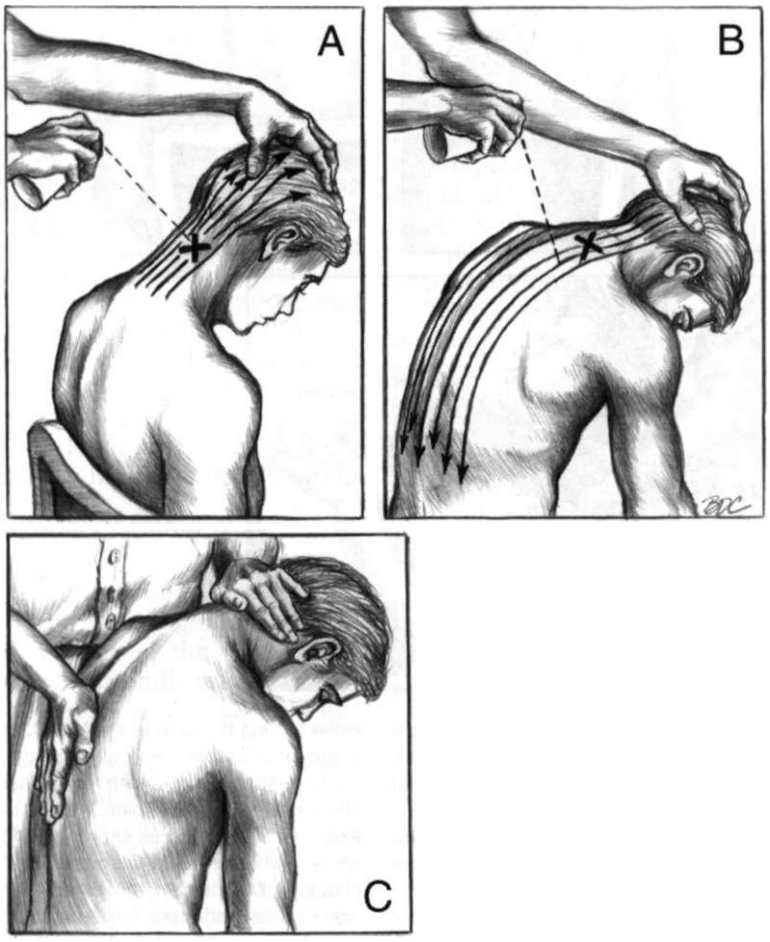


Figure 16.6. Spray pattern *(arrows)* and release technique for trigger points (Xs) in predominantly longitudinal posterior cervical and upper thoracic muscles. A, upper posterior cervical spray and release of the longitudinal semispinalis capitis muscles bilaterally and the splenius capitis, using head and neck flexion with an up-sweep spray pattern. During and after spray, the patient breathes *out,* relaxes, and looks down as the operator's left hand guides and takes up slack only. **B,** prespray of the lower posterior cervical (splenii and semispinalis) and upper thoracic longissimus muscles bilaterally with the neck and upper thoracic spine in a comfortable flexed position, using a down-sweep spray pattern *(down arrows).* **C,** manual release of longitudinal lower posterior cervical and upper thoracic muscles immediately following spray. The operator's hand placement localizes the region selected for release. Release is obtained using a contract-relax technique. As the patient looks up and gently breathes in, the operator lightly resists the contraction of the posterior cervical musculature with one hand (left in this example). Then the patient looks down, breathes out, and relaxes completely, letting the head fall forward. The operator's left hand stabilizes and the right hand applies downward pressure to release the muscles between the hands. **Caution:** The operator should not apply forceful downward pressure to the head in the position shown as it can stress the cervical spine enough to cause complications in spines that are medically compromised (see Text).

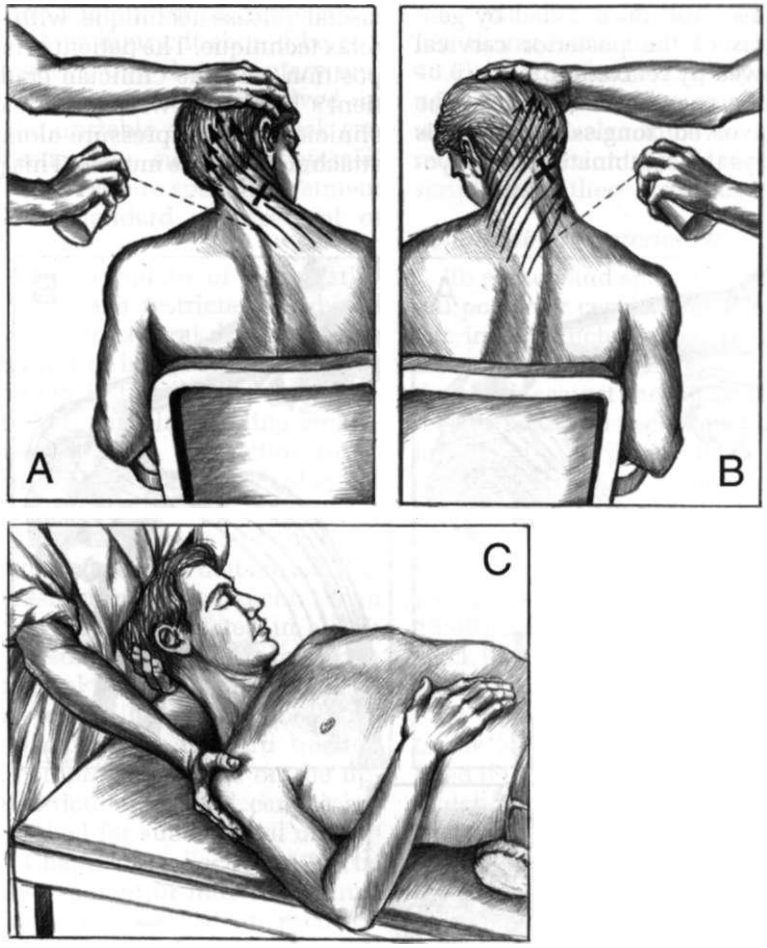


Figure 16.7. Spray pattern *(arrows)* for trigger points (Xs) in the more diagonal posterior cervical muscles. **A,** passive stretch primarily of the *right* "^" diagonal muscles (e.g., multifidi and rotatores) and the *left* "V" diagonal (e.g., splenius) by flexing the head and neck, while turning the face toward the right. See Figure 16.2 for the muscle fiber directions of cervical muscles. The skin over the muscles being stretched is covered with an up-sweep pattern of the vapocoolant. **B,** stretch of the "V" diagonal muscles (e.g., splenius) on the *right* and the "A" diagonal fibers (e.g., multifidi and rotatores) on the *left,* by flexing the head and neck while turning the face to the left. C, stretch-release of diagonal posterior cervical muscles *following* vapocoolant application, using positioning that reduces strain on the cervical spine. Patient is supine and the examiner's hand (left as shown here) cradles the head while the other hand stabilizes at the level of the shoulder. Direction of movement with traction is toward the left with neck flexion and left rotation, which is particularly effective for the right "V" diagonal muscles (e.g., splenius). The corresponding procedure is done toward the opposite side with a change of hand position for the remaining diagonal muscles.

is similar to that of Figure 16.7C; however, for the longissimus capitis release the operator's hand needs to be at the base of the neck, as in Figure 20.11 for first rib and scalene muscle release *[see* Chapter 20). Next, the clinician sidebends the patient's head away from the involved longissimus capitis muscle and "fine tunes" the muscle release with small amounts of head/neck rotation, taking up any slack in the muscle. When the barrier is encountered and the area of the costotransverse junction seems to elevate against the monitoring thumb, that same hand applies gentle **downward pressure** for release, while the clinician's other hand stabilizes the patient's head.

This stretch technique for the longissimus capitis is then integrated with a contract-relax technique. When it appears that the endpoint of the stretch has been reached, the patient is asked to take an easy shallow breath and then exhale slowly and fully during relaxation to augment the stretch. The longissimus capitis release described here can also release the scalene muscles (which *directly* elevate the first rib, *see* Chapter 20) if one then guides the neck toward slight extension rather than flexion.

The treatment technique for the longissimus capitis that was described above for the supine position can be adapted for use in releasing other posterior neck muscles that can be involved. This adaptation requires "fine tuning" of the release by adding small degrees of sidebending and rotation in line with specific tight fibers. The amount of motion can be determined by the patient's response to stretch (with minimal discomfort) and by the clinician's palpating hand. Prespray for this latter technique is shown in Figure **16.7 A** and **B.**

The More Diagonal Posterior Neck Muscles

Figure **16.7A** illustrates stretch and spray of the right "A" diagonal posterior neck muscles, including the right semispinalis cervicis, multifidi, and rotatores muscles, and the more superficial right upper trapezius.

To stretch and spray the right "V" diagonal posterior neck muscles, including the right splenius capitis and splenius cervicis, the patient gently flexes the neck and rotates the face to the opposite side with manual monitoring by the clinician as illustrated and described in Figure **16.7B.** During this stretch, vapocoolant is applied bilaterally in a diagonal upsweep pattern that follows the line of the stretched fibers on *both* sides of the neck, since stretch of these "V" diagonal muscles on the right also stretches "A" diagonal muscles on the left, and *vice versa.*

Figure **16.7C** illustrates and describes a manual release technique for these diagonal muscles using positioning that reduces strain on the cervical spine; intermittent cold (icing or spray) can be used prior to this release. An alternate way of placing the hands in a crossed position for releasing posterior cervical muscles is illustrated and described by Ehrenfeuchter, *et al.*[32]

13. TRIGGER POINT INJECTION
(Figs. 16.8, 16.9, and 16.10)

Injection of the active trigger points (TrPs) should be considered only after stretch and spray or other noninvasive treatment has been tried, and the patient's TrP pain and restricted neck motion persist. However, patients with fibromyalgia are relatively intolerant of manual release techniques, so injection may be the preferred TrP therapy for some of them. In patients with fibromyalgia, adjunctive but specific injections of myofascial TrPs can produce dramatic results.[64]

Trigger point injection is followed immediately by spray and stretch (or another method of gentle muscle release and lengthening) of the injected muscle, and then by full *active* range of motion. A hot pack can then be applied to rewarm the skin over the muscle. Injection of posterior cervical muscles also has been described and illustrated by Kraus[52] and by Rachlin.[62]

Trigger points in the posterior cervical muscles are frequently bilateral, so it is often necessary to inject them on both sides of the body. A common mistake is the failure to inject deeply enough because of the possibility of penetrating the vertebral artery in the posterior cervical triangle or the dura mater of the spinal cord. These are significant concerns, so these deep TrPs should not be injected by beginners and should never be injected in a hurry. The vertebral artery is avoided by noting carefully the spinal level and *avoiding* injections deep into the lateral posterior neck at, or above, the level of the C_2 spinous process (Fig. **16.5).**

The vertebral artery is vulnerable to needle penetration as the vessel emerges from its path through the transverse processes of the vertebrae to enter the cranial vault (Fig. 16.5).

A number of disturbing experiences have occurred during injection deeply at the level of the spinous process of the atlas (C_1), which is normally less prominent

than C_2. One report[77] was based on the impression that numbness, tingling, and weakness which developed in the opposite arm during the TrP injection may have been due to vertebral artery spasm and spinal cord or brain ischemia. Months later, the patient, apparently malingering, was receiving compensation for the complaints while working full time elsewhere, without evident disability. Apparently, the symptoms had cleared up spontaneously.

A second patient, during posterior cervical TrP injection, developed similar contralateral arm symptoms, which suggested cerebral or spinal cord ischemia. The symptoms disappeared spontaneously in 3 days.

A third patient developed similar symptoms of persistent tingling and pain in the contralateral upper limb in the course of this TrP injection, and was reexamined meticulously 3 days following the onset. He was found then to have marked activation of TrPs in the scalene muscles on the side of the symptoms. Inactivation of these scalene TrPs by procaine injection promptly eliminated the upper limb pain, without recurrence through several years of follow-up. Apparently, latent scalene TrPs on the contralateral side were activated, possibly as satellites of the posterior cervical TrPs.

In general, penetration into the spinal canal is avoided by always angling the needle slightly laterally when injecting the deeper paraspinal muscles. However, in some patients, the cervical spinal cord may not be covered by bone between vertebrae as far as 1 cm or more lateral to the edge of a cervical spinous process. Penetration of the dura in this space can be avoided by establishing the depth of the lamina at 2 cm lateral to the lateral edge of a cervical spinous process, and *not* inserting the needle to a greater depth whenever it must be directed more medially. When testing for the depth of the lamina in this way it is important to be sure that contact with bone has not bent the tip of the needle. If this happens, a scratchy sensation develops on retraction of the needle. The needle must be replaced immediately.

Semispinalis Capitis

The upper portion of the semispinalis capitis muscle lies deep to the upper trapezius medially and the splenius capitis laterally (Fig. 16.5). After confirming by palpation that pain is originating from location 1 in the attachment region of the upper semispinalis capitis (Fig. 16.1 A and **B),** the tender area may be injected by angling the needle upward, directing it toward the occipital bone, not below the bony margin. This avoids the vertebral artery, which lies deep and below the lower margin of the occipital bone (Fig. 16.5). Immediate restoration of full neck flexion may follow treatment at this attachment point, but the scalp pain and hyperesthesia of prior occipital nerve entrapment by the muscle may last from a few days to several weeks, diminishing gradually.

A TrP near location 2 in the upper semispinalis capitis (Fig. 16.1 A and **B)** should NOT BE INJECTED because of its proximity to the vertebral artery. Intermittent cold and stretch, trigger point pressure release, and deep massage can be employed to inactivate TrPs. This TrP may be responsible for the attachment tenderness at location 1 and should not be neglected.

The middle portion of the semispinalis capitis muscle lies deep to both the upper trapezius and splenius capitis muscles (Figs. 16.5 and 16.8), and therefore requires relatively deep penetration for injection. Injection of its TrPs near the C_3-C_4 region (Fig. 16.9)-the most likely location of TrPs in this muscle (Fig. 16.10)-usually does not pose a serious threat to the vertebral artery. However, the needle should *not* penetrate the area superior to C_2 where the artery is vulnerable. Rachlin[62] presents in his Figure 10-40 a clear illustration of the anatomical relations and his injection technique for the semispinalis and multifidi muscles near the C_4 level.

Longissimus Capitis

A TrP region in this long, relatively narrow muscle can be injected in the lateral part of the neck (deep to the splenius capitis muscle and lateral to the semispinalis capitis) at approximately the level of C_3, which is a common location for TrPs in this muscle (Fig. 16.10). At a level inferior to C_4 (as seen in the C_5 level cross section

of Fig. 16.8), the muscle is too deep to be reliably identified.

Injecting this muscle at the C_3 level should pose no danger to the vertebral artery if the needle is directed laterally and the muscle is not injected superior to C_2.

Semispinalis Cervicis

This muscle is deep to the semispinalis capitis, superficial to the multifidi, and has longer fibers than the multifidi. Trigger points in the semispinalis cervicis are not likely to be found above the level of the spinous process of C_4 (Fig. 16.10). Any existing TrPs would be found at various levels about halfway between the spinous processes and the transverse processes of each vertebral segment spanned by digitations of the multifidi that harbor TrPs. Figure 16.8 presents in cross section the location of this muscle near the cephalad end of expected TrPs. This same figure illustrates that needle penetration to about 5 cm (2 in), nearly halfway through the diameter of the neck, may be required to reach these deep paraspinal muscles. It may be simpler

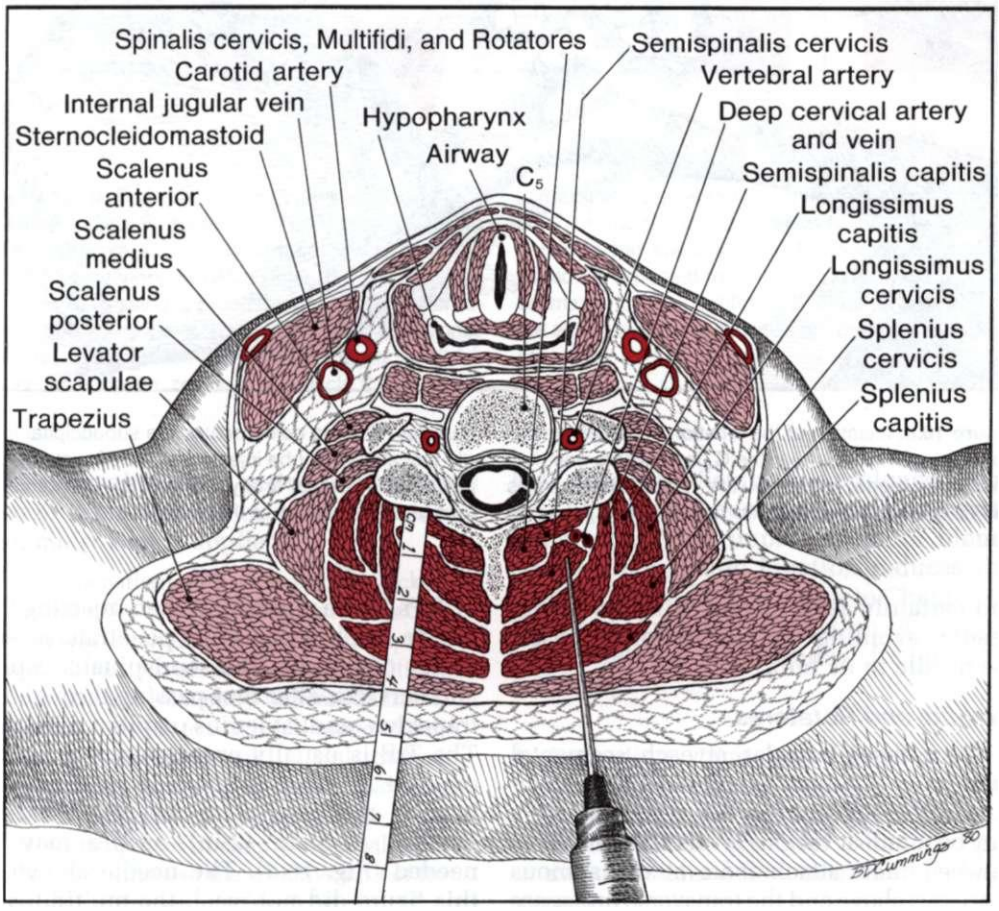


Figure 16.8. Cross section of the neck through the C_5 vertebra, which corresponds to the approximate level of the TrP region at location 3 in Figure 16.1. The bony parts of the vertebra are stippled black and are outlined by a *dark line surrounding black stipples.* The ruler shows that the 5-cm (2-in) needle cannot penetrate the full depth of the posterior cervical muscles without compression of the skin. The vertebral artery is surrounded by the vertebral transverse processes. It travels anterior to, and along the lateral border of the posterior cervical muscles. Paraspinal muscles and major blood vessels are *dark red;* other muscles are *light red.*

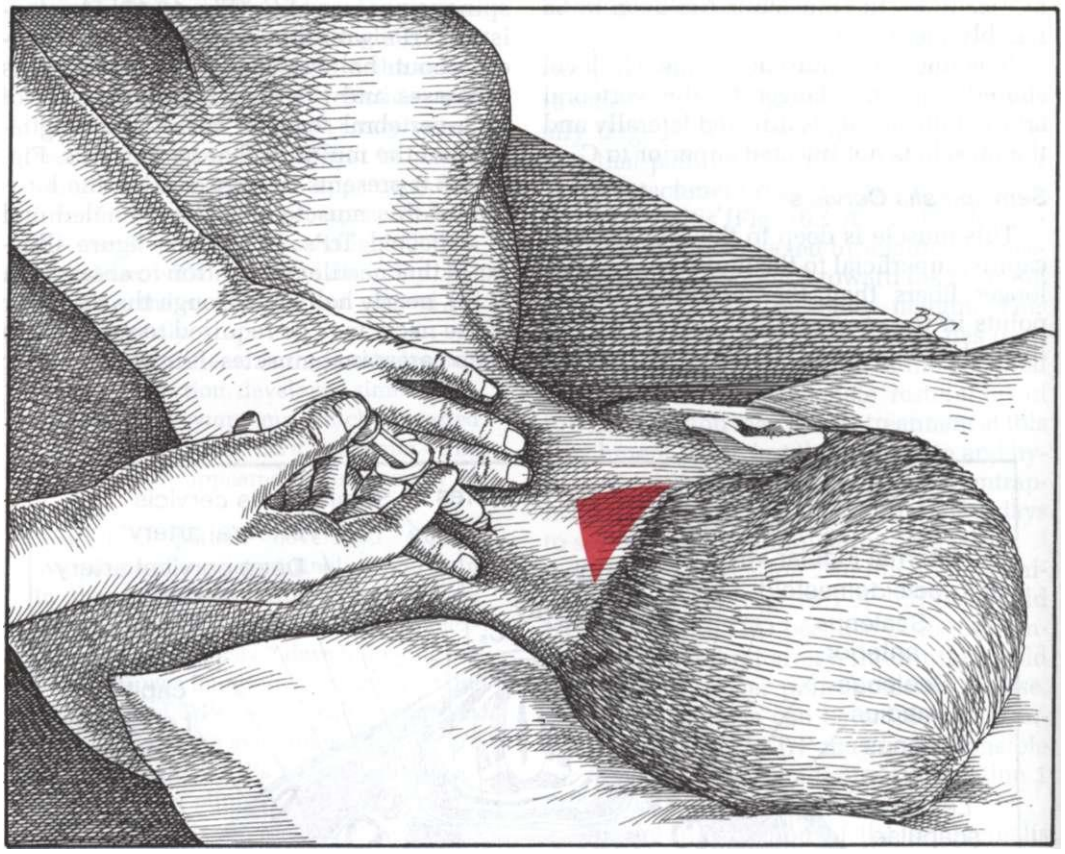


Figure 16.9. Injection of the location in the left posterior cervical muscles near the C_4 level where one may encounter trigger points of the middle semispinalis capitis, semispinalis cervicis, multifidi, and rotatores muscles. The *red color* locates the suboccipital triangle that should *not* be injected, so as to avoid the unprotected vertebral artery. Figure 16.5 illustrates the muscular boundaries of the triangle.

and certainly safer to start with the longer needle, avoiding the temptation to insert the needle to its hub.

Multifidi and Rotatores

Since these muscles at each segmental level have different lengths of fibers spanning different numbers of vertebrae, TrPs may be found at various levels about halfway between their attachments at the spinous processes above and the transverse processes below. The rotatores are the deepest muscles and lie directly over the laminae of vertebrae so that they are rather readily identified as the muscular layer immediately superficial to needle contact with the lamina.

Location 3 of Figure 16.1A and **D** illustrates a common location and pain pattern of TrPs in the multifidi. When injecting this TrP, to reach it one must penetrate several layers of muscle (the semispinalis capitis and cervicis, after first passing through the trapezius and splenius capitis muscles). The TrP is usually encountered at least 2 cm (3/4 in) deep to the skin, and may lie beyond the reach of a 3.8-cm (1 1/2-in) needle. A 5-cm (2-in) needle may be needed (Fig. 16.8). The needle shown in this figure did not reach the multifidus. It helps to depress the skin on both sides of the needle while injecting. The pain response to injection may seem out of proportion to the tenderness elicited by palpation, because of the depth of the TrPs. Following injection, passive rotation stretch during vapocooling is performed;

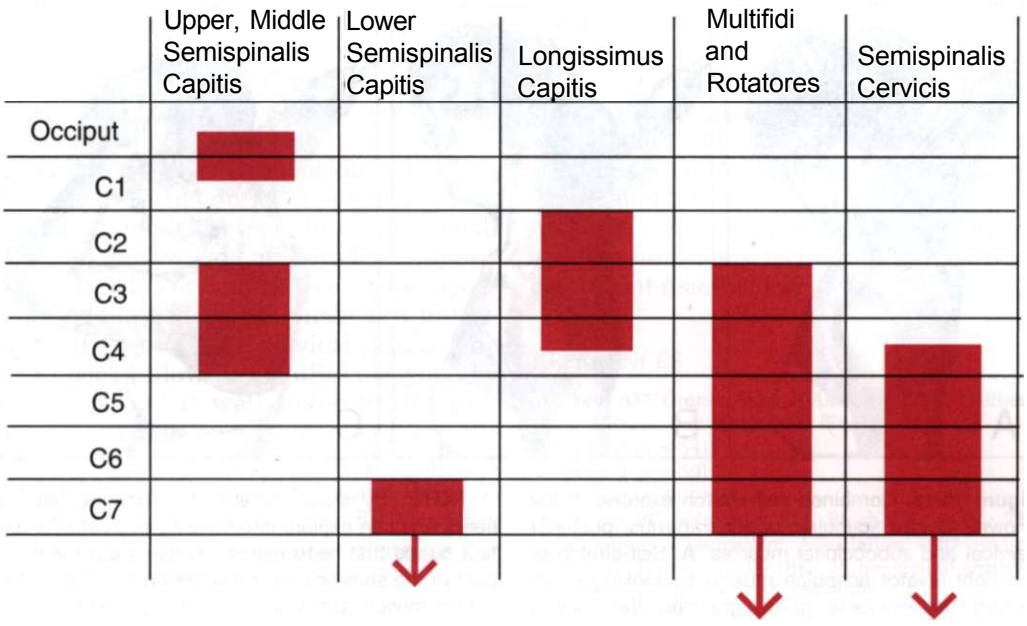


Figure 16.10. Possible locations (many not palpable) of trigger points in posterior cervical muscles based on attachments and expected locations of endplate zones for posterior cervical muscles. Segmental levels correspond to spinous processes (or the posterior tubercle of C_1). The expected location of endplate zones is based on the anatomy of Figure 16.3 and the distribution of endplate zones in a muscle.[28] The upper semispinalis capitis corresponds to location 2 in Figure 16.1 and the middle portion to location 3. Trigger points in the lower third of the semispinalis capitis would be expected to range from C_7 to about T_2. The estimates for the multifidi and rotatores only apply to the extent that these muscles are present in the cervical region.

the patient then does active full rotations (two or three times in each direction), and then moist heat is applied.

> In one patient with a chronically locked hypomobile cervical-occipital junction who was receiving osteopathic manipulation, injection of the cervical multifidi and rotatores bilaterally increased left lateral rotation 45° to reach full range of motion and increased right lateral rotation 25° to reach full range of motion (Gerwin, 1996, personal communication). This shows the power of deep paraspinal muscle shortening and the effectiveness of inactivating the responsible TrPs.

14. CORRECTIVE ACTIONS (Fig. 16.11)

Postural Stress

Chronic strain activates posterior longitudinal cervical TrPs as these muscles checkrein the weight of the head when it is held in partial flexion for prolonged periods. Optimizing posture to reduce gravitational stress[53] or improvement of biomechanical/ergonomic function reduces this strain. The reader is referred to Chapter 41, Section C for a full discussion of postural considerations. Corrections include the following:

1. A reading stand or adjustable music stand to change the angle of, or to raise, the reading and work materials and to approximate eye-level contact and avoid sustained flexion of the head and neck.
2. Elevation of the computer monitor when it is used continuously for prolonged periods and when it requires a downward gaze.
3. Eyeglasses with adequate focal length so that the patient can see clearly with the head held in a balanced upright position.

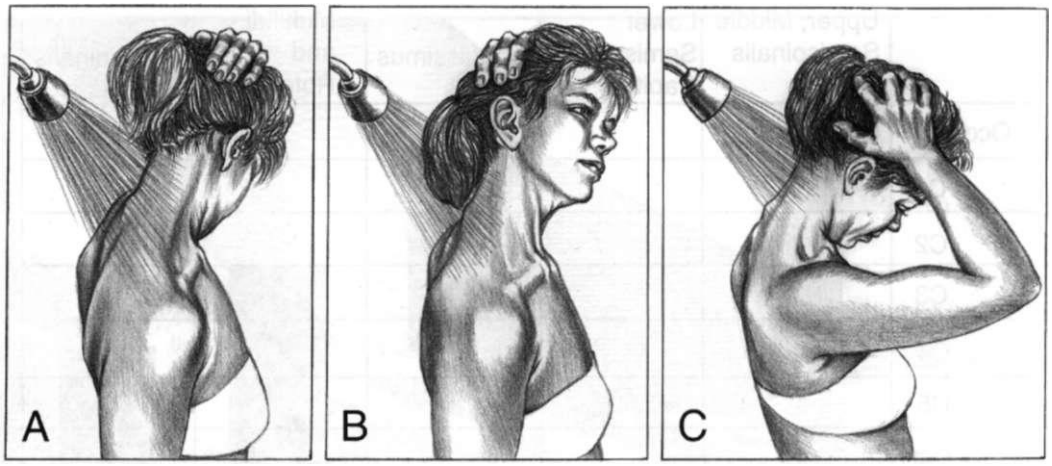


Figure 16.11. Combined self-stretch exercise in the shower: levator scapulae, upper trapezius, posterior cervical and suboccipital muscles. **A,** Self-stretch of the right levator scapulae muscle by looking down toward the opposite axilla, grasping the rotated head above the mastoid area and taking up slack in the muscle, while reaching downward toward the floor with the free hand to lengthen the muscle. **B,** Self-stretch of the right upper trapezius muscle by sidebending the neck to the opposite side, and rotating the face as far as is comfortable to the same side as the involved muscle; the patient slowly exhales and allows the weight of the arm to take up slack. As the muscle relaxes, the free hand reaches downward toward the floor. **C,** Self-stretch of the posterior cervical muscles. The occipital region is grasped by the thumbs as the hands assist active head flexion, while the patient looks down and slowly exhales.

NOTE: By *slowly* sidebending and turning the head, one can explore intermediate positions for any taut bands that need release. In every case, the impact of the shower of warm water on the skin overlying the muscle assists in relaxation and release of the muscle. This exercise may be done seated as well as standing. Since the levator scapulae and the upper trapezius are attached to the scapula and the clavicle, respectively, reaching the arm downward lowers their distal attachments and stretches those muscles; it also provides helpful reciprocal inhibition of them. Since stretching of a muscle on one side of the neck puts the contralateral muscle in a shortened position, it can activate a latent TrP in that muscle and produce reactive cramping. Therefore, these stretches in **A** and **B** for the right levator scapulae and right upper trapezius should each then be performed for the corresponding muscles on the left side. *Active* range of motion should follow each stretch.

Otherwise, a new prescription for longer focal length lenses ("card playing or computer glasses") should be obtained.

4. Selection of bifocal insets that are large, fully half the height of the entire lens, when needed for close work such as reading or sewing.
5. Adjustment of eyeglass frames so that the lower portion of the rim does not occlude the line of sight on looking down (Fig. 16.4A and B).
6. Exercising on a stationary bicycle by sitting upright with the arms swinging freely or placed on the hips, and *not* hunched over holding low handlebars that do not steer the machine.
7. Placing a cloth roll or pillow behind the thoracolumbar junction while sitting to maintain the normal lumbar lordotic curve and lift the sternum, improving head and neck posture.
8. Inactivation of pectoralis major or minor TrPs (see Chapters 42 and 43) that induce round- shouldered posture and a functional thoracic kyphosis.

These last two corrections permit the erect head and neck to assume a balanced relaxed position over the thoracic spine (as in Fig. 16.4D). In summary, as emphasized by Tichauer,[72] the patient must comfortably maintain a balanced head posture.

Another simple correction to promote erect balanced sitting posture is provided by placing a small pad under the ischial tuberosities. The pad should *not* extend under the upper thigh.

Excessive cervical extension at night is corrected by obtaining a slightly softer (non-sagging) mattress, or by using a small soft neck pillow that comfortably supports the normal cervical curve. Chattopadhyay[21] described the rationale and importance of a well-fitting cervical pillow. The small neck pillow (Cervipillo) designed by Ruth Jackson[44] is well suited to this purpose. A versatile and adaptable pillow, the Wal-pil-o(r), was described by Walpin.[79] A jiggly foam rubber pillow must be discarded and replaced with one filled with a non-springy material, like feathers or shredded dacron.

Other Factors

The neck muscles of patients with posterior cervical TrPs may be particularly vulnerable to chilling and, if so, can be kept covered at night by a turtle-neck sweater worn in bed, or by a loose scarf draped around the neck. Similarly, the neck must be protected from cold drafts during the day. Long hair offers natural protection against this cold exposure.

To temporarily relieve neck strain after an acute exacerbation, one may prescribe a soft collar to be worn loosely as a *chin rest* when riding in a car or working at a desk. The collar is NOT tightly adjusted for immobilization of the neck, but applied loosely. For instance, a Thomas plastic collar may be worn upside down and loosely enough to allow space for head rotation and to look down at the sides, yet tight enough to support the chin so that the head is in the neutral position.

Exercise Therapy

A primary form of self-therapy for patients with posterior cervical TrPs is presented in Figure 16.11 as part of the combined self-stretch exercise in the shower. Details are presented in the caption. The patient may place a lightweight sandbag on the head during periods of the day for posture training.[16] Head-rolling exercises, or other movements which hold the head in extreme positions while changing the direction of stretch, should be *avoided.*

Keep in mind that if cervical joints are hypermobile, the patient will learn to do stabilizing exercises instead of stretching. The patient can use TrP pressure, self-massage, self-positioned counterstrain, and the hold-relax technique to inactivate or prevent reactivation of the TrP.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (Figs. 4- 51, 4-54).
2. *Ibid.* (Fig. 4-54).
3. *Ibid.* (Fig. 4-59).
4. *Ibid.* (Fig. 4-56).
5. Arnett FC, Edworthy SM, Bloch DA, *et al:* The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum* 32:315-324, 1988.
6. Baker BA: The muscle trigger: evidence of overload injury. /*Neurol Orthop Med Surg* 7:35- 44, 1986.
7. Bardeen CR: The musculature. Section. 5. In *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (pp. 449-452).
8. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 355, 360).
9. *Ibid.* (p. 468).
10. Bates T: Myofascial pain. Chapter 14. In *Ambulatory Pediatrics II: Personal Health Care of Children in the Office.* Edited by Green M, Haggerty RJ. W.B. Saunders, Philadelphia, 1977 (Fig. 14-1, p. 148).
11. Beal MC: Viscerosomatic reflexes: a review. / *Am Osteopath Assoc* 85:786-801, 1985.
12. Beal MC, Morlock JS: Somatic dysfunction associated with pulmonary disease, *f Am Osteopath Assoc* 84:179-183, 1984.
13. Bogduk N, Simons DG: Neck pain: joint pain or trigger points? Chapter. 20. In: *Progress in Fibromyalgia and Myofascial Pain,* Vol. 6 of Pain *research and Clinical Management.* Edited by Vaeray H, Mersky H. Elsevier, Amsterdam, 1993 (pp. 267-273).
14. Bonica JJ, Sola AE: Neck pain. Chapter 47. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990 (p. 858).
15. Bovim G, Bonamico L, Fredriksen TA, *et al.:* Topographic variations in the peripheral course of the greater occipital nerve. Autopsy study with clinical correlations. *Spine* i6(4/:475-478, 1991.
16. Cailliet R: *Soft Tissue Pain and Disability.* F.A. Davis, Philadelphia, 1977 (pp. 131- 133).
17. Carter BL, Morehead J, Wolpert SM, *et al: Cross-Sectional Anatomy: Computed Tomography and Ultrasound Correlation.* Appleton-Century-Crofts, New York, 1977 (Sect. 15).
18. *Ibid.* (Sect. 14).
19. *Ibid.* (Sect. 13).
20. *Ibid.* (Sect. 16).
21. Chattopadhyay A: The cervical pillow. *J Indian Med Assoc 75(l):6-9,* 1980.

22. Chu J: Dry needling (intramuscular stimulation) in myofascial pain related to lumbosacral radiculopathy. *Eur JPhys Med Rehabil 5(4).*106-121, 1995.
23. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 469- 471).
24. *Ibid.* (p. 1194, Fig. 12-28).1985
25. *Ibid.* (pp. 466-469, 472, Fig. 6-21).
26. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 524, 525).
27. *Ibid.* (Fig. 526).
28. Coers C, Woolf AL: *The Innervation of Muscle, A Biopsy Study.* Blackwell Scientific Publications, Oxford, 1959.
29. Cyriax J: Rheumatic headache. *Br Med J* 2:1367-1368, 1938.
30. D'Alonzo GE Jr, Krachman SL: Respiratory system. Chapter 37. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997 (pp.441-458).
31. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 534).
32. Ehrenfeuchter WC, Heilig D, Nicholas AS: Soft Tissue Techniques. Chapter 56. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997 (pp.781-794, *see* p. 783).
33. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena 1912 (pp. 401, 404, 406, 420, Figs. 56, 57).
34. *Ibid.* (p. 405, Fig. 58).
35. *Ibid.* (p. 426, Figs. 59, 61).
36. Erhardt CC, Mumford PA, Venables PJ, *et al.:* Factors predicting a poor life prognosis in rheumatoid arthritis: an eight year prospective study. *Ann Rheum Dis 48:7-13,* 1989.
37. Gerwin R: A study of 96 subjects examined both for fibromyalgia and myofascial pain [Abstract]./ *Musculoske Pain 3(Suppl* 1j.121, 1995.
38. Gerwin R: Personal communication, 1996.
39. Glover JC, Yates HA: Strain and counterstrain techniques. Chapter 58. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997:809-818 (p. 810).
40. Granges G, Littlejohn G: Prevalence of myofascial pain syndrome in fibromyalgia syndrome and regional pain syndrome: a comparative study. / *Musculoske Pain l(2):19-35,* 1993.
41. Halla JT, Hardin JG Jr.: Atlantoaxial (C1-C2) facet joint osteoarthritis: a distinctive clinical syndrome. *Arthritis Rheum* 30f5j:577-582, 1987.
42. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. / *Musculoske Pain* 2(l):29-59, 1994.
43. Hubbell SL, Thomas M: Postpartum cervical myofascial pain syndrome: review of four patients. *Obstet Gynecol* 65.-56S-57S, 1985.
44. Jackson R: *The Cervical Syndrome.* Ed. 3. Charles C Thomas, Springfield, 111., 1977 (pp. 310-314).
45. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia 9(Suppl* 3J.157-64, 1989.
46. Jaeger B, Reeves JL, Graff-Radford SB: A psychophysiological investigation of myofascial trigger point sensitivity vs. EMG activity and tension headache. *Cephalalgia 5(Suppl* 3j:68, 1985.
47. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W.B. Saunders, Philadelphia, 1991 (p. 201).
48. *Ibid.* (p. 203).
49. Jones LH: *Strain and Counterstrain.* American Academy of Osteopathy, Colorado Springs (now Newark, OH), 1981.
50. Kappler RE, Ramey KA: Head, diagnosis and treatment. Chapter 44. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997 (pp. 515-540, *see* p. 530).
51. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993.
52. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (pp. 104, 105)
53. Kuchera ML: Gravitational stress, musculoligamentous strain and postural realignment. *Spine* 9(2):463-490, 1995.
54. Kuchera WA, Kuchera ML: *Osteopathic Principles in Practice.* Ed. 2. Greyden Press, Columbus, OH, 1994 (p. 360).
55. Kuchera ML, McPartland JM: Myofascial trigger points, an introduction. Chapter 65. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997 (pp. 915-918).
56. Lockhart RD, Hamilton GF, Fyfe FW: *Anatomy of the Human Body.* Ed. 2. J.B. Lippincott, Philadelphia, 1969 (pp. 169, 274, Fig. 278).
57. Middaugh SJ, Kee WG, Nicholson JA: Muscle overuse and posture as factors in the development and maintenance of chronic musculoskeletal pain. Chapter 3. In: *Psychological Vulnerability to Chronic Pain.* Edited by Grezesia R, Ciccone D. Springer Publishing Co., New York, 1994 (pp. 55-89).
58. Modell W, Travell JT, Kraus H, *et al.:* Contributions to Cornell Conferences on Therapy. Relief of pain by ethyl chloride spray. *NY State J Med* 52.1550-1558, 1952.
59. Pauly JE: An electromyographic analysis of certain movements and exercises: 1. Some deep muscles of the back. *AnatRec* 255:223-234, 1966.
60. Pernkopf E: Arias *of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 30).
61. *Ibid.* (Fig. 35).
62. Rachlin ES: Injection of Specific Trigger Points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 305-308, Fig. 10- 40).
63. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981.
64. Rubin BR: Rheumatology. Chapter 38. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997, pp. 459-466.
65. Shapiro R: Personal Communication, 1996.
66. Sola AE: Trigger point therapy. Chapter 47. In: *Clinical Procedures in Emergency Medicine.* Edited by Roberts JR, Hedges JR. Saunders, Philadelphia, 1985 (Fig. 47-8).
67. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (pp. 308, 311).
68. *Ibid.* (p. 312).
69. *Ibid.* (p. 313).
70. Sunderland S: The nerve lesion in the carpal tunnel syndrome, / *Neurol Neurosurg Psych* 39:615-626, 1976.

71. Takebe K, Vitti M, Basmajian JV: The functions of semispinalis capitis and splenius capitis muscles: An electromyographic study. *Ant Rec* 2 79:477-480, 1974.
72. Tichauer ER: Industrial engineering in the rehabilitation of the handicapped. *JIndEng* 29:96-104,1968 (p. 98 Fig. 2, p. 99 Table 2).
73. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2. Vol. 1. Macmillan, New York, 1919 (p. 272).
74. Travell J: Rapid relief of acute "stiff neck" by ethyl chloride spray, *f Am Med Worn Assoc* 4:89-95,1949.
75. Travell J: Pain mechanisms in connective tissue. In: *Connective Tissues, Transactions of the Second Conference, 1951.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1952 (pp. 119, 120).
76. Travell J: Referred pain from skeletal muscle: the pectoralis major syndrome of breast pain and soreness and the sternomastoid syndrome of headache and dizziness. *NY State J Med* 55:331-339, 1955.
77. Travell J, Bigelow NH: Role of somatic trigger areas in the patterns of hysteria. *Psychosom Med* 9:353-363, 1947 (p. 361, Figs. 7, 8).
78. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 22:425-434, 1952.
79. Walpin LA: Bedroom posture: the critical role of a unique pillow in relieving upper spine and shoulder girdle pain. *Arch Phys Med Rehabil* 58:507, 1977.
80. Wolfe F, Smythe HA, Yunus MB, *et al:* American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia: Report of the Multicenter Criteria Committee. *Arthritis Reumatol* 33:160-172, 1990.
81. Wolff HG: *Wolff's Headache and Other Head Pain.* Ed. 3. Oxford University Press, New York, 1972 (pp. 549, 554).

CHAPTER 17
Suboccipital Muscles: Recti Capitis Posteriores Major and Minor, Obliqui Inferior and Superior

HIGHLIGHTS: **REFERRED PAIN** from these muscles is "ghostly" in the poor definition of the deep head pain that radiates from the occiput toward the region of the orbit. However, these muscles are a common source of headache. **ANATOMICAL** attachment of three of these four muscles is to the occiput. The other connects to the spinous process of the axis, and to the transverse process of the atlas, affecting only rotation of the head. **FUNCTION** of these four deeply placed, bilateral suboccipital muscles is to help provide and control movements of rocking (nodding), rotation, and side bending the head. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are caused by a forward-head posture with a posteriorly rotated occiput, by abuse of the checkrein (control) function during sustained head flexion, by abuse of the extension function during sustained upward head tilt, and by sustained head rotation combined with tilt. The suboccipital muscles are prone to develop active TrPs as satellites of TrPs in other neck muscles, and from chilling of the neck when the muscles are fatigued. **PATIENT EXAMINATION** reveals restriction of head flexion, rotation, and/or side bending at the *top* of the neck post. **TRIGGER POINT EXAMINATION** reveals only tenderness to pressure on the deep suboccipital muscles through the overlying semispinalis capitis and trapezius. By direct palpation alone, it is rarely possible to distinguish TrPs in the individual suboccipital muscles. Which muscles are likely to be involved can be identified by specific movement restriction. **DIFFERENTIAL DIAGNOSIS** of occipitoatlantal, atlantoaxial, and C_2 articular dysfunctions requires specific examination techniques. **TRIGGER POINT RELEASE** is first applied to the other, more superficial, neck muscles that are likely to be responsible for activating satellite TrPs in the suboccipital group. Stretch and spray of the suboccipital muscles using an up-sweep pattern must relate to all fiber directions. Trigger point pressure release and deep massage are also effective. Associated suboccipital articular dysfunction should be treated. **TRIGGER POINT INJECTION** is generally not recommended. If injection is considered, full understanding of the relation of these muscles to the vertebral artery is essential. **CORRECTIVE ACTIONS** include correction of forward-head posture, the elimination of muscle overload, and the use of a home program that includes muscle stretch/lengthening.

1. REFERRED PAIN
(Fig. 17.1)

These paired suboccipital muscles (4 on each side) are the most deeply-placed muscles just below the base of the skull. Their trigger points (TrPs) are a common source of head pain that seems to penetrate inside the skull, but is difficult to localize. Patients are likely to describe the headache as hurting "all over," but on careful questioning, most describe the pain extending forward unilaterally to the occiput, to the eye

and the forehead, with a lack of clearly definable limits (Fig. 17.1). The pain of suboccipital muscles does not have the straight-through-the-head quality of the pain referred from the splenius cervicis muscle.

Hypertonic saline injected into the suboccipital muscles produced pain felt deeply in the head, and it was described as "headache."[14]

Travell[24] reported the management of a patient with an unusual referred pain pattern from suboccipital TrPs. The patient also showed evidence of conversion hysteria.

Rosomoff, *et al.*[19] found that 67.6% of 34 patients with chronic neck pain who had been given the waste-basket diagnosis of Chronic Intractable Benign Pain had TrPs or tender points in the suboccipital muscles when examined for them. Another study by Levoska[15] reported that, among 160 female office employees, 63% of the 72 subjects with disturbing neck symptoms had suboccipital tenderness to palpation. The tenderness could be related to TrP tenderness of suboccipital muscles or to cervical joint tenderness.

2. ANATOMY
(Fig. 17.2)

Three of these short suboccipital muscles connect the first two cervical vertebrae with the occipital bone,[4,13] and the fourth, the obliquus capitis inferior, connects the upper two cervical vertebrae with each other (Fig. 17.2).

Rectus Capitis Posterior Minor

This short, nearly vertical muscle converges ***below*** to attach to the tubercle on the posterior arch of the atlas. It spreads ***above*** to attach along the medial half of the inferior nuchal line of the occiput just above the foramen magnum.[4]

Rectus Capitis Posterior Major

The fibers of this muscle skip the atlas and attach ***below*** to the spinous process of the axis. ***Above*** they fan out, attaching to the lateral part of the inferior nuchal line of the occiput (and to the bone inferior to the line), lateral to the rectus capitis posterior minor.

Obliquus Capitis Superior

The fibers of this "oblique" muscle run almost vertically. They attach ***below*** to the transverse process of the atlas, and they

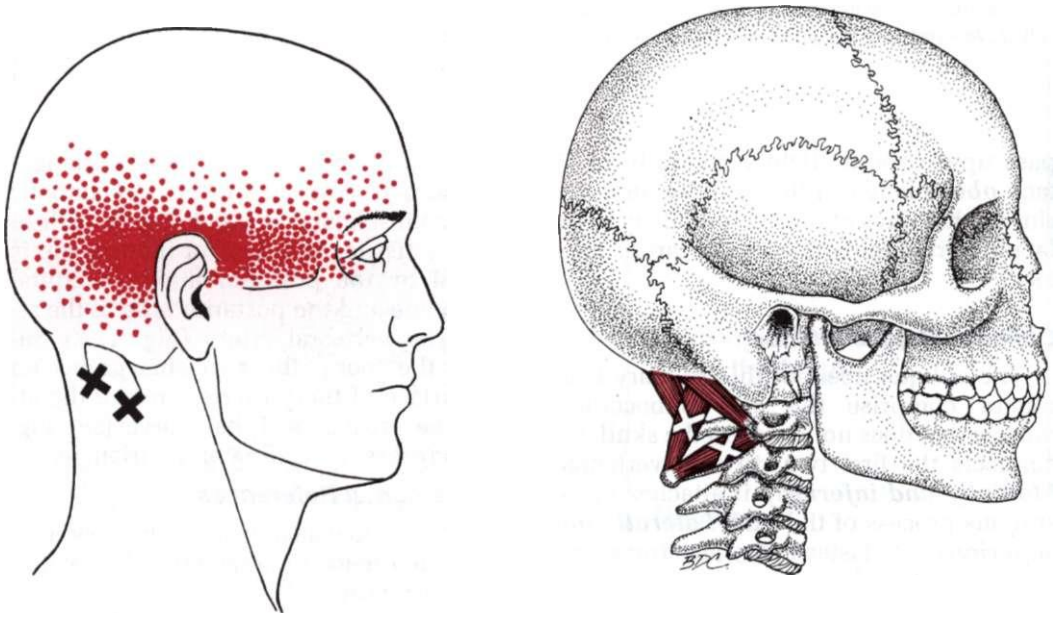


Figure 17.1. Referred pain pattern *(dark red)* of trigger points (Xs) in the right suboccipital muscles *(medium red).*

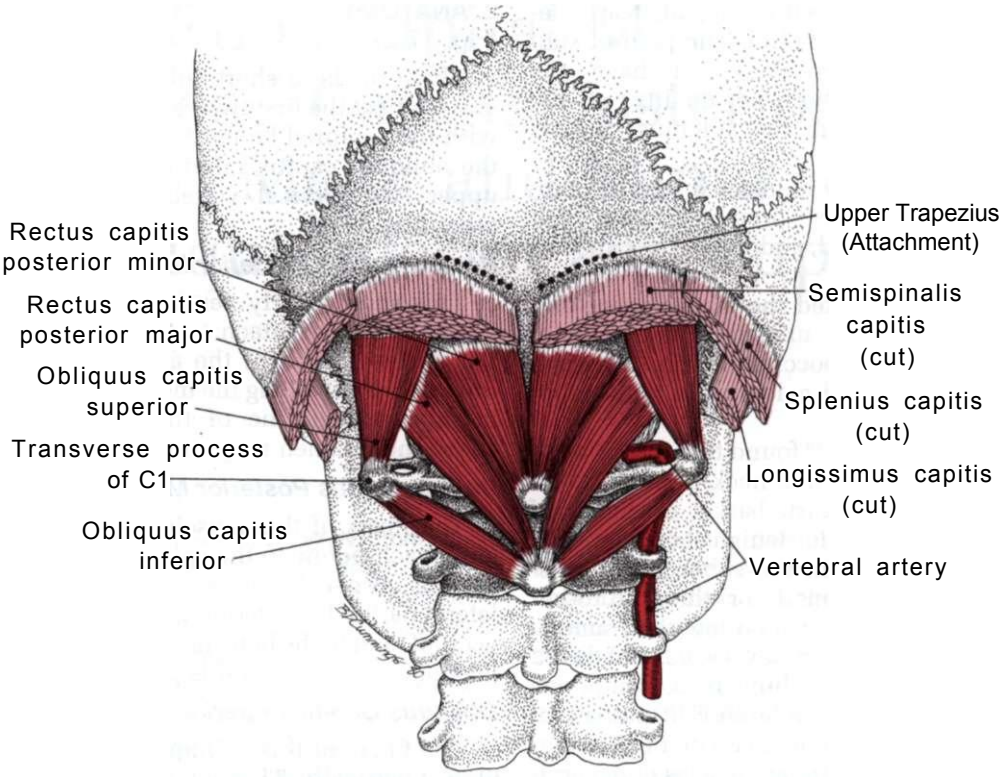


Figure 17.2. Attachments of the deep suboccipital muscles *(medium red)*. The most lateral three of these four muscles define the suboccipital triangle. This triangle surrounds the transverse portion of the vertebral artery *(dark red)* and should be avoided when injecting TrPs in the posterior neck muscles. The more superficial overlying muscles are *light red*. The *black dotted lines* indicate the location of attachment of the upper trapezius, which is the most superficial posterior neck muscle.

pass upward and slightly medially to attach ***above*** between the superior and inferior nuchal lines of the occiput, deep to the lateral part of the semispinalis capitis muscle.[14]

Obliquus Capitis Inferior

The oblique fibers of this primary head rotator comprise the only suboccipital muscle that does not fasten to the skull, but connects the first two cervical vertebrae. ***Medially and inferiorly*** it attaches to the spinous process of the axis. ***Laterally and superiorly*** it fastens to the transverse process of the atlas.[14]

Suboccipital Triangle

This triangle is bounded by three suboccipital muscles: the two obliqui and the rectus capitis posterior major. The triangular space is covered by the semispinalis capitis muscle and is filled largely with fibrofatty tissue. The floor of the triangle is formed by the posterior atlanto-occipital membrane and the posterior arch of the atlas.[4] The vertebral artery (Fig. 17.2) traverses the floor of this space in a groove on the surface of the posterior arch of the atlas. The greater occipital nerve *(see* Fig. 16.5) crosses the ceiling of the triangle.

Supplemental References

The suboccipital muscles have been well illustrated in posterior[1,5-7,10,22] and in side[23] views.

In 20 autopsy cases examined bilaterally, the greater occipital (part of the second cervical) nerve penetrated the inferior oblique muscle in 7.5% of cases.[2]

3. INNERVATION

The suboccipital muscles are supplied by branches of the dorsal primary division of the suboccipital (first cervical) nerve.

4. FUNCTION

(Fig. 17.3)

The first two joints at the top of the spinal column are highly specialized joints that provide head mobility. The articulation between the occiput and the first cervical vertebra (atlas) provides predominately flexion-extension (rocking or nodding) with only a small amount of lateral bending; the atlantoaxial articulation provides head rotation. The suboccipital muscles specifically control movement at these two joints and help to stabilize the head. This head movement on the spinal column is distinctly different from movement of the cervical spine itself.

The occipitoatlantal (OA or C_0-C_1 joints are spheroid articulations with the possible movements limited by very tight capsules.[17a] A current committee report,[17a] citing the most recent studies, concluded that the dominant movement is flexion-extension with a total range of approximately 22° to 24°. Lateral bending is 5° to 10°. The committee report[17a] cites recent investigators as showing some axial rotation at this level in both *in vitro* and *in vivo* studies.

A study of 150 healthy asymptomatic volunteers found that with increasing age from 20 to over 60 years there was a progressive decrease in all ranges of head and neck motion except for rotation from the position of full flexion.[17a] The suboccipital muscles that connect the atlas with the skull across this joint (the rectus capitis posterior minor and the obliquus capitis superior) function as extensors of the head.[4] The obliquus capitis superior has been reported to bend the head laterally[4,13] and clearly has the best leverage to do so (Fig. 17.3). Figure 17.3 graphically summarizes the actions of all four muscles.

The atlantoaxial (AA) joints provide 45°-47° of axial rotation to each side.[3,1a] The two suboccipital rotators (the inferior oblique muscle, which connects the axis to the atlas, and the rectus capitis posterior major, which connects the axis to the skull) rotate the head toward the side of muscular activity. Only the rectus capitis

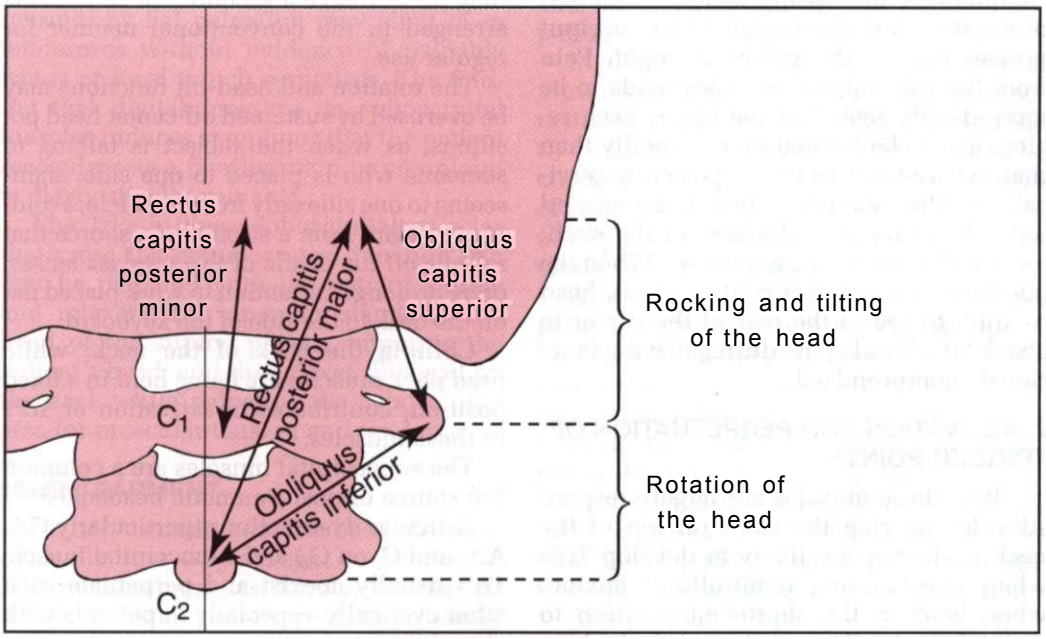


Figure 17.3. Graphic summary of the actions of the right suboccipital muscles.

posterior major provides both extension and rotation.[4] Refer also to Figure 17.6B which presents functional information regarding stretching in a more anatomical form.

5. FUNCTIONAL UNIT

For extension, the major synergist of the suboccipital muscles is the semispinalis capitis. Antagonists for extension are the longus capitis and rectus capitis anterior.

For rotation, the major synergists are the splenius capitis on the same side and the sternocleidomastoid on the opposite side. The major antagonists to the suboccipital muscles for rotation are the contralateral mates to the obliquus capitis inferior and the rectus capitis posterior major.

For the minimal lateral bending, the synergist is the rectus capitis lateralis; the antagonists are the contralateral counterparts of the obliquus capitis superior and the rectus capitis lateralis.

6. SYMPTOMS

Pain evoked by TrPs in the suboccipital muscles blurs indistinguishably with pain referred from the semispinalis. It is rare that the suboccipital muscles develop TrPs without associated involvement of other major posterior cervical muscles. Patients complain of distressing headache caused promptly when the weight of the occiput presses against the pillow at night. Pain from the suboccipital muscles tends to be more deeply seated in the upper neck region, and to be located more laterally than that experienced from the posterior cervical muscles. Patients often poke around with their fingers at the base of the skull, locating "a sore spot right there." When the obliquus capitis inferior is involved, head rotation to see to the rear of the car or to check "the blind spot" during driving is seriously compromised.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Since these muscles are largely responsible for moving the skull on top of the neck post, they are likely to develop TrPs when checkreining (controlling) flexion, when held in the shortened position to maintain extension while one is looking upward (e.g., when a person lies prone on the floor, propped up in the elbows to support the head while watching television), or when held in a shortened position while one is looking to the side for a prolonged period. Excessive anterior head positioning (forward-head posture) is often accompanied by a posteriorly-rotated occiput to accommodate the line of vision. This position activates and perpetuates TrPs in the suboccipital muscles and in other posterior cervical muscles.

If upward gaze is maintained by tilting the entire cervical spine, the cbeckrein function of the sternocleidomastoid muscles is abused. If, instead, a person rocks the head on top of the cervical spine, the suboccipital extensors are strained by prolonged contraction. The checkrein function of the suboccipital extensors is overloaded by sustained forward flexion of the head and neck which is often due to problems with the visual apparatus: maladjusted eyeglass frames, uncorrected nearsightedness, lenses with too short a focal length, and the use of trifocal lenses that require frequent or sustained fine adjustment of head position. People who use inverted eyeglasses to do fine overhead work, with their bifocals above rather than below, are in serious trouble with head positioning at other times if they do not have a second pair of bifocals arranged in the conventional manner for regular use.

The rotation and head-tilt functions may be overused by sustained off-center head positions, as when the subject is talking to someone who is placed to one side, sightseeing to one side only from a vehicle, avoiding the glare from a strong light source that reflects off the inside of the eyeglass lenses, or by prolonged attention to work placed flat on the desk to the side of the keyboard.

Chilling the back of the neck, while tired neck muscles are being held in a fixed position, contributes to activation of TrPs in these muscles.

The suboccipital muscles are a common TrP source of post-traumatic headache.[23]

Articular dysfunctions (particularly OA, AA, and C_2 on C_3) and suboccipital muscle TrPs usually coexist and perpetuate each other cyclically, especially in patients with chronic pain.

8. PATIENT EXAMINATION
(Figs. 17.4 and 17.5)

Myofascial TrPs in the suboccipital muscles can produce moderate restriction of the range of motion of the head. When the TrPs in the suboccipital muscles remain untreated, flexion (Fig. 17.4B) and side bending (Fig. 17.4C) are incomplete by the distance of one or two finger-breadths. Rotation may be reduced 30°. On examination for head mobility, the examiner feels increased resistance in the suboccipital region sooner than normal, causing early movement between successively lower cervical vertebrae.

With the patient in the seated position, it is difficult to isolate restriction of head rotation specifically due to the suboccipital muscle tightness. Figure 17.5 illustrates and describes how to examine for restriction of rotation of the head with the patient supine on a treatment table.

The patient's posture and movement should be observed, particularly for forward-head posture with a posteriorly-rotated occiput *(see* Chapter 5, Section C for assessment of anterior head positioning).

9. TRIGGER POINT EXAMINATION

Because of the intervening superficial musculature, examination of these deep muscles by flat palpation may elicit deep tenderness without evidence of palpable bands or local twitch responses. The finding that digital pressure on suboccipital muscles induces symptoms that the patient recognizes as a familiar pain or complaint is diagnostically valuable.

The TrPs in these suboccipital muscles that cross the craniocervical junction can best be palpated with the patient supine and relaxed. The operator stands at the head of the supine patient, supports the patient's head, and then flexes the head on the neck while palpating the suboccipital area for muscular tension and tenderness.

10. ENTRAPMENT

No nerve entrapment has been observed clinically that was thought to be due to TrPs in these muscles. Rarely, TrPs in the inferior oblique muscle potentially could entrap the greater occipital nerve. This entrapment was seen in one of the 7.5% of cases where the nerve penetrated the muscle.[2]

11. DIFFERENTIAL DIAGNOSIS

with contribution by
Roberta Shapiro, D.O.

The reader is referred to Chapter 16, Section 11 for an extensive discussion of arthritic disorders that can affect this region as well.

Patients with head and neck pain caused by suboccipital TrPs are commonly mistakenly diagnosed as having tension-type headache, cervicogenic headache,[12] occipital neuralgia,[8] or chronic intractable benign pain. Chronic intractable benign pain is defined as "non-neoplastic pain of greater than 6 months duration without objective physical findings and known nociceptive peripheral input."[19] One study of patients having the "diagnosis" of chronic intractable benign pain of the neck[19] reported TrPs or tender points in suboccipital muscles in 67.6% of 34 patients. The authors questioned the validity of the intractable benign pain diagnosis whenever the examination was based only on the usual routine physical examination and procedures and the examination did not include palpation of the muscles for relevant TrPs.

Myofascial TrPs in these suboccipital muscles usually coexist with articular (somatic) dysfunctions at the OA, AA, and the C_2 on C_3 levels. These areas all need to be checked and treated. Although *treatment* of joint dysfunction is not within the scope of this text, differential diagnosis must include joint dysfunction.

An OA dysfunction is assessed with the patient in the supine position. The operator's fingers are placed directly under the base of the occiput and the patient attempts an active chin-tuck (head retraction) in this position, or the operator can passively initiate a gliding type of chin-tuck motion at this level. If there is asymmetry at the OA joint, then it will look as though the patient's chin is turned away from the side of the articular dysfunction.

Atlantoaxial restrictions, which clinicians frequently overlook, are assessed

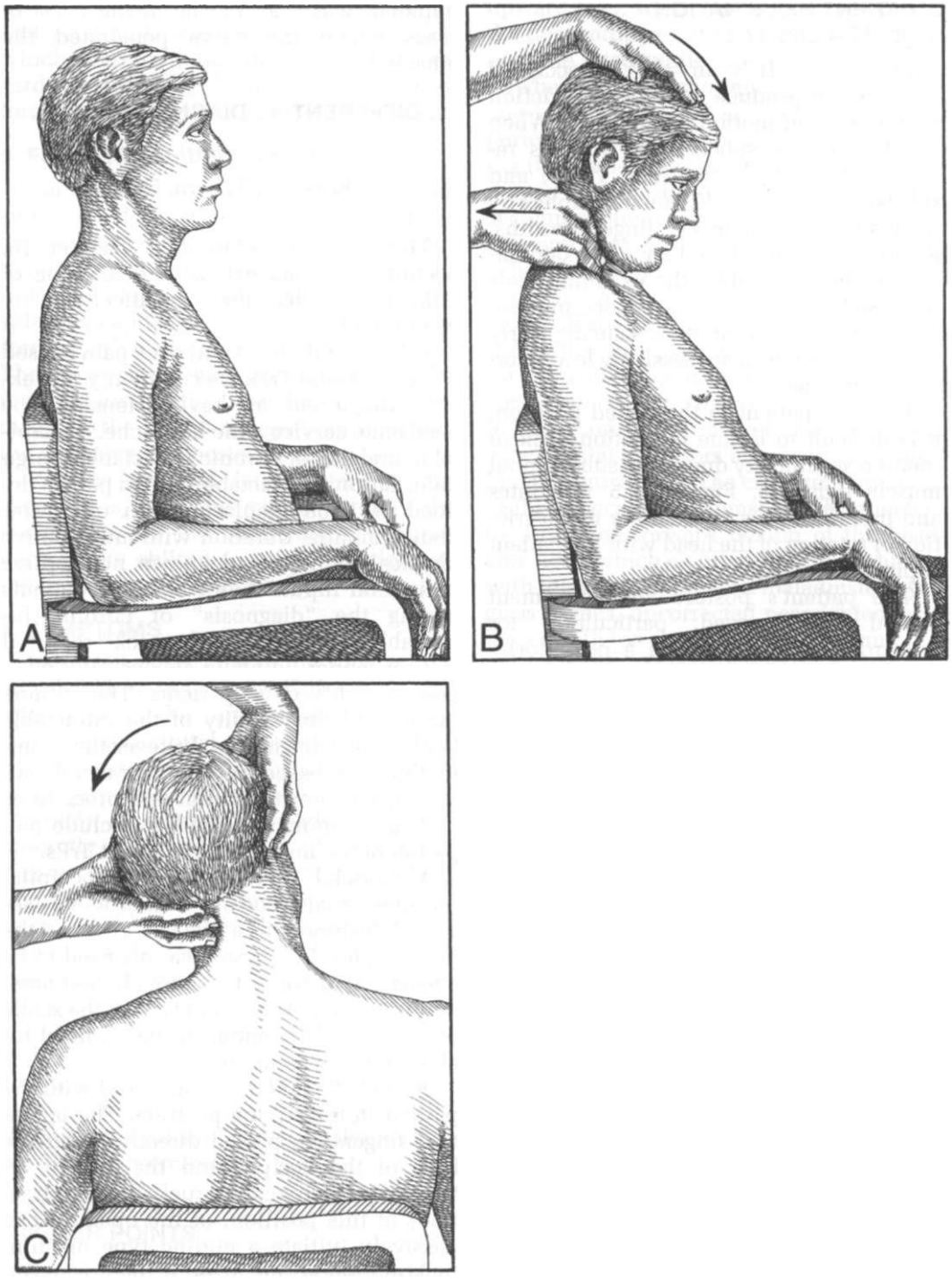


Figure 17.4. Tests for restricted motion of the head on top of the neck post due to taut suboccipital muscles. Restricted movement of the head on the neck is found by stabilizing the cervical spine and noting early motion between cervical vertebrae caudal to the atlantoaxial joint. **A,** resting seated position. **B,** testing of flexion requires that one hand monitor the upper cervical spine to detect separation of spinous processes below C_3. **C,** testing combined sidebending of the head and neck. The supine position is preferred for optimum muscle relaxation to distinguish between muscle tightness and joint restriction.

with the patient in the supine position and the head and neck positioned in full flexion to isolate the AA joint (Fig. 17.5). Then rotation is tested to each side. The end of range restriction from muscular involvement is softer and more pliable than the hard rigid end feel of joint restriction. The consistent finding with suboccipital muscle involvement is restricted rotation toward the side opposite the involved muscles (obliquus capitis inferior and possibly the rectus capitis posterior major) that are shortened because of TrPs. Crepitus is a very common finding in patients with osteoarthritis of the C_1-C_2 (AA) joint.[9] In these patients, pain is often partly due to suboccipital TrPs.

The C_2 on C_3 segment is an easy one to delineate; the axis is the most cephalad midline structure that one palpates since it is the first cervical vertebra that has a spinous process. This segment is isolated and examined in the supine position.

Patients with any of the dysfunctions described above present with severe pain that is associated with suboccipital TrPs and headache. Characteristically, the patient's head is tilted to one side and rotated to the other side.

Figure 17.5. Isolation and testing for restricted movement of the atlantoaxial (AA) articulation. The examiner positions the supine patient's cervical spine in full flexion to fix the lower cervical spine and isolate the atlantoaxial joint. The patient's head is supported completely against the examiner's body, and the examiner's hands apply only a rotation movement to the head, testing rotation to each side. This picture shows testing of rotation to the left; restriction could indicate trigger point tightness of right suboccipital muscles. The same position can be used for treatment utilizing postisometric relaxation techniques.

12. TRIGGER POINT RELEASE
(Figs. 17.6 and 17.7)

The head must be tilted on top of the neck post in specific directions to stretch those muscles that either extend the head, side bend it, or rotate it (Figs. 17.3 and 17.6B). In every case, prespray is applied to extend upward well above the hair line (Fig. 17.6A). With thick hair, the effectiveness of the vapocoolant spray may be increased by separating the hairs to make a track through them. A roll of bandage is handy to tie up long hair and lift it off the neck. A wig should be removed.

Prespray is followed by manual release of suboccipital trigger points (TrPs) as described and illustrated in Figure 17.7. The advantage of the operator holding the patient's head between the palms and fingers with the thumbs below the occiput is that, particularly during the patient's exhalation, it permits the operator to exert an upward traction force that gently releases compressive forces on the cervical articulations and suboccipital muscles. To lengthen the suboccipital muscles, upward traction is applied, and then the head is flexed on the cervical spine (as in nodding); the cervical spine itself is not flexed unless one wishes to release all of the posterior cervical musculature. The process is repeated until there is no further gain, or until full normal range of motion is reached. As described in the legend for Figure 17.7, augmented postisometric relaxation utilized in different directions of movement, including rotation, can release all of these muscles.

This release technique is similar to that shown in Figure 15.5B for the splenius capitis; however for the suboccipital release, upward traction is first applied to the occiput, and the stretch movement does not include the lower cervical region.

> Lewit[17] describes and illustrates basically the same procedure. The patient sits on the treatment table, with the therapist behind, and leans back against the therapist's chest. The therapist places both thumbs on the patient's occiput, with fingers placed on the malar bones from above. To take up the slack, the therapist

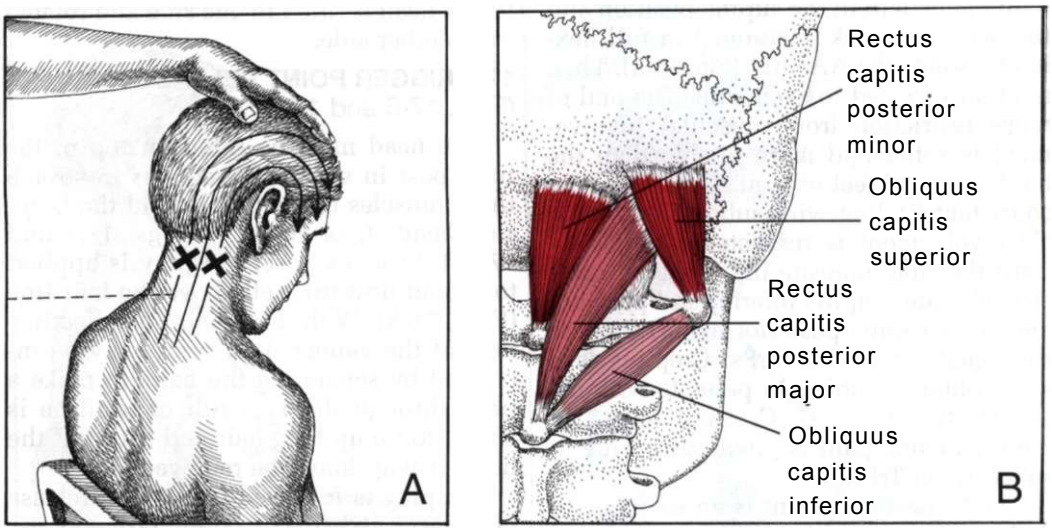


Figure 17.6. Prespray and Muscle Stretch. Prespray for stretch of suboccipital muscles and schematic identifying what muscles are released by various head movements. **A,** location of trigger points *(black* Xs) and the spray lines *(arrows)* for prespray of suboccipital muscles preparatory to stretch-release, which is shown in Figure 17.7. **B,** the *dark red* muscles (rectus capitis posterior minor and obliquus capitis superior) are stretched primarily by flexion of the head on the neck. The *medium red* muscle (rectus capitis posterior major) is released by the combination of flexion and rotation of the head to the opposite side. The *light red* muscle (obliquus capitis inferior) is stretched and released by rotation of the face to the opposite side. All four muscles can be released using a combined flexion and rotation of the head on the neck by turning the face toward the opposite side and then lowering the chin (nodding the head down).

> tilts the head slightly forward so as to draw the patient's chin in to the neck. The clinician then tells the patient to look up and breathe in slowly, while resisting the patient's tendency to raise the head; the patient is then told to look down and breathe out slowly, leaning back to allow the chin to drop ever closer to the throat *[without* bending the neck forward). This maneuver is repeated about three times.

The stretch-and-spray procedure should be followed by a hot pack that adequately covers the lower occiput and the posterior neck region. This is helpful, but may be difficult because the patient frequently does not want the hair to get wet, and the pack tends to slide down.

Trigger point pressure release can be used to inactivate TrPs in the suboccipital muscles as can deep massage. However, very *deep* massage is required to penetrate the overlying trapezius, semispinalis, and splenius capitis muscles.[21] The suboccipital triangle at the C_1 level, which is where the vertebral artery traverses horizontally, should be avoided if massage there causes any symptoms suggestive of brain ischemia.

Often OA, AA, and C_2 on C_3 dysfunctions coexist and must all be checked and treated. Manual techniques for gentle release of TrP tightness of muscles and for treatment of articular restriction are often similar enough to release both. One such technique is suboccipital decompression (traction), which is a relaxing, tension-release procedure for the upper cervical region. This procedure is performed with the patient in the supine position and with the examiner's fingertips placed in the suboccipital recess bilaterally. The patient's head is supported on the pads of the examiner's fingers. Initially, pressure is applied anteriorly to induce regional extension at the OA, AA and C_2 articulations. When relax-

ation of the suboccipital muscles is detected, the examiner applies traction in a cephalad direction with the fingertips, while slightly spreading the fingers apart directly against the base of the occiput. This upward traction force at the occiput releases compressive forces on the cervical articulations, induces regional flexion in a chin-tuck position, and helps to release tension in the suboccipital muscles.

If normal joint motion has been restored and the TrPs are resistant to noninvasive methods, it may be necessary to consider injection with full precautions.

It is important to correct forward-head posture when it exists and to teach maintenance of good posture (see Chapter 41, Section C)

13. TRIGGER POINT INJECTION

Before injection is considered, noninvasive treatment should be tried. Repeated applications of stretch and spray with deep massage generally are effective in eliminating suboccipital TrP irritability. With appropriate precautions, daily application of up to 1.5 watts/cm^2 of pulsed ultrasound using the moving head technique can be

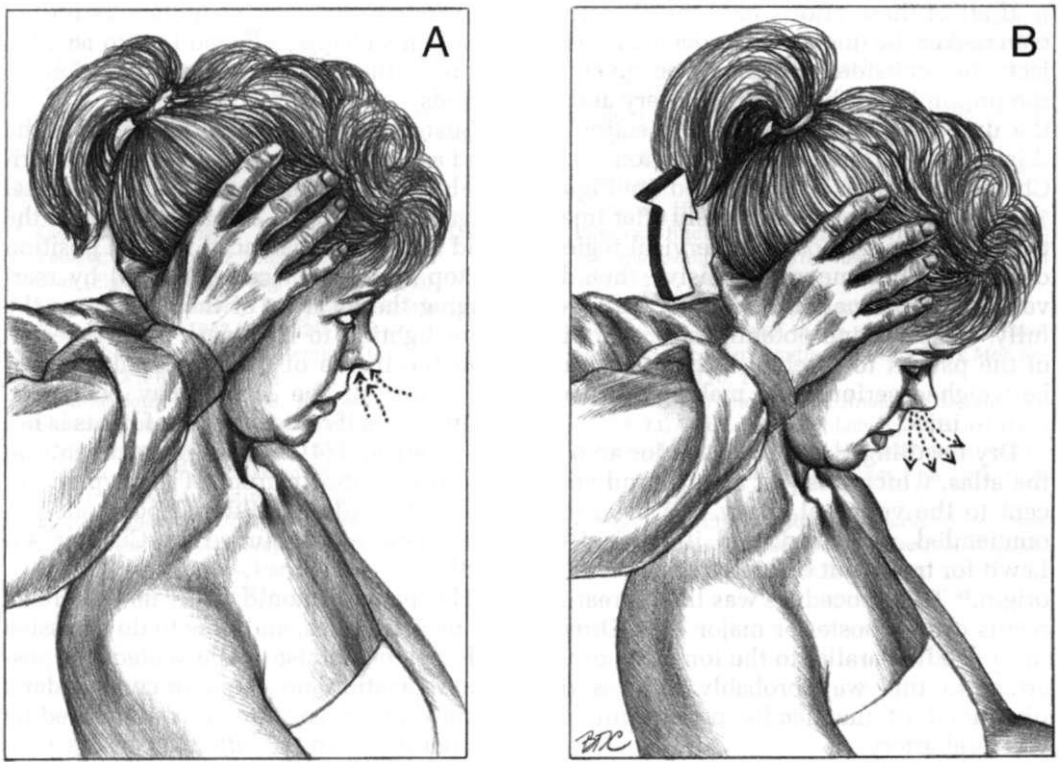


Figure 17.7. Release of suboccipital muscles using augmented postisometric relaxation. This approach can be used following prespray, as shown in Figure **17.6A.** It can be used also without operator assistance as a self-stretch for a home program. **A,** operator flexes the patient's head gently to take up any slack in the suboccipital muscles; then, while the patient looks up and slowly takes in a deep breath, the therapist holds the head position and gently resists the patient's tendency to extend. **B,** the patient then slowly exhales fully, looks down, and allows the head to flex, relaxing the posterior muscles, while the operator exerts upward traction at the occiput (releasing compressive forces on the cervical articulations) and takes up the slack that develops. With the same hand placement and procedure, additional release of the more diagonal muscles can be obtained (similar also to Fig. **15.5B** for the splenius). The clinician should first apply upward traction at the occiput, and then rotate the patient's head toward the opposite side (for the obliquus capitis inferior); rotate contralaterally *and* flex the head to release the rectus capitis posterior major.

helpful, but may require 2 weeks of treatment to produce results. Upper cervical joint dysfunction should be treated

One can also approach releasing these suboccipital TrPs by meticulously inactivating all other active TrPs (by injection if desired) in the posterior cervical muscles. Sometimes, a TrP in another muscle (for example, in the splenius capitis or the semispinalis) is acting as a key TrP that produces satellite TrPs in the suboccipital muscles. Inactivating these key posterior cervical TrPs often also inactivates their satellite suboccipital TrPs without further treatment of the satellites. Hong[11] has described and illustrated this principle for numerous other muscles in this region.

If all of these efforts fail, and the clinician makes the questionable decision to inject, due consideration must be given to the proximity of the vertebral artery and to the untoward results of local injection in this region, as described in Section 13 of Chapter 16, and as illustrated in Figure 16.9. For example, immediately after injection in the upper posterior cervical region, one patient became unresponsive, then developed grand mal seizures, but recovered fully.[20] The age and potential susceptibility of the patient to cerebral ischemia should be weighed seriously in making the decision to inject.

Dry needling along the posterior arch of the atlas, which lies just caudad and adjacent to the vertebral artery, has been recommended, described, and illustrated by Lewit for treatment of headache of cervical origin.[16] This procedure was likely treating rectus capitis posterior major TrPs. Directing a needle parallel to the long axis of the artery in this way probably reduces the likelihood of the needle penetrating the vertebral artery.

Rachlin[18] described and illustrated injection of the obliquus capitis superior muscle emphasizing the importance of directing the needle toward the occiput. This would require remarkably precise localization of needle placement.

14. CORRECTIVE ACTIONS

For patients who develop active TrPs in the suboccipital muscles, it is critically important to keep this part of the neck warm by covering the neck in some way, such as by wearing a turtle-neck sweater indoors and a hood that covers the head *and* neck outdoors. Nightwear rarely provides a collar high enough to cover the suboccipital area adequately; therefore, the patient should wear something like an old-fashioned nightcap, a soft hooded jacket, or drape a scarf in such a way as to protect the suboccipital skin from cooling.

Sustained upward gaze with the head tilted up must be avoided by revising the individual's activity to whatever extent is necessary. In a case seen by Dr. Travell, a stage director learned to direct the performance from farther back in the theater, instead of from the front row where he had been below the level of the actors on the stage. This change allowed him to face the actors without looking up for prolonged periods.

Sustained and strained positions of the head are reduced by (1) avoiding use of trifocals; (2) using lenses with adequate focal length for the task at hand to allow the head to rest in a balanced upright position on top of the cervical spine; (3) by rearranging the location of the patient, or the room lighting to eliminate glare reflected from the inside of the lenses (alternately, the inside of the lenses may be coated against glare if repositioning of lights is not practical); and (4) by placing documents on a vertical stand in front of the typist, not flat to one side. Additional postural considerations are included in Chapter 41, Section C.

The patient should learn how to relax the neck muscles, and how to do a passive self-stretch exercise while *seated* (for postural relaxation) on a stool or chair under a warm shower. The stretch is performed by the patient doing a sei/-assist of his own nodding motion (flexion of the head on the neck), similar to the stretch shown in Figure 17.7B, but with the *patient's* fingers under the occiput. The patient uses his own fingers under the occiput to exert upward traction prior to directing the movement of the head. A comparable self stretch for the suboccipital muscles is described and illustrated by Lewit.[17] A series of passive stretches should be applied separately in unidirectional movements (no head

rolling) with successive degrees of head rotation to fully stretch *all* of the suboccipital muscles. Passive stretching should be followed by full active range of motion, contracting and stretching muscles in both the agonist and antagonist directions. This cycle of movements is repeated several times, slowly without jerking.

Patients with suboccipital TrPs usually find that a cervical collar is more annoying and irritating than helpful, due to its direct pressure on these muscles.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991(p. 241, Fig. 4.58).

1a. Bogduk N. Biomechanics of the cervical spine. In: *Physical Therapy of the Cervical and Thoracic Spine.* Ed. 2. Edited by Grant R. New York: Churchill Livingstone, 1994.

2. Bovim G, Bonamico L, Fredriksen TA, *et al.:* Topographic variations in the peripheral course of the greater occipital nerve: autopsy study with clinical correlations. Spine **J6**(4j:475-478, 1991.

3. Cailliet R: *Soft Tissue Pain and Disability.* F.A. Davis, Philadelphia, 1977 (pp. 107-110).

4. Clemente CD: Gray's Anatomy., Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 473- 475).

5. *Ibid.* (Fig. 6-22, p. 474; Fig. 12-28, p. 1194).

6. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 526, 529).

7. Eisler R: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (Fig. 63, p. 433).

8. Graff-Radford S, Jaeger B, Reeves JL: Myofascial pain may present clinically as occipital neuralgia. *Neurosurgery* 19(4j.-610-613, 1986.

9. Halla JT, Hardin JG: Atlantoaxial (C1-C2) facet joint osteoarthritis: a distinctive clinical syndrome. *Arthritis Rheum 30(5):577-582,* 1987.

10. Hollinshead WH: *Anatomy for Surgeons.* Ed. 3. Vol. 1, *The Head and Neck.* Harper & Row, Hagerstown, 1982 (Fig. 1-51, pp. 69-71).

11. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. / *Musculoske Pain 2(1* J.-29-59, 1994.

12. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 9:157-164, 1989.

13. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 202).

14. Kellgren JH: Observations on referred pain arising from muscles. *Clin Sci* 3.175-190, 1938 (pp. 180, 210, 212).

15. Levoska S: Manual palpation and pain threshold in female office employees with and without neck-shoulder symptoms. *Clin J Pain* 9:236-241, 1993.

16. Lewit K: The needle effect in the relief of myofascial pain. *Pain* 6:83-90, 1979.

17. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991.

17a. Panjabi M, Dvofdk J, Sandler A, *et al.* Cervical spine kinematics and clinical instability. In: *The Cervical Spine.* Ed. 3. Philadelphia, Lippincott-Raven, 1998.

18. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360.

19. Rosomoff HL, Fishbain DA, Goldberg M, *et al.:* Physical findings in patients with chronic intractable benign pain of the neck and/or back. *Pain* 37:279-287, 1989.

20. Rubin D: Personal communication, 1979.

21. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Rehabil* 62:107-110, 1981.

22. Spalteholz W: *Handatlas derAnatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 314).

23. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (pp. 278, 279).

24. Travell J, Bigelow NH: Role of somatic trigger areas in the patterns of hysteria. *Psychosom Med* 9:353-363, 1947 (Case 3, pp. 360, 361).

PART 3
UPPER BACK, SHOULDER AND ARM PAIN

CHAPTER 18
Overview of the Upper Back, Shoulder, and Arm Region

This third part of the *Trigger Point Manual* includes muscles of the upper back, shoulder, and arm that refer pain into the torso and upper limb. It includes the scalene and levator scapulae neck muscles, most of the muscles that attach to the scapula, all the muscles that cross the glenohumeral joint, and the anconeus, which is included as an extension of the triceps brachii muscle. The trapezius muscle was included in Part 2.

This chapter is divided into Section A: Pain and Muscle Guide, and Section B: Diagnostic Considerations and Treatment Techniques. The pain guide of Section A lists the muscles that may be responsible for pain in the areas shown in Figure 18.1. The muscles most likely to refer pain to each specific area of the body are listed below under the name of that area. One uses this chart by first locating the name of the area of the body that hurts and by then looking under that heading for all the muscles that are likely to refer pain to that area. Then, reference should be made to the individual muscle chapters; the number for each chapter follows in parenthesis.

In a general way, the muscles are listed in the order of the frequency in which they are likely to cause pain in that area. This order is only an approximation; the selection process by which patients reach an examiner greatly influences which of their muscles are most likely to be involved. **Boldface** type indicates that the muscle refers an essential pain pattern to that pain area, meaning that the pattern is present in nearly every patient when the trigger point (TrP) is active. Normal type indicates that the muscle refers a spillover pattern to that pain area (pain that some, but not all, patients experience).

Section B presents an overview of considerations that apply to more than one of the muscles included in this part of the *Trigger Point Manual.* These comments are not focused on any one muscle, but rather on how to recognize and deal with multiple-muscle involvement and the interaction of muscles with related conditions.

SECTION A

PAIN AND MUSCLE GUIDE

UPPER-THORACIC BACK PAIN

Scaleni (20)
Levator scapulae (19)
Supraspinatus (21)
Trapezius (TrP$_2$ and TrP$_3$)(6)
Trapezius (TrP$_8$) (6)
Multifidi (48)
Rhomboidei (27)
Splenius cervicis (15)
Triceps brachii (TrP$_1$) (32)
Biceps brachii (30)

BACK-OF-SHOULDER PAIN

Deltoid (28)
Levator scapulae (19)
Scaleni (20)
Supraspinatus (21)
Teres major (25)
Teres minor (23)
Subscapularis (26)
Serratus posterior superior (47)
Latissimus dorsi (24)
Triceps brachii (TrP$_1$) (32)
Trapezius (TrP$_3$ and TrP$_6$) (6)
Iliocostalis thoracis (48)

BACK-OF-ARM PAIN

Scaleni (20)
Triceps brachii (TrP$_1$ and TrP$_3$) (32)
Deltoid (28)
Subscapularis (26)
Supraspinatus (21)
Teres major (25)
Teres minor (23)
Latissimus dorsi (24)
Serratus posterior superior (47)
Coracobrachialis (29)
Scalenus minimus (20)

MID-THORACIC BACK PAIN

Scaleni (20)
Latissimus dorsi (24)
Levator scapulae (19)
Iliocostalis thoracis (48)
Multifidi (48)
Rhomboidei (27)
Serratus posterior superior (47)
Infraspinatus (22)
Trapezius (TrP$_4$) (6)
Trapezius (TrP$_8$) (6)
Serratus anterior (46)

FRONT-OF-SHOULDER PAIN

Infraspinatus (22)
Deltoid (28)
Scaleni (20)
Supraspinatus (21)
Pectoralis major (42)
Pectoralis minor (43)
Biceps brachii (30)
Coracobrachialis (29)
Sternalis (44)
Subclavius (42)
Latissimus dorsi (24)

FRONT-OF-ARM PAIN

Scaleni (20)
Infraspinatus (22)
Biceps brachii (30)
Brachialis (31)
Triceps brachii (TrP$_8$) (32)
Supraspinatus (21)
Deltoid (28)
Sternalis (44)
Scalenus minimus (20)
Subclavius (42)

SECTION B

with contributions by
Roberta Shapiro, D.O.

DIAGNOSTIC CONSIDERATIONS

Thoracic Outlet Syndrome (TOS)

Chapter 20 provides a major review and analysis of the thoracic outlet syndrome under Section 11, **Differential Diagnosis.** This syndrome can involve numerous muscles that are covered in this part of the *Trigger Point Manual.*

Myofascial Pseudothoracic Outlet Syndrome

The dictionary definition of thoracic outlet syndrome is "compression of brachial plexus and subclavian artery by attached muscles in the region of the first rib and the clavicle."[2] This makes it, by definition, an entrapment syndrome. Myofascial TrPs in the scalene, pectoralis minor, and subclavius muscles can produce a true (entrapment) thoracic outlet syndrome. The contributor to this section has identified a myofascial variant that mimics the thoracic outlet syndrome. This **pseudothoracic out-**

let syndrome typically involves a quadrad of muscles: the pectoralis major, latissimus dorsi, teres major, and subscapularis. When at least three of these muscles have active TrPs, the patient presents with the myofascial pseudothoracic outlet syndrome (pseudo-TOS). The syndrome can mimic many diagnoses in addition to the thoracic outlet syndrome, including a multiple-level cervical radiculopathy, various types of bursitis, and tendinitis in the shoulder region. Routinely, the frustrated patient who has been referred with one of these misleading diagnoses has not been successfully treated because the muscular source of the patient's pain was not identified. All of these muscles are relatively strong medial rotators. The pseudo-TOS can be seen typically in patients who have suffered cerebrovascular accidents (CVA) or "strokes," as they tend to have selective spasticity of medial rotators and adductors and therefore tightness in these same four muscles, which is especially critical in the subscapularis. Because of the severe restriction of shoulder mobility caused by the

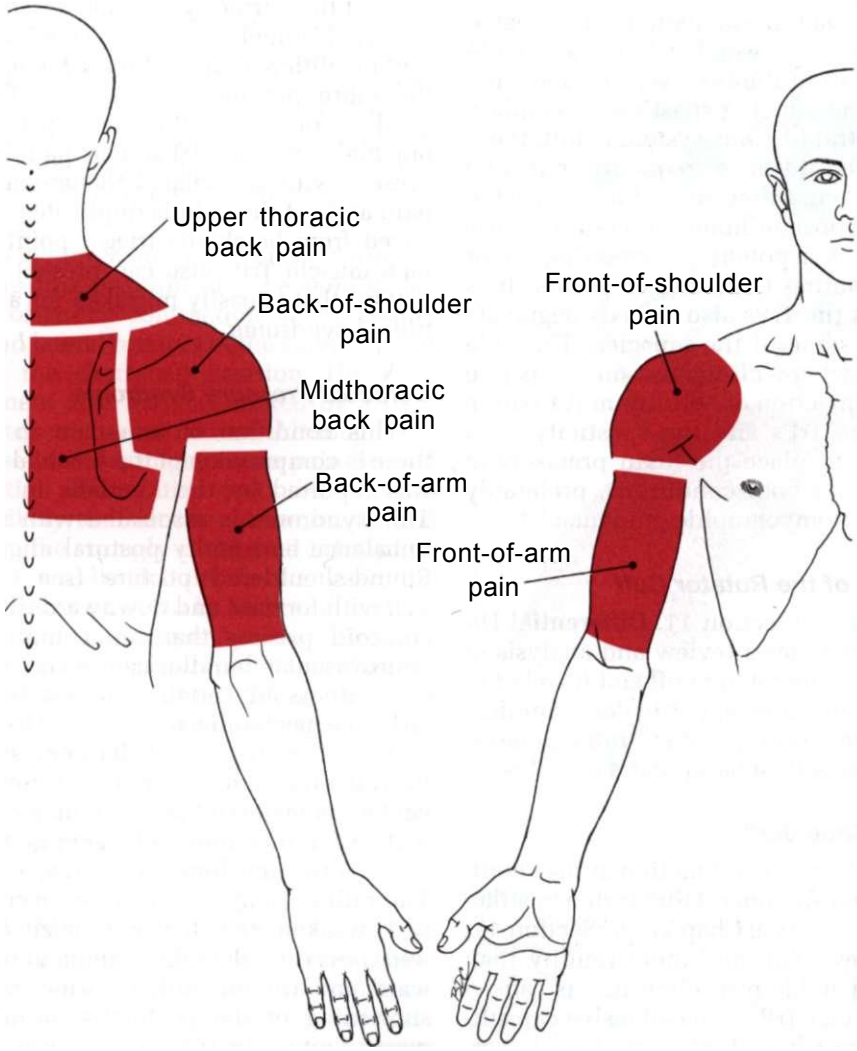


Figure 18.1. The designated areas *(red)* within the upper back, shoulder, and arm that may encompass pain referred there by myofascial trigger points. See text for listing of muscles that may refer pain to each area.

TrPs in this group of muscles, the patient is often identified as having a "frozen shoulder," which is discussed in more detail in Chapter 26, Subscapularis.

Patients who have suffered a cerebrovascular accident are often positioned in medial rotation and adduction of the glenohumeral joint, which tends to activate TrPs in these four muscles. The increased tension caused by the combination of TrPs and spasticity in these four muscles can cause the shoulder subluxation typical of these patients. In addition to causing pain, the subluxation and TrP activity facilitate the development of reflex sympathetic dystrophy. It is vital therefore, that these patients be treated early with myofascial TrP release techniques to all of the involved muscles. Because of the ongoing spasticity secondary to the central nervous system insult, these patients should have *frequently repeated* treatments, and they should be instructed in an appropriate home program because spasticity is a potent perpetuating factor for reactivating their trigger points. It is likely that the TrPs also reflexly aggravate spasm in some of the muscles. This is a situation where clinicians may wish to consider injection of botulinum A toxin to control the TrPs and the spasticity. It is important to place the toxin precisely at endplates for both conditions, preferably using electromyographic guidance.[1,3]

Problems of the Rotator Cuff

Chapter 21 (Section 11, **Differential Diagnosis)** includes a review and analysis of problems of the rotator cuff and its relation to muscle imbalance, particularly applicable to the supraspinatus, infraspinatus, teres minor, and subscapularis muscles.

"Frozen Shoulder"

Painfully restricted motion at the shoulder ("frozen shoulder") that is due to adhesive capsulitis *(see* Chapter 26, Section 11) exhibits less pain and more rigidity than does comparable restriction that is caused by myofascial TrPs. True adhesive capsulitis often requires short-term steroid therapy, which may be given orally.[4,5] However, myofascial TrPs often respond well to noninvasive therapy. Multiple TrPs in the rotator cuff muscles, especially in the *subscapularis* muscle, can mimic the symptoms of adhesive capsulitis. When a patient has not responded well to treatment for the diagnosis of adhesive capsulitis, the clinician needs to consider TrP sources for the patient's symptoms. When TrPs are producing the symptoms, appropriate TrP therapy (without steroids) should be initiated. It is not unusual for both conditions to coexist. Both need treatment.

Carpal Tunnel Syndrome

Some TrP sources can mimic the symptoms of the currently overdiagnosed entity of carpal tunnel syndrome. This is an important differential diagnosis for many of the more proximal upper limb TrPs. A good example is the pain pattern of brachialis muscle TrPs. The patient may present with an isolated thenar-eminence pain and yet the pain is duplicated and referred from brachialis trigger points. Scalene muscle TrPs also can present a pain pattern that is easily mistaken for a carpal tunnel syndrome.

Coracoid Pressure Syndrome

This condition of arm pain in which there is compression of the brachial plexus was reported by the Kendalls in 1942.[1a] This syndrome is associated with muscle imbalance and faulty postural alignment. Round-shouldered posture (see Chapter 41), with forward and downward tilt of the coracoid process that can compress the neurovascular bundle, can occur because of *tightness* in certain muscles (particularly the pectoralis minor, the latissimus dorsi indirectly through its depression of the humerus, and the sternal portion of the pectoralis major). One of the major factors that can induce muscle tension and shortening is the development of trigger points. The pull of tight pectorals can overstretch and weaken the lower trapezius. This *weakness* can allow the scapula to ride upward and tilt forward, favoring adaptive shortening of the pectoralis minor (and perpetuating any TrPs in it). Trigger points also can inhibit muscular activity (for example, in the lower trapezius). Clinicians

need to be aware that TrPs can produce dysfunction and not only pain.

Screening Test

The Mouth Wrap-around Test is useful to screen for involved shoulder-girdle muscles. This test requires full active abduction and lateral rotation of the arm at the glenohumeral joint. It also requires normal scapular mobility. If the clinician looks closely at *how* the subject raises the arm, scapulohumeral rhythm can also be assessed. The patient does this test (Fig. 18.2) by bringing the hand and forearm *behind* (not above) the head and sliding the hand as far forward as possible trying to cover the mouth. The head should be turned no more than 45°, and should not be tilted. Normally, the fingertips can cover the mouth nearly to the midline in most persons, just to the corner of the mouth if the subject has short upper arms, and over the entire mouth with hypermobile joints.

Moving the hand to the end position or holding this position may be *painful* because of strong contraction of the abductors and lateral rotators of the shoulder that are in the shortened position. However, movement also may be *limited* by a tight adductor or medial rotator. Although any of these muscles might cause pain-limited restriction during this test, the muscles most likely to limit the movement in this way are the strongly contracted infraspinatus and middle deltoid. In this case, the pain is most likely to be in the immediate vicinity of the trigger points. The test movement also passively stretches the subscapularis muscle and if that muscle has TrP tightness it is likely to refer pain behind the shoulder and to the wrist. The TrPs of a tight latissimus dorsi muscle would cause pain at the end of its extensive range of motion only if no other muscle were restricting the movement.

TREATMENT TECHNIQUES

Treatment techniques for specific muscles are presented in the individual muscle chapters of this volume. Presented here are two treatment techniques, each of which can release a number of muscles in the upper thoracic and interscapular regions.

Scapular Mobilization and Interscapular Muscle Release

Figure 18.3 illustrates and describes a scapular mobilization technique that directly releases tight interscapular musculature. It releases the middle and lower trapezius, the rhomboid muscles, and abnormal tension of associated fascial tissues. In addition, the technique helps to release other muscles that attach to the

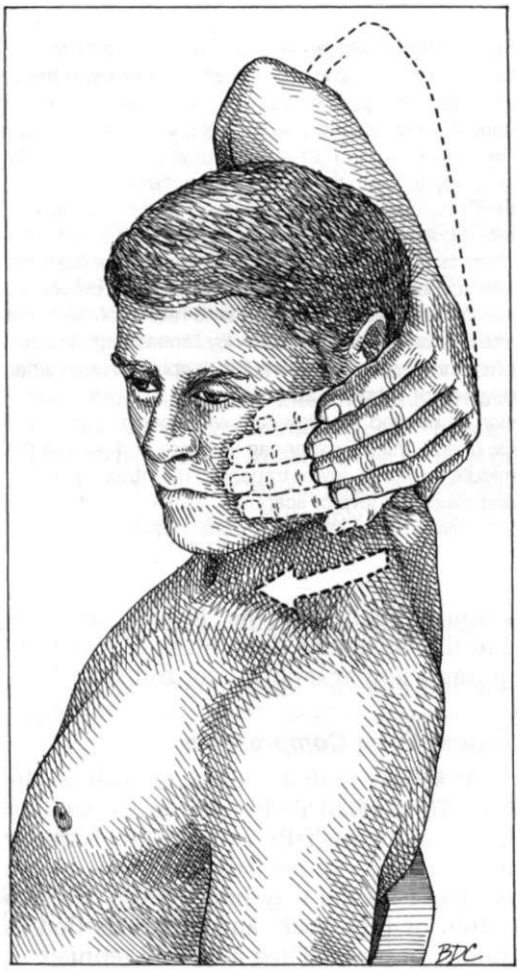


Figure 18.2. Screening test (Mouth Wrap-around Test) of abduction and lateral rotation of the arm. The *fully rendered hand* shows restricted range of motion. The *dotted white arrow* and *dotted outline* show the additional reach that would have been normal for this particular subject who had congenially short upper arms. Most persons can normally cover half the mouth; individuals with hypermobile joints normally cover the entire mouth with the hand.

Figure 18.3. Interscapular muscle release and scapular mobilization technique. Patient is sidelying with the affected side up. The examiner stands in front of the patient, reaches over the patient's shoulder to grasp the upper portion of the vertebral border of the scapula, and with the other arm reaches under the patient's humerus to grasp the lower portion of the vertebral border of the scapula. The examiner then slowly abducts the scapula to mobilize it and to release the interscapular muscles and fasciae. This procedure can be followed by postisometric relaxation for more effective and specific release. By "fine tuning" the mobilization (that is, by moving the scapula through small degrees of rotation, abduction, elevation, or depression as needed for alignment with specific tight muscle fibers), the examiner can lengthen and release the middle trapezius, lower trapezius, rhomboideus major and minor, the levator scapulae, and also can facilitate full release of the latissimus dorsi muscle.

scapula, including the levator scapulae and the latissimus dorsi muscle, which requires scapular mobility for full stretch.

Trigger Point Compression

A self-treatment technique called the Cold Tennis-ball Technique can be used for inactivation of TrPs in the interscapular muscles. Two tennis balls are needed. One is placed all the way into the toe end of a cotton or tube sock, and a knot is tied to hold it in place. Then a second tennis ball is placed in the sock and a knot is tied on the other side of this second ball (Fig. 18.4). The sock is then placed in the freezer.

Once a day, or when the patient has discomfort in the affected interscapular muscles, the patient removes the cold tennis balls from the freezer and lies supine on them, initially placing the tennis balls below the level of the scapula and on each side of the vertebral column. The patient then slides downward so that the tennis balls in effect roll up over the interscapular muscles. This movement produces a trigger point compression and/or a self-massage effect that is completely under the patient's control. Whenever the patient hits a "hot spot" or a painful trigger point, he or she can maintain that position and control the pressure by starting gently and gradually increasing body-weight pressure on the tennis balls until that TrP releases. The procedure should be continued to locate any additional TrPs. One can use this technique with or without freezing the tennis balls. The patient should try it each way to see which is more effective. Most patients prefer the cold application because it seems to be more effective. A well-controlled research study to explore why patient reactions differ would be helpful.

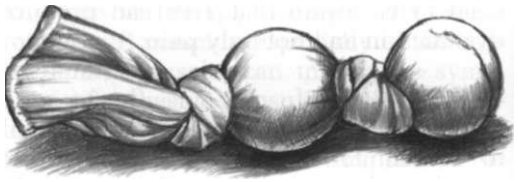


Figure 18.4. Sock enclosing two tennis balls that can be chilled and used for self-treatment of interscapular muscles. Refer to text for details on the Cold Tennis-ball Technique.

REFERENCES

1. Hubbard DR. Chronic and recurrent muscle pain: pathophysiology and treatment, and review of pharmacologic studies. *JMusculoske Pain* 1996;4 (1/2):123-143.

1a. Kendall FP, McCreary EK, Provance PE: *Muscles: Testing and Function.* Ed. 4. Baltimore: Williams & Wilkins, 1993 (p.343).

2. McDonough JT, ed. *Stedman's Concise Medical Dictionary,* 2nd ed. Baltimore: Williams & Wilkins, 1994:995.

3. Simons DC Clinical and etiological update of myofascial pain from trigger points. *J Musculoske Pain* 1996;4 (1/2):97-125.

4. Travell J, Rinzler SH. Pain syndromes of the chest muscles: Resemblance to effort angina and myocardial infarction, and relief by local block. *Can Med Assoc J* 1948;59:333-338.

5. Webber TD. Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J Am Osteopath Assoc* 1973;72:697-710.

CHAPTER 19
Levator Scapulae Muscle

HIGHLIGHTS: When a patient suffers from a "stiff neck" (markedly limited rotation), trigger points (TrPs) in the levator scapulae muscle are frequently responsible. **REFERRED PAIN** from the levator scapulae concentrates in the angle of the neck and along the vertebral border of the scapula. It may project to an area posterior to the shoulder joint. **ANATOMY:** this muscle attaches above to the transverse processes of the first four cervical vertebrae, and attaches below to the region of the superior angle of the scapula. **FUNCTION:** The levator scapulae rotates the scapula (glenoid fossa) downward and elevates the scapula. With the scapula fixed, this muscle assists rotation of the neck to the same side and both muscles together can checkrein (control) flexion of the neck. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are most likely to occur as a result of keeping the shoulder elevated and the muscle in a sustained shortened position, particularly when the muscle is fatigued and exposed to cold. **PATIENT EXAMINATION** reveals primarily restriction of neck rotation. **TRIGGER POINT EXAMINATION** by palpation of the muscle as it emerges from beneath the trapezius at the angle of the neck discloses its most important, central TrP that may be difficult to locate. Palpation just above the superior angle of the scapula often locates a second region of marked tenderness, the attachment TrP. **DIFFERENTIAL DIAGNOSIS** includes the scapulocostal syndrome, zygapophysial joint pain, and bursitis. **TRIGGER POINT RELEASE** may be performed in two steps: application of vapocoolant spray or icing over the muscle and painful areas while taking up slack in the muscle. This step is followed immediately with bimanual release of tightness in the vertical and diagonal muscle fibers. **TRIGGER POINT INJECTION** requires careful positioning of the patient and often needling at both the central and attachment TrP regions to be successful. **CORRECTIVE ACTIONS** call for relief of muscular strain and for regular passive stretching of the muscle at home, preferably while the patient is seated under a warm shower.

1. REFERRED PAIN
(Fig. 19.1)

Myofascial pain due to trigger points (TrPs) is one of the most important causes of neck pain[21] or neck and shoulder pain,[15] and the levator scapulae is one of the most commonly involved shoulder-girdle muscles. In a study of shoulder-girdle muscles in 200 normal young adults, Sola *et al.*[33] found latent TrPs in more levator scapulae muscles (20% of subjects) than in any other muscle except the upper trapezius. In a clinical study of active TrPs,[32] the levator scapulae was the most commonly involved shoulder-girdle muscle.

Both trigger areas shown in Figure 19.1 project pain to the essential reference zone at the angle of the neck,[5, 34, 36] with a spillover zone along the vertebral border of the scapula,[5,36] and to the shoulder posteriorly.[5,20,35,37] This "stiff neck" muscle, when involved, consistently limits neck rotation due to pain on movement. If the TrPs are active enough, they refer severe pain even at rest.

2. ANATOMY
(Fig. 19.2)

The fibers of the levator scapulae attach ***above*** to the transverse processes of the first four cervical vertebrae (posterior tubercles of the C_3 and C_4 transverse processes); and ***below*** to the vertebral border of the scapula between the superior angle and the root of its spine (Fig. 19.2).

The twist of the muscle fibers is rarely noted or illustrated.[16] The C_1 digitation is

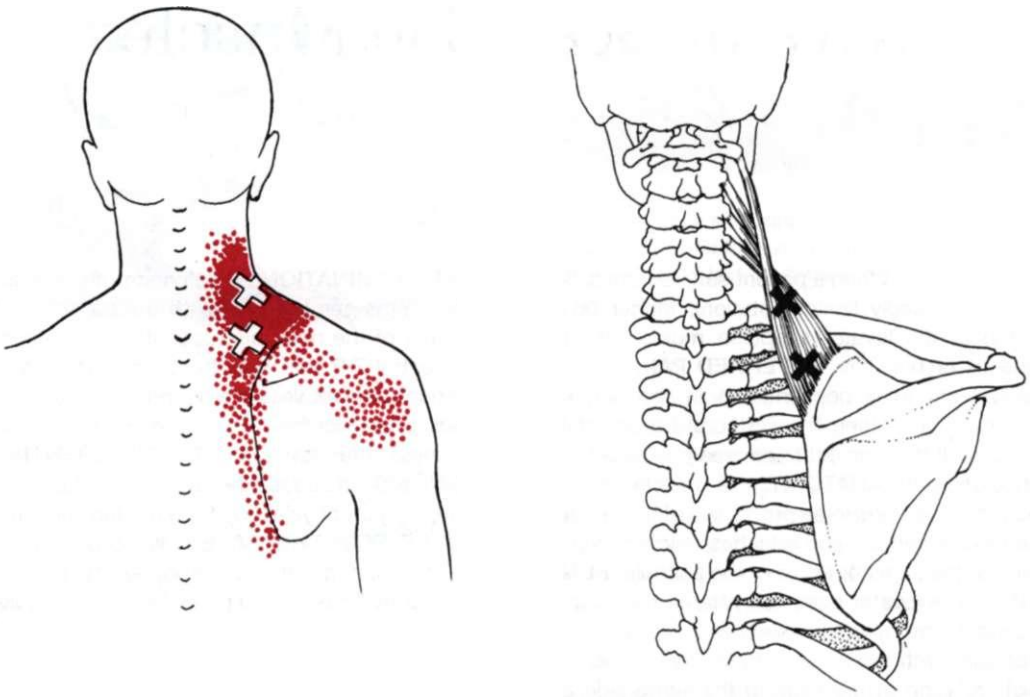


Figure 19.1. Consolidated referred pain pattern for trigger point regions (Xs) of the right levator scapulae muscle. The essential pain pattern is *solid red,* and the spillover pattern is *stippled red.* The upper *X* locates TrPs in the midportion of the muscle (often overlooked). The lower *X* locates the much more obvious trigger area tenderness commonly found near the region of the muscle's scapular attachment, which often is enthesopathy secondary to taut band tension associated with the TrPs.

superficial to the others and passes more vertically to the vertebral border of the scapula. The C_4 digitation lies deepest and passes diagonally to a lateral attachment on the superior angle of the scapula.

Menachem, *et al.*[22] studied the anatomical structures in the region of the scapular attachment of the levator scapulae muscle in 30 cadavers. In 63%, the levator scapulae was inserted on the scapula in two layers enfolding the medial border of the scapula. Thus, in the majority of bodies, some of the scapular attachment was on the under side of the scapula and not readily palpable. In nearly half of those bodies, a bursa was found in the areolar tissue between the two layers. In 13 (43%), a narrow band of the serratus anterior was reflected over the medial border of the scapula around its upper angle, close to the attachment of the levator scapulae. In 5 of these 13 (38%) another bursa occurred between the serratus anterior, the angle of the scapula, and the levator scapulae. These bursae are a potential source of tenderness in this region.

Supplemental References

Other authors have illustrated the muscle as seen from the front,[9, 10] the side,[8, 11] and from behind.[1,12, 16, 30]

3. INNERVATION

The levator scapulae muscle is supplied by branches of the third and fourth cervical nerves *via* the cervical plexus and sometimes, in part, by fibers from the dorsal scapular nerve derived from the C_5 root.[7]

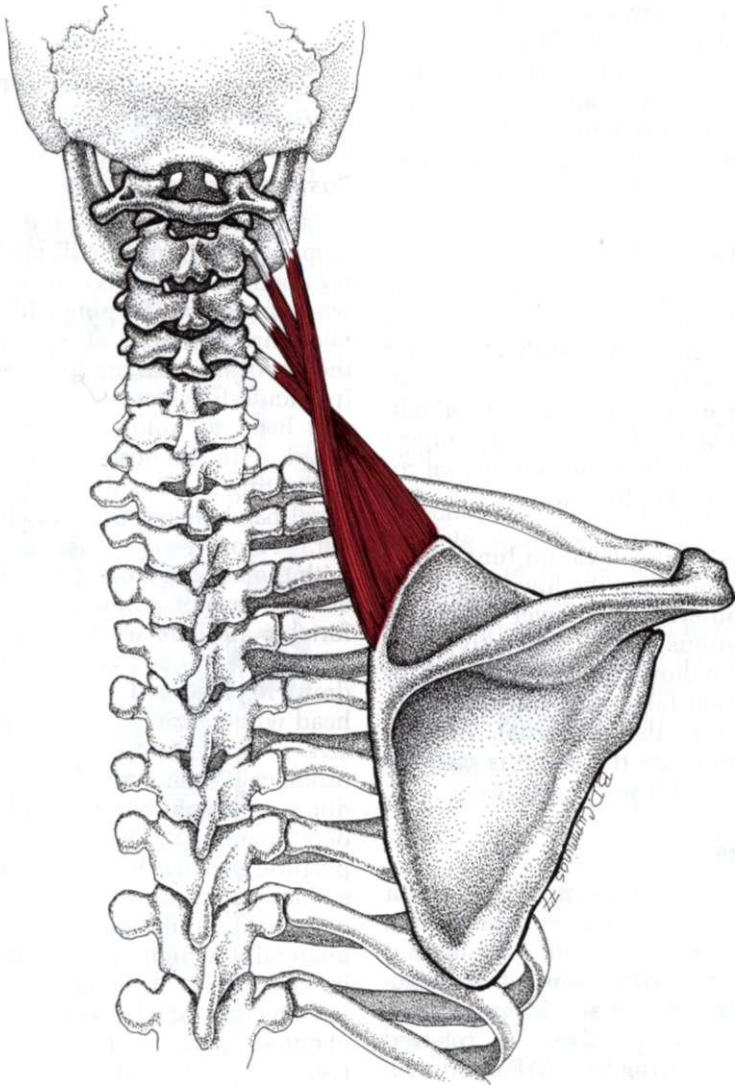


Figure 19.2. Attachments of the levator scapulae muscle. Note how the upper and lower digitations twist in their course from the superior attachment to the inferior attachment.

4. FUNCTION

When the neck is stabilized, the levator scapulae muscle first helps to rotate the scapula, facing the glenoid fossa downward, and then elevates the scapula as a whole.[7] When the scapula is anchored, this muscle helps to complete neck rotation to the same side.[7] Bilateral levator scapulae muscles acting together assist extension of the neck and checkrein (control) neck flexion. During arm movements in normal subjects, the levator scapulae was recruited during arm extension and not during scapular plane arm elevation or during arm abduction.[13]

In conjunction with the upper trapezius and uppermost fibers of the serratus anterior, the levator scapulae helps to elevate the scapula during activities such as shrugging the shoulders, supporting weight directly on the shoulder girdle (e.g., counteracting the pull of a heavy purse or letter

carrier's bag), and lifting a weight with the upper extremity.[3] The levator scapulae, rhomboidei major and minor, and the latissimus dorsi together rotate the glenoid fossa of the scapula downward. This pulls the inferior angles of the scapulae closer together posteriorly.[3,18]

5. FUNCTIONAL UNIT

The splenius cervicis and the scalenus medius muscles are synergistic with the levator scapulae in neck stabilization and may develop active TrPs in association with it because of some common attachments. The rhomboid muscles are important synergists for elevation and medial rotation of the scapula (downward rotation of the glenoid fossa).

Antagonists to the elevation function of the levator scapulae are the lowest fibers of the serratus anterior, the lower trapezius, and the latissimus dorsi (indirectly). The latissimus dorsi, however, is a synergist for scapular rotation (also indirectly, through its attachment to the humerus). Antagonists for rotation are the serratus anterior and the upper and lower trapezius.

6. SYMPTOMS

With severe involvement of the levator scapulae alone, patients complain of pain at the angle of the neck and of a painful "stiff neck." The diagnoses of scapulocostal syndrome[6,23,24.,28] and levator scapulae syndrome[22] emphasize the referred pain symptoms arising from TrPs in the levator scapulae muscle. The diagnosis of "stiff neck syndrome," or torticollis,[34, 35] emphasizes the restriction of range of motion, since tension in the levator scapulae is a common cause of neck stiffness[34,35] *(see* Chapter 7, Section 11 for the differential diagnosis of stiff neck and torticollis). Patients with active TrPs in the levator scapulae are unable to turn the head fully to the same side because of pain on contraction, and not fully to the opposite side because of painful increase in muscle tension. To look behind, they must turn the body instead.

Neoh[27] reported on 75 patients complaining of shortness of breath and nuchal soreness. Ninety percent of them were relieved of their symptoms after dry needling of their levator scapulae TrPs.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

(Fig. 19.3)

Postural Stress

Patients are likely to develop levator scapulae TrPs and a "stiff neck" because of occupational stresses, such as secretarial work in general,[22] typing with the head and neck turned to look at work placed beside the keyboard, making long telephone calls (particularly when laterally flexing the neck and head to hold the phone), talking at length with the head turned toward someone sitting to one side,[6] and carrying a personal bag hanging from the affected shoulder.[22] Another activating stress is sleeping with the neck in a tilted position that shortens the levator scapulae, as in an uncomfortable airplane seat, especially when the muscle is fatigued and exposed to a cold draft. Recreational stress from tilting the head while gazing fixedly at a stage, movie screen, or television can also precipitate the problem. Psychological stress, which produces a "weight-of-the-world-on-my-shoulders" reaction or a tense, hostile, aggressive posture, also may be contributory.[6] Sitting in a chair with armrests that are too high elevates the scapulae and shortens the muscle bilaterally, which encourages activation of its latent TrPs. Walking with a cane that is too long, so that it forces unnatural elevation of one shoulder, tends to activate TrPs in the levator scapulae on the same side (Fig. 19.3).

Activity and Overload Stress

Myofascial TrPs in the levator scapulae muscle can be activated and perpetuated by overexercise, such as in playing vigorous tennis, swimming the crawl stroke when out of condition, or rotating the head repeatedly back and forth. An example of repeated head rotation is "spectator neck" that is caused by sitting near the net at a tennis court and repeatedly turning the head and neck to follow the ball from side to side.

The levator scapulae muscle can be overloaded and develop active TrPs when function of the serratus anterior is inhibited by serratus TrPs. A motor vehicle acci-

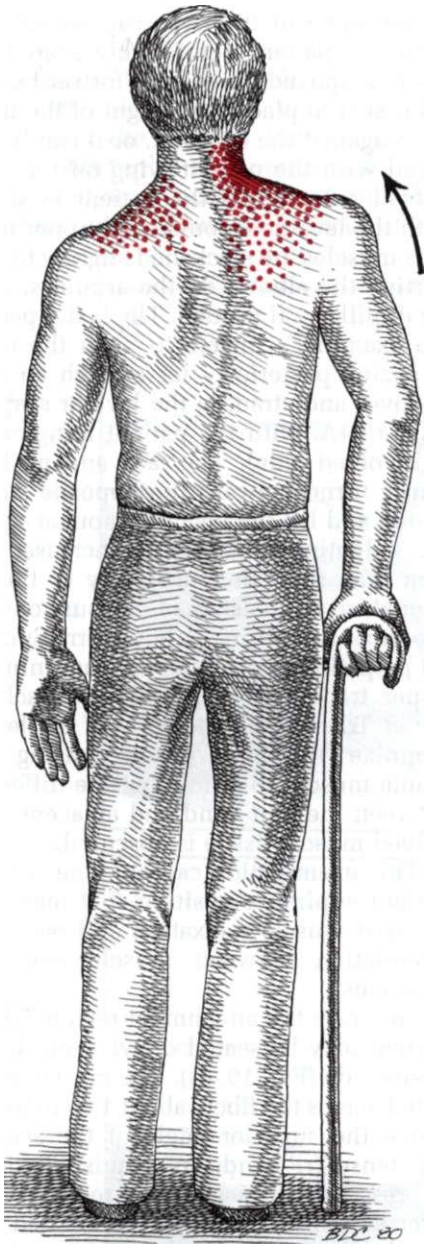


Figure 19.3. Activation primarily of right levator scapulae trigger points, and secondarily of other muscles on the left, by walking with a cane that is too long, held in the right hand. The patient's resultant pain distribution is shown in *red*. The *arrow* indicates the undesirable lift of the right shoulder so the long cane can clear the ground when walking.

dent in which the vehicle is struck from any direction commonly activates levator scapulae TrPs due to acute overload stress.[2] Sometimes TrPs in this muscle can arise reflexly from the activity of a key TrP in the functionally related upper trapezius muscle.[17]

Trigger points in the levator scapulae muscle can be activated and perpetuated by stresses imposed on it from asymmetries in the lower part of the body. For example, if there is a lack of normal push-off during walking, the levator may contract excessively during each gait cycle in a futile attempt to "lift the body" and in an attempt to preserve momentum for weight transition to the other foot. Push-off can be affected by many conditions such as calf muscle weakness, lower limb-length inequality, or a foot with a flattened arch. Asymmetry induced by quadratus lumborum shortening also can impact the levator scapulae. It may be that the (twisting) diagonal structure of the levator scapulae makes it particularly vulnerable.

Infection

During the prodromal stage of an acute upper respiratory infection, the levator scapulae becomes vulnerable to activation of its TrPs by mechanical stresses that are usually well within its tolerance. This susceptibility to activation by ordinary loads may start a day or two before the fully developed symptoms of a head cold or sore throat appear, and may last for several weeks thereafter. A stiff neck syndrome also often begins during an attack of oral herpes simplex.

8. PATIENT EXAMINATION

The examiner first observes the patient and looks for neck and shoulder postural asymmetries, then asks the patient to turn the head fully in each direction.

The patient tends to hold the neck rigid, looking to either side by turning the eyes or body but not the neck. The head may be tilted slightly toward the involved side.[35] If the patient's head is strongly tilted to one side (wry neck), sternocleidomastoid TrPs are more likely to be responsible than are levator scapulae TrPs. Whereas levator scapulae involvement reduces neck movement, a patient with active upper trapezius

TrPs moves the neck frequently trying to stretch the trapezius.

Active neck rotation is most restricted as the face turns toward the side of the pain. The degree of restriction depends upon the severity of involvement. When both sides are involved, as commonly occurs, rotation can be markedly restricted in both directions. Neck flexion is blocked only at the end (extreme range) of the movement; extension is relatively unaffected. If rotation of the neck is unrestricted, active TrPs in the levator scapulae are unlikely.

There is minimal limitation of shoulder motion. Full abduction requires full upward rotation of the scapula which can be painfully restricted by TrP tightness of the levator scapulae. The Hand-to-shoulder-blade Test is normal *(see* Fig. 22.3). The Mouth Wrap-around Test *(see* Fig. 18.2) is restricted chiefly by marked lack of head/neck rotation.

9. TRIGGER POINT EXAMINATION (Fig. 19.4)

The levator scapulae develops TrP tenderness in two locations: a central TrP area at the angle of the neck where the muscle emerges from beneath the anterior border of the upper trapezius;[24, 35] and a much more readily identified secondary area near where the muscle attaches to the superior angle of the scapula.[23,24,29] This distal attachment tenderness is likely enthesopathy resulting from sustained TrP tension and is commonly associated with the palpable induration of a ventral TrP and its palpable taut band that causes the excessive tension. For the anatomical relations to adjacent muscles, see Figure 20.7. Sola and Williams[34] reported locating the lower TrP tenderness by electrical stimulation which produced pain referred to the neck and back of the head. Michele *et al.*,[23] in an initial article, described in great detail how to locate the area of tenderness at the angle of the scapula, but did not identify the central TrP at the base of the neck. Later, Michele and Eisenberg[24] identified the tenderness of both the upper TrP and the lower trigger area of the levator scapulae, and illustrated how to palpate the upper TrP as the prime source of the scapulocostal syndrome.

The central TrP in the levator scapulae at the angle of the neck can be palpated with the patient comfortably seated and the hips moved far enough forward on the chair seat to place the weight of the upper torso against the backrest, or it can be palpated with the patient lying on the uninvolved side. When the patient is sitting, both the levator scapulae and upper trapezius muscles are slackened slightly by supporting the elbows on the armrests, using small pillows if needed. The laxity permits the examiner's fingers to push the upper trapezius posteriorly far enough so as to uncover and straddle the levator scapulae (Fig. 19.4A, with the patient lying on the uninvolved side). The face and neck are gently turned toward the opposite side to tauten and lift the levator scapulae against the palpating fingers. The increased tension may raise the sensitivity of the TrP enough so that sustained pressure on it reproduces its referred pain pattern. Successful palpation depends upon slackening the upper trapezius sufficiently to reach the upper TrPs within the belly of the levator scapulae muscle without tensing that whole muscle so much that the difference between the taut band and adjacent uninvolved muscle tissue is obscured.

This examination can be done with the patient supine, a position that may yield the best muscle relaxation and better differentiation between muscle and joint problems.

To locate the attachment region TrP, the patient may be seated or lying on the opposite side (Fig. 19.4B). The muscle is palpated across the fibers about 1.3 cm (0.5 in) above the superior angle of the scapula. The tense TrP bands are exquisitely tender to pressure, but local twitch responses and referred pain are not readily elicited from this lower TrP area, which is covered by the trapezius muscle. The region of attachment feels indurated and tender and can be rocked back and forth between the fingers when they straddle it. When the attachment has been stressed for a period of time, the area may feel gritty (like gravel) or like a scar.

Of 22 patients seen in an orthopedic clinic for shoulder pain over the upper medial angle of the scapula,[22] 95% of

them had maximum tenderness within 2 cm of the upper angle. Pressing on the tender spot reproduced or aggravated the typical pain. In 73% small nodules or crepitation was palpable at the tender spot, which the authors identified as a TrP. Thermography was performed in 19 of the 22 patients. Increased heat emission was observed in about half (58%) of the symptomatic shoulders and in none of the contralateral shoulders. Thermography was considered an unreliable diagnostic test in these patients.[22]

10. ENTRAPMENT

No primary nerve or vascular entrapments due to TrPs have been recognized

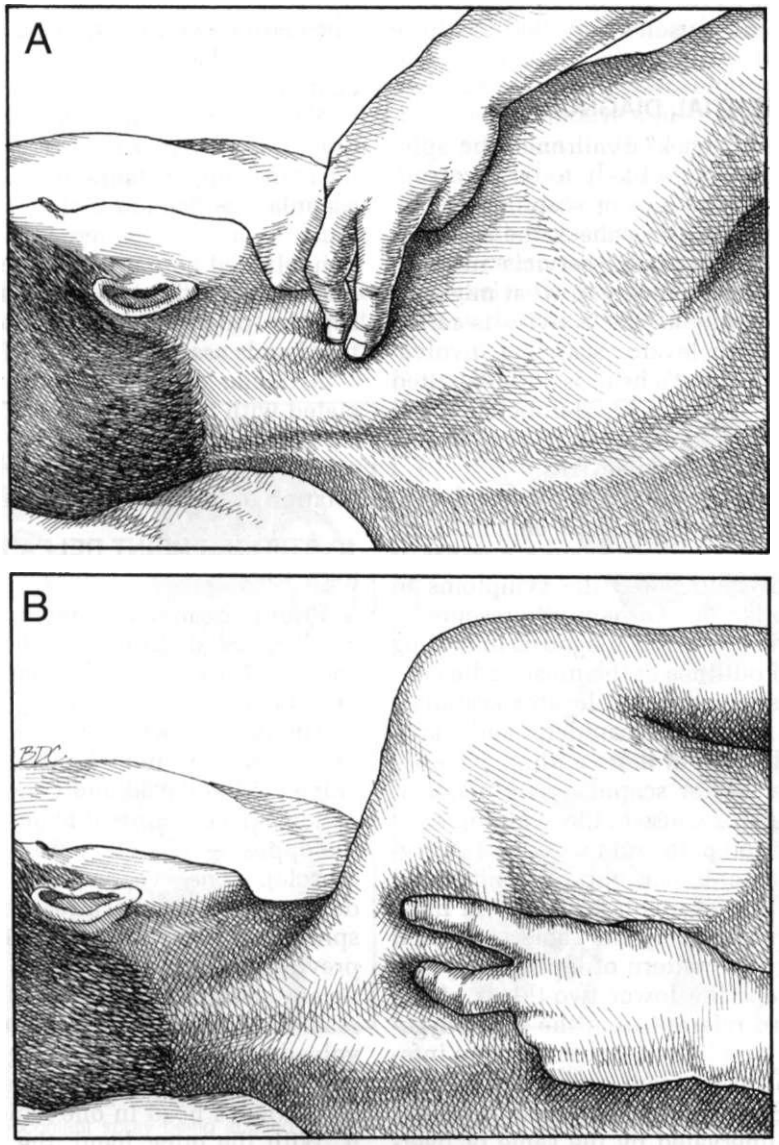


Figure 19.4. Examination of the levator scapulae muscle, patient lying on the uninvolved side. **A,** pressing the free border of the upper trapezius aside with the index finger to straddle the tense levator scapulae muscle and localize the upper trigger point between the fingers. **B,** straddling the lower trigger area just cephalad to the muscle's attachment to the superior angle of the scapula.

in vessels or nerves penetrating this muscle. However, as noted by Andrew Fischer, M.D. (personal communication), the levator scapulae is in a strategic position to aggravate a cervical radiculopathy caused by narrowed cervical neural foramina. The increased muscle tension associated with TrPs can further occlude the compromised foramina, thereby increasing nerve compression effects. One of these effects is to facilitate the activation of TrPs in the muscles supplied by those nerves.

11. DIFFERENTIAL DIAGNOSIS

In the "stiff neck" syndrome, the splenius cervicis also is likely to be involved. When TrPs in the levator scapulae are active, it is wise to also check the scalenus medius and iliocostalis cervicis muscles for TrP activity. Contrary to what might be expected, rhomboid TrP activity is rarely associated with levator scapulae involvement. If the patient's head is *strongly* tilted to one side (wry neck), sternocleidomastoid TrPs are more likely to be responsible than are levator scapulae TrPs.

The etiology of the **scapulocostal syndrome** has been considered enigmatic by many authors in the past, but a number of authors have attributed the symptoms to trigger points.[6,23,24] Ormandy[28] presented a scholarly review of this diagnosis including anatomical outlines of the muscles he considered responsible: the levator scapulae, rhomboid minor, subscapularis and trapezius muscles. All of these authors have included the levator scapulae as a major, if not the primary, cause of the symptoms.

The referred pain and tenderness of zygapophysial joints can appear confusingly similar to that of myofascial TrPs in muscles at approximately the same segmental level. The pain pattern of levator scapulae TrPs overlaps the lower two-thirds of the pain pattern referred from the C_4-C_5 zygapophysial joint but also extends more inferiorly.[4] However, there are important differences. Even though joints and muscles often are innervated by the same or overlapping neural segments, myofascial pain referral patterns can be distinctively different for different muscles innervated by the same neural segments. The patterns are not always limited to the sclerotomes or myotomes of the segments that innervate the muscle. (Sources of deep pain are rarely referred to the dermis, so dermatomes are not applicable.) A TrP is confirmed by physical examination of the muscle for palpable TrP characteristics. Exceptional skill is needed to accurately identify painful zygapophysial joint dysfunction by manual examination. The reliable confirmation of joint dysfunction by anesthetic block is a complicated procedure requiring unusual skill and detailed knowledge of spinal anatomy.[19]

The physical finding of crepitation, and the relatively frequent presence of bursae near the upper (superior) angle of the scapula (*see* Section 2 of this chapter), indicate that the tenderness and referred pain elicited here may be caused by a bursitis instead of, or in addition to, enthesopathy caused by unrelieved tension of taut bands associated with TrPs.

Articular dysfunctions commonly associated with levator scapulae TrPs can be at C_3, C_4, C_5, or C_6, or at several of these levels. Typically, one sees side bending and rotation of the head to the same side.

12. TRIGGER POINT RELEASE (Fig. 19.5)

Prior to treatment, X-ray films of the cervical spine should be reviewed for any condition that would preclude passive neck flexion and rotation.

The patient sits relaxed in a chair, with the pelvis level, and with the arm on the involved side relaxed and hanging free. The patient's face is turned about 30% toward the opposite side (away from the involved muscle). The vapocoolant is sprayed downward in parallel sweeps following the spray lines shown in Figure 19.5A, and as previously described.[26,35] The patient takes up any slack that develops in the muscle by reaching downward with the arm on the involved side (right arm in Fig. 19.5A). **Immediately** (Fig. 19.5B) the operator cradles the patient's head in one hand to stabilize it. With the other hand, the clinician applies gentle, steady traction on the vertical fibers and then on the diagonal fibers of the levator scapulae muscle and on related soft tissues; this gentle pressure is applied

downward and anteriorly around the rib cage to depress and abduct the scapula until the soft tissue resistance barrier is reached. While this position is held, the patient inhales while gently contracting the levator scapulae muscle against mild resistance supplied by the operator's hand on the scapula. Then the patient **slowly** exhales and relaxes the muscle while the operator again takes up all the slack that develops. This release can be done with the patient supine, incorporating postisometric relaxation.

The patient should learn to identify the sense of tension when the direction of pull affects the most shortened (most tense) muscle fibers. When that direction of pull is reached, the more skillful operators can "feel" this tension without the patient telling them.

To reach full stretch length of the levator scapulae muscle, it is necessary to abduct the scapula. When this movement is restricted by tightness in other muscles, it can be released by applying the interscapular release maneuver illustrated in Figure 18.3. An alternative, similar technique is described by Lewit[21] that includes postisometric relaxation with respiratory augmentation.

Full release of the levator scapulae muscle is frequently complex and difficult to achieve by stretch and spray. Parallel myotatic muscles, including the splenius cervicis, scalenus medius, scalenus posterior, and the posterior cervical muscles, must be released to achieve a full stretch of the levator scapulae. If the scalene muscles require stretch and spray, it also is desirable to stretch and spray the upper pectoralis major fibers, since their trigger points (TrPs) cause disagreeable chest pain and are likely to become involved in association with scalene TrP activity.

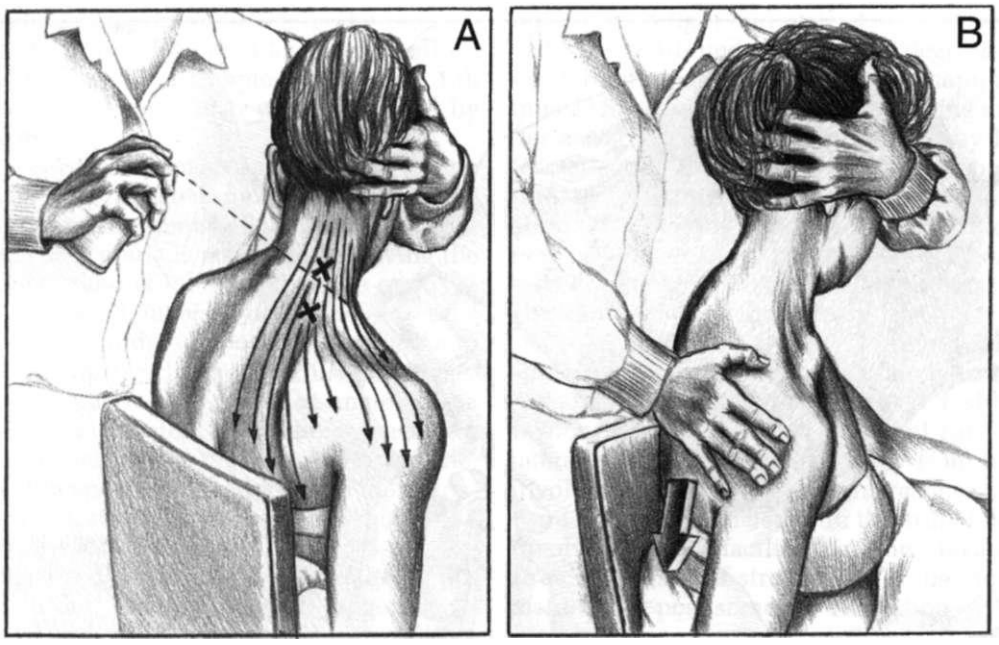


Figure 19.5. Spray and stretch release of trigger points in the right levator scapulae muscle, patient seated. **A**, Vapocoolant spray being applied in slow repeated sweeps *(arrows)* while the patient takes up any slack in the levator scapulae muscle by reaching downward with the right arm. **B,** Release of trigger points **immediately** following spray by elongating the muscle while the patient slowly exhales. The heel of the operator's right hand takes up slack in the muscle and stretches the most vertical and longest fibers *(thick arrow);* the palm and fingers stretch the diagonal fibers as the operator presses downward and slightly forward to abduct the scapula while the operator's left hand stabilizes the patient's head. See text for details of incorporating postisometric relaxation.

Sometimes, the antagonistic neck muscles also must be stretched and sprayed to coax the levator scapulae to lengthen. This stretch of a restricted levator scapulae shortens the ipsilateral serratus anterior muscle more than usual and may activate any latent TrPs in it, producing a painful reactive cramp with chest pain. This problem is prevented, or readily relieved, by stretching and spraying the serratus anterior muscle *(see* Chapter 46).

If the pain shifts to the other side of the neck, the procedure has uncovered a lesser, but significant degree of TrP activity in the contralateral levator scapulae that requires application of the same procedures to that muscle.

If the patient has concurrent articular dysfunction in the C_3-C_6 vertebral segments, treatment begins with TrP and myofascial release as described above with the patient in the seated position. The patient is then repositioned supine for treatment using muscle energy techniques to restore mobility of cervical articulations. Muscle energy techniques are described in principle by Mitchell Jr.[25] and in operational detail for the cervical spine by Greenman.[14] These procedures are followed, if necessary, by inactivation of any residual trigger points.

13. TRIGGER POINT INJECTION
(Fig. 19.6)

The lower trigger area near the scapular attachment of the levator scapulae (Fig. 19.1) is more readily located than the mid-muscle trigger point (TrP), which is the critical one. Injection of the upper TrP may eliminate tenderness in the region of the inferior trigger area, but not *vice versa.*

For injection of the upper TrP (Fig. 19.6), the patient lies on the unaffected side (affected side up), with the back toward the operator, and the patient's body angled across the treatment table by plac-

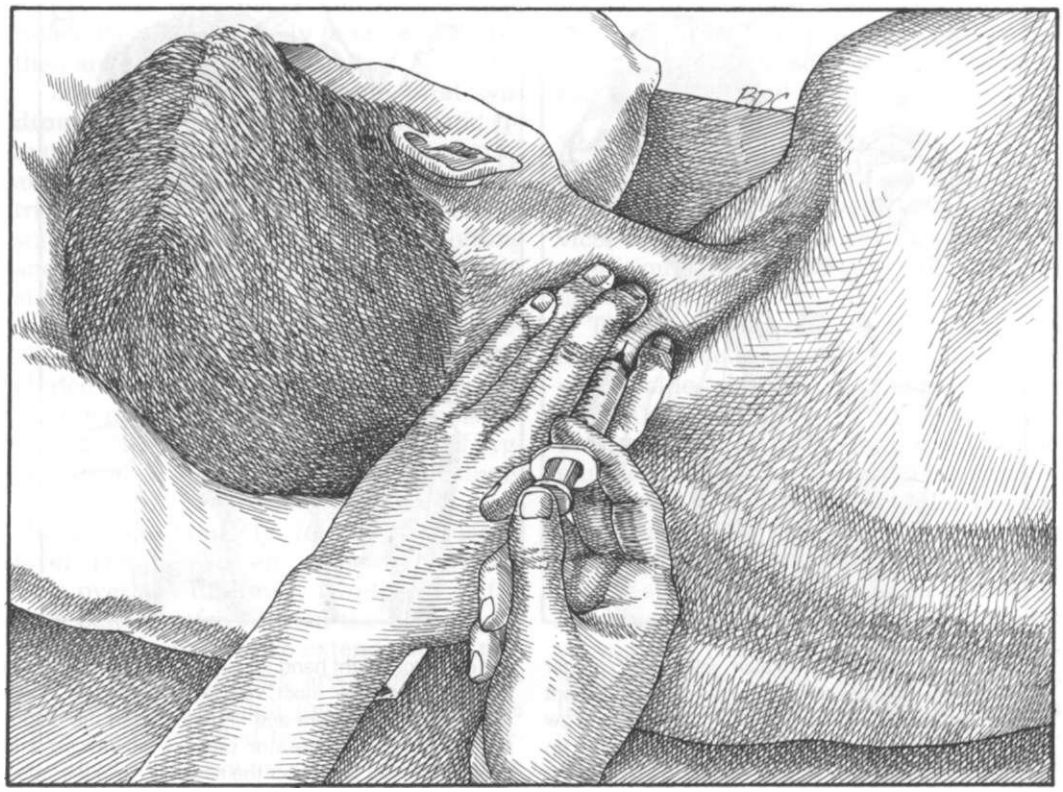


Figure 19.6. Injection of the upper trigger point in the right levator scapulae muscle at the base of the neck where the muscle emerges from beneath the upper trapezius.

ing the shoulder close to the edge of the table near the clinician. A pillow supports the head. The patient rests the uppermost upper limb on his or her body, with the elbow bent to balance it. If more tension is desired in the levator scapulae to be injected, the uppermost arm can be placed in full medial rotation with the hand across the back to produce scapular winging. The operator presses aside the free upper border of the trapezius muscle and palpates the levator scapulae as it emerges from beneath the trapezius *(see* Fig. 20.7, Regional Anatomy, and Fig. 16.8, Cross Section). The TrP (the spot of maximum tenderness in the taut muscle fibers) is fixed against a transverse process for injection. The needle is directed anteriorly toward the TrP but away from the rib cage (Fig. 19.6). This muscle frequently has multiple taut bands and multiple TrPs in its numerous fascicles, which may make it necessary to do more extensive needling than in most muscles. Dry needling with an acupuncture needle or injection with a hypodermic needle using procaine or lidocaine are all effective here *(see* Chapter 3, Section 13). This technique is also well illustrated by Rachlin.[31]

If soreness persists in the lower trigger area, it is injected next, just above the scapular attachment of the levator scapulae. The scapula is abducted by having the patient, who is lying on the side opposite the involved muscle, bend forward in a "round-shouldered" posture to stretch and thin out the overlying trapezius. The trigger area just above the superior angle of the scapula is located by rubbing the finger transversely across the fibers of the muscle. The fingers of one hand localize the palpable induration. The other hand holds the syringe and directs a 3.8-cm (1.5-in), 22-gauge needle upward just above the scapular border. Needle insertion tangential to the rib cage avoids penetrating between the ribs where it might cause a pneumothorax. A 27-gauge needle or acupuncture needle can also be effective with the alternate needling technique described in Chapter 3, Section 13. Addition of corticosteroid in refractory cases may be helpful in this lower location, but is not recommended when injecting the more cephalad TrP.

Injection is followed by stretch and spray, moist heat, and, finally, active range of motion.

14. CORRECTIVE ACTIONS

Patients who are prone to develop active levator scapulae TrPs must learn how to release and lengthen a tight levator scapulae muscle for themselves. Release is most effective under a warm shower, preferably sitting on a chair or stool that has a low backrest. The patient relaxes the neck muscles as much as possible and drops the shoulder on the affected side, letting the arm hang free. The contralateral hand assists in turning the head toward the unaffected side and down toward the axilla to take up slack in the muscle, while the free hand reaches downward toward the floor to provide additional muscle lengthening (see Fig. 16.11A). When seated, the patient can stabilize the scapula by sitting on the hand of the affected side and then can apply a slow steady stretch (without jerking) in each direction that the muscle feels tight and restricted. This procedure is continued with varying degrees of head rotation to release levator scapulae muscle fibers of all directions. Standing under a warm shower is helpful but may be less effective than sitting because of postural reflexes that inhibit muscular relaxation. Lengthening this muscle on one side may produce reactive cramping in the contralateral muscle. The contralateral muscle also should be stretched gently.

Greenman[14] illustrates a useful self-stretch for the levator scapulae muscle. The patient lies on the unaffected side, *without* a pillow (so that the head can be laterally flexed and rotated away from the involved muscle). The patient reaches toward the foot of the bed with the arm of the involved side, facilitating the levator stretch. This self-stretch could be augmented by postisometric relaxation with deep breathing.

Lewit[21] illustrates a gravity-assisted self-stretch using postisometric relaxation with respiratory augmentation that is gentle and effective and releases both the levator scapulae and the upper trapezius muscles. A multi-purpose self-stretch that includes the levator scapulae, upper trapezius, and posterior cervical

muscles is the Combined Self-stretch Exercise illustrated in Figure 16.11. This is a particularly useful exercise because these muscles are so frequently involved as a group.

If the patient sits at a desk when talking with visitors, he or she should turn the chair (not just the head) to face the visitor squarely, or rearrange the furniture so that the visitor's chair is in front of the desk.

To avoid posterior cervical and levator scapulae strain when reading, writing, or typing, myopia must be corrected by eyeglasses with a sufficiently long focal length. Material to be read should be in focus when placed upright on a book-holder or copy stand, or on a music stand. The use of one of these supports relieves sustained neck flexion. For a card player, one pair of eyeglasses should be adjusted for focus at arm length ("card-playing glasses").

If a keyboard is too high but cannot be lowered, and the chair provides inadequate adjustment, 2 or 3 cm (about an inch) or so of firm cushion, folded newspapers, or a magazine may be placed on the rear two-thirds of the chair seat bottom. The front one-third of the seat is not raised, to avoid compression of the thighs and to open the angles at the hips and knees. The backrest should provide adequate thoracolumbar support (see Fig. 16.4D).

Individuals who have trouble with levator scapulae (or upper trapezius) TrPs and are obliged to use a telephone frequently or for long calls **must** find a way to relieve the muscle strain of holding the phone to one's ear. The most effective solution is a headset with a microphone positioned near the mouth, such as the one telephone operators use. A cradle for the phone that lets it rest on the shoulder can help only **if** the cradle does not require continuous shrugging of the shoulder to hold it in place. Usually a shoulder cradle still requires muscular effort and is undesirable. Changing hands frequently from one side to the other or resting the elbow on a desk top to position the phone can provide some temporary relief and provide more pain-free working time.

The patient should apply a hot pack or a moist heating pad to the TrP areas, especially at the end of the work day or on retiring for the evening.

When in bed, the patient should position the pillow to avoid shortening and cramping of the muscle (see Fig. 7.7).

If a walking cane is used, its length should provide a level shoulder-girdle axis during walking (*not* as in Fig. 19.3).

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (pp. 234, 381; (Figs. 4-48, 6-32).
2. Baker BA: The muscle trigger: evidence of overload injury. *J Neurol Orthop Med Surg* 7:35-44, 1986.
3. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 267, 268).
4. Bogduk N, Simons DG: Neck pain: joint pain or trigger points? Chapter 20. In: *Progress in Fibromyalgia and Myofascial Pain.* Vol. 6 of *Pain research and Clinical Management.* Edited by Vaer0y H, Mersky H. Elsevier, Amsterdam, 1993 (pp. 267-273).
5. BonicaJJ: Neck pain. Chapter 47. In: *The Management of Pain.* Ed. 2, Vol 1. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, 1990 (pp. 848-867).
6. Cailliet R: *Neck and Arm Pain.* F.A. Davis, Philadelphia, 1964 (p. 97).
7. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 516).
8. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 576).
9. *Ibid.* (Fig. 594).
10. Eisler P: Die *Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (Fig. 49).
11. *Ibid.* (Figs. 50, 52).
12. *Ibid.* (Fig. 51).
13. Eliot DJ: Electromyography of levator scapulae: new findings allow tests of a head stabilization model. *J Manipul Physiol Ther 19(1):19-25,* 1996.
14. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 195-196, 498).
15. Grosshandler SL, Stratas NE, Toomey TC, *et al:* Chronic neck and shoulder pain, focusing on myofascial origins. *Postgrad Med* 77:149-158, 1985.
16. Hollinshead WH: *Anatomy for Surgeons.* Ed. 3, Vol. 3, *The Back and Limbs.* Harper & Row, New York, 1982 (p. 305, Fig. 4-36).
17. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain 2(l):29-59,* 1994.
18. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 83).
19. Jull G, Bogduk N, Marsland A: The accuracy of manual diagnosis for cervical zygapophysial joint pain syndromes. *Med J Aust* 348:233-236, 1988.
20. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (p. 98).
21. Lewit K: *Manipulative Therapy in Behabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 195, 196).

22. Menachem A, Kaplan O, Dekel S: Levator scapulae syndrome: an anatomic-clinical study. *Bull Hosp Joint Dis* 53(1):21-24, 1993.
23. Michele AA, Davies JJ, Krueger FJ, *et al.:* Scapulocostal syndrome (fatigue-postural paradox). *NY State J Med* 50:1353-1356, 1950 (p. 1355, Fig. 4).
24. Michele AA, Eisenberg J: Scapulocostal syndrome. *Arch Phys Med Rehabil* 49:383-387, 1968 (pp. 385, 386, Fig. 4).
25. Mitchell FL Jr: Elements of muscle energy technique. Chapter 12. In: *Rational Manual Therapies.* Edited by Basmajian JV, Nyberg R. Williams & Wilkins, Baltimore, 1993 (pp. 285-321).
26. Modell W, Travell JT, Kraus H, *et al.:* Contributions to Cornell conferences on therapy. Relief of pain by ethyl chloride spray. *NY State J Med* 52:1550-1558, 1952 (p. 1551).
27. Neoh CA: Treating subjective shortness of breath by inactivating trigger points of levator scapulae muscles with acupuncture needles. *J Musculoske Pain* 4(3):81-85, 1996.
28. Ormandy L: Scapulocostal syndrome. *Va Med Q 121(2):105-108,* 1994.
29. Pace JB: Commonly overlooked pain syndromes responsive to simple therapy. *Postgrad Med* 58.107-113, 1975 (p. 110).
30. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 28).
31. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (p. 315).
32. Sola AE, Kuitert JH: Myofascial trigger point pain in the neck and shoulder girdle. *Northwest Med* 54:980-984, 1955.
33. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med Rehabil* 34:585-590, 1955.
34. Sola AE, Williams RL: Myofascial pain syndromes. *Neurol* 6:91-95, 1956 (p. 93, Fig. 1).
35. Travell J: Rapid relief of acute "stiff neck" by ethyl chloride spray. *J Am Med Wom Assoc* 4:89-95, 1949 (pp. 92-93, Fig. 3, Case 1).
36. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med 11*:425-434, 1952.
37. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little, Brown & Company, Boston, 1988 (Fig. 12-1).

CHAPTER 20
Scalene Muscles

HIGHLIGHTS: Scalene muscle trigger points (TrPs) and their associated thoracic outlet ***entrapment*** syndrome are often overlooked sources of pain in the shoulder-girdle region and upper limb. Scalene TrPs are among the more demanding of skill for identification and treatment but are also among the most important myofascial TrPs. **REFERRED PAIN** from all three of the major scalene muscles can radiate anteriorly, laterally, and/or posteriorly. Posteriorly, pain is referred to the upper vertebral border of the scapula and the area medial to it. Anteriorly, aching pain is referred to the pectoral region; laterally, it is referred down the front and back of the arm, skips the elbow to reappear on the radial forearm, and may extend to the thumb and index finger. On the left side of the thorax, this pain is easily mistaken for angina pectoris during activity or at rest. **ANATOMY:** above, the three major scalene muscles attach to transverse processes of cervical vertebrae; below, the scalenus anterior and scalenus medius attach to the first rib, and the scalenus posterior attaches to the second rib. **FUNCTION:** the scalene muscles stabilize the cervical spine against lateral movement and are well situated to elevate and stabilize the first and second ribs during inhalation. **SYMPTOMS** may be primary myofascial pain, or secondary sensory and motor disturbance due to neurovascular entrapment. Pain on the radial side of the hand indicates a referred myofascial source; pain on its ulnar side with puffiness of the hand suggests brachial plexus and subclavian vein entrapment. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS:** They can be activated by pulling, lifting and tugging, and by overuse of these respiratory muscles as in coughing. They can be perpetuated by chronic muscle strain due to a tilted shoulder-girdle axis caused by a lower limb-length discrepancy or an asymmetrical pelvis and by any systemic perpetuating factor. **PATIENT EXAMINATION** is assisted diagnostically by the Scalene-cramp Test, the Scalene-relief Test, and the Finger-flexion Test. **TRIGGER POINT EXAMINATION** requires skill and an understanding of local anatomy. Flat palpation locates most scalene TrPs against the underlying transverse processes of the vertebrae. **ENTRAPMENT** of the lower trunk of the brachial plexus is commonly due to increased tension of the scalenus anterior and the scalenus medius muscles that is caused by taut bands of TrPs in those muscles. This entrapment causes ulnar pain, tingling, numbness, and dysesthesia. TrP activity in the scalenus anterior can cause hand edema. **DIFFERENTIAL DIAGNOSIS** includes the carpal tunnel syndrome, C_5-C_8 radiculopathy, and cervical spine articular dysfunctions. The close relation between the diagnosis of thoracic outlet syndrome and scalene TrPs is especially important because the TrPs often cause the pain symptoms and entrapment signs. The TrPs are commonly overlooked and can be effectively treated without surgery. **TRIGGER POINT RELEASE** using spray and stretch employs neck sidebending to the side away from the scalene TrPs, while the operator applies down sweeps of vapocoolant spray over the muscle and its pain reference zones. **TRIGGER POINT INJECTION** may be necessary for complete relief, but must be done with full understanding of, and respect for, local anatomy. **CORRECTIVE ACTIONS** are usually essential for continued relief and often require: performance of the Neck-stretch exercise, adoption of normal coordinated respiration instead of paradoxical breathing, elimination of postural muscle strain, correction of seating and lighting arrangements, elevation of the head of the bed at night, proper selection of a neck pillow, and maintenance of adequate body warmth.

1. REFERRED PAIN
(Fig. 20.1)

Scalene muscle trigger points (TrPs) are a common (and commonly overlooked) source of back, shoulder, and arm pain. Although these TrPs rarely refer pain to the head, they are commonly associated with TrPs that do. More than half of 11 patients with cervicogenic headache also had associated active scalene TrPs that were contributing to their pain.[28]

Active TrPs in the anterior, medial, or posterior scalene muscles may refer pain anteriorly to the chest, laterally to the upper limb, and posteriorly to the medial scapular border and adjacent interscapular region (Fig. 20.1A).[40,71,75] It is important to remember that any one of the scalene muscles can produce any part of the referred pain pattern.

Posteriorly, pain is commonly referred from TrPs in the scalenus anterior to the back, over the upper half of the vertebral border of the scapula and to the adjacent interscapular region.[7] When the patient presents with posterior shoulder pain, particularly along the border of the scapula, one should be sure to check for scalene TrPs. *Scalene muscles are among the most common sources of this back pain.*

Anteriorly, persistent aching pain is referred in two finger-like projections over the pectoral region down to about the nipple level;[73] this pattern commonly originates in the lower part of the scalenus medius or scalenus posterior.

Scalene pain referred to the anterior shoulder region is not characteristically described as deep in the joint, as is the pain referred from the infraspinatus muscle. Scalene muscle pain extends down the front and back of the arm (over the biceps and triceps brachii muscles).[7] The referred pain usually skips the elbow and reappears in the radial side of the forearm, the thumb, and the index finger. This upper limb pattern arises from TrPs in the upper part of the scalenus anterior and from the scalenus medius. On the left side of the thorax, this TrP referred pain may be mistaken for angina pectoris since it is likely to be associated with muscular activity.

In an upper-extremity amputee, this referred pattern of upper limb pain produced severe phantom limb pain that was relieved by one author (JGT) by inactivation of scalene TrPs. Sherman[60] lists elimination of TrPs as one treatment for relief of phantom limb pain.

Experimental injection of 0.2 to 0.5 ml of a 6% solution of sodium chloride into the scalenus anterior in 7 subjects evoked referred pain primarily in the shoulder region in all subjects, pain down the arm in one subject, and a superficial hyperesthesia radiating upward over the neck in two subjects.[63]

The less frequently seen pain referred from TrPs in the variable scalenus minimus muscle projects strongly to the thumb (Fig. 20.1B). This pain covers the lateral aspect of the arm from the deltoid insertion to the elbow but skips the elbow to cover the dorsum of the forearm, wrist, hand and all five digits, accenting the thumb. Myofascial TrPs may refer a sensation that the patient describes as "numbness" of the thumb with or without demonstrable hypoesthesia to cold or touch.

2. ANATOMY
(Figs. 20.2 and 20.3)

Scalenus Anterior
(Fig. 20.2)

The anterior scalene muscle attaches ***above*** to the anterior tubercles on the transverse processes of vertebrae C_3 to C_6; ***below,*** it attaches by a tendon to the scalene tubercle on the inner border of the first rib and on the upper surface anterior to the groove for the subclavian artery (Fig. 20.2).[10] Vertebra C_7 is unlikely to have an anterior tubercle unless an anomalous slip of the scalenus anterior or the presence of a scalenus minimus muscle requires it.

Scalenus Medius
(Fig. 20.2)

The scalenus medius is the largest of the scalene muscles and attaches ***above*** to the posterior tubercles on the transverse processes usually of vertebrae C_2 through C_7 (sometimes to the processes of only the 4th and 5th cervical vertebrae).[4] The muscle slants diagonally and attaches ***below*** to the cranial surface of the first rib, posterior and part of it deep to the groove for the subclavian artery (Fig. 20.2 and *see* Fig. 20.9). A slip of the muscle sometimes extends to the second rib.

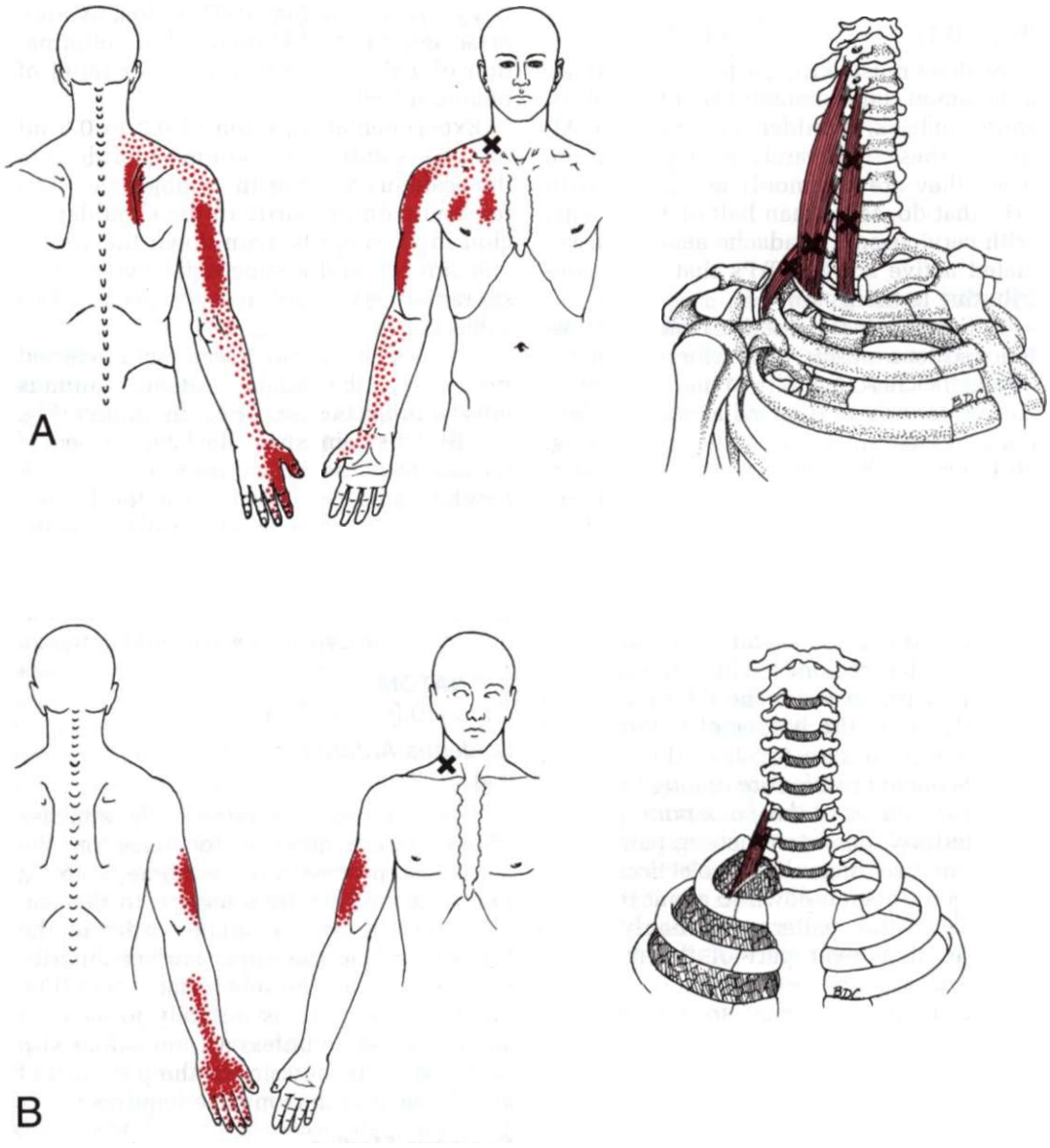


Figure 20.1. Composite pain patterns *(solid red* areas are the essential pain reference zones, and *stippled red* areas are the spillover reference zones) with location of some trigger points (Xs) in the right scalene muscles *(medium red).* **A,** scalenus anterior, medius, and posterior. Some trigger points may have only one essential reference zone. **B,** scalenus minimus,

Scalenus Posterior
(Fig. 20.2)

This muscle attaches ***above*** to the posterior tubercles on the transverse processes of the lowest two or three cervical vertebrae, and ***below*** to the lateral surface of the second rib and sometimes of the third rib (Fig. 20.2). The scalenus posterior crosses the first rib posterior to the scalenus medius and deep to the anterior borders of the upper trapezius and levator scapulae muscles (*see* Fig. 20.7).

Scalenus Minimus
(Fig. 20.3)

All the scalene muscles are variable in their attachments. The most variable is the scalenus minimus, which occurred on at

least one side of the body in one-half to three-quarters of the bodies studied.[5, 20] This muscle usually extends ***above*** to the anterior tubercle on the transverse process of vertebra C_7, sometimes also of C_6. ***Below,*** it attaches to the fascia supporting the pleural dome and beyond to the inner border of the first rib. The muscle lies behind (deep to) the scalenus anterior and attaches behind (posterior to) the groove for the subclavian artery (Fig. 20.3).[10] The pleural dome, or cupola, is strengthened by Sibson's fascia and anchored by this fascia to the anterior tubercle of C_7 and to the inner border of the first rib. The scalenus minimus reinforces this fascia and can be a strong, thick muscle.[11,20]

The scalenus minimus passes beneath and behind the subclavian artery to attach to the first rib, whereas the anterior scalene

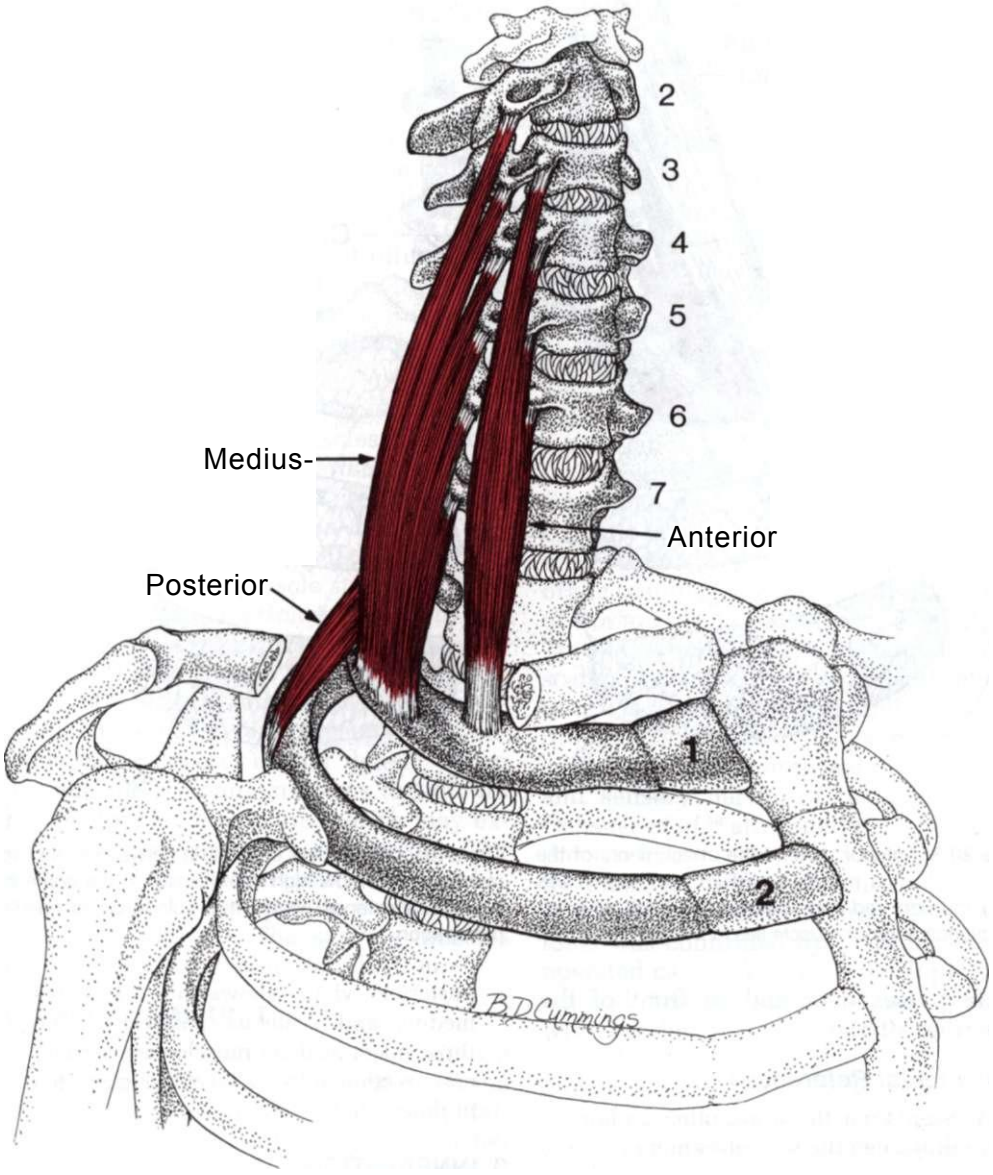


Figure 20.2. Oblique view of the attachments of the three major scalene muscles to the cervical vertebrae and to the first and second ribs. The clavicle has been cut and the section that overlies the scalene muscles removed.

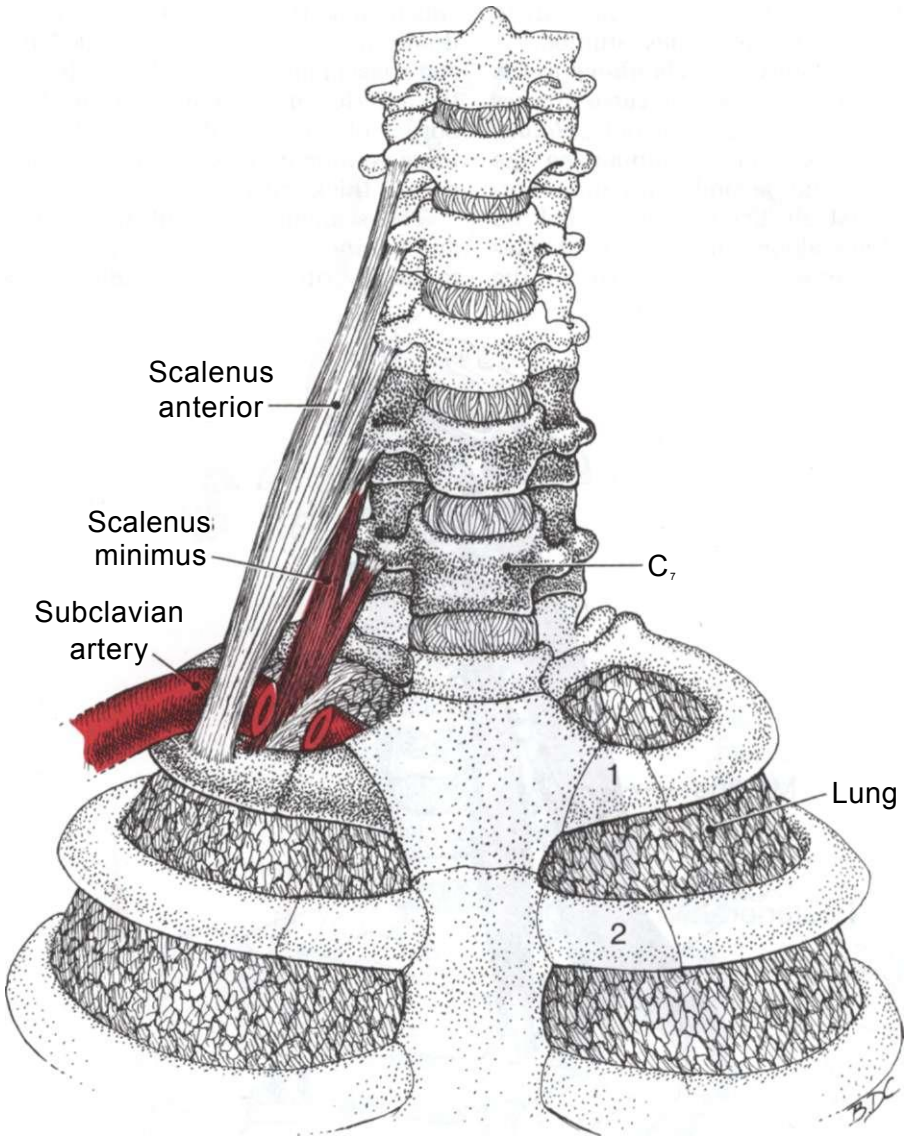


Figure 20.3. Anterior view of the attachments of the scalenus minimus muscle (*medium red),* which lies *behind* the *dark red* subclavian artery (cut), whereas the scalenus anterior muscle lies *in front* of the artery. The artery passes over the first rib between these two muscles. Note how high into this region the dome of the pleura extends, where it is vulnerable to needle penetration.

muscle passes over and in front of the artery (Fig. 20.3).[20]

Supplemental References

As seen from the front, other authors have illustrated the scalenus anterior,[10,13,20,46] the scalenus medius,[10, 20, 21] the scalenus posterior,[10,13,21] and the scalenus minimus muscles.[2,20] The three major scalene muscles are shown from the side.[14] A **posterior view shows the scalenus medius and scalenus posterior.[15] The three major scalene muscles are seen in cross section at the C_5 level in Figure 16.8 of this volume.**

3. INNERVATION

All the scalene muscles are innervated by motor branches of the anterior primary divisions of spinal nerves C_2 through C_7,

according to the segmental level of muscular attachment.

4. FUNCTION

These muscles function to stabilize the cervical spine against lateral movement, and they serve a primary role in respiration.

Fixed From Below

Acting unilaterally, the scalene muscles laterally flex the cervical spine[11,30,55] and, when stimulated, they flex the head obliquely forward and sideways.[18] All four scalene muscles are poorly placed to influence rotation of the neck significantly. Acting bilaterally, the anterior scalene muscles assist in neck flexion.[10,30] The much flatter angle of the scalenus posterior makes it especially suited to stabilizing the base of the neck by preventing, or controlling, side sway, in a manner similar to the lowest diagonal fibers of the quadratus lumborum muscle at the base of the lumbar spine.

Fixed From Above

Clearly, the scalene muscles have long been recognized as important auxiliary muscles of respiration and are more commonly used for respiration than are the sternocleidomastoid muscles.[8,40] Electromyographic and muscle stimulation evidence supports a primary function, not just an accessory inspiratory function.[6,19] The scalenes are active in normal quiet inhalation.[17] Scalenotomy causes an immediate decrease in the vital capacity, but considerable recovery occurs later.[8] When present, the scalenus minimus too should be effective for inhalation, which may explain its hypertrophy in some persons. The scalene muscles often contract (possibly unnecessarily) when people carry, lift, or pull heavy objects. The scalene muscles may respond to provide stabilization.

5. FUNCTIONAL UNIT

The scalene muscles on one side are synergistic with each other and with the sternocleidomastoid for sidebending (lateral flexion) of the neck. They are assisted in this function by a few posterior cervical muscles including the longissimus capitis and the multifidi. During inhalation they are synergistic with the diaphragm and intercostal muscles and are assisted by both divisions of the sternocleidomastoid muscle.

During labored breathing, the upper trapezius,[8] levator scapulae, and omohyoid muscles can assist inhalation by elevation of the shoulder that helps to lift the weight of the shoulder girdle off the chest wall. The pectoralis minor muscle has a synergistic myotatic function with the scalene muscles for elevation of the ribs when the scapula is stabilized.[8] The contralateral scalene muscles are antagonists for lateral flexion and are likely to be synergists for stabilization.

6. SYMPTOMS

Referred pain from the scalene muscles, especially from the scalenus anterior, is seen frequently among patients with shoulder and upper limb pain syndromes.[40] Nearly half of several classes of physical therapy students had tender scalene muscles due to latent TrPs on at least one side.[48] This is considerably higher than the 11% prevalence reported by Sola *et al.*[62] in a population of Air Force inductees.

The **scalenus anticus (anterior scalene) syndrome** was identified as early as 1935 by pain in the anterior or posterior aspect of the arm and at the upper medial border of the scapula, as well as by the tenderness of the muscle to palpation.[40,47,50] In 1942, Travell, *et al.*[70] reported signs caused by scalene TrPs that included venous obstruction, vasomotor changes, and, if the syndrome was severe, evidence of arterial insufficiency with compression of the motor and sensory nerves of the affected arm. Ochsner *et al.*[50] attributed the symptoms of the syndrome to contraction and spasm of the muscle, which abnormally elevated the first rib. The scalene source of the symptoms was confirmed by the finding, in all operated cases, of an overdeveloped, spastic, and stiffened scalenus anterior muscle with sudden and marked descent of the first rib following surgical division of the muscle. True spasticity was not distinguished from contracture and taut bands of TrPs. The literature is clear that a scalene muscle problem is primarily responsible for neural or vascular entrapment in many patients who are commonly diagnosed as having a thoracic outlet syndrome (see Sec-

tion 11 of this chapter for review of this subject). However, it usually fails to identify the cause of the muscle problem.

Relief of pain by infiltrating the scalene muscles was used by Adson[1] as a diagnostic test to distinguish the scalenus anterior syndrome from structural causes of cephalobrachialgia. The TrP nature of the syndrome was not recognized. After an initial wave of enthusiasm for scalenotomy following Adson's report, interest waned as emphasis shifted to the carpal tunnel syndrome and to radiculopathy from nerve root compression by a protruded cervical disc. As the over-enthusiasm for these diagnoses fades, the abundant evidence that the scalenus anterior tension causes serious compressive syndromes in many patients is regaining attention. Scalenus anterior TrP tension secondary to compression of a cervical nerve root supplying that muscle may produce additional neurocirculatory signs that overshadow the typical clinical features of discogenic disease.

The back, shoulder, upper limb, and chest pain patterns characteristic of scalene TrPs are described in Section 1. When the patient complains of pain in the upper back just medial to the superior angle of the scapula, the most likely myofascial source is a scalene TrP. Patients with scalene TrPs sometimes speak of their "shoulder" pain while rubbing the upper half of the arm. Sleep is often disturbed by pain. When night pain is severe, the patient is likely to sleep sitting up on a sofa or propped up on pillows for relief. This helps to prevent the sustained shortening of the scalene muscles that tends to occur when the patient lies flat and the chest and shoulders ride up around the neck during sleep.

Neurological symptoms of numbness and tingling in the hand (chiefly in the ulnar distribution) and the unexpected dropping of objects from the hand can result from entrapment of the lower trunk of the brachial plexus as it exits the thorax by hooking over the first rib.

Edema of the hand, when present, appears diffusely distal to the wrist, particularly over the bases of the four fingers and dorsum of the hand. Patients are likely to experience puffiness of the dorsum of the hand, stiffness of the fingers, and tightness of rings on fingers, especially in the morning on awakening. When these are due to scalene TrPs, the symptoms are likely to be caused by entrapment of the subclavian vein and/or lymph duct as they pass across the first rib in front of the attachment of the scalenus anterior. The puffiness disappears later in the day. The associated stiffness of the fingers is not due solely to the edema, but also to myofascial tautness of the finger extensors, which may have an autonomic reflex component. A test for this stiffness is illustrated later in Figure 20.6.

Scalene TrP activity alone causes minimum restriction of neck rotation, whereas active TrPs in the levator scapulae and splenius cervicis muscles markedly limit cervical rotation.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Scalene TrPs may be activated by the following:

Accidental trauma
Pulling or lifting (when hauling ropes in sailing)
When handling and riding horses
Playing a game of tug-of-war
Participating in competitive swimming[22]
While carrying awkwardly large objects
Playing certain musical instruments
Overuse of these respiratory muscles in paradoxical breathing
Hard paroxysms of coughing (due to allergy, pneumonia, bronchitis, asthma, or emphysema)
Sleeping with the head and neck low when the head of the bed is slightly lower or level with the foot of the bed (as when a thick rug is placed only under the foot of the bed)
A tilted shoulder-girdle axis due to a lower limb-length inequality when standing
A small hemipelvis when seated
Loss of an upper limb or surgical removal of a heavy breast
Idiopathic scoliosis
An awkward leaning position assumed when seated in order to compensate for short upper arms that do not reach the armrests of most chairs, or assumed because of a need to position the head to look at someone.[69]

Apparently and understandably, a whiplash-type injury from a motor vehicle accident is likely to activate TrPs in the scalene muscles. Eighty-one percent of whiplash patients with a pain complaint had at least one active scalene TrP.[27] Although no specific control data of pre- accident incidence are available, and although scalene TrPs are common, it is very unlikely that 80% of those experiencing this trauma already had been suffering pain from *active* TrPs in their scalene muscles.

Scalene TrPs are often activated secondary to TrPs in the sternocleidomastoid (SCM) muscle, with which the scalene muscles form a functional unit. The severe "stiff neck" syndrome of the levator scapulae muscle sometimes includes active scalene TrPs.[68]

The scalene muscles can be affected by anything that produces a severe deviation from the normal pattern of gait. Limping on a weightbearing limb (with resultant torso adjustments) and lack of normal push-off at the end of the stance phase can activate TrPs in the scalene (and levator scapulae and sternocleidomastoid) muscles because those muscles contract excessively in their reflex attempt to "help the movement" and/or maintain equilibrium.

When any of these activating conditions persist, they can also perpetuate scalene TrPs, as can any of the systemic perpetuating factors (*see* Chapter 4).

8. PATIENT EXAMINATION
(Figs. 20.4-20.6)

Patients with a scalene myofascial pain syndrome tend to move the arm and neck restlessly, as if trying to relieve a "sore" muscle. Lateral bending of the neck to the opposite side is usually restricted by at least 30°. Neck rotation is painful only at the extreme range of motion to the same side, especially when the chin is then dipped down toward the shoulder, as described below for the Scalene-cramp Test. Scalene involvement itself causes no restriction of motion at the glenohumeral joint, and pain is not significantly increased by tests of shoulder motion. However, horizontal abduction at the shoulder may be limited by *associated TrPs* in the pectoral muscles.

Cervical range of motion should be assessed, using appropriate caution to avoid vertebral artery compromise. While the subject's neck is sidebent, the examiner should slowly and gently move the patient's head and neck into varying degrees of rotation. This maneuver often brings out a patient response of added pain or a "tight feeling." If the patient is then instructed to point to the troublesome area, the examiner can use that as a starting point to palpate for TrPs.

Scalene-cramp Test
(Fig. 20.4)

To perform this test, the patient rotates the head fully to the side of the pain and actively pulls the chin down into the hollow above the clavicle by flexing the head and neck (Fig. 20.4). During the last part of this movement, the anterior and middle scalene muscles strongly contract while in the shortened position, which evokes a local cramp-like pain in the region of the TrP and may further activate the TrP causing continuing moderate or severe pain referred from it. This pain can be relieved by prompt appli-

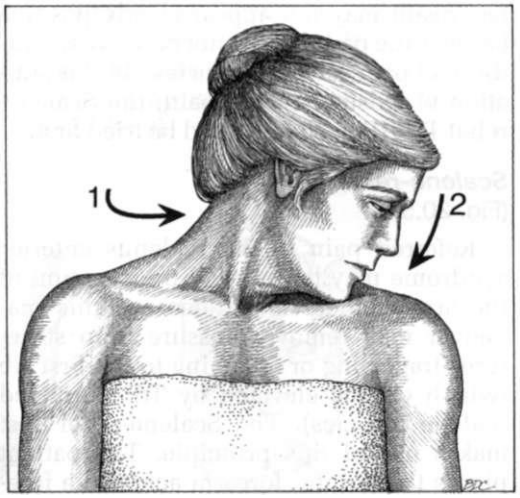


Figure **20.4.** The Scalene-cramp Test elicits or increases pain from active trigger points in the scalene muscles. *1,* the head rotates fully to the left side to test left scalene muscles. 2, the chin dips down into the hollow behind the clavicle. This hard contraction in the shortened position of scalene muscles (with active trigger points) causes a local ache at the TrP and pain that may be referred to a distance, as illustrated in Figure 20.1.

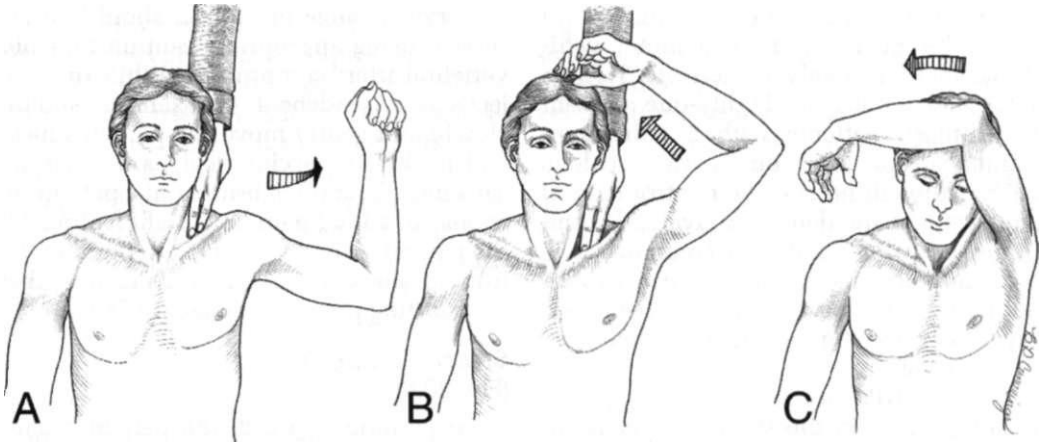


Figure 20.5. The Scalene-relief Test helps to identify a scalene TrP source of referred pain that is caused or aggravated by clavicular pressure on the nerves passing over the elevated first rib or on an involved muscle. **A,** examiner's fingers demonstrate tightness of the space between the clavicle and scalene muscles. **B,** the fingers demonstrate the increased clearance behind the clavicle provided by raising the shoulder and arm. **C,** clearance beneath the clavicle is maximized by swinging the shoulder forward, which protracts the scapula and pivots the clavicle forward and upward to fully relieve clavicular pressure on thoracic outlet structures. Pain relief by this test should occur immediately or within a few minutes.

cation of spray and stretch to the activated TrP. If the patient was already in severe pain before attempting the test movement, the test result may not appear clearly positive because the patient does not perceive the additional pain caused by the test. In this situation of existing severe pain, the Scalene-relief Test (Fig. 20.5) should be tried first.

Scalene-relief Test
(Fig. 20.5)

Referred pain of the scalenus anterior syndrome may be relieved by elevation of the arm and clavicle,[50] because this maneuver may remove pressure from structures traversing or attaching to the first rib (which can be elevated by TrP-shortened scalene muscles). The Scalene-relief Test makes use of this principle. The patient places the painful forearm across the forehead while raising and pulling the shoulder *forward* to lift the clavicle off the underlying scalene muscles and brachial plexus (Fig. 20.5C). Pain relief, when it occurs, ensues immediately or within a few minutes. The examiner's two fingers in Figure 20.5A and B demonstrate how the movement increases clearance beneath and behind the clavicle. None of the positions in Figure 20.5 should affect pain due to cervical radiculopathy.

Finger-flexion Test
(Fig. 20.6)

To be valid, this test of finger flexion must be performed with the metacarpophalangeal (MCP) joints actively held straight, in full extension. This position requires forceful contraction of the extensor digitorum muscle, but the tightly closed fist does not. The test is normal when the fingertips can firmly touch the volar pads of the MCP joints (Fig. 20.6A). If one or more compartments of the extensor digitorum muscle harbor active TrPs, each corresponding finger fails to flex completely. Figure 20.6B shows a positive test for TrPs in the extensor of the index finger. Voluntary hyperextension of the MCP joints strongly loads the finger extensors, increasing the activity of these TrPs. This TrP activity apparently reflexly limits simultaneous distal interphalangeal (DIP) flexion by inhibiting the corresponding finger flexor.

The test also is positive when active TrPs are present in the scalene muscles. In this case, all four fingertips may fail to touch the MCP volar pads (Fig. 20.6C).

However, there is no difficulty in making a tight fist when the MCP joints are allowed to flex. Apparently, TrPs in the scalene muscles similarly inhibit finger flexors when the MCP joints are extended. Scalene muscle TrPs are frequently the *key* to forearm extensor digitorum TrPs. The referred motor effects of TrPs frequently are independent of, and can affect different locations than, referred sensory effects.

A positive test is not simply due to edema, since this test of distal interphalangeal flexion is frequently restored to normal immediately after stretch and spray of the involved scalene muscles. Furthermore, edema is more likely to occur only with involvement of the scalenus anterior, whereas active TrPs in any of the scalene muscles may be responsible for an abnormal Finger-flexion Test.

9. TRIGGER POINT EXAMINATION
(Figs. 20.7, 20.8 and 20.9)

In the authors' experience, the scalene muscles harbor active TrPs in the following order of frequency: anterior, middle, posterior, and minimus. To determine the most useful diagnostic criteria, Gerwin *et al.*[23] tested the reliability with which four experienced physicians following a *3-hour training session* could identify five characteristics of TrPs in five pairs of muscles in 10 subjects. They found the most reliable diagnostic criteria to be the detection of a taut band, the presence of spot tenderness, the presence of referred pain, and reproduction of the patient's symptomatic pain. Manual determination of the presence or absence of a local twitch response (LTR), although very helpful diagnostically when observed, was highly reliable only in the most accessible and readily palpated muscles. The scalene muscles were not included in this study. The LTR is moderately difficult to elicit manually in the anterior and middle scalene muscles and very difficult in the posterior scalene muscle. It can be depended on only as a diagnostically confirmatory finding. Local twitch responses are, however, characteristically elicited when a needle encounters the TrP.

When trying to locate the anterior and middle scalene muscles, it is helpful to remember that the digitations of the anterior scalene muscle attach to the anterior tubercles of cervical vertebrae, the brachial plexus emerges between the anterior and posterior tubercles, and the fibers of the middle scalene muscle attach to the posterior tubercles. The brachial plexus descends in a palpable groove between the two muscles and becomes progressively more superficial to emerge from between the two muscles in order to exit the neck and thorax by crossing over the first rib (*see* Fig. 20.9).

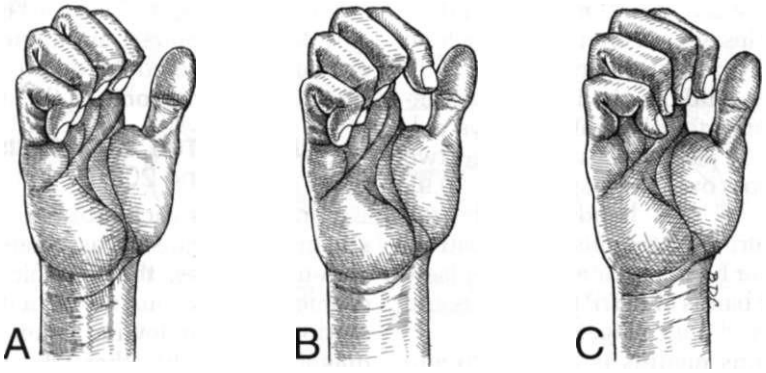


Figure 20.6. Finger-flexion Test with the proximal phalanges extended. **A,** normal finger closure with *all* fingertips pressed tightly against the metacarpophalangeal joint volar creases. **B,** positive extensor digitorum test. Incomplete index finger flexion can indicate a trigger point in the part of the extensor digitorum muscle that controls the index finger, or in the extensor indicis muscle. **C,** positive scalene test. Incomplete flexion of all fingers may indicate more general involvement and inhibition of the long flexor muscles, which can occur when scalene trigger points are active on the same side.

The TrPs in the scalenus anterior are found by palpating the muscle behind the posterior border of the clavicular division of the sternocleidomastoid muscle (Fig. **20.7).** The posterior sternocleidomastoid border can be approximated by locating and briefly occluding the external jugular vein with finger pressure just above the clavicle (Fig. **20.8A).** This vein usually crosses the scalenus anterior muscle at about the level of its active TrPs. A simpler method may be to identify the sternocleidomastoid muscle (on the right side, for example) by palpation while resisting sidebending of the head and neck toward the ipsilateral (right) side with the face turned to the contralateral (left) side.

If the inferior belly of the omohyoid muscle has a tender TrP and taut bands, it can easily be mistaken for the anterior scalene, although these muscles have different fiber directions. The omohyoid muscle is more superficial than the scalene muscles, comes out from behind the sternocleidomastoid muscle, and crosses diagonally over the anterior scalene muscle[12] (Fig. **20.7** and *see* Chapter **12).** It can cross at about the same level as the scalene TrPs depending on which scalene digitation is involved and depending on head position.

The **scalenus anterior** can be identified by positioning the patient's head to take up any slack in the muscle and then palpating its anterior and posterior borders (Fig. **20.8).** Its posterior border is confirmed by locating the groove between the anterior and middle scalene muscles, which cradles the brachial plexus bundle of nerve fibers (Fig. **20.9).** In this groove, behind the clavicle, the pulsating subclavian artery is nearly always palpable where it passes between these two muscles to cross over the first rib (Fig. **20.9).** The fingers of one hand straddle the scalenus anterior to establish its location, while the other hand palpates and precisely localizes taut bands and TrP tenderness and induces referred pain. (Fig. **20.8B).**

The **scalenus medius** is parallel to and on the posterior side of the groove described above that contains the bundle of brachial plexus nerve fibers. It is larger than the scalenus anterior and lies anterior to the free border of the upper trapezius (Fig. **20.7).** It can be palpated against the posterior tubercles of the transverse processes of the vertebrae, to which its digitations are attached.

The **scalenus posterior** is difficult to reach. It lies more horizontal than, and dorsal to, the scalenus medius. It passes anterior to the levator scapulae, which must be pushed aside at the point where the levator scapulae emerges near the anterior free border of the upper trapezius (Fig. **20.7).** Finding TrP tenderness requires palpation posterior to the scalenus medius and to the depth of the first rib.

Scalenus minimus TrP activity is usually discovered only after inactivation of TrPs in the other scalene muscles. Involvement of this variable muscle is then recognized by residual tenderness deep to the mid-portion of the scalenus anterior (*see* Section **2).**

10. ENTRAPMENT

A primary entrapment of nerve fibers traversing one of the scalene muscles has been reported, but is relatively rare. However, if the muscle develops active TrPs in this situation, the increased tension of the taut bands might cause neurological symptoms. Much more common is the secondary entrapment effect (from the elevation of the first rib) as neurovascular structures exit the thoracic outlet. Entrapment of the lower trunk of the brachial plexus is commonly due to TrP tautness of the scalenus anterior and the scalenus medius. This entrapment causes ulnar pain, tingling, numbness, and dysesthesia. Trigger point activity in the scalenus anterior often causes hand edema. Additional secondary entrapment phenomena are considered in detail under **Thoracic Outlet Syndrome** in the next section.

11. DIFFERENTIAL DIAGNOSIS
(Figs. 20.10, and 20.11)

Because it is such an important diagnosis and is so closely associated with the scalene muscles, the thoracic outlet syndrome (TOS) is considered in detail in this section after reviewing other differential diagnoses. Finally, other TrPs that are associated with scalene TrPs are identified.

The **carpal tunnel syndrome** may occur as a concurrent entrapment with the TOS, or the symptoms of a carpal tunnel syndrome may be caused by scalene TrPs. Loss of normal mobility of the structures forming the carpal tunnel often make a major

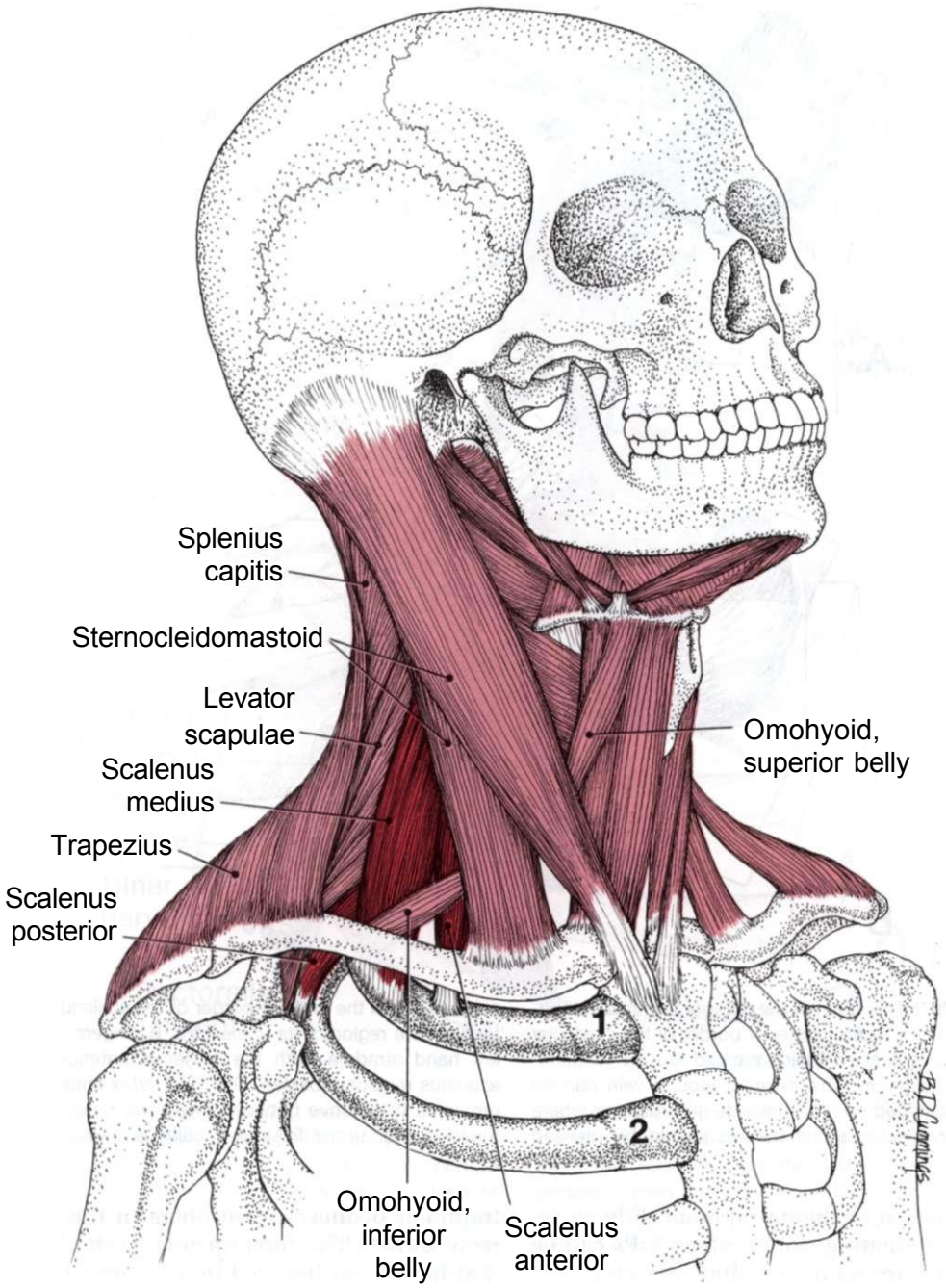


Figure 20.7. Neighboring muscles *(medium red)* that are useful landmarks in locating the scalene muscles *(dark red).* The inferior belly of the omohyoid muscle is easily mistaken for the anterior scalene muscle although they do not have the same fiber direction. It is superficial and is located where one could expect to find the scalene muscle.

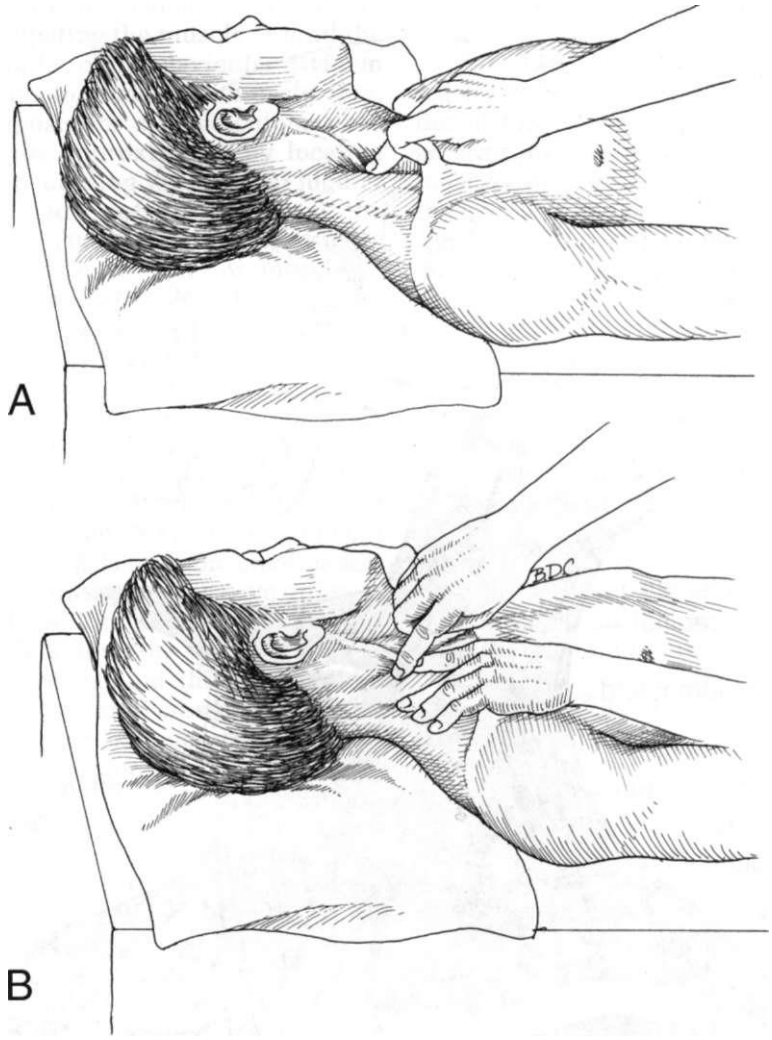


Figure 20.8. Palpation of the anterior and middle scalene muscles. **A,** the posterior border of the clavicular division of the sternocleidomastoid muscle is identified by palpation. The external jugular vein can be briefly occluded so that it stands out, marking where the sternocleidomastoid muscle should be pressed aside to reach the anterior border of the scalenus anterior in the region of its usual TrPs. **B,** fingers of the left hand straddle both the scalenus anterior and scalenus medius muscles. The right index finger approaches the groove between these two muscles at the level of an upper TrP in the scalenus medius.

contribution to the entrapment. Edema reflexly originating from scalene TrPs can be another important contributing factor.

A **C_5-C_6 radiculopathy** can produce a pain complaint very similar to that reported by patients with active scalene TrPs. Both may be present because the neuropathy encourages the development of forearm TrPs that refer pain to the wrist region, and middle scalene TrPs may be causing entrapment of these nerve fibers in the thoracic outlet. The forearm myofascial TrPs that have been induced by the nerve compression, are likely to persist following successful surgery to relieve the radiculopathy; in this case the forearm TrPs must be inactivated for lasting relief of symptoms.

Anterior and/or middle scalene TrPs are commonly associated with **C_4, C_5, and C_6 articular dysfunctions.** Treatment employs

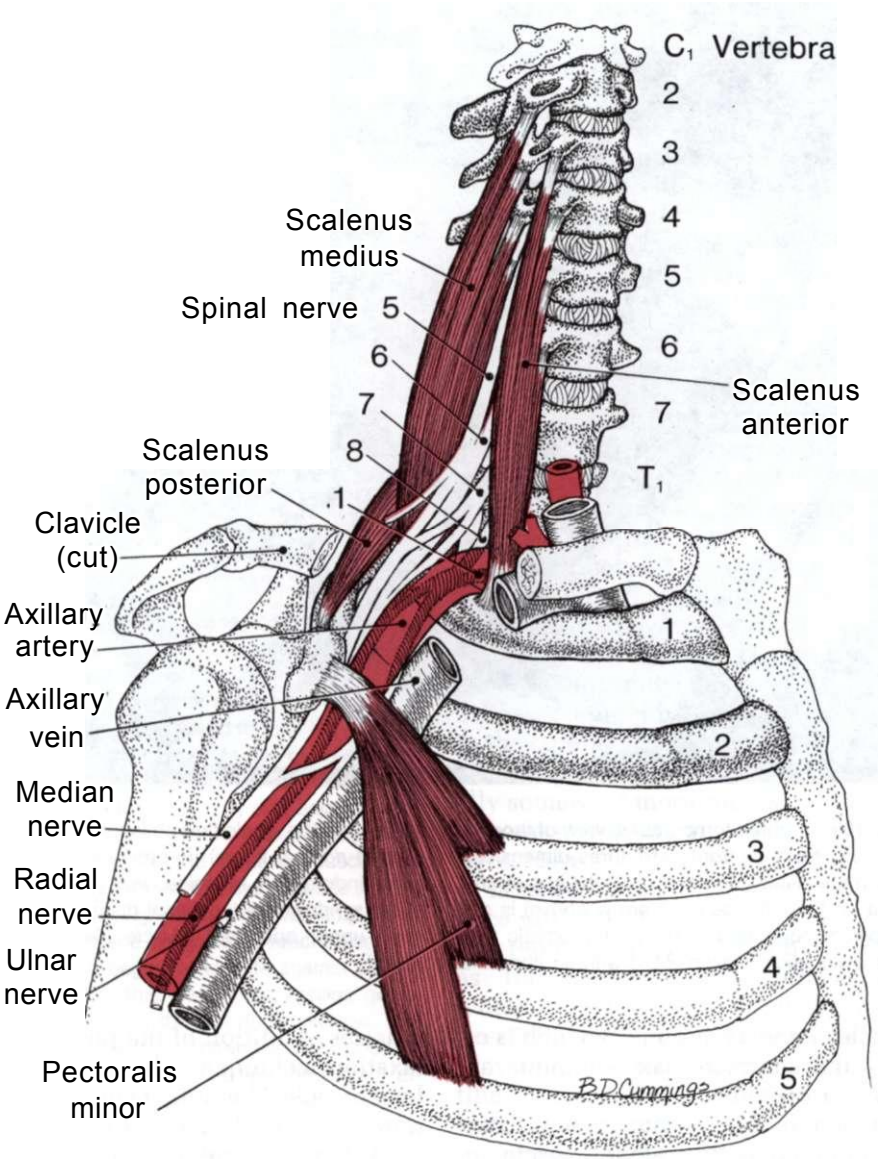


Figure 20.9. Thoracic outlet entrapment by the *medium red* scalene muscles. The neurovascular bundle is spread out to show the relations of its component parts. A portion of the clavicle has been removed. The brachial plexus and *dark red* subclavian artery emerge above the first rib and behind the clavicle between the scalenus anterior and scalenus medius muscles. The spinal nerves are numbered on the *left,* the vertebrae on the *right.* The T, nerve lies dorsal to and beneath the subclavian artery. These structures crossing over the first rib can be compressed when the rib becomes elevated. Trigger points in the scalenus anterior and/or the scalenus medius are associated with taut bands that increase muscle tension and elevate the first rib, compressing the neurovascular structures.

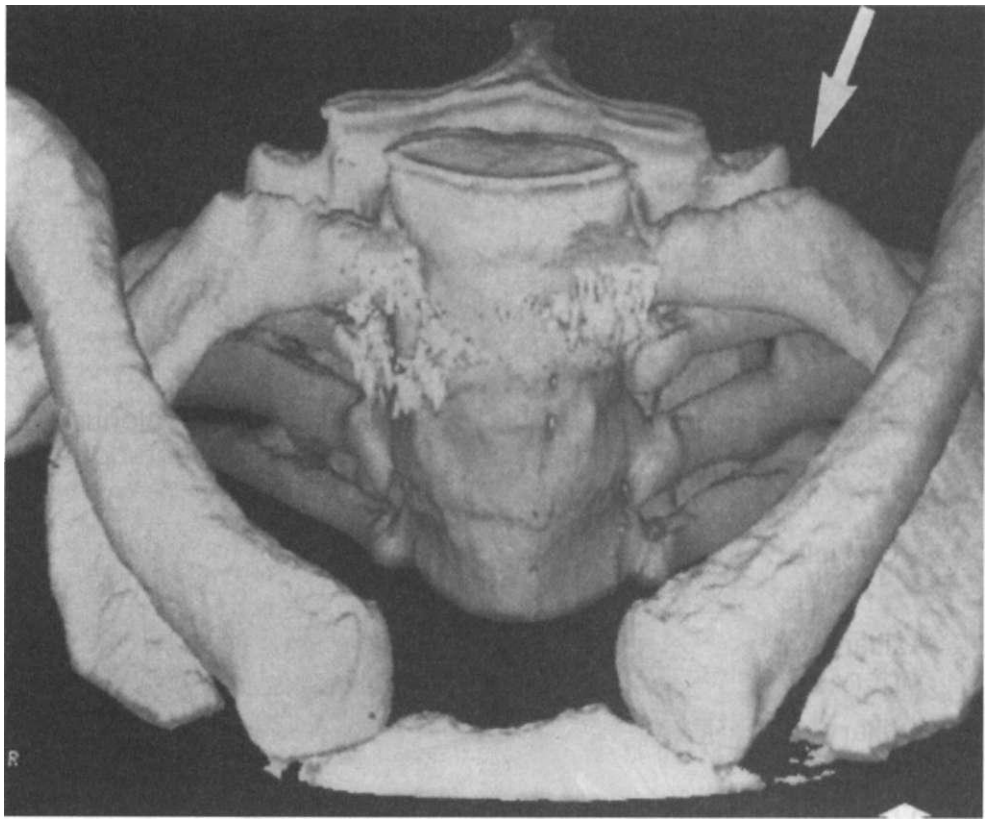


Figure 20.10 Computed tomographic view of thoracic outlet viewed from in front with three-dimensional shaded surface display. The first rib at the costotransverse joint on the left side *(long arrow above)* is displaced upward compared to the asymptomatic right side. This dislocation is associated with an abnormal position of the whole first rib *(short white arrow at the bottom right* of figure) (Reproduced with permission from Lindgren KA, Manninen H, Rytkonen H: Thoracic outlet syndrome-a functional disturbance of the thoracic upper aperture? *Muscle Nerve* 78/526- 530, 1995.)

the muscle energy technique,[24] which is essentially the contract-relax technique applied to mobilize joints.

An **articular dysfunction** that is commonly observed with scalene muscle involvement is elevation of the first rib (Fig. 20.10). Because of the first rib's location under the clavicle, it can be palpated most easily at the head of the rib, posteriorly, at its articulation with the first thoracic vertebral segment. The technique for treating first rib fixation and elevation is shown in Figure 20.11. The clinician applies pressure to an elevated first rib on the right side with the right thumb beneath the upper trapezius muscle over the head of the first rib. This technique also helps to release any remaining TrP tightness of the scalene muscles. Addition of the postisometric relaxation technique helps to relax the scalene muscles. Including controlled respiration as part of the technique augments relaxation and release.

An **apparent** elevation of the firs rib concurrent with T_1 articular dysfunction" may result from rotation of the vertebra by a longissimus capitis muscle *(see* Chapter 16) that is shortened from TrP tension. This muscle, through its attachment to the transverse process, may indirectly affect the first rib through its pull on the costotransverse junction.

Thoracic Outlet Syndrome

The thoracic outlet syndrome is a collection of symptoms. Like low back pain, it

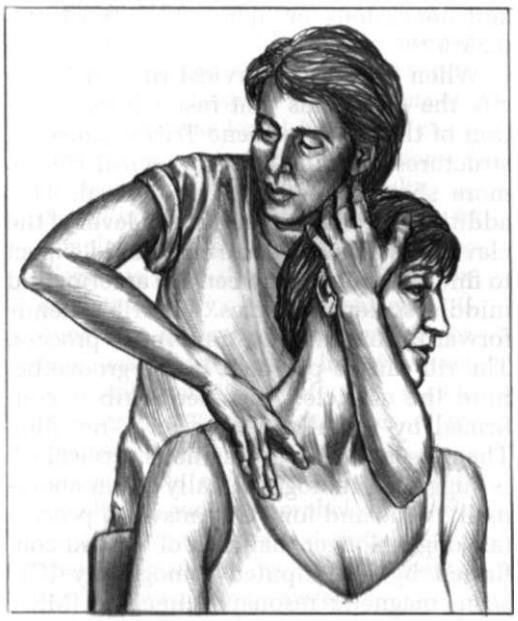


Figure 20.11. Technique to release an elevated first rib and/or release anterior and middle scalene muscles. Treatment is performed with the patient in the seated position and with the examiner's left arm cradling and fully supporting the patient's head to treat the right anterior and middle scalene muscles and/or an elevated right first rib. The examiner's right thumb palpates and monitors the head of the first rib by displacing the upper trapezius posteriorly, while the examiner's other hand takes up slack in the scalene muscles by sidebending the patient's neck to the opposite side (to the left side here, for right-sided involvement). When the end point of scalene elongation is reached and the head of the first rib begins to elevate against the examiner's right hand, that hand then applies gentle downward pressure over the first rib for release. This release is combined with a contract-relax (or postisometric relaxation) technique whereby the patient attempts right sidebending of the neck against light resistance provided by the examiner's left hand, followed by relaxation. Full release is accomplished through sidebending of the neck with fine tuning, utilizing rotation to isolate the precise muscle fibers that are shortened. The patient facilitates the release first by breathing in and looking upward to the right, which also contracts the right scalene muscles. During the relaxation phase, the patient looks down to the left and breathes out; the examiner takes up slack by bringing the patient's scalene muscles to the new length barrier, while maintaining gentle downward pressure on the head of the first rib. This procedure can be repeated 3 to 5 times for complete release of the scalene muscles and treatment of the elevated first rib. The area can be presprayed with vapocoolant as shown in Figure 20.12.

is not a well-defined diagnosis but often is reported as if it were a specific disease. Abnormal tension of scalene muscles is frequently implicitly or explicitly identified as responsible for the symptoms of TOS, but why the muscle has become abnormally tense remains enigmatic in most of the current TOS literature. Myofascial TrPs are not considered in that literature. The TOS is a source of much frustration and controversy, partly because there is no one clearly recognized set of symptoms that define it. The variety of etiologies that are proposed is a major source of confusion.[39]

The following literature review and commentary makes it clear that surgeons are frustrated because only about half of operative interventions for TOS are successful. Some are dramatically successful and some are disastrously unsuccessful. There is little agreement as to how one can reliably predict the postoperative outcome. Apparently a piece of the puzzle is missing. The fact that a major contributing cause for the pain and entrapments-myofascial TrPs-is commonly overlooked contributes to the confusion and frustration.

Nonoperative interventions are generally somewhat more successful than operative ones, with much less danger to the patient. However, there remains a large group of nonoperative patients who also experience unsatisfactory results. The nonoperative reports often identify physical therapy as the treatment, but rarely mention whether that included specific examination for TrPs, and if TrPs were found, how they were treated. Unfortunately, no scientific studies were found that critically tested the myofascial TrP approach as a nonoperative intervention. Such a study is urgently needed and would require experienced and trained examiners.[23]

Because the thoracic outlet is anatomically the superior border of the thorax, authors have included a variety of syndromes and conditions that they have identified as TOS. A number of issues are considered individually here: definition of TOS, cervical rib and other developmental anomalies, scalenus anticus (anterior) syndrome, myofascial pseudothoracic outlet syndrome, costoclavicular syndrome, diagnostic considerations, and treatment.

Definition of Thoracic Outlet Syndrome. A medical dictionary[45] defines thoracic outlet syndrome as "compression of brachial plexus and subclavian artery by attached muscles in the region of the first rib and clavicle," which reflects the usual structures that receive primary clinical attention. The anatomical relations of these structures is illustrated in Figure 20.9 (from which a portion of the clavicle has been removed). Both the brachial plexus and the subclavian artery emerge through the interscalene triangle bounded by the anterior and middle scalene muscles and the first rib, where nerves of the brachial plexus and the subclavian artery pass over the first (or rarely cervical) rib. The subclavian vein accompanied by a lymphatic duct passes over the first rib anterior (medial) to the attachment of the anterior scalene muscle. Entrapment symptoms may be of neural, vascular, and/or lymphatic origin.

The lower trunk of the brachial plexus is formed from spinal nerves C_8 and T_1. The T_1 nerve exits the spinal foramen between the first and second thoracic vertebrae, and courses cephalad to hook over the first rib where its fibers and those of the C_8 spinal nerve are wedged between the subclavian artery and the rib attachment of the scalenus medius. When positional changes or TrP activity in the scalenus anterior or medius elevate the first rib, fibers of the lower trunk must angulate more sharply over it. This elevation also wedges the subclavian artery more tightly against the lower trunk.

Cervical Rib and Other Developmental Anomalies. Over the past 50 years a remarkably extensive literature has accumulated describing in exquisite detail a myriad of minor and some major anatomical variations that can occur at the thoracic outlet. These variations have received so much attention because their presence, or suspected presence, is commonly used as justification for surgery.

Although cervical ribs and deformed first ribs are dramatic radiographic findings, they are relatively rare; among 40,000 consecutive chest X-ray examinations of army recruits, completely articulated cervical ribs were found in 0.17% and anomalous or deformed first ribs in 0.25%.[43]

When present, a cervical rib can intensify the symptoms that result from elevation of the rib by scalene TrPs because all structures crossing over a cervical rib are more sharply angulated than usual. This additional rib is palpated at the level of the clavicle as a bulge where one would expect to find the groove between the anterior and middle scalene muscles. The rib extends forward from the C_7 transverse process. The rib can be palpated in the groove behind the clavicle. An osseous rib is confirmed by visualization in an X-ray film. The presence of a cartilaginous cervical rib is suggested radiographically by an abnormally wide and long C_7 transverse process (as long or longer than that of T_1) and confirmed by a computed tomography (CT) scan, magnetic resonance imaging (MRI), or ultrasound imaging. Sometimes, a vestigial cervical rib that consists of a fibrous band may have a sharp edge that needs to be excised.

A number of congenital anomalies would be expected to increase the likelihood of entrapment at the thoracic outlet. A congenital abnormally narrow space between the attachments of the two scalene muscles at the first rib will restrict the opening and make the neurovascular structures more vulnerable to compression. An additional space-occupying structure, such as an accessory muscle or fibrous band, that passes through the interscalene triangle will have the same effect. Fibrous sharp edges of the scalene muscles or fibrous bands bordering or within the interscalene triangle can make components of the brachial plexus more vulnerable to compression damage. It is an interesting observation that TOS usually first appears in early adulthood[38] (when individuals begin to develop active TrPs) and that patients with documented cervical ribs have obtained relief from symptoms of thoracic outlet entrapment with inactivation of their scalene TrPs.

Regardless of the presence or absence of congenital anomalies, patients with more severe symptoms seem more likely to benefit from surgery than patients with less severe symptoms.

Makhoul and Machleder[43] analyzed **200** consecutive surgically treated cases of TOS for developmental anomalies and reviewed the literature. A developmental abnormality was found in **66%** of cases, higher than in unselected populations. A cervical rib or first rib abnormality appeared in 8.5% of cases. A C_7 rib may be complete or only a rudimentary fibrocartilaginous band detectable on X-ray examination only by an enlarged C_7 transverse process. Supernumerary scalene muscles were found in **10%,** developmental variations of the scalene muscles in 43%, and variations of the subclavius muscle in 19.5% of cases. However, the only correlation between the clinical and the morphologic characteristics was stricture and thrombosis of the subclavian vein due to enlargement of the subclavius muscle system.[43]

Roos[57] evaluated **1,120** operations for TOS and found 9 types of soft-tissue anomalies not visible on X-ray examination. The fibrous bands that were most commonly found at operation in patients with severe TOS were associated with nerve roots of the brachial plexus.

Scalenus Anticus (Anterior) Syndrome and First Rib Involvement. The term scalenus anticus syndrome originated with surgeons[50] who were convinced that increased tension of the anterior scalene muscle was responsible for the entrapment of neurovascular structures in the interscalene triangle that caused the patient's symptoms. The reason for this increased muscle tension remains enigmatic in the current surgical literature. The scalene muscles are identified by clinicians as prone to hyperactivity and increased muscle tension,[33] which likely reflects a reflex response of scalene muscles with TrPs to the TrP activity in other muscles. The scalenus anticus syndrome is often considered synonymous with TOS as shown by the dictionary definition of TOS.[45]

Lewit[33] observed that blockage (immobility) of the first rib goes hand in hand with reflex spasm (increased tension) of the scalene muscle on the same side, which is abolished by treatment of the first rib. Whether his observed "spasm" is truly reflex spasm or muscle tension from TrP contracture of the muscle fibers must be resolved by electromyographic studies, which have not been reported. The question arises because the technique he describes for releasing the first rib also would be expected to release TrPs in the scalene muscles.

Lindgren has written a series of papers over a period of 8 years[34,35,37,39] that emphasizes the important relation between the TOS and dislocation or subluxation of the first rib. He presented imaging of a first rib dislocation[38] (Fig. 20.10) that shows *elevation* of the head of the first rib at the costotransverse joint. The treatment, which he found to successfully restore normal relations of the first rib (and relieve the patient's symptoms) and which he illustrated and described clearly,[36] is essentially an isometric contract-relax technique specifically for the three scalene muscles. The contract-relax technique he used is a well-recognized and effective treatment for release of scalene TrPs. This raises the question of whether the manipulation- release of first rib elevation and immobilization isn't primarily a matter of effectively inactivating scalene TrPs and releasing the abnormal tension that they induce in the scalene muscles. One would expect that downward pressure applied to the posterior portion of the first rib as the scalene tension is released would facilitate restoration of the normal anatomical relations at the costotransverse joint.

Thomas *et al.*[67] emphasized the middle scalene muscle as being just as important as the anterior scalene in producing the TOS. Since the middle scalene is usually a larger more powerful muscle and has leverage as good as, if not better than, the anterior muscle for elevating the first rib, the middle scalene muscle likely is more important. Among 108 patients operated on for TOS, 35 had no bony abnormality, but 23% of these patients did have an anterior insertion of the middle scalene that placed the lower trunk of the brachial plexus and the subclavian artery in direct contact with the muscle's anterior margin. This would make the nerves and artery more vulnerable to abnormal sustained tension of the middle scalene caused by TrPs. In a study of 56 cadavers, the lower trunk of the

brachial plexus rested on the inferior portion of the margin of the middle scalene muscle in practically all cases.[67]

Myofascial Pseudothoracic Outlet Syndrome. In addition to the scalene muscles (which can produce true TOS), other muscles can have TrPs that refer pain in locations that mimic TOS symptoms. The four primary muscles that can mimic TOS symptoms, and that are particularly confusing if several of them develop TrPs at the same time, are the pectoralis major, latissimus dorsi, teres major, and the subscapularis muscles. This quadrad is described in detail in Chapter 18. Other authors have identified additional muscles whose TrPs produce symptoms that may be diagnosed as TOS. These include the pectoralis minor,[27, 64, 66] trapezius,[64] and levator scapulae muscles.[64]

Since these muscles all commonly develop TrPs and are infrequently, if ever, examined by surgeons as a likely source of TOS symptoms, it is not surprising that a large percentage of patients operated on for TOS, in whom no anatomical abnormality is found, experience limited benefit from their operations. Also, practitioners of conservative treatment of TOS often overlook the possibility of TrPs in many of these muscles and apply general therapy not specifically directed to the inactivation of identified TrPs. These factors help to account for many of those patients who don't respond well to conservative treatment.

Costoclavicular Syndrome. This syndrome is attributed to compression of the neurovascular bundle between the clavicle and the first rib when the shoulders are drawn backward and down as in the position of a military brace or when carrying a heavy back pack.[29] Any muscle tightness that tends to elevate the first rib would aggravate this syndrome. In addition to the scalene muscles, increased TrP tension of the pectoralis minor[64] can contribute indirectly to first rib elevation when the third through fifth ribs (sometimes also the first and second ribs) are displaced upward.

Makhoul and Machleder[43] reviewed the surgical findings in patients operated on for costoclavicular syndrome and found numerous references to compression of the subclavian vein against the first rib because of enlargement of the subclavius muscle system. An abnormality in this system was found in 19.5% of their 200 surgical TOS patients, and an exostosis at the subclavius tubercle was observed in 15.5% of them (which suggests abnormal increased tension of that muscle).

These observations may relate to the type of first rib dislocation at the costotransverse joint that was discussed above. The subclavius muscle attaches laterally to the middle third of the clavicle and medially to the first rib and its cartilage at their junction.[11] Prolonged shortening of this muscle could produce a force that would tend to elevate the rib, as illustrated in Figure 20.10.

In this connection, it is interesting to note that Greenman[25] illustrates a stretch technique for correcting respiratory dysfunction of the first rib that could be effective for inactivating TrPs in the subclavius muscle with relatively little effect on the scalene muscles. It strongly retracts the acromion (and lateral clavicle) while stabilizing the anterior part of the first rib with no effort to sidebend the neck.

Coracoid Pressure Syndrome. This syndrome is described by Kendall, ef *al.* as "a condition of arm pain in which there is compression of the brachial plexus... [that] is associated with muscle imbalance and faulty postural alignment."[30] Forward depression of the coracoid process tends to narrow the space available for the three cords of the brachial plexus, the axillary artery, and the axillary vein to pass between the attachment of the pectoralis minor (to the coracoid process) and the rib cage. As a cause of the forward and downward tilting of the coracoid process, the authors[30] implicate some muscles that are weak (like the lower trapezius) and some that are tight, chiefly the pectoralis minor. However, those authors[30] do not mention TrPs and their taut bands, which commonly shorten the pectoralis minor muscle and most likely contribute to this syndrome *(see* Chapter 43, Pectoralis Minor).

Diagnostic Considerations. The history and physical examination have proven to be the most useful for making the diagnosis of TOS. Further testing may help to confirm that there is entrapment and may indicate where it is, but usually tells the examiner little about what is causing the entrapment,

which is what the surgeon needs to know. The exception to this is venous entrapment, which implicates the subclavius muscle system. Physical signs may reflect entrapment of the brachial plexus, subclavian artery, subclavian vein, or the lymph duct from the arm. Electrodiagnostic procedures test for compromise of nerve function, and provocative maneuvers are commonly used to detect both arterial and nerve involvement. Neural involvement is reported to be much more common than arterial involvement,[43,57] and the literature rarely mentions venous and lymphatic compromise except in connection with the costoclavicular syndrome.

Commonly recognized **neurological** signs and symptoms of TOS appear chiefly in the ulnar distribution[16,32,58,70] and sometimes (sensory loss) in the territory of the medial antebrachial cutaneous nerve.[16] Entrapment of the lower trunk of the brachial plexus affects nearly all fibers of the ulnar nerve, and some fibers of the median nerve. Patients with this lower trunk compression complain chiefly of numbness, tingling, and dysesthesias in the 4th and 5th digits, ulnar side of the hand, and occasionally of the forearm. Patients show mild hypesthesia to light touch, pinprick and temperature change in the little finger.

The increased angulation of the neurovascular bundle over a cervical rib instead of the first rib will increase its vulnerability to entrapment. An increase in tension caused by myofascial TrPs will likely cause more severe symptoms when a cervical rib is present, but release of the TrPs may also relieve the symptoms they precipitated, if the TrPs have not been allowed to persist for too long a time and if the tension has not produced permanent nerve damage.

Entrapment of the **axillary artery** is more often due to TrP activity and tautness of the pectoralis minor (see Chapter 43) than to TrP activity of the scalene muscles. The artery also may be entrapped by costoclavicular compression and by forward depression of the coracoid process of the scapula, which are often aggravated by a forward-slumped, round-shouldered posture. Since pectoralis TrPs are likely to be associated with scalene TrPs, the arterial flow may suffer a double entrapment where the subclavian artery emerges from the thorax wedged between the first rib and the tendon of the scalenus anterior, and where the axillary artery hooks behind the pectoralis minor muscle (Fig. 20.9).

Compromise of **venous or lymph** drainage due to entrapment of the subclavian vein and/or the lymph duct at the thoracic outlet may cause edema of the fingers and dorsum of the hand, as noted in Section 6. Reflex suppression of peristaltic contractions of the lymph duct due to scalene TrP activity may contribute to the edema. Subclavian vein compression due to TrP tension and shortened scalene muscles has been observed clinically, and in one case[44] the compression between the first rib and the clavicle resulted in a clot which required surgical removal.

Electrodiagnostic tests have been disappointingly unreliable for diagnosing TOS except in more severe cases.[59] On the other hand, electrodiagnostic tests should be negative in the case of myofascial pseudothoracic outlet syndrome. Needle EMG was the most sensitive to a neuropathy caused by TOS, but was positive only in more chronic and severe cases.[51] They found that F-wave testing was the next most sensitive, and nerve conduction studies were of little value or useless, except to diagnose peripheral neuropathies that were suspected of being TOS.[52,59] Other authors did not find F-wave studies[51] or somatosensory evoked potentials (SEP) helpful in diagnosing TOS.[31,51]

Provocative maneuvers, especially for vascular responses, have been disappointingly unhelpful guides for deciding whether an operation will relieve the symptoms or not. Roos[57] reported that the only maneuver which he found helpful was a neurological test that required the patient to hold the hands up with arms abducted to 90% and the elbows bent at 90% as if told to "stick 'em up." A study of 200 normal-population volunteers[56] found that vascular responses were too common to be a reliable indicator of TOS. The Adson maneuver produced 13.5% positive responses (other authors have reported much higher values depending on exactly how the test is performed);[57] the costoclavicular maneuver produced positive responses in 47%,

and the hyperabduction maneuver in 57% of normal extremities.[56] On the other hand, evaluation of neurological responses produced positive results to the Adson maneuver in only 2% of normal extremities; to the costoclavicular maneuver in 10%; and to the hyperabduction maneuver in 16.5% of normal extremities.[56] Identification of the structure(s) suffering compression does not by itself identify the cause of compression. The cause may still be of anatomical or muscular TrP origin. These maneuvers are considerably more reliable as indicators of neurological entrapment than of vascular entrapment.

Therapy for Thoracic Outlet Syndrome. SURGICAL APPROACH. A review of surgical reports[41] noted that surgery for TOS has a controversial reputation and emphasized the importance of understanding neuromuscular physiology for its evaluation. The surgical approach usually aims to eliminate the problem by removing any aberrant structures such as a cervical rib or fibrous bands, but is mainly done on the basis of clinical symptoms. If no anatomic abnormalities are found, which is commonly the case, tenotomy of one or both scalenes forming the interscalene triangle, removal of one or both scalene muscles, or excision of at least the portion of first rib to which these muscles attach is usually performed. Surgery articles claim success rates ranging from 24%-90%[35] depending on the criteria for success, the criteria for what constitutes an operable case of TOS, and the skill of the surgeon.

Lindgren,[35] in charge of a rehabilitation service, reviewed the results of 48 surgeries for TOS and found that less than half of the 20 patients with first rib resections and less than half of the 7 patients receiving cervical rib resections became asymptomatic. Thirteen of the patients with rib resections had residual idiopathic hypesthesia or dysesthesia due to nerve lesions. Occasionally the results can be tragic, especially so in 5 cases where there were significant surgical sequellae, the pain was the only complaint, and little or no clinical abnormality could be demonstrated before operation.[9] There was no indication that a TrP cause of the pain had been considered or investigated.

A pain clinic evaluation of the results of surgery in 32 patients diagnosed as suffering from TOS following a motor vehicle accident showed very good results in less than half.[42] Although musculotendinous or osseous anomalies compromising primarily the lower trunk of the brachial plexus were identified in 87% of patients at operation, it is difficult to see how these anomalies could have been caused by the accident. However, this type of accident very commonly activates TrPs in muscles that produce symptoms of TOS.[3,27] The increased tension, especially of scalene muscles, could cause TrP pain and compression of vulnerable but previously asymptomatic structures.

Although 98% of the surgery patients for TOS reported by Roos[57] had neurological rather than vascular symptoms, only 22% of his total group obtained good relief by surgical decompression. One of his key tests for selecting patients for operation was reproduction of their symptoms by applying supraclavicular pressure [on the scalene muscle] suggesting that they may have had unrecognized scalene TrPs.

When the lower components of the brachial plexus or vessels must pass over a sharp fibrous edge rather than the normally smooth first rib, additional pressure on neurovascular structures by shortened scalene muscles is likely to initiate symptomatic compression. One can only wonder how many of Roos'[57] successfully operated patients and how many of the surgical failures would have been relieved by inactivating scalene TrPs.

Another surgical review[32] of 50 patients who received an extensive, comprehensive evaluation for *possible* TOS could establish the diagnosis of TOS in only 12, of whom 7 were operated on. Only 4 of the 7 obtained complete relief. Of the remaining non-TOS patients with long-term follow-up, 20 (57%) reported good results from physical therapy and nonoperative management. The authors concluded that sufficiently careful selection of patients can yield good results. Most surgical reviews of the TOS end with the admonition to fully explore conservative treatment before resorting to surgery. The nature of the partially successful physical therapy was not stated.

It is noteworthy that there seems to be no satisfactory correlation between the

abnormality encountered or the structure removed surgically and the success of the outcome. Not one of the surgical reports reviewed indicated that patients were examined preoperatively for TrPs that might be making a major contribution to the patients' symptoms.

CONSERVATIVE APPROACH. Again, rates of successful treatment are variable among authors and range from 9% to 83% (and usually are 50% or better). Successful treatment was strongly and inversely related to the severity of symptoms initially.[34]

Conservative treatment for TOS almost always includes a treatment procedure that would be likely to release scalene muscle tightness, usually a stretching exercise[34] or a myofascial release procedure. Both can be effective ways of inactivating TrPs if applied in a suitable manner to release TrPs in the *involved* muscles. Effective management also may need to include correction of poor posture (particularly forward, round-shouldered posture-refer to Chapters 5, Section C and Chapter 41, Section C), elimination of unnecessary activity stress on the muscles, education of the patient on proper care of the muscles, mobilization of articular dysfunctions, and attention to life stresses and coping strategies. A few patients with symptoms of TOS will have anatomical abnormalities that require surgical correction for complete relief.

Members of many disciplines recognize the importance of diagnosing and treating TrPs in patients with symptoms of a TOS. An osteopathic physician[64] reported that in most cases of TOS scalene or pectoral TrPs are responsible and treated them with myofascial release and self stretching.[65] A physician practicing physical medicine and rehabilitation[66] noted that scaleni TrPs commonly mimic the symptoms of a C_6 radiculopathy component of a TOS and that pectoralis minor TrPs will create symptoms of medial cord compression. A physical therapist[72] identified TrPs in the scaleni, supraspinatus, infraspinatus, and pectoral muscles as most commonly mimicking TOS. A neurologist[53] reported that of 198 patients diagnosed as having TOS, 11 were operated on and the remaining 187 (94%) *were relieved by injections of novocaine in their anterior scalene muscles.*

Not uncommonly, authors describe conservative treatment that releases tight muscles in patients with TOS but do not mention the possibility of TrPs. Usually, the treatments described are known to release myofascial TrPs in scalene,[33,38,49] levator scapulae,[49] and pectoral[49] muscles.

Related Trigger Points

The scalenus anterior and medius muscles are often involved together. If the scalenus minimus harbors active TrPs, all four **scalene muscles** usually are affected. The **sternocleidomastoid** muscle, which is also an important part of the functional unit for vigorous or labored inhalation, is likely to become involved if the scalene TrPs have been active for a considerable period of time.

Active TrPs in the scalenus medius are likely to be found, and have been reported, in association with TrPs in the **upper trapezius, sternocleidomastoid, and splenius capitis** muscles.[74]

Satellite TrPs may develop in several of the areas to which the scalene muscles refer pain. Both the **pectoralis major and minor muscles** commonly develop TrPs in regions that correspond to the scalene-referred pattern of anterior chest pain. Satellite TrPs in the **long head of the triceps brachii** correspond to the scalene pattern of posterior arm pain and those in the **deltoid** muscle[26] correspond to the anterior pattern. Although the dorsal forearm is a less common site of scalene pain, *secondary TrPs* tend to develop in the extensores carpi radialis, extensor digitorum, and extensor carpi ulnaris[26] and also in the brachioradialis muscle.

When TrPs in the lateral part of the brachialis muscle are induced as satellites of scalene TrPs, both the brachialis and scalene muscles refer pain to the thumb, making this digit especially painful.

When the omohyoid muscle *(see* Chapter 12) develops TrPs and becomes tense, it can act as a constricting band across the brachial plexus.[62] Because the tense muscle stands out prominently when the head is tilted to the other side, it can be mistaken for the upper trapezius or a scalene muscle. When the omohyoid harbors TrPs, it can prevent full stretch of the trapezius and scalene muscles, and therefore also must be released.

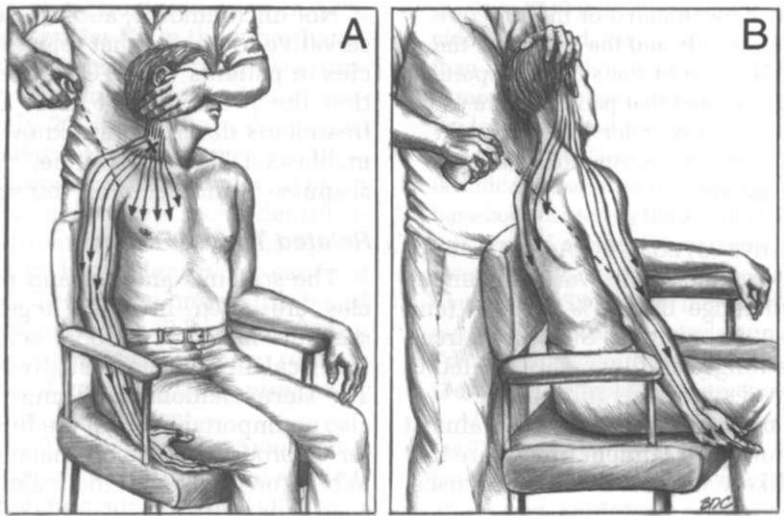


Figure 20.12. Stretch positions and spray pattern for the right scalene muscles. The patient anchors the right hand under the thigh. **A,** front view showing the anterior and anterolateral part of the spray pattern. **B,** side view showing the posterior and posterolateral part of the spray pattern. The total spray pattern should be used for each of the scalene muscles. For lengthening of the scalene muscles, the patient's neck is sidebent **away** from the involved muscle. The clinician cradles the patient's head and moves the neck in different positions of rotation, in line with the tight fibers, "fine tuning" the movement to lengthen the appropriate shortened fibers. Rotation of the face is away from the side of trigger points in the scalenus posterior, as in **A.** The face looks forward for a tight scalenus medius and the face is turned toward the muscle in the case of trigger point tightness of the scalenus anterior, as shown in Figure **20.14D** for self stretch. The technique for release of an elevated first rib shown in Figure **20.11** can effectively release all three scalene muscles.

12. TRIGGER POINT RELEASE
(Fig. 20.12)

Positioning

If the patient is seated for treatment, the operator first needs to make sure that the pelvic- and shoulder-girdle axes are level. A small hemipelvis should be corrected by an ischial lift under the ischial tuberosity on the small side to straighten the spine and level the patient's shoulders. This is important for relaxation of overloaded neck muscles. The patient should slide the hips forward slightly on the chair seat, lean back comfortably against the backrest, and slip the fingers under the thigh to anchor the pectoral girdle and rib cage on the side to be stretched. The other arm may rest in the lap or on the armrest (Fig. 20.12). The patient is encouraged to relax and let the shoulders drop. When needed, relaxation is facilitated by having the patient take a few slow, full breaths while concentrating on *diaphragmatic breathing* and slow relaxed exhalation.

Spray and Stretch

Scalenus Anterior. To stretch this muscle after a few initial sweeps of spray, the head and neck of the seated patient is tilted toward the contralateral side and *slightly* back, resting against the clinician's body. Vapocoolant or icing is applied along the lines of the scalenus anterior fibers and over the referred pain pattern of the chest (Fig. 20.12A). Then the spray again is swept over the muscle to the front and back of the arm and continued downward to include the thumb and index finger (Fig. 20.12B). The clinician then turns the patient's face toward the side of the involved anterior scalene, in the position shown in Figure 20.14D, to take up additional slack. Finally, the spray is again directed downward over the muscle and continued over

the back to cover the referred pain area around the upper and medial borders of the scapula (Fig. 20.12B).

Scalenus Medius and Scalenus Posterior. To stretch and spray the scalenus medius and the scalenus posterior, the seated patient's head and neck are sidebent away from the muscle involved with trigger points (TrPs) (toward the contralateral shoulder) and supported against the operator's body (Fig. 20.10). To specifically stretch the scalenus *posterior,* the patient's face should be turned away from the involved muscle. To stretch the *medius,* the face should be turned forward in a neutral position. If the patient's head is cradled between the operator's hand and body, vertical neck traction can be applied simultaneously. This feeling of support and the release of pressure on cervical structures helps the patient to relax the neck muscles and quiets interfering weight-bearing postural reflexes.

The sweeps of spray or icing should follow the direction of the muscle fibers being stretched. As a scalene muscle and the complete referred pain pattern are covered with parallel sweeps of the spray, the operator should be careful to fully include those areas where the patient has been experiencing spontaneous pain. A greater range of neck motion, and therefore more complete and lasting relief, usually occurs if *all* parts of the composite referred pain pattern (Fig. 20.1) are included in the vapocoolant application. Stretch and spray are followed at once with moist heat.

Stretch and spray of the scalene muscles should be applied bilaterally to avoid activating latent TrPs that might cause reactive cramping on the untreated side. Stretching a tense muscle on one side of the neck causes an unaccustomed degree of shortening of its partner on the other side. If such new and severe contralateral pain ensues, it should be treated by immediate stretch and spray of the reacting shortened muscles. The scalenes may be involved with TrPs bilaterally because of their alternating roles in stabilizing the neck, as well as their bilateral role in respiration.

In an alternate approach, the *patient* lies supine with instructions to do passive *self-stretch* of the scalene muscles as described for the Side-bending Neck Exercise in Section 14 *(see* Fig. 20.14). At the same time, the spray can be applied as described above, over the scalene muscle being stretched and then over the referred pain pattern of the chest and arm. To reach the posterior spray pattern, the patient must turn to one side during application of the spray and return to the relaxed supine position to continue self-stretch. This position makes it more difficult to spray the upper back and scapular pain reference zone, but usually provides more relaxation and effective stretch, and also trains the patient in the self-stretch technique for home use.

Other Release Techniques and Additional Considerations

Lewit[33] describes and illustrates a *gravity-assisted release* of the scaleni that is particularly effective for the middle and posterior muscles with the patient sidelying and the TrP-involved muscle uppermost. This technique of postisometric relaxation (PIR) is gentle, effective, and readily adapted to a self-stretch program for use at home. The patient is instructed to look upward and breathe in (contraction phase for scalenes in this position), hold the breath and the position about 6 seconds, then slowly breathe out and let the head and neck sink back to the table (relaxation phase of PIR). This should be repeated three times. If breathing is faulty, the pattern should be corrected with training that establishes normal diaphragmatic breathing. (Refer to Section 14 of this chapter).

Trigger point pressure release, complemented by instructing the patient to do slow relaxed breathing, is another technique for inactivating scalene TrPs. **Indirect techniques** may also be effective, particularly when combined with TrP pressure release *(see* Chapter 3, Section 12).

To fully lengthen the scalene muscles, it is often necessary to **release parallel muscles** that are also tense due to TrPs and which restrict sidebending of the neck. Examples are the upper trapezius and both the clavicular and sternal divisions of the sternocleidomastoid muscle. Less frequently, a tense omohyoid muscle stands

out under the skin like a rope as it stretches over other neck structures and attaches to the scapula. If it harbors TrPs, it can prevent full stretch of the scaleni and should itself be released (see Chapter 12).

As a rule, patients sleep more comfortably lying on the side of the involved scalene muscles. If TrPs in the posterior scapular musculature (e.g., the infraspinatus), prevent this, these TrPs should be inactivated so that the patient can sleep comfortably on the preferred side.

13. TRIGGER POINT INJECTION (Fig. 20.13)

Effective needle contact with an active trigger point (TrP) results in a local twitch response (LTR) that may be detected visually and/or by palpation. The clinician should watch carefully for an LTR when injecting scalene TrPs to confirm effective placement of the needle.

Scalenus Anterior and Medius

Long[40] recommended injection with procaine for relief of myofascial pain due to TrPs in the scalene muscles. In a surgical study, testing for pain relief obtained by infiltrating the scalenus anterior with 1.0% lidocaine did not predict the results of scalenotomy.[61] However, in this surgical study, the final therapeutic effect of the injection was not studied, and injection was not directed specifically to TrPs.

For injection of TrPs in the anterior and middle scalene muscles, the patient should lie supine and turn the head slightly away from the side to be injected (Fig. 20.13). In addition, it may help to elevate both the head and shoulder slightly by a pillow to slacken the sternocleidomastoid and trapezius muscles.

The vertical groove between the anterior and middle scalene muscles which locates the brachial plexus is identified by palpation as described earlier in Section 9, and the needle should be directed away from the nerves and upward toward either the anterior or middle scalene muscle. Farther cephalad, at the level where one finds the scalene central TrPs, the groove may be difficult to identify unless one has followed it up from the first rib.

The most common TrP in the scalenus anterior is found either under, or near the external jugular vein (Fig. 20.8). The operator's free hand presses the clavicular division of the sternocleidomastoid muscle and jugular vein aside, and palpates the scalene muscle for tense muscular nodules in taut bands and tender spots that, when compressed, reproduce the patient's pain complaint. Several individual taut bands that contain active TrPs are usually palpable. A band is pinned down between the index and middle fingers at a TrP to localize it for injection and to provide hemostasis during and after injection. The needle should be inserted well above the apex of the lung, which ordinarily extends about 2.5 cm (1 in) above the clavicle.[2] *All* scalene injections are made at least 3.8 cm (l 1/2-in) above the clavicle.

Active scalene TrPs are usually found about halfway between the clavicle and the mastoid process. Two fingers straddle the nodule of the TrP to be injected, with one finger in the groove for the brachial plexus. The needle should be angled in front of (ventral to) the groove to inject the scalenus anterior and behind (dorsal to) it to inject the scalenus medius (Fig. 20.13).

As seen from the operator's side view of the neck, if the needle penetrates too deeply, too caudad, and a little too far toward the front, the stellate ganglion or sympathetic trunk may be anesthetized, producing a transient Horner's syndrome. The stellate ganglion usually lies ventral to the origin of the first rib at the junction of its head and neck.[11]

The roots of spinal nerves C_4 to C_7 and the lower trunk of the brachial plexus emerge between the anterior and middle scalene muscles and then cross in front of the lower portion of the middle scalene (Fig. 20.9). Care and patience are needed to inject the TrPs in the multiple thin bands of involved scalenus medius muscle above the level of these nerves. The patient should be warned of possible transient numbness and weakness of the arm due to infiltration of the nerve trunks by the local anesthetic. When penetrated by the needle, scalene TrPs often refer sharp intense pain to the arm and hand strongly suggestive of neurogenic pain. This reproduction of the referred pain pattern is characteristic of TrPs and need not signal needle contact with brachial plexus nerve fibers. Effective penetration of a TrP consistently produces an LTR; penetration of a nerve does not. A

2.5-cm (1-in), 23- or 24-gauge needle may be used. After injection, pressure is maintained for hemostasis because bleeding within the scalene muscles causes local irritation and marked afterpain.

The illustration of injecting middle scalene TrPs by Rachlin[54] presents an unrealistic anatomical drawing of the relationship between the scalene muscles and the brachial plexus. To safely inject these muscles, these anatomical relationships must be clearly understood and key features must be palpated to assure safe positioning of the needle.

Scalenus Posterior

For injection of the scalenus posterior, the patient should be sidelying with the involved muscle uppermost, with the back toward the operator, and with the head tilted slightly toward the involved side to slacken the upper trapezius, which should be pushed aside (Fig. 20.7 shows why). The technique is similar to that described by Kraus.[32] The levator scapulae muscle is located as it emerges from beneath the trapezius at the root of the neck (Fig. 20.7). The scalenus posterior is then found anterior to the levator scapulae. The scalenus posterior TrPs are approached from behind. Because of its submerged position among other muscles, a 22-gauge, at least 3.8-cm (l 1/2-in) needle is used. To avoid introducing the needle between the ribs, it should be directed tangential to them and posteriorly. The scalenus posterior can be injected through the same skin puncture as that used to inject the upper TrP of the levator scapulae. When a scalenus posterior TrP is encountered, the patient usually reports pain referred to the region of the triceps brachii muscle.

For TrPs in any of these scalene muscles, injection is followed by spray and stretch, moist heat, and active side-bending movements to full range on both sides, with the patient lying supine.

Scalenus Minimus

When the scalenus minimus muscle is present, its TrPs, as a rule, are not inactivated by spray and stretch. Local injection is indicated if local tenderness and referred pain characteristic of TrPs in the scalenus minimus persist and the other scalene muscles are free of TrPs. The minimus may be injected through the same skin puncture that is used for the lower TrP of the scalenus anterior. The needle is inserted at least 3.5 cm (1 1/2-2 in) above the clavicle, straight in rather than upward (and *not* angled downward toward the apex of the lung), through the scalenus an-

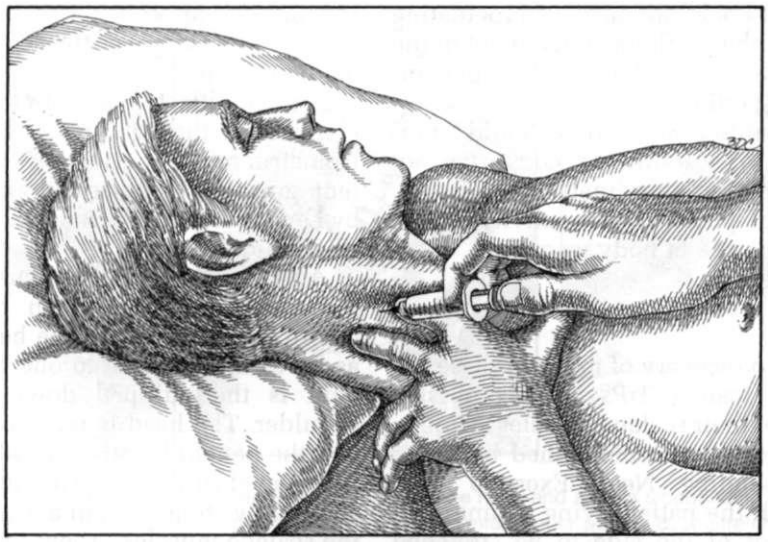


Figure 20.13. Injection of the scalenus medius with the patient supine. Fingers straddle the middle scalene muscle with the index finger in the groove between the scaleni anterior and medius muscles to locate the brachial plexus. The needle is directed posteriorly away from the groove to avoid nerve fibers of the plexus,

terior and toward a transverse process. The needle passes through the space above the subclavian artery before it encounters the scalenus minimus muscle (muscle shown in Fig. 20.3). Care must be taken not to inject below the TrP tenderness of this muscle. The clinician should become thoroughly familiar with the anatomical relations of this muscle before attempting to inject it. A 3.8-cm (1 1/2-in), 23-gauge needle is used. Following the injection, spray and stretch, and moist heat are applied.

This muscle can be hypertrophied in patients accustomed to paradoxical breathing. Because of this overload, its TrPs can be making a significant contribution to a thoracic outlet syndrome.

14. CORRECTIVE ACTIONS

(Figs. 20.14, 20.15, and 20.16)

Correction of poor posture is of paramount importance, as is the use of safe and efficient body mechanics, for long-term relief from muscle pain. Postural correction and body mechanics are discussed in Chapter 41 of this volume.

In most patients, multiple factors contribute to the activation and reactivation of scalene TrPs. Elimination of one factor may result in some improvement. Identification and correction of all major perpetuating factors, together with local treatment of the affected muscles, is often required for complete lasting relief.

Patients with scalene TrPs should be instructed in the following corrective actions: stretching, coordinated respiration, reduction of postural and activity strain, and maintenance of body warmth.

Stretching

Critical to recovery of many of these patients with scalene TrPs is daily passive stretching of their scalene muscles at home. Stretching can be accomplished by doing the Side-bending Neck Exercise (Fig. 20.14). With the patient lying supine, first the shoulder of the side to be stretched (right side in this illustration) is lowered and the hand anchored under the buttock (Fig. 20.14A). The patient must learn to reach over the head to the ear with the hand of the contralateral side, assisting the head and neck to tilt it to the side away from the involved muscles while concentrating on relaxation of the neck muscles. The head is drawn smoothly down toward the shoulder. The degree of head rotation determines which of the three major scalene muscles is specifically placed on stretch.

To stretch the *scalenus posterior* (Fig. 20.14B), the patient uses the assistive hand to gently pull the head and neck into sidebending *away* from the side of the TrPs, then turns the face away from the affected muscle. To stretch the *scalenus anterior,* the patient turns the face toward the affected muscle. For the *scalenus medius,* the supine patient looks straight up toward the ceiling (neutral position), or slightly toward the pulling arm. The patient concentrates the stretch on those directions in which the muscles feel tightest, holds each stretch for a slow count of six while inhaling and slowly exhaling to give the stretched muscles time to release, and then gently takes up any slack that develops. The head is returned to the neutral mid-position. A pause, with deep *diaphragmatic* breathing between each passive stretch, helps to reestablish complete muscular relaxation. The exercise should always be done bilaterally. It is more effective if performed after application of moist heat has warmed the skin over the scalene muscles for 10-15 min.

Another effective self-stretch can be accomplished through gravity-assisted post-isometric relaxation for release of the scalene muscles, as described and illustrated by Lewit[33] and as described in section 12 of this chapter.

An effective active scalene exercise is the movement utilized in the Scalene-cramp Test (Fig. 20.4). The head is turned as far as it can rotate to one side, and the chin is then dipped down toward the shoulder. The head is returned to neutral, and the patient breathes deeply. The cycle is repeated in the opposite direction. This alternately stretches and actively contracts the scalene muscles. About four cycles are performed daily. This is useful as an active range of motion follow-up to the passive Side-bending Neck Exercise.

Coordinated Respiration
(Figs. 20.15 and 20.16)

Coordinated respiration should be taught to those who are accustomed to using paradoxical breathing. Paradoxical respiration is a *common* source of abuse and overload of the scalene muscles and is frequently adopted by patients following abdominal surgery and by people who constantly retract a protruding abdomen to improve their appearance. People who do paradoxical breathing often complain that they are "always out of breath," or that they "run out of breath" when talking on the telephone.

Normal contraction of the diaphragm pushes the abdominal contents down toward the pelvis, causing protrusion of the abdomen and increased lung volume in the lower chest during inhalation. Normal resting inhalation involves coordinated contraction of the diaphragm with expansion of the *lower* thorax and elevation of the rib cage, all of which increase lung volume. In paradoxical respiration, these chest and abdominal functions oppose each other; on inhalation, the chest expands (moves up and out) while the abdomen moves in, elevating the diaphragm and decreasing lung volume. On exhalation, the reverse occurs. Consequently, a normal effort produces inadequate tidal volume, and the muscles of the *upper* chest, and especially the scalene muscles, overwork to exchange sufficient air. This muscular overload results from poor coordination of the major components of the respiratory apparatus. The mechanics of normal respiration are presented in detail in Chapter 45 and illustrated in Figures 45.8 and 45.9.

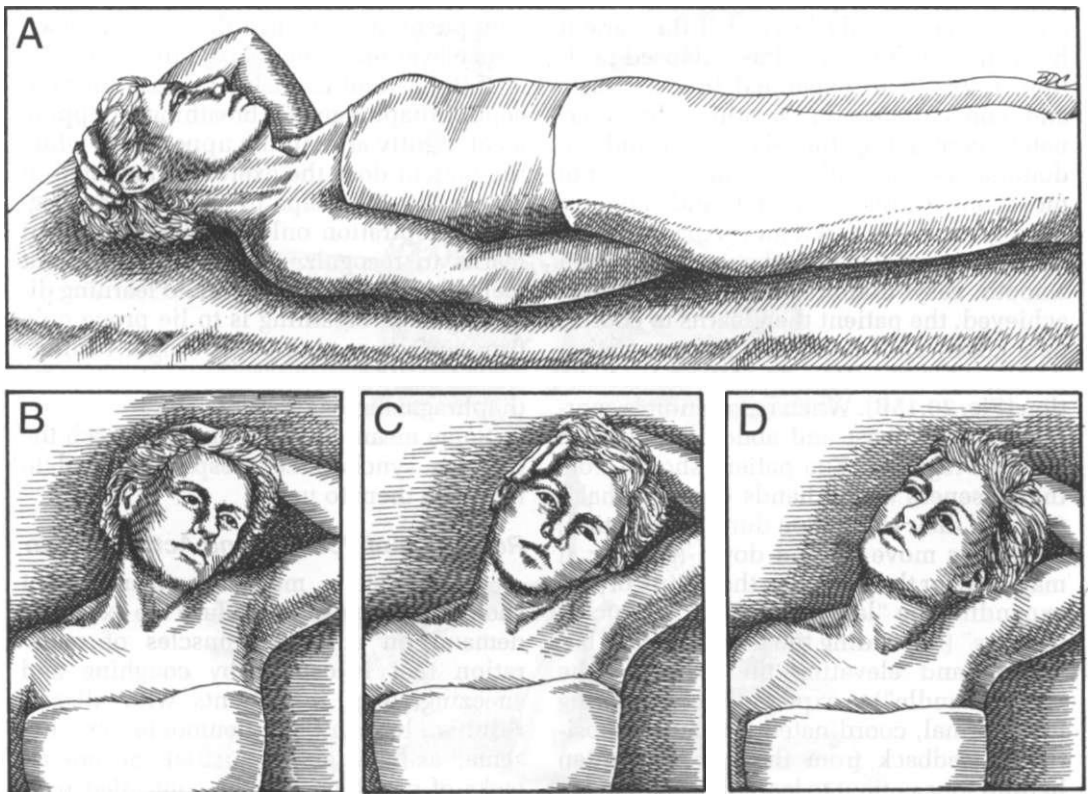


Figure 20.14. The Side-bending Neck Exercise is performed with the patient supine. Each position passively stretches one of the three major scalene muscles. The exercise should always be done bilaterally. **A,** the hand on the side to be stretched is anchored under the buttock. The contralateral hand is placed over the head and assists sidebending away from the muscle with trigger points. **B,** to stretch the scalenus posterior, the face is turned away from the involved muscle. **C,** the face looks forward to stretch the scalenus medius. **D,** the face is turned toward the involved muscle to stretch the scalenus anterior.

The patients who breathe paradoxically must learn to synchronize diaphragmatic (abdominal) and chest breathing if they are to relieve the scalene muscle overload (Fig. 20.15). The top part of the figure (Fig. 20.15A) illustrates the poor inhalation pattern, showing the abdomen moving in while the chest moves out. The patient can learn to identify his paradoxical pattern by placing one hand on the abdomen and one on the chest while taking a deep breath. This paradoxical pattern moves air mostly between the upper and lower chest and moves little air in and out of the lungs. The diaphragm-abdominal muscle component and the intercostal muscles are literally fighting each other instead of working together.

To learn normal diaphragmatic breathing, the patient exhales fully with one hand on the chest and the other on the abdomen (Fig. 20.15B). Diaphragmatic respiration alone is most easily learned if the patient holds the chest fixed in the collapsed position, rather than expanded (Fig. 20.15C), and concentrates on breathing by alternately contracting the diaphragm and abdominal muscles (allowing the abdomen to move out during inhalation and move in during exhalation) without expanding the upper chest or elevating the sternum. When smooth easy diaphragmatic breathing is achieved, the patient then learns to coordinate costal and diaphragmatic respiration during inhalation (Fig. 20.15D) and exhalation (Fig. 20.15B). When respiration is coordinated, the chest and abdomen move in and out together. The patient should note the closeness of the hands during exhalation and their separation during inhalation; the hands move up and down *together.* It may help for the patient to then think of also expanding the "lateral bellows" or "bucket handles" (expanding the lower rib cage laterally), and elevating the sternum (the "pump handle") to expand the chest during full, normal, coordinated inhalation. Positional feedback from the hands is often helpful for a patient to learn this technique.

The patient should practice *coordinated* breathing at intervals throughout the day and on retiring. Taking each breath to the count of "4 in," and a count of "4 out," then a pause, "hold-and-relax" for a count of 4 improves pacing and provides rhythm. The patient should become aware of using this coordinated breathing throughout the day.

Having learned to breathe properly while recumbent, the patient must transfer this learning to the upright posture. A few patients learn coordinated respiration more readily in the seated position than when recumbent. The patient sits in a chair with a firm flat seat (Fig. 20.16), tilts the front of the pelvis forward and down (exaggerating the lumbar lordosis), and draws in a slow deep breath. This anterior pelvic tilt separates the anterior chest from the symphysis pubis, making it easy and natural to contract the diaphragm and to protrude the abdomen while inhaling. Then, by rocking the pelvis backward (posterior pelvic tilt or abdominal curl movement) and leaning slightly forward during slow exhalation, the abdomen moves in and the increased intra-abdominal pressure pushing up against the diaphragm assists elevation of the relaxed diaphragm.

If the patient is unable to grasp the concept of diaphragmatic breathing, strapping a belt tightly around the upper chest while the patient does the exercise illustrated in Figure 20.15C, helps to enforce diaphragmatic respiration only, so that the patient learns to recognize what that movement feels like. Another approach to learning diaphragmatic breathing is to lie prone on a firm surface so that body weight restricts chest breathing and assures predominantly diaphragmatic respiration.

Some means must be found to teach the patients synchronized respiration and to motivate them to use it.

Reduction of Postural and Activity Strain

Appropriate medical management should be employed to reduce the excessive demand on auxiliary muscles of respiration that is caused by coughing and sneezing, (e.g., in patients with allergic rhinitis, bronchitis, pneumonia, emphysema, asthma, and sinusitis). Severe attacks of coughing may be controlled with adequate antitussive medication and with the patient learning to suppress and avoid the cough reflex by clearing the throat instead of coughing.

Body Asymmetry. A tilted shoulder-girdle axis, sometimes caused by the func-

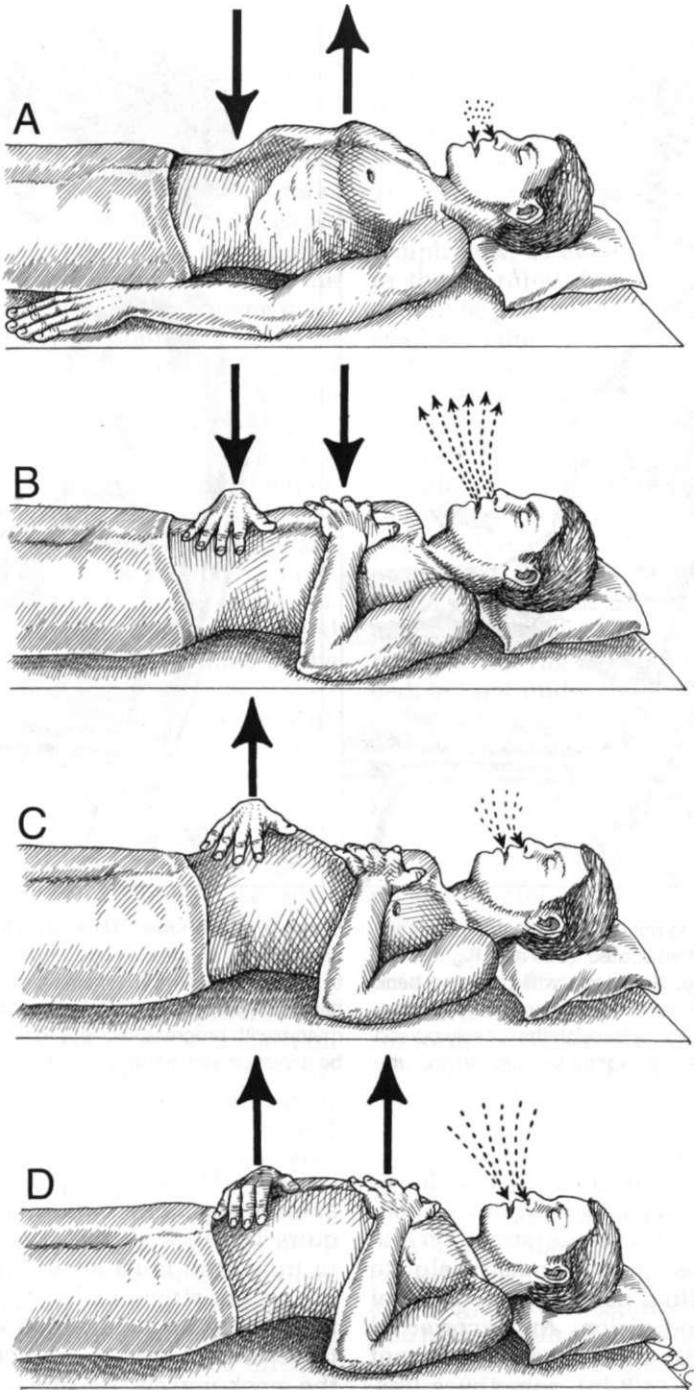


Figure 20.15. Learning a normal pattern of respiration, patient supine. The patient is trained to become aware of the respiratory mechanism by feeling the position and movement of the hands. **A,** erroneous paradoxical breathing *(red arrows):* abdomen in, chest out. **B,** first step, complete exhalation. **C,** then, inhalation by using the diaphragm only, protruding the abdomen, and keeping the chest collapsed. **D,** finally, synchronize chest and diaphragm by taking deep breaths while concentrating on moving the chest and abdomen in and out together. This is the pattern of normal coordinated respiration.

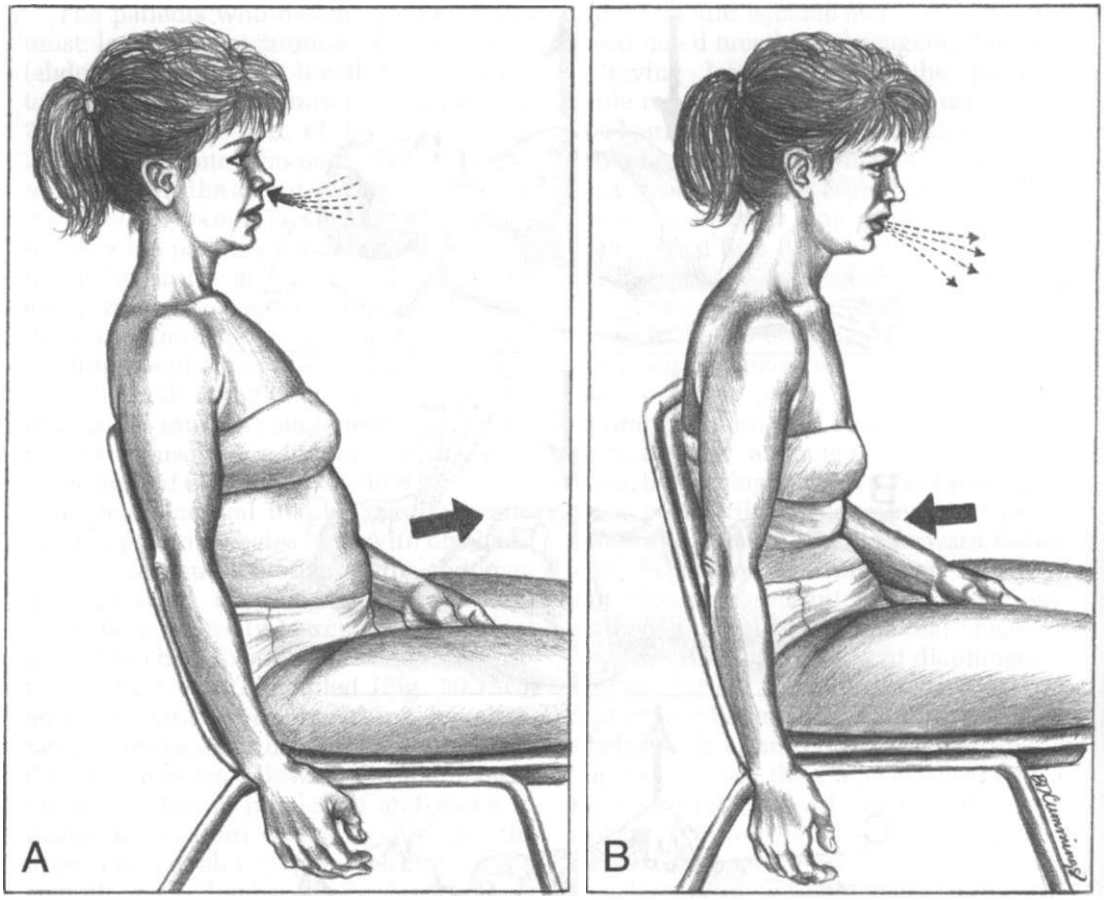


Figure 20.16. Learning normal, coordinated diaphragmatic breathing, patient seated. (See also Fig. 20.15). **A,** breathe in through the nose while leaning back slightly, allowing the abdomen to move out and forward. **B,** breathe out easily through the loosely pursed lips, while slowly leaning slightly forward, so that the pressure on the lower abdomen helps to push the diaphragm up and the air out. Slowly lean back slightly to begin another cycle. When a regular, relaxed rhythm has been established, try to retain the same respiratory rhythm with progressively less rocking. This effect can be achieved very naturally in a rocking chair.

tional scoliosis associated with a lower limb-length discrepancy and/or a small hemipelvis, places chronic strain on the scalene muscles, which must help to straighten the tilted neck in order to level the eyes for good vision. An uncorrected lower-limb length or pelvic discrepancy of as little as 1 cm (3/8-in), sometimes less, can perpetuate scalene TrPs despite all other efforts in management. For identification and correction of these asymmetries, see Chapter 4 and Chapter 48 (Section 14) of this volume, and see Chapter 4 of Volume 2.

Body Mechanics. The patient must avoid carrying awkward packages that require lifting with the arms extended out in front, and must avoid hauling, pulling or tugging strenuously. Whenever undertaking any such vigorous effort, the patient must learn to reduce consciously the neck-muscle tension caused by unnecessarily elevating the shoulders and projecting the head forward. Scalene muscle strain due to increasing intra-abdominal pressure when closing the glottis, as when straining during lifting or defecation, may be reduced by panting

through the open mouth and dropping the shoulders, which inhibits scalene contraction.

When turning over in bed, the patient should *roll* the head without lifting it off the pillow.

The patient with active scalene TrPs, who has been instructed to do the In-doorway Stretch Exercise (*see* Fig. 42.9) because of active TrPs in other muscles, should start with the arms-high position and avoid the arms-down position until the scalene muscles are TrP- and symptom-free.

Postural Strain. The lower rims of thick-rimmed eyeglasses may occlude the line of vision for writing or reading when the head is held in the balanced erect position. When that happens, the person then tilts the head forward and down to see clearly over the lower rims, causing persistent shortening of the anterior neck muscles and strain of the posterior neck muscles. The correction for this is illustrated in Figure 16.4, by tilting the plane of the eyeglasses.

Active scalene TrPs in patients who have a unilateral hearing impairment often appear to be refractory to treatment when they persistently rotate the head and neck to face the "good ear" toward the speaker. The patient should turn the entire body, not just the head, and should take other measures to improve the hearing (e.g., a hearing aid), if possible.

Seating and Lighting. The patient with persistent scalene TrPs should provide and use an appropriate elbow rest, especially on the affected side, when sitting and reading, writing, sewing, driving, riding in a car, or telephoning. The telephone receiver should be held in the hand on the *unaffected* side, with occasional change of hands (not ears) on long calls. An executive (speaker) phone or headset eliminates the problem of holding the telephone receiver for a long time. Use of the shoulder to hold the handset must be avoided by these patients.

The reading light should shine directly on reading material from overhead and *not* from the affected side, which causes the head to be turned that way. For those who frequently read in bed, a light that clips on the head of the bed or is attached anywhere overhead may be essential to recovery.

Bed Elevation. Tilting the bed frame so that the head end is higher prevents the chest from riding up around the neck at night and creates mild steady traction on the scalene muscles. This avoids placing the scalene muscles in a cramped position of sustained shortening (which aggravates TrP activity in any muscle) and, in this case, may impede venous drainage and lymph flow, as evidenced by hand edema in the morning. Frequently, scalene TrPs *cannot be permanently relieved* without elevation of the head end of the bed.

The head of the patient's bed should be raised 8 to 9 cm (*3-3 1/2* in) by placing blocks or telephone books under the legs at the head-end of the bed to tilt the bed frame. Telephone books are practical to use because the height of the head-end of the bed can be adjusted by selecting the number of pages used; they soon become indented, so the legs of the bed do not slip off as they may with blocks. Additional correction is required if a rug lies under the foot of the bed, but not under the head of the bed.

Patients may try sleeping on two pillows to obtain the same raised effect, or to improve "sinus drainage." The result may be increased pain because while the pillows do elevate the patient's head, they also flex the neck, which causes anterior scalene shortening that can aggravate these TrPs.

Bed Pillow. The patient should use only one soft comfortable pillow of the right thickness to maintain a normal cervical lordosis. When the patient lies on the affected side, the pillow under the head should be thick enough and should be bunched up between the neck and shoulder to prevent tilting of the head that would cause sustained shortening of the involved scalene muscles.

A foam rubber pillow should be discarded. The jiggle of the head and neck on a springy pillow aggravates scalene TrPs. The patient with allergies may select a foam rubber pillow to avoid allergenic fillers, and should be warned against that mistake. Sensitive patients may wish to carry their "safe" home pillow with them on trips.

When lying on the back (supine), the patient should pull the corners of the pillow forward between the shoulder and the

cheek on each side. This ensures that the shoulders rest on the bed and not on the pillow and that the cervical spine is in line with the thoracic spine. It also encourages a shoulder-down position, which avoids shortening and cramping of the chest-elevator (scalene) and scapula-elevator (levator scapulae and upper trapezius) muscles. This pillow position supports the head in the midline and encourages bilateral scalene relaxation *(see* Fig. *7.7A).*

Maintenance of Body Warmth

Chilling the body, especially when resting, reduces peripheral blood flow and can lead to increased skeletal muscle irritability. In bed, an electric blanket can be invaluable. It is helpful in other rooms, also, when sitting or lying on a sofa in cold climates, during inclement weather, or when the thermostat is set low.

If the bedroom is drafty, a high-necked sweater or warm scarf should be worn in bed. Such neck protection is often helpful on airplane flights.

The patient can apply a moist heating pad over the scalene TrPs on the front of the neck for 10-15 min before going to sleep at night. However, for some people, the neutral warmth (just keeping the body heat in) that is provided by a wool scarf or "baby" blanket produces greater comfort.

REFERENCES

1. Adson AW: Cervical ribs: symptoms, differential diagnosis and indications for section of the insertion of the scalenus anticus muscle. *J Int Coll Surg* 26:546-559, 1951 (p. 548).
2. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 557, Fig. 8.7).
3. Baker BA: The muscle trigger: evidence of overload injury. *J Neurol Orthop Med Surg* 7:35-44, 1986.
4. Bardeen CR: The Musculature. Sect. 5. In: *Morris's Human Anatomy.,* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (p. 388).
5. *Ibid.* (p. 389).
6. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 409, 412, 426).
7. Bonica JJ, Sola AE: Other painful disorders of the upper limb. Chapter 52. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, 1990 (pp. 947-958).
8. Campbell EJ: Accessory muscles. In: *The Respiratory Muscles: Mechanics and Neural Control.* Ed. 2. Edited by Campbell EJ, Agostoni E, Davis JN. W.B. Saunders, Philadelphia, 1970 (pp. 181-183, 186).
9. Cherington M, Happer I, Machanic B, *et al.:* Surgery for thoracic outlet syndrome may be hazardous to your health. *Muscle Nerve* 9(7):632-634, 1986.
10. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (Fig. 6-17).
11. *Ibid.* (pp. 463, 521).
12. *Ibid.* (Fig. 6-15).
13. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 576, 594).
14. *Ibid.* (Fig. 524).
15. *Ibid.* (Fig. 233).
16. Dawson DM, Hallett M, Millender LH: *Entrapment Neuropathies.* Little, Brown & Co, Boston, 1983 (pp. 103, 171).
17. De Troyer A: Actions of the respiratory muscles or how the chest wall moves in upright man. *Bull Eur Physiopathol Respir 20(5):*409-413, 1984.
18. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 511).
19. *Ibid.* (pp. 479-480).
20. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (pp. 308-310, Figs. 39, 40).
21. *Ibid.* (Fig. 4).
22. Frankel SA, Hirata I Jr.: The scalenus anticus syndrome and competitive swimming. *JAMA 215:1796-*1798, 1971.
23. Gerwin RD, Shannon S, Hong CZ, *et al:* Interrater reliability in myofascial trigger point examination. Pain 69:65-73, 1997.
24. Goodridge JP, Kuchera WA: Muscle energy treatment techniques for specific areas. Chapter 54. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997 (pp. 697-761).
25. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 124, 146, 147).
26. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(1):29-59, 1994.
27. Hong CZ, Simons DG: Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. *J Musculoske Pain 1 (1):*89-131, 1993.
28. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 9:157-64, 1989.
29. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 76).
30. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 317, 343).
31. Komanetsky RM, Novak CB, Mackinnon SE, *et al:* Somatosensory evoked potentials fail to diagnose thoracic outlet syndrome. *J Hand Surg 21(4):662-*666, 1996.
32. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (pp. 104, 105).
33. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (p. 24; p. 196, Fig. 6.91; 197, 244, 245).
34. Lindgren KA: Thoracic outlet syndrome with special reference to the first rib. *Annates Chirurgiae et Gynaecologiae 82(4):218-230,* 1993.

35. Lindgren KA: Reasons for failures in the surgical treatment of thoracic outlet syndrome. *Muscle Nerve* 28:1484-1486, 1995.
36. Lindgren KA: Conservative treatment of thoracic outlet syndrome: a 2-year follow-up. *Arch Phys Med Rehabil* 78:373-378, 1997.
37. Lindgren KA, Leino E: Subluxation of the first rib: a possible thoracic outlet syndrome mechanism. *Arch Phys Med Rehabil* 69(9):692-695, 1988.
38. Lindgren KA, Manninen H, Rytkbnen H: Thoracic outlet syndrome-a functional disturbance of the thoracic upper aperture? *Muscle Nerve* 38:526-530,1995.
39. Lindgren KA, Manninen H, Rytkbnen H: Thoracic outlet syndrome [a reply]. *Muscle Nerve* 29:254-256, 1996. (Letter)
40. Long C: Myofascial pain syndromes: part 2—syndromes of the head, neck and shoulder girdle. *Henry Ford Hosp Med Bull* 4:22-28, 1956.
41. Mackinnon SE, Patterson. GA, Novak CB: Thoracic outlet syndrome: a current overview. *Semin Thorac Cardiovasc Surg* 8(2):176-182, 1996.
42. Mailis A, Papagapiou M, Vanderlinden RG, *et al.:* Thoracic outlet syndrome after motor vehicle accidents in a Canadian pain clinic population. *Clin J Pain* 3 3(4j:316-324, 1995.
43. Makhoul RG, Machleder HI: Developmental anomalies at the thoracic outlet: an analysis of 200 consecutive cases. *J Vase Surg* 36(4):534-542, 1992.
44. Maloney M: Personal communication, 1993.
45. McDonough JT Jr: *Stedman's Concise Medical Dictionary.* Ed. 2. Williams & Wilkins, Baltimore, 1994 (p. 995).
46. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy,* Ed. 3. Mosby-Year Book, St. Louis, 1993 (p. 191).
47. Naffziger HC, Grant WT: Neuritis of the brachial plexus mechanical in origin. The scalenus syndrome. *Surg Gynecol Obstet* 67:722-730, 1938.
48. Nielsen AJ: Personal communication, 1980.
49. Novak CB: Conservative management of thoracic outlet syndrome. *Sem Thorac Cardiovasc Surg* 8(2):201-207, 1996.
50. Ochsner A, Gage M, DeBakey M: Scalenus anticus (Naffziger) syndrome. *Am J Surg* 28:669-695, 1935.
51. Passero S, Paradiso C, Giannini F, ef *al:* Diagnosis of thoracic outlet syndrome. Relative value of electrophysiological studies. *Acta Neurologica Scand* 90(3):179-185, 1994.
52. Poole GV, Thomae KR: Thoracic outlet syndrome reconsidered. *Am Surg 62(4):287-291,* 1996.
53. Popelianskii II, Kipervas IP: [On the clinical basis of infiltration and operative treatment of patients with scalenus anticus syndrome]. *Vopr-Neirokhir 32(2):22-25,* 1968.
54. Rachlin ES: Injection of Specific Trigger Points. Chapter 10. In: *Myofascial Pain and Fibromyalgia,* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360.
55. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 233, 258).
56. Rayan GM, Jensen C: Thoracic outlet syndrome: provocative examination maneuvers in a typical population. *J Shoulder Elbow Surg* 4(2),1:113-117, 1995.
57. Roos DB: Pathophysiology of congenital anomalies in thoracic outlet syndrome. *Acta Chir Belg* 79(5):353-361, 1980.
58. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Rehabil* 62:107-110, 1981.
59. Schnyder H, Rosier KM, Hess CW: [The diagnostic significance of additional electrophysiological studies in suspected neurogenic thoracic outlet syndrome]. *Schweizerische Medizinische Wochenschrift. J Suisse Med* 324(9):349-356, 1994.
60. Sherman RA: Published treatments of phantom limb pain. *Am J Phys Med Rehabil* 59:232-244,1980.
61. Sivertsen B, Christensen JH: Pain relieving effect of scalenotomy. *Acta Orthop Scand* 48:158-160, 1977.
62. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med* 34:585-590, 1955.
63. Steinbrocker O, Isenberg SA, Silver M, *et al:* Observations on pain produced by injection of hypertonic saline into muscles and other supportive tissues. *J Clin Invest* 32:1045-1051, 1953.
64. Sucher BM: Thoracic outlet syndrome—a myofascial variant: Part 1. Pathology and diagnosis. *J Am Osteopath Assoc* 90(8):686-704, 1990.
65. Sucher BM: Thoracic outlet syndrome—a myofascial variant: Part 2. Treatment. *J Am Osteopath Assoc* 90(9):810-812, 817-823, 1990.
66. Tardif GS: Myofascial pain syndromes in the diagnosis of thoracic outlet syndromes. *Muscle Nerve* 33:362, 1990. (Letter)
67. Thomas GI, Jones TW, Stavney LS, et *al:* The middle scalene muscle and its contribution to the thoracic outlet syndrome. *Am J Surg* 345(5):589-592, 1983.
68. Travell J: Rapid relief of acute "stiff neck" by ethyl chloride spray. *J Am Med Worn Assoc* 4:89-95,1949.
69. Travell J: *Office Hours: Day and Night.* The World Publishing Company, New York, 1968 (pp. 271-272).
70. Travell J, Rinzler S, Herman M: Pain and disability of the shoulder and arm, treatment by intramuscular infiltration with procaine hydrochloride. *JAMA* 320:417-422, 1942.
71. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 3 3:425-434, 1952 (p. 428).
72. Walsh MT: Therapist management of thoracic outlet syndrome. *J Hand Ther 7 (2):131-* 144, 1994.
73. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J Am Osteopath Assoc* 72:697-710, 1973 (p. 706, Fig. 30).
74. Wyant GM: Chronic pain syndromes and their treatment. II. Trigger points. *Can Anaesth Soc J* 26:216-219, 1979 (Patients 1 and 2).
75. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little, Brown & Co, Boston, 1988 (p. 211, Fig. 12-2).

CHAPTER 21
Supraspinatus Muscle

HIGHLIGHTS: This muscle is often a major actor in diagnoses relating to the **rotator cuff. REFERRED PAIN** from trigger points (TrPs) in this muscle is felt as a deep ache in the mid-deltoid region of the shoulder and usually extends part way down the arm. The pain also may concentrate at the lateral epicondyle and, rarely, may extend to the wrist. **ANATOMICAL** attachments of the supraspinatus muscle are to the supraspinous fossa medially and to the greater tubercle of the head of the humerus laterally. **FUNCTION** of this muscle is to position the humeral head accurately in the glenoid fossa and to stabilize the head of the humerus firmly in the fossa when the arm is used. It abducts the arm at the glenohumeral joint. Its **FUNCTIONAL UNIT** includes the middle deltoid and upper trapezius as synergists during abduction, and includes the other three rotator cuff muscles for stabilizing the humeral head. **SYMPTOMS** include chiefly referred pain that is aggravated by forceful abduction of the arm at the shoulder joint and by passive stretching when fully adducting the arm. Patients report difficulty in reaching up above the shoulder, and may experience pain at night that disturbs sleep. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are likely to result when heavy objects are carried with the arm hanging down by the side, and when the subject is working or lifting above shoulder height. **PATIENT EXAMINATION** demonstrates reduced range of motion in the Mouth Wrap-around Test. **TRIGGER POINT EXAMINATION** of the mid-muscle TrP region by flat palpation elicits exquisite spot tenderness, but the lateral trigger area, adjacent to the acromion, is so deeply placed that firm palpation may reveal only minimal tenderness. The region of attachment of the supraspinatus tendon to the head of the humerus can also be exquisitely tender. **DIFFERENTIAL DIAGNOSIS** includes rotator cuff tears, subdeltoid or subacromial bursitis, C_5-C_6 radiculopathy, and related TrPs in shoulder-girdle muscles. **TRIGGER POINT RELEASE** employing spray and stretch begins with the patient seated and the ipsilateral hand placed behind the back. Then, while vapocoolant spray or icing is applied from medial to lateral over the muscle fibers and referred pain pattern, the clinician takes up slack in the muscle as it develops. **TRIGGER POINT INJECTION** is carried out with the patient lying on the uninvolved side and the needle directed into one of the three areas of spot tenderness: in the TrPs located midfiber, in the musculotendinous junction region deep in the lateral part of the supraspinous fossa, or in the region of terminal tendon attachment to the joint capsule under the acromion. **CORRECTIVE ACTIONS** require avoiding continued overload of the muscle, and performing a stretch exercise at home while seated under a warm shower.

1. REFERRED PAIN

(Fig. 21.1)

Active trigger points (TrPs) in the supraspinatus muscle cause a deep ache of the shoulder, concentrating in the mid-deltoid region. This ache often extends down the arm and the forearm, and sometimes focuses strongly over the lateral epicondyle of the elbow (Fig. 21.1).[48] This epicondylar component helps to distinguish supraspinatus TrPs from infraspinatus TrPs, which do not concentrate pain at the elbow.[48, 51] Rarely, pain is referred to the wrist from the supraspinatus. The tenderness and pain that it projects to the mid-deltoid region are easily mistaken for subdeltoid bursitis.

Other authors have described the pain referred from the supraspinatus as traveling toward, or into, the shoulder,[6,26,27,30] to the outer side of the arm,[6,25,26] and from the scapula to midhumerus.[28]

Experimental injection of 6% hypertonic saline into normal supraspinatus muscles caused referred pain to the shoulder (3 subjects), to the upper back (2 subjects) and to the elbow (1 subject).[46]

2. ANATOMY
(Fig. 21.2)

The supraspinatus muscle arises from the medial two-thirds of the supraspinous fossa of the scapula[33] and attaches ***laterally*** to the superior facet of the greater tubercle of the humerus (Fig. 21.2). Figure 21.2A also identifies the attachments of the other three muscles that comprise the rotator cuff. These muscles can also be seen elsewhere in frontal section at the shoulder joint.[10]

SUPPLEMENTAL REFERENCES

Other authors have clearly illustrated the supraspinatus muscle from behind,[8],

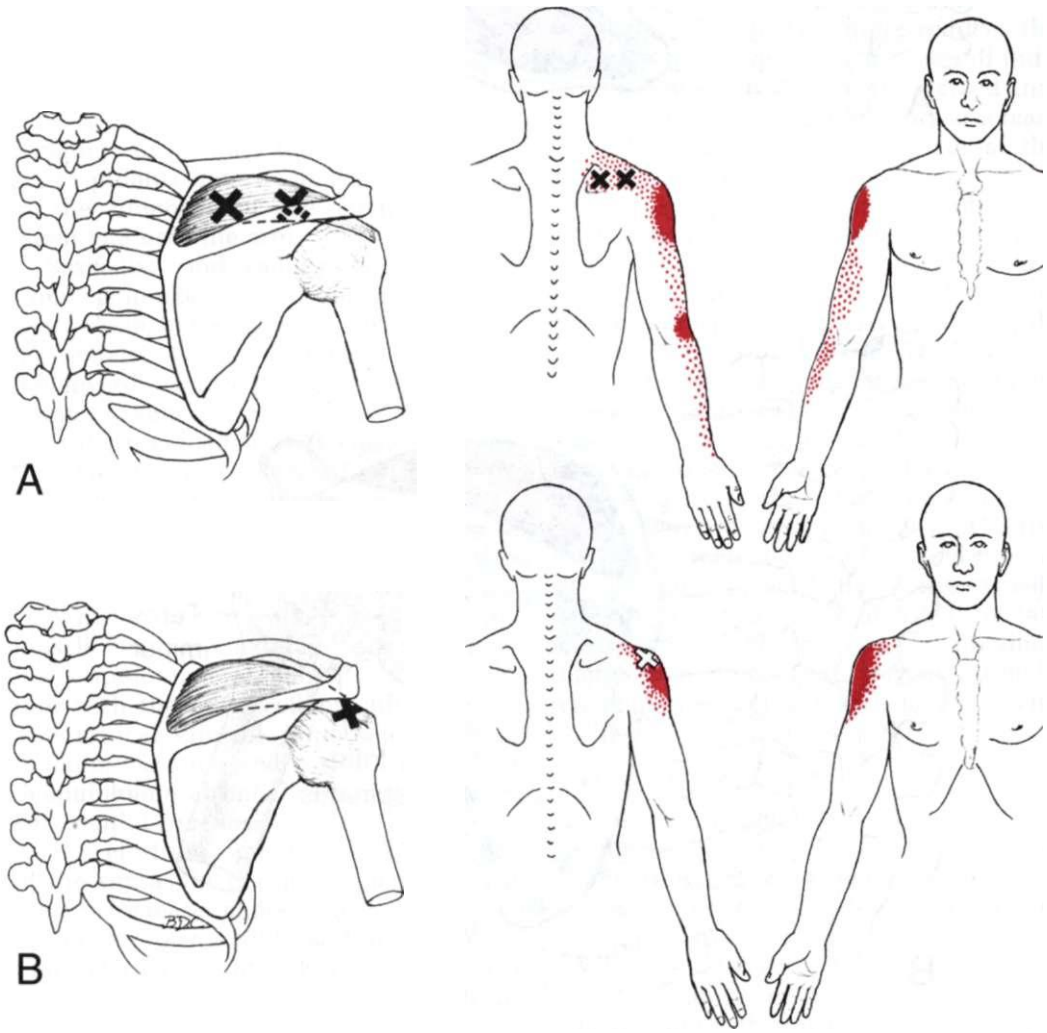


Figure 21.1. Referred pain patterns (essential reference zone *solid red,* spillover zone *stippled red)* of trigger locations (Xs) in the right supraspinatus muscle and tendon. **A,** The medial X represents the TrP location that is close to midmuscle. The lateral X is the trigger area located in the region of the musculotendinous junction. **B,** tender trigger area located in the region of attachment of the supraspinatus tendon to the capsule of the glenohumeral joint.

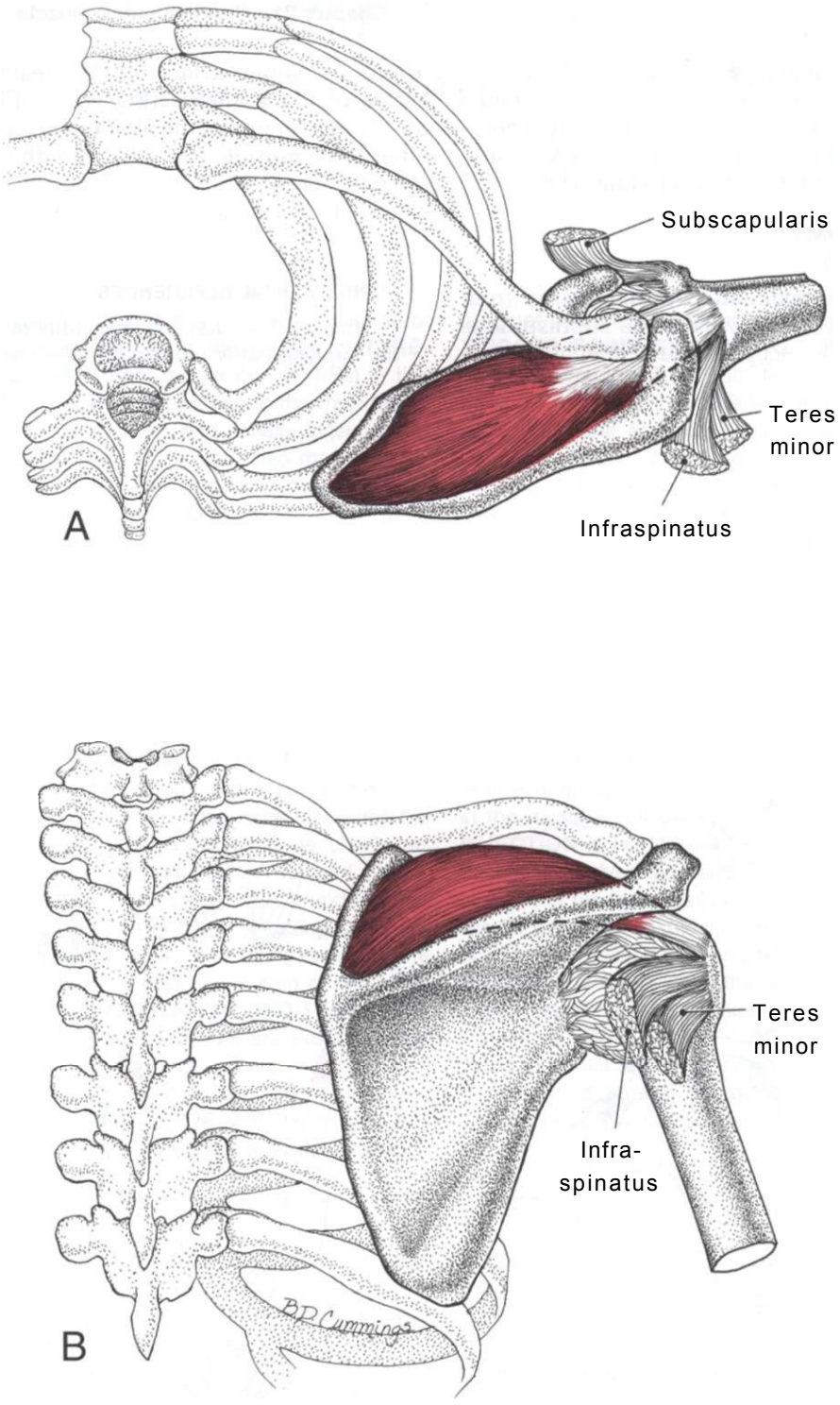


Figure 21.2. Attachments of the supraspinatus muscle *(red)*. The other rotator cuff muscles are cut and reflected to show their attachments more clearly. **A,** seen from above, including the relation of the humeral attachment of the supraspinatus muscle to the attachments of the other three rotator cuff muscles. **B,** seen from behind. It becomes obvious why such a long needle is required to reach the deep, lateral trigger point area through the overlying trapezius muscle, if one envisions the trapezius attachments to the superficial surface of the surrounding bones, the clavicle, acromion, and spine of the scapula.

[11,36,45] from above,[2] from in front,[47] in longitudinal section of the muscle in anatomical cross section of the shoulder region that shows the fiber arrangement,[14] and in sagittal section.[3,37]

3. INNERVATION

The supraspinatus muscle is innervated by the suprascapular nerve through the upper trunk of the brachial plexus, from the C_5 spinal nerve.[8]

4. FUNCTION

The supraspinatus muscle abducts the arm and pulls the head of the humerus inward toward the glenoid fossa,[7,8,29] which prevents downward displacement of the humeral head when the arm is hanging down at the side.[5,13] The supraspinatus muscle stabilizes the head of the humerus in the glenoid cavity when one uses the arm.

Basmajian and Deluca[5] showed electromyographically that supraspinatus activity alone, in the absence of other muscular activity at the shoulder, prevented downward displacement of the head of the humerus when the upper limb, hanging at the side, was loaded to exhaustion with a 7-kg weight or was loaded with sudden downward jerks. The wedge action due to the angulation of the glenoid fossa and the cartilaginous labrum help to make this mechanism so effective.[5,7]

A major function of the supraspinatus muscle is to help maintain balance among the scapulohumeral muscles in cooperation with the other rotator cuff muscles. The critical clinical importance of this function[32] is reviewed under the heading **Scapulohumeral Imbalance** in Section 11, **Differential Diagnosis.**

The electromyographic (EMG) activity of the supraspinatus during abduction of the arm increases almost linearly from resting to vigorous activity at 150° of abduction. During flexion, EMG activity increases rapidly at first, reaches a plateau, and again increases as flexion approaches 150°.[23] During sustained flexion or abduction to 90°, the supraspinatus muscle was the first to show evidence of fatigue (within a fraction of a minute) compared with other shoulder muscles. After 5 minutes both amplitude and frequency changes indicated advancing fatigue of the supraspinatus muscle.[19] Supraspinatus tendinitis is common in people doing work that demands elevated arms,[18] indicating vulnerability of this muscle to overuse in this position.

In the past, some believed that the supraspinatus is more effective than the deltoid muscle for the initiation of abduction when the arm is at the side. However, the clinical observations of Duchenne,[13] the EMG studies of Inman, *et al.*,[22] and the fact that experimental paralysis of the supraspinatus muscle simply reduces the force and endurance of abduction[5] all indicate otherwise. It is now recognized that this muscle and the deltoid work as a team throughout abduction of the arm at the glenohumeral joint.[5,23,29]

Although *Gray's Anatomy*[3] attributes weak lateral rotation of the arm to this muscle, no EMG data were found to support a function of medial or lateral rotation.

The supraspinatus is active during walking, while the arm is swinging either forward or backward, but not at the ends of the swing. This activity helps to prevent downward dislocation of the head of the humerus. During the golf swing in right-handed golfers, the right supraspinatus muscle starts out with moderate EMG activity (approximately 25% of manual muscle strength test [MMT]) that progressively fades to less than 10% of MMT by late follow-through. The left muscle maintained relatively moderate EMG activity throughout the entire swing,[38] with more activity during early and late follow-through.

5. FUNCTIONAL UNIT

Electromyography has shown that the middle deltoid, upper trapezius, and rhomboid muscles are synergistic with the supraspinatus during abduction of the arm (the latter two acting on the scapula); these muscles are also active at varying degrees during flexion.[23] Although not part of the previous study, the lower trapezius and the serratus anterior also are considered to rotate the scapula during arm elevation. The remaining three muscles of the rotator cuff, the infraspinatus, teres minor, and subscapularis assist the supraspinatus to posi-

tion and stabilize the head of the humerus accurately in the glenoid fossa during abduction.[41] Duchenne reported that the serratus anterior is an essential partner for stabilizing the scapula during abduction.[13]

The latissimus dorsi, teres major, and lower fibers of the pectoralis major muscles can act as antagonists to the supraspinatus.

6. SYMPTOMS

The chief complaint of patients with supraspinatus TrPs is referred pain that is usually felt strongly during abduction of the arm at the glenohumeral joint and is felt as a dull ache when pain is present at rest. Supraspinatus TrPs alone rarely cause severe, sleep-disturbing nocturnal pain, although other authors have noted stiffness of the shoulder[28] and night-time ache[26,28] due to involvement of the supraspinatus.

Supraspinatus TrPs may produce ache or pain at rest or pain throughout movement, but they usually do not produce a severe pain in any particular small arc of motion.

Some patients complain of snapping or clicking sounds around the shoulder joint, which disappear when the supraspinatus TrPs that are causing these symptoms are inactivated. Tautness of supraspinatus fibers due to TrP activity probably interferes with the normal glide of the head of the humerus in the fossa, a mechanism that is well described by Cailliet.[7]

When the supraspinatus muscle on the dominant side is affected, the patient reports difficulty in reaching the head to comb the hair, brush the teeth or shave and complains of restricted shoulder motion during sports activities that require arm elevation, such as serving a tennis ball. When TrPs are located on the nondominant side, the patient may be unaware of moderate restriction of these motions, since the dominant arm usually performs these arm-elevation activities.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Supraspinatus TrPs can be activated by carrying heavy objects, such as a suitcase, briefcase, or package with the arm hanging down at the side, and by regularly walking a large dog that pulls hard on a leash. The TrPs of this muscle also may be activated by lifting an object to, or above, shoulder height with the arm outstretched and by doing a task at work that demands repeated and/or moderately prolonged elevation of the arms.[17]

8. PATIENT EXAMINATION

Sola and colleagues found the supraspinatus muscle to be one of the less frequently involved shoulder-girdle muscles both in patients[43] and in young healthy adults.[44] We find that this muscle is seldom involved by itself, but usually in association with the infraspinatus or the upper trapezius, which very commonly harbor TrPs.

Range of motion of the glenohumeral joint should be examined. The Mouth Wrap-around Test (see Fig. 18.2) is restricted by supraspinatus TrPs. In the upright position, the patient is unable to hold the arm fully abducted because this contracts the supraspinatus in the shortened position and compresses any enthesopathy at its humeral attachment. When lying supine, the patient with supraspinatus TrPs has less difficulty performing the Mouth Wrap-around Test because the muscle is not lifting the weight of the arm.

The examiner should note when and where pain occurs. Supraspinatus TrPs can produce pain at rest or during movement, particularly abduction. The pain usually occurs throughout abduction. If pain occurs only in one small arc of motion, evaluate for a rotator cuff injury.

In addition to examining scapular mobility, the clinician should examine accessory joint movements (joint play) in the glenohumeral, acromioclavicular, and sternoclavicular joints. Joint play is described by Mennell.[33] Accessory movements in these joints are needed for full range of motion of the arm. The elbow complex also should be included in the examination since the supraspinatus often refers pain to that region.

Patients with supraspinatus TrPs may be aware of, and be concerned about, clicking in the shoulder during movement. The clicking can be heard and palpated when the patient moves the arm at the glenohumeral joint in a way that activates the in-

volved fibers of the supraspinatus muscle. Inactivating the supraspinatus TrPs eliminates the symptoms. The mechanism of this clicking is unknown but may relate to enthesopathy since the palpable source is also tender.

The humeral attachment of the supraspinatus tendon is most easily palpated if the hand of the upper limb being examined is placed behind the back at waist level to medially rotate the arm and bring the tendon within reach from beneath the acromion.

Palpation often reveals marked tenderness beneath the deltoid at the attachment of the supraspinatus tendon, especially in people who have abused the abduction function of this muscle. Degenerative tendinitis of the supraspinatus was commonly found among older welders.[19] Welders are particularly prone to develop shoulder pain.[20] One author (DGS) has seen early calcific deposits at the insertion of the tendon resolve with inactivation of TrPs in the supraspinatus muscle. Michele, *et* ai.[34] also noted this calcification in patients with tenderness deep in the region of this muscle. These deposits may be evidence of chronic tendon strain caused by TrP tautness of the involved supraspinatus muscle fibers. The tendinous attachment region of the supraspinatus muscle has a relatively avascular zone which makes it vulnerable to enthesopathy when subjected to sustained tension which further reduces circulation, producing ischemic hypoxia.[18]

9. TRIGGER POINT EXAMINATION (Fig. 21.3)

The patient sits comfortably, or lies on the uninvolved side, with the affected arm close to the body and relaxed. In the case of less active TrPs, it may be desirable to place the arm in the stretch position, as for stretch and spray. The supraspinatus muscle must be palpated through the trapezius muscle. The approximate locations of the midmuscle TrP and the lateral trigger area are shown in Figure 21.1 A. Other authors also have identified the lateral[49] and more medial[26] areas of tenderness associated with supraspinatus TrPs. Both tender regions lie in the supraspinous fossa of the scapula underneath a relatively thick part of the trapezius muscle. Therefore, a local twitch response of the supraspinatus is unreliably elicited by palpation, and is not always perceived by needle penetration. The medial TrP is located by flat palpation (Fig. 21.3) just above the spine of the scapula several centimeters (about 1 to 1.5 inches) lateral to the vertebral border of the scapula in the midregion of the muscle fibers. TrP tenderness located in the midregion of the

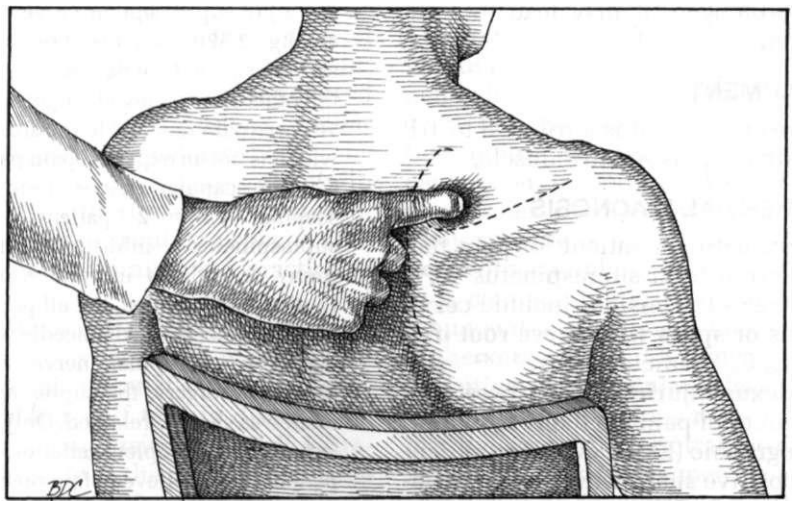


Figure 21.3. Application of digital pressure to the medial trigger point region in the supraspinatus muscle. Sufficient pressure on an active trigger point elicits a pain that the patient recognizes.

supraspinous fossa can be either a central TrP (the midportion of some fibers pass here at about half the thickness of the muscle) or an attachment TrP, which can occur anywhere in the supraspinous fossa since supraspinatus fibers attach throughout the medial two-thirds of the fossa.[3a] In the middle portion of this deep muscle, central and attachment TrPs are not distinguishable by palpation. The lateral region of spot tenderness is palpated in the space between the spine of the scapula and the clavicle, just medial to the acromion. This tenderness most likely represents enthesopathy of the musculotendinous junction secondary to increased muscle tension associated with central TrPs in that muscle.

The severity and extent of the referred pain evoked by needling TrPs in the lateral muscular area are usually out of proportion to the slight degree of tenderness to deep palpation reported by the patient, probably because of the depth of muscle that the palpation pressure must penetrate.

A third tender spot may lie in the region of the tendinous attachment to the head of the humerus, where the tendon of the muscle blends with the joint capsule to form part of the rotator cuff under the acromion (Fig. 21.IB). This tender region corresponds to the poorly vascularized area described by Hagberg[18] that is particularly vulnerable to sustained or repeated overload. When local hypoxia is sufficiently severe and prolonged, it may lead to local calcification.

10. ENTRAPMENT

No nerve entrapment is attributed to TrP tension in the supraspinatus muscle.

11. DIFFERENTIAL DIAGNOSIS

When evaluating a patient for pain that may be referred from supraspinatus TrPs, other diagnoses to consider include **cervical arthritis or spurs with nerve root irritation,**[26] C_5-C_6 radiculopathy,[26, 42] and **brachial plexus injuries.** All of these neurogenic sources of pain are likely to exhibit electromyographic (EMG) evidence of denervation (positive sharp waves and fibrillation potentials) in the muscles supplied by the compromised nerves. Muscles with only myofascial TrPs show no EMG evidence of denervation because EMG evidence of a muscle entrapment of a nerve shows up in the muscles that are distal to the muscle causing the entrapment.

In addition, **subdeltoid bursitis, rotator cuff tears** and supraspinatus TrPs all may cause tenderness at the tendinous attachment to the rotator cuff (capsule) beneath the acromion. Only the TrPs, however, cause spot tenderness in the midportion of the supraspinatus muscle. Rotator cuff diseases including tears are considered in more detail below.

For a small group of patients with shoulder pain, surgical release of **entrapment of the suprascapular nerve** at the suprascapular notch is needed, and with appropriate electrodiagnostic evaluation this group can be clearly identified. Surgeons agree that conservative treatment should be tried before surgery, especially in less involved cases. Patients with nerve entrapment can also have TrPs that contribute significantly to their pain. A patient should not be considered for surgical release of the suprascapular ligament without positive electrodiagnostic findings. Magnetic resonance imaging (MRI) facilitates the diagnosis of suprascapular nerve entrapment in patients who have shoulder pain of unclear origin, with a perineural mass and atrophy of the spinatus musculature.[15]

Post and Mayer[39] reported 10 cases (0.4%) of suprascapular nerve entrapment among 2,520 patients presenting with shoulder pain. One patient had a sarcoma, the other nine received surgery. In 6 of the 10 cases pain radiated to the arm and neck, which is not an expected pain pattern from a suprascapular nerve lesion at the scapula. Only 2 patients evidenced supraspinatus or infraspinatus muscle atrophy, 6 patients initially had negative routine EMG studies, but all patients were positive when a coaxial needle was used to obtain suprascapular nerve conduction times. At surgery, the suprascapular ligament was sharply released. Only 4 patients experienced complete relief of pain. Four patients were relieved of preoperative pain complaint, but still had persistent shoulder pain of unidentified origin, and one had persistent bilateral bicipital tendinitis.

Although all of these patients had a demonstrable nerve entrapment that required attention, it is also apparent that the entrapment was not the only source of their pain. Myofascial TrPs are likely candidates for much of the unexplained residual pain, but apparently were not considered. There is need for a well-designed research study to critically examine the role of TrPs in such patients with shoulder pain before and after operation.

Another example indicating the importance of identifying suprascapular nerve entrapment was reported by Hadley, *et* a/.[16]

Rotator Cuff Lesions

Two orthopedic surgeons carefully analyzed the results of arthroscopic examinations under anesthesia of 123 patients with painful shoulders to better understand the nature of rotator cuff lesions.[1] Despite a thorough surgical clinical examination and arthroscopy, 55% of the patients remained with an "unclear" diagnosis. Impingement syndrome was identified in 32% of patients, although only 16% showed thickening and fibrosis with or without inflammation. It was not indicated what finding warranted that diagnosis in the remaining 16%. Inflammatory changes that mostly affected the supraspinatus without thickening, fibrosis, or rupture were seen in 6% of patients. (This is suggestive of enthesopathy secondary to TrPs, but TrPs were not included in the description of examination). One can only wonder how many of the majority of the patients who had unclear or unsubstantiated diagnoses suffered from pain caused by TrPs. Authors familiar with myofascial TrPs consider them to be among the most common causes of pain in the shoulder.[6,50] Unfortunately, recognition of TrPs by palpation requires a special skill that usually takes training and practice to learn.

Neither rotator cuff disease nor impingement syndrome, as each term is commonly used, is a specific or satisfactory diagnosis. However, **rotator cuff tear** is diagnosable with high accuracy using MRI.[12,35] Ultrasound was reliable in the diagnosis of large tears, less so for small ones, and of little value for tendinitis.[12] Patients treated conservatively showed continuing improvement through 18 months of follow-up if a rotator cuff tear was <1 cm^2, had a duration of <1 year before treatment, and there was no significant functional impairment initially.[4] These patients should respond well to inactivation of corresponding TrPs (utilizing non-stretching measures), especially if the TrPs contributed significantly to the overload that caused the tear. Unfortunately, no controlled research studies were found that specifically addressed the contribution of TrPs to these rotator cuff problems. Identifying and treating the TrP component promptly should save patients much misery and save the health care system much expense. It should be worth research investigation.

Scapulohumeral Imbalance

One indicator of the stabilizing effect of the muscles surrounding the shoulder joint is the finding in patients under anesthesia of 8 unstable joints among 123 patients with painful shoulders that were clinically stable before anesthesia.[1] Lippitt and Matsen[32] reported a sophisticated and well-illustrated analysis of glenohumeral stability that was based on cadaver dissections. They identified unbalanced muscular forces that produced net reaction forces which direct the humeral head outside of the glenoid cavity and create an unstable joint that is prone to anatomical damage. The margin for error is relatively small for large angles of elevation under heavy load. They emphasize that normal glenohumeral function depends strongly on good dynamic balance of the muscles.[32]

As discussed in Chapter 2, Section B of this volume, TrPs can cause increased muscle tension, incoordination, and inhibition of muscles in the same functional unit. They provide a potent source for disturbance of scapulohumeral muscular balance.

A common clinical symptom of scapulohumeral muscle imbalance is "catching" of the joint with sudden severe pain when executing a particular elevation movement. The acute pain is relieved by returning the arm to a neutral position. This "catching" tends to happen repeatedly and is relieved by inactivating the TrP or TrPs in the muscle(s) that often cause the dynamic muscle imbalance. The unbalanced

tension would facilitate the humeral head "climbing the wall" of the glenoid cavity, and that could cause pinching of the synovium. Pinching of the synovium in this way might be demonstrable in a research study using video-recorded ultrasound imaging *during* joint movement before and after TrP release that relieves pain.

Previous (Referral) Diagnoses

Clinicians who are skillful in dealing with TrPs often receive referrals of "problem" patients who are dissatisfied with the diagnosis(es) and treatment of their shoulder pain. These patients commonly arrive with essentially no diagnosis or one of those mentioned below. The problem often turns out to be contributed to, if not largely caused by, supraspinatus (and other) TrPs. In every case, an essential step is to identify the TrPs that reproduce the patient's pain.

One such diagnosis that is commonly seen is **bursitis,** sometimes identified more specifically as subdeltoid or subacromial bursitis. The fact that TrPs refer tenderness as well as pain to the region of these bursae adds to the diagnostic confusion. The subdeltoid bursa is large and lies beneath the deltoid muscle against the joint capsule.[9] The tenderness of this bursa is more diffuse than the spot tenderness of deltoid TrPs, which also exhibit relatively easily palpated taut bands.

The subacromial bursa is more superficial and lies between the deep surface of the acromion and the tendon of the supraspinatus muscle overlying the joint capsule.[9] This bursitis is diagnosed by palpation of tenderness directly under the acromial process with the arm in the neutral resting position at the patient's side and duplicating the patient's pain at the point of pressure. However, by palpation, this bursitis alone is indistinguishable from supraspinatus enthesopathy. The other diagnostic test for subacromial bursitis is reproducing only the same local pain by application of resistance at 90° of arm abduction. Supraspinatus TrPs could also reproduce this finding. The presence of both conditions is not unlikely. The tendinous attachment region of the supraspinatus muscle is in direct contact with this bursa. Enthesopathy (nociceptor sensitization) of that attachment caused by the sustained tension induced by TrPs in the muscle belly may become an inflammatory enthesitis that, by its direct contact, causes inflammatory changes in the subacromial bursa. Experimental evidence supporting this interpretation appears under "**Frozen Shoulder**" in Chapter 26, Section 11. When present, active supraspinatus TrPs should be treated immediately. If both conditions are present, both must be treated for prompt relief.

Additional diagnoses that can be confused with supraspinatus TrPs are rotator cuff disease (reviewed above), **supraspinatus tendinitis, "frozen shoulder,"** and **brachial neuritis.**

Related Trigger Points

The shoulder pain of the supraspinatus myofascial pain syndrome does not have the deep, aching quality of the pain referred by infraspinatus TrPs, which goes deep into the shoulder joint and is easily mistaken for arthritis of the glenohumeral joint.[42]

In our experience, both the supraspinatus and infraspinatus muscles frequently develop TrPs, and the trapezius muscle may become involved as part of the functional unit.

Since the deltoid muscle lies in the pain reference zone of the supraspinatus, it may develop satellite TrPs.

If the latissimus dorsi muscle has become involved as an antagonist, inactivating its TrPs will increase abduction of the arm by release of this adductor muscle tension.

12. TRIGGER POINT RELEASE
(Fig. 21.4)

If there is any suspicion of rotator cuff damage, the supraspinatus should not be stretched. Treatment can include nonstretching methods such as TrP pressure release, deep massage to the taut band, gentle hold-relax (no range of movement involved), indirect techniques (e.g., using the principles of Hoover[21] or Jones[24]), and/or injection. Application of vapocoolant or icing can precede any of these techniques. These techniques are described in Chapter 3, Section 12.

When muscle lengthening is needed and additional tension is not contraindicated,

spray and stretch can be used. The forearm of the seated patient is placed behind the back at waist level (Fig. 21.4). After a few preliminary sweeps of spray, the arm is moved across the back to take up the slack. The patient is encouraged to relax in this lengthened position by leaning back and pinning the arm against the chair back. The stream of vapocoolant spray is applied in unhurried parallel sweeps from medial to lateral in line with and over the supraspinatus muscle fibers, across the acromion and over the deltoid, down the arm to the elbow, and over the forearm (Fig. 21.4). By having the patient bend the head and neck toward the opposite side and by spraying upward over the upper trapezius, TrP tension in this muscle can be released, since it also often is involved. Hot packs are applied, followed by full range of active motion of the treated muscles.

If both the supraspinatus and infraspinatus TrPs are extremely sensitive and the patient has difficulty in placing the

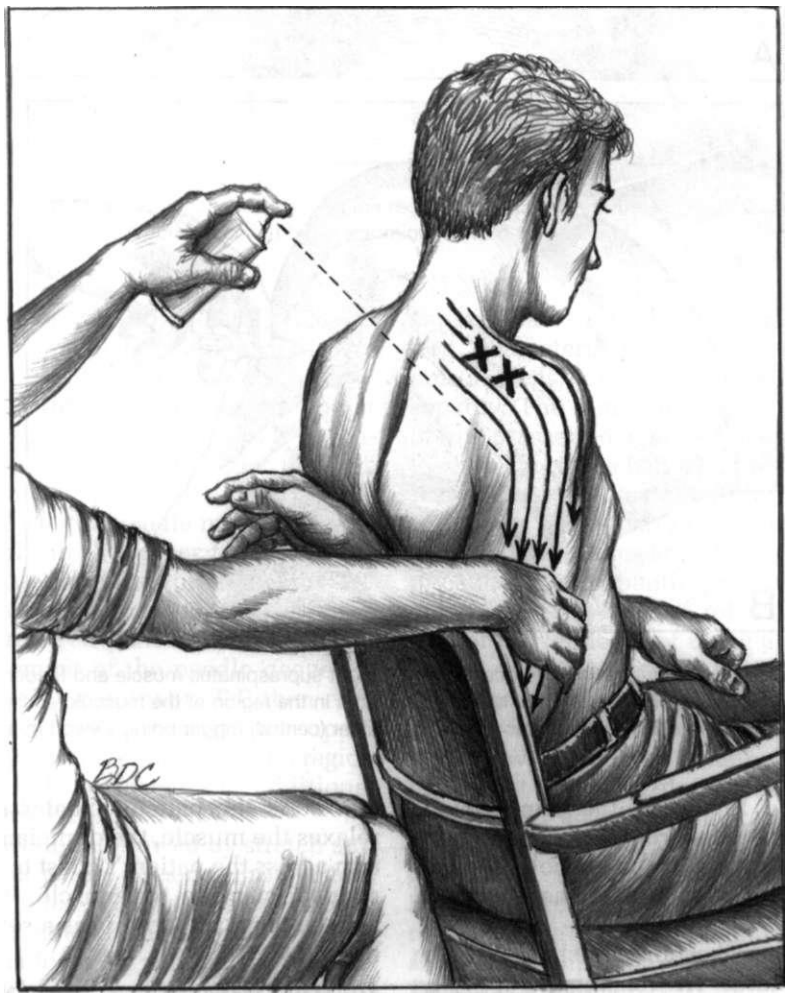


Figure 21.4. Stretch position and spray pattern *(arrows)* for trigger points (Xs) in the right supraspinatus muscle. The operator positions the patient's arm in medial rotation, exerts downward pressure on the arm, and then directs the arm across the patient's back. See text for augmentation techniques. An alternate stretch is done by bringing the patient's arm across the front of the body instead of behind it, but this alternate stretch does not provide as much medial rotation at the glenohumeral joint. If there is a suspected rotator cuff tear, the supraspinatus should NOT be stretched. Trigger points can be treated by trigger point pressure release, massage, indirect techniques, and/or injection instead of stretch.

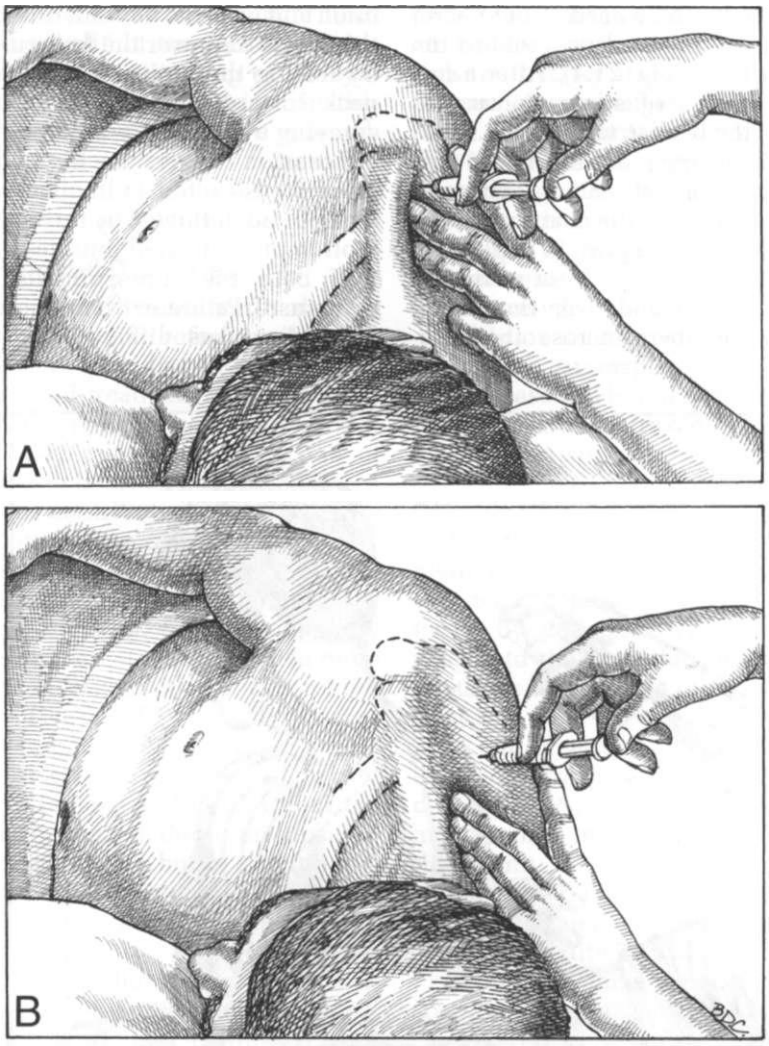


Figure 21.5. Injection of sensitive trigger locations in the right supraspinatus muscle and tendon with the patient lying on the left side. **A,** injecting the lateral trigger area in the region of the musculotendinous junction as seen from above. **B,** injecting a more medial midfiber (central) trigger point, viewed from above.

hand behind the back, the arm may be brought across the front of the chest instead. Either way, the vapocoolant traces the pattern of Figure 21.4, as described above.

Lewit[31] describes and illustrates release of supraspinatus TrP tension by applying postisometric relaxation to the arm. It is held at the elbow by the clinician and moved across the patient's chest to take up slack. The patient breathes in and contracts the muscle by gently pressing the elbow laterally against the clinician's light resistance. As the patient exhales slowly and relaxes the muscle, the clinician moves the arm across the patient's chest to take up additional slack in the muscle. Patients can also be taught to do this as a self-stretch at home.

13. TRIGGER POINT INJECTION
(Fig. 21.5)

Midfiber Supraspinatus Trigger Point

With the patient lying on the uninvolved side, the medial (central) trigger point (TrP) is located by palpation and in-

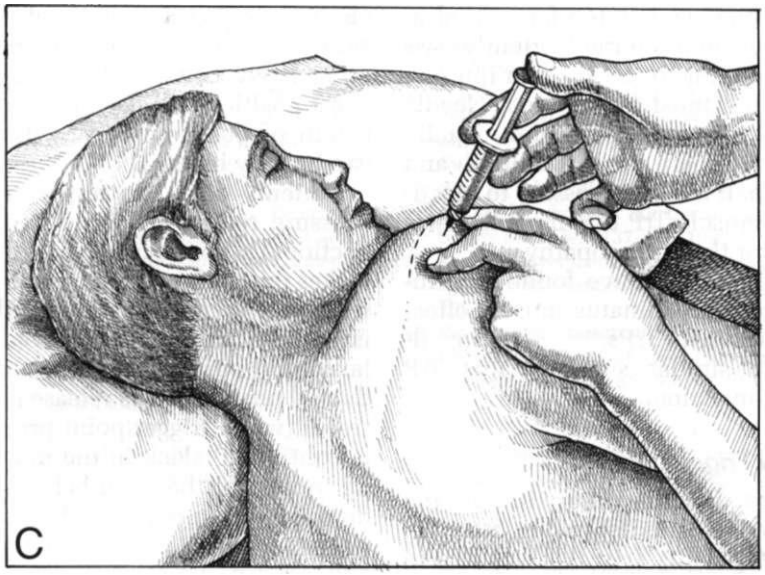


Figure 21.5.—*continued* **C,** injecting the region of attachment of the supraspinatus tendon beneath the acromion, viewed from behind.

jected, using a 3.2- to 3.8-cm (1.25- to 1.5-in) needle, which is directed downward *into the bony fossa* of the scapula below and behind the edge of the upper trapezius (Fig. 21.5B). If the needle is inserted lateral to this TrP in order to angle the needle medially, instead of vertically as in Figure 21.5B, the needle may encounter an active TrP_2 in the upper trapezius (see Fig. 6.2). Penetration of this trapezius TrP produces a visible local twitch response and elicits referred pain to the neck. Continued movement of the needle deeper to penetrate the supraspinatus TrP then elicits its referred pain pattern to the upper limb. The operator should probe the region with the needle to locate any additional supraspinatus TrPs.

If this injection, followed by stretch and spray and hot packs, does not fully restore shoulder motion, the operator should check just medial to the acromion in the supraspinous fossa for another spot of tenderness in the region of the musculotendinous junction. One should also check for subacromial tenderness.

Lateral Supraspinatus Trigger Area

If pressure on a well localized tender spot deep in the lateral portion of the supraspinatus muscle refers pain in a pattern characteristic of the supraspinatus muscle, it is likely to be caused by enthesopathy. The tenderness is elicited by applying pressure deep into the supraspinous fossa in the space between the spine of the scapula and the clavicle, just medial to the acromion. This location is beyond reach of massage techniques and is marginal for application of therapeutic pressure. The tenderness is usually best relieved by injection of the tender spot using a needle that is long enough to reach it through the overlying upper trapezius muscle. The authors usually have found procaine to be effective. However, to the extent that the tenderness at this musculotendinous junction is caused by a sterile tissue reaction due to prolonged overload, injection with analgesic and a corticosteroid (with proper limitations of frequency and dose) may hasten recovery. We do NOT recommend use of steroid for injecting the central midfiber TrPs.

In a large person, injection of the lateral trigger area may require a 5 cm (2 in) needle directed deep into the supraspinous fossa (Fig. 21.5A). It is important to direct the needle precisely to the spot of deep tenderness. If the injection is directed caudally

from behind the clavicle too far medial to the trigger area, one can inadvertently pass the needle anterior to the scapula into the rib cage, which must be avoided. Needle contact with this sensitive region usually flashes referred pain to the deltoid and down the arm. It is also necessary to inactivate the midmuscle TrP that most likely is responsible for the enthesopathy.

Other authors also have found that injection of the supraspinatus muscle effectively inactivates its TrPs.[25-27] Rachlin[40] illustrates two similar supraspinatus TrP locations for injection.

Subacromial Trigger Area

Tenderness beneath the tip of the acromion that remains following inactivation of supraspinatus TrPs is likely due to enthesopathy of the humeral attachment of the supraspinatus tendon, which is often identified as supraspinatus tendinitis. This tenderness should respond to injection of a local anesthetic (Fig. 21.5C). Injection is followed by passive stretch of the muscle during a few sweeps of the spray, and then by a hot pack.

14. CORRECTIVE ACTIONS

The patient should avoid supraspinatus overload by not carrying a heavy object, such as an overloaded briefcase, in the hand with the arm hanging down at the side, and by not lifting heavy things overhead. The patient also should avoid sustained contraction of the muscle, as when maintaining the arm in abduction or flexion (e.g., holding the arms up continuously for several minutes to put curlers in the hair, or to do overhead work) and should drop the arms occasionally to relax the muscles so they can replenish their blood supply.

The patient must learn to release the TrP tightness by slowly, firmly stretching the supraspinatus muscle, pulling the forearm across and upward behind the *back* with the other hand in order to position the involved arm as in Figure 21.4. This passive stretch may be done most effectively while the patient sits on a stool under a warm shower with the water beating on the muscle. The patient also may stretch the muscle by bringing the elbow of the involved side across the *front* of the chest with the other hand. Lewit[31] illustrates this across-chest position and describes patient application of postisometric relaxation, which can be very helpful.

Patients also can apply trigger point pressure release (described in Chapter 3, Section 12) for themselves. A Theracane® makes this self-treatment much easier. This treatment is most effective if the pressure is applied while the involved arm is relaxed and *supported* in a comfortably adducted position. This release is more effective if (while trigger point pressure release is continued) slack in the muscle is taken up by sliding the hand behind the back as muscle tension is reduced.

REFERENCES

1. Adolfsson L, Lysholm J: Arthroscopy for the diagnosis of shoulder pain. *Int Orthop* 15(4):275-278,1991.
2. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991(p. 383, Fig. 6.35).
3. *Ibid.* (p. 395, Fig. 6.52).
3a. *Ibid.* (p. 391, Fig 6.45).
4. Bartolozzi A, Andreychik D, Ahmad S: Determinants of outcome in the treatment of rotator cuff disease. *Clin Orthop* 308.-90-97, 1994.
5. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 185, 240-242, 263, 268, 274, 275, 385).
6. Bonica JJ, Sola AE: Other painful disorders of the upper limb. Chapter 52. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, 1990 (pp. 947-958).
7. Cailliet R: *Soft Tissue Pain and Disability.* F.A. Davis, Philadelphia, 1977 (pp. 149-151, Fig. 122).
8. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 523, Fig. 6-46).
9. *Ibid.* (p. 373).
10. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 35).
11. *Ibid.* (Fig. 524).
12. D'Erme M, De Cupis V, De Maria M, *et al.:* [Echography, magnetic resonance and double-contrast arthrography of the rotator cuff. A prospective study in 30 patients]. *Radiol Med (Torino) 86(l-2):72-80,*1993.
13. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 59-63).
14. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sect. 30).
15. Fritz RC, Helms CA, Steinbach LS, *et al.:* Suprascapular nerve entrapment: evaluation with MR imaging. *Radiology 182(2):437-444,* 1992.
16. Hadley MN, Sonntag VK, Pittman HW: Suprascapular nerve entrapment. A summary of seven cases. *J Neurosurg* 64(6);843-848, 1986.

17. Hagberg M: Electromyographic signs of shoulder muscular fatigue in two elevated arm positions. *Am J Phys Med* 60(3):111-121, 1981.
18. Hagberg M: Local shoulder muscular strain—symptoms and disorders. J *Human Ergol* 13:99-108,1982.
19. Herberts P, Kadefors R: A study of painful shoulder in welders. *Acta Orthop Scand* 47(4):381-387, 1976.
20. Herberts P, Kadefors R, Andersson G, Petersen I: Shoulder pain industry: an epidemiological study on welders. *Acta Orthop Scand* 52(3):299-306, 1981.
21. Hoover HV: Functional technic. In: *Yearbook, Academy of Applied Osteopathy.* Carmel, CA, 1958, (pp. 47-51).
22. Inman VT, Saunders JB, Abbott LC: Observations on the function of the shoulder joint. *J Bone Joint Surg* 26:1-30, 1944 (pp. 18, 21).
23. Ito N: Electromyographic study of shoulder joint. *J fpn Orthop Assoc* 54.1529-1540, 1980.
24. Jones LH: *Strain and Counterstrain.* The American Academy of Osteopathy, Colorado Springs, 1981.
25. Kellgren JH: A preliminary account of referred pains arising from muscle. *Br Med J* 1:325-327, 1938 (Case 3).
26. Kelly M: New light on the painful shoulder. *Med J Aust* 1:488-493, 1942 (Cases 2 and 8, Figs. 2B and 3C).
27. Kelly M: The nature of fibrositis. III. Multiple lesions and the neural hypothesis. *Ann Rheum Dis* 5:161-167, 1946 (Case 2).
28. Kelly M: Some rules for the employment of local analgesia in the treatment of somatic pain. *Med J Aust* 3:235-239, 1947 (Table 1 No. 4).
29. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 272).
30. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (p. 98).
31. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 203-205).
32. Lippitt S, Matsen F: Mechanisms of glenohumeral joint stability. *Clin Orthop Res 291:20-28,* 1993.
33. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
34. Michele AA, Davies JJ, Krueger FJ, et *al.:* Scapulocostal syndrome (fatigue-postural paradox). *NY State J Med* 50:1353-1356, 1950 (p. 1355).
35. Morrison DS, Ofstein R: The use of magnetic resonance imaging in the diagnosis of rotator cuff tears. *Orthopedics* 13(6):633-638, 1990.
36. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 28).
37. *Ibid.* (Fig. 45).
38. Pink M, Jobe FW, Perry J: Electromyographic analysis of the shoulder during the golf swing. *Am J Sports Med 18*(2):137-140, 1990.
39. Post M, Mayer J: Suprascapular nerve entrapment: Diagnosis and treatment. *Clin Orthop Res* 223:126-136, 1987.
40. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360, (pp. 320- 322).
41. Rasch PJ: *Kinesiology and Applied Anatomy.* Ed. 7. Lea & Febiger, Philadelphia, 1989 (pp. 127-131).
42. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981 (Tables 1 and 2).
43. Sola AE, Kuitert JH: Myofascial trigger point pain in the neck and shoulder girdle. *Northwest Med* 54:980-984, 1955.
44. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med* 34:585-590, 1955.
45. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 324).
46. Steinbrocker O, Isenberg SA, Silver M, *et al.:* Observations on pain produced by injection of hypertonic saline into muscles and other supportive tissues. *J Clin Invest* 32:1045-1051, 1953 (Table 2).
47. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 313).
48. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 11:425-434, 1952.
49. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J Am Osteopath Assoc* 72:697-710, 1973 (Fig. 28 Part 1, p. 10).
50. Weed ND: When shoulder pain isn't bursitis. The myofascial pain syndrome. *Postgrad Med 74(3)101-*102, 1983.
51. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Little, Brown & Company, Boston, 1988 (p. 211, Fig. 12-2).

CHAPTER 22
Infraspinatus Muscle

HIGHLIGHTS: **REFERRED PAIN** from the usual trigger point (TrP) locations in this *"Shoulder Joint Pain"* muscle concentrates deeply in the anterior deltoid region and in the shoulder joint, extending down the front and lateral aspect of the arm and forearm, and sometimes including the radial half of the hand. Pain occasionally may be referred to the suboccipital and posterior cervical areas. **A** trigger area near the vertebral border of the scapula may refer pain over the adjacent rhomboid muscles. **ANATOMICAL** attachments are, medially, to the infraspinous fossa of the scapula and, laterally, to the greater tubercle of the humerus. **FUNCTION** of this muscle includes stabilization of the head of the humerus in the glenoid cavity during movement of the arm, with its chief action being lateral rotation of the arm at the glenohumeral joint. **SYMPTOMS** are referred pain when sleeping on either side, inability to reach behind to a back pocket or to brassiere hooks in back, and inability to reach to comb the hair or brush the teeth. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** usually result from acute overload while reaching backward and up. **PATIENT EXAMINATION** reveals restriction of medial and lateral rotation at the glenohumeral joint, demonstrated by the Hand-to-shoulder Blade Test. **TRIGGER POINT EXAMINATION** locates active TrPs midmuscle 1 or 2 cm (0.5 or 1 in) below the spine of the scapula, or occasionally more caudally. **DIFFERENTIAL DIAGNOSIS** includes suprascapular nerve entrapment, bicipital tendinitis, C_5-C_6 radiculopathy and arthritis in the shoulder joint. **TRIGGER POINT RELEASE** of this muscle with the spray and stretch technique may be done by adducting the arm across the front of the chest, or behind the back, while directing the vapocoolant or icing strokes laterally over the muscle and down the arm over its referred pain pattern, including the hand. Separate cooling sweeps are directed upward over the suboccipital area. **TRIGGER POINT INJECTION** begins with the patient lying on the side opposite the involved muscle while the TrP is localized between palpating fingers. Injection is followed by passive stretching, active range of motion, and hot packs. **CORRECTIVE ACTIONS** include elimination of recurrent overload on the muscle, proper positioning in bed at night, self-administered trigger point pressure release, and self-stretch exercises.

1. REFERRED PAIN
(Fig. 22.1)

We have found that, when the patient feels referred pain from myofascial trigger points (TrPs) intensely deep in the front of the shoulder, the infraspinatus muscle is the major source.[46]

Most reports of the referred pain pattern from this muscle identify the front of the shoulder as the major target area (Fig. 22.1A).[21,33,39,42,45,47,49,50,51,53] In 193 cases of infraspinatus referred pain, all patients identified the front of the shoulder as painful.[45] The shoulder pain is usually felt *deep* within the joint.[47] The pain is described as also projecting down the anterolateral aspect of the arm,[21,24,30,33,39,45,47,49,51,53] to the lateral forearm,[33,39,42,45,47,49,51,53] to the radial aspect of the hand,[30,33,39,42,47,51,53] and occasionally to the fingers,[30,45] or to the upper posterior cervical region (Fig. 22.1A). Patients usually identify the most painful area by covering the front of the shoulder with the hand.

A few authors located the pain in the back of the shoulder,[21,24] which we find can be referred simultaneously from TrPs that also are present in the adjacent teres minor

muscle. Bonica and Sola[4] described aching pain referred primarily to the region of the deltoid muscle. Rachlin[37] emphasized pain to the back of the shoulder and also included referral along the vertebral border of the scapula and to the base of the neck in the region of the levator scapulae.

Much of the variation among these reports is probably due to the appearance of referred pain in the variable spillover zones. Among 193 subjects, 46% experienced pain in the deltoid and biceps brachii regions, none reported elbow pain, 21% reported pain in the radial forearm, 13% in the radial side of the hand, and 14% in the suboccipital posterior cervical area.[45] The three Xs in Figure 22.1A give common examples of where TrPs are found

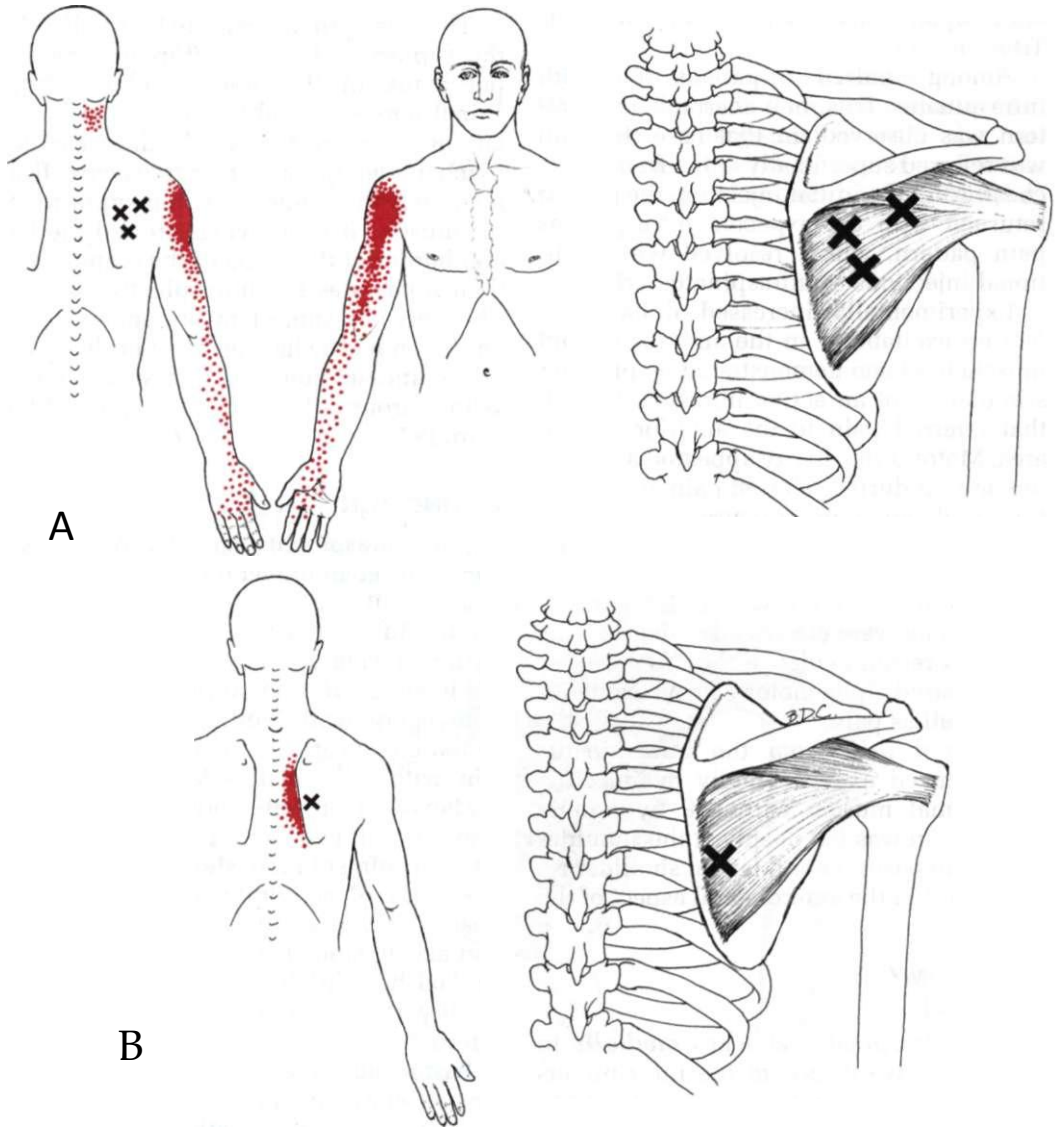


Figure 22.1. Referred pain patterns *(red)*, and location of corresponding trigger points (Xs) in the right infraspinatus muscle. *Solid red* shows essential referred pain zones, *stippled red* areas show spillover zones. **A,** three common locations of trigger points. **B,** location of tenderness at a trigger area in the region of the musculotendinous junction and the corresponding referred pain pattern.

in the midportion (endplate zone) of this muscle. No distinction is made in the pain patterns arising from these TrPs.

Occasionally, a trigger area of spot tenderness occurs close to the vertebral border of the scapula (Fig. 22.1B) that is located near the musculotendinous junction region and refers pain to the adjacent interscapular rhomboid muscles. This pain pattern is difficult to distinguish from that of the trapezius TrP_4 (see Fig. 6.3) and is likely enthesopathy secondary to infraspinatus TrPs.

Among hundreds of patients seen with infraspinatus TrPs, one aberrant pain pattern was observed; in that case the pain was referred superficially to the front of the chest. After the initial injection, the patient returned with the expected infraspinatus pain pattern, which resolved with additional injections of infraspinatus TrPs.[47]

Experimentally, increased alpha motor neuron excitability in the anterior deltoid muscle has been demonstrated by pressure stimulation of an active infraspinatus TrP that referred pain to the anterior deltoid area. Motor unit activity appeared at rest in the deltoid during referred pain elicited by this application of pressure. The patient was unable to eliminate this motor unit activity by relaxation, although surrounding muscles that were not within the pain reference zone were electrically silent.[48] This supports recent evidence that TrPs can refer increased alpha motor neuron excitability as well as pain.

Referred pain from the infraspinatus was induced experimentally by injecting the normal muscle with 6% hypertonic saline. Pain was felt deeply at the shoulder tip, in the posterior and lateral shoulder regions, and in the anterolateral aspect of the arm.[22]

2. ANATOMY
(Fig. 22.2)

The infraspinatus attaches ***medially*** to the medial two-thirds of the infraspinous fossa below the spine of the scapula and to adjacent fascia. ***Laterally*** it fastens to the posterior aspect (middle facet) of the greater tubercle of the humerus[6] (Fig. 22.2), and the tendon blends superiorly and posteriorly with the shoulder joint capsule.[10]

The upper medial portion of the muscle is covered by the lower trapezius.

Supplemental References

Other authors have illustrated the infraspinatus muscle in dorsal view without its artery and nerve supply,[1,6,8,34,43] and with them,[9] and in cross section.[13,35]

3. INNERVATION

The infraspinatus muscle is supplied by the suprascapular nerve, through the upper trunk of the brachial plexus from spinal nerves C_5 and C_6.[6] The suprascapular nerve passes through the scapular notch under the superior transverse ligament and then innervates the supraspinatus muscle. It next swings around the lateral border of the spine of the scapula and then innervates the infraspinatus muscle. The nerve is subject to entrapment as it passes under the ligament that bridges over the scapular notch and also where it swings around the end of the spine of the scapula.[7]

4. FUNCTION

The infraspinatus laterally rotates the arm at the glenohumeral joint with the arm in any position,[12] and helps to stabilize the head of the humerus in the glenoid cavity during movements of the arm.[5,6,25]

Inman, *et al.*[w] demonstrated that, electromyographically, infraspinatus activity increased linearly with increasing abduction, with additional peaks of activity during flexion. In a well-conducted study, Ito[19] showed that, compared to the supraspinatus, the infraspinatus showed a relatively low level of activity that gradually and steadily increased throughout both abduction and flexion. The one exception was a marked but variable increase at 140° that usually reached only moderate levels of contraction.

Basmajian and De Luca[3] clearly described how the angulation of the glenoid fossa, together with the activity of horizontal fibers in several muscles, provides a wedge action that prevents downward displacement of the head of the humerus. They showed that activity of the

supraspinatus muscle and of posterior fibers of the deltoid prevented downward displacement of the humeral head, even with considerable downward loading of the adducted arm. However, in other positions, additional protection of the joint by rotator cuff muscular activity, which includes contraction of the infraspinatus, becomes critical.[3] In abduction, multiple muscles contribute both to the abductive force and to stabilization of the humeral head in the glenoid fossa.

Electrical activity of the infraspinatus is not mentioned in conjunction with adduction of the shoulder, and Duchenne[12] found no adduction component on stimulation.

At the beginning of a golf swing in right-handed golfers, the right infraspinatus exhibits barely 30% of the EMG activity seen during maximum voluntary contraction and quickly fades down to around 10%. The left infraspinatus starts with a low level of activity and reaches a peak of nearly 60% of maximum contraction during early follow-through.[36]

5. FUNCTIONAL UNIT

The infraspinatus and teres minor have nearly identical actions, but different innervations. The infraspinatus muscle functions in parallel with the teres minor and posterior deltoid for lateral rotation of the arm. The infraspinatus also functions synergistically with the supraspinatus and other rotator cuff muscles by stabilizing the head of the hu-

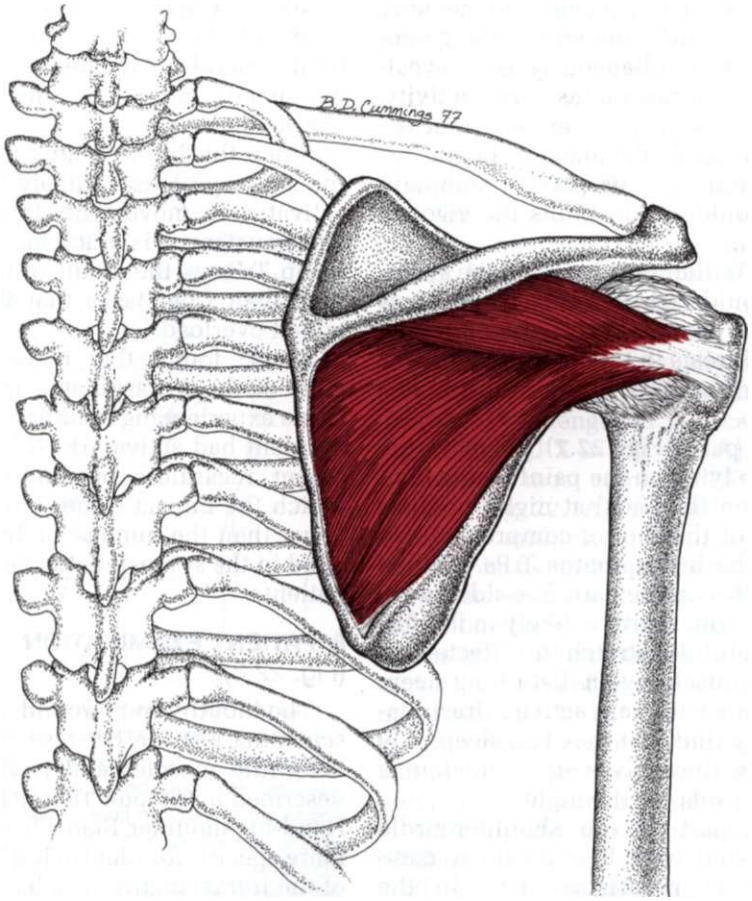


Figure 22.2. Visible attachments of the infraspinatus muscle, showing the direction of muscle fibers.

merus in the glenoid cavity during abduction and flexion of the arm.[3]

The subscapularis, pectoralis major and anterior deltoid muscles act as antagonists to the infraspinatus and posterior deltoid for rotation of the arm.

6. SYMPTOMS

We agree with other authors that when myofascial pain is referred to the shoulder joint, the infraspinatus, supraspinatus, and sometimes the levator scapulae muscles are its most likely muscular sources.[23,40]

Various patients with an infraspinatus TrP commonly complain: "I can't reach into my back pants pocket; I can't fasten my brassiere behind my back; I can't zip up the back of my dress; I can't get my sore arm into my coat sleeve last, but must put it in first; or I can't reach back to the night stand beside my bed." Inability to medially rotate and to adduct the arm at the glenohumeral joint simultaneously is a revealing sign of infraspinatus TrP activity. Patients are likely to complain that attempting to comb the hair or brush the teeth is painful. Tennis players complain that this shoulder pain limits the vigor of their strokes.

Sola and Williams[42] identified the symptoms of shoulder-girdle fatigue, weakness of grip, loss of mobility at the shoulder, and hyperhidrosis in the referred pain area as due to TrP activity in the infraspinatus muscle.

Referred pain (Fig. 22.1) prevents the patient from lying on the painful side (and sometimes on the back) at night, because the weight of the thorax compresses and stimulates the infraspinatus TrPs.[47] When the patient lies on the pain-free side for relief, the uppermost arm is likely to fall forward and painfully stretch the affected infraspinatus muscle, again disturbing sleep. Thus, patients with very active infraspinatus TrPs may find that they can sleep only by propping themselves up, seated in a chair or on a sofa for the night.

A major part of the shoulder-girdle pain associated with hemiplegia is commonly due to myofascial TrPs in the trapezius, scalene, supraspinatus, infraspinatus, and subscapularis muscles. In the absence of spasticity at rest, the TrPs in these muscles usually respond well to local treatment.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Infraspinatus TrPs are usually activated by an acute stress or by multiple overload stresses, such as the stress of frequently reaching out and back to a bedside stand (especially during an acute illness when muscles may be "below par"), grabbing behind for support to regain balance (e.g., grasping the railing when slipping on stairs), twisting the arm that holds a ski pole during a fall, excessive poling when skiing, delivering an especially hard tennis serve when off balance, or an experienced ice skater dragging a novice skater around by the arm for a long period of time. The onset of shoulder pain is usually within a few hours of the initiating trauma. The patient generally can identify exactly what happened and when the muscle was overloaded.

Since the infraspinatus muscle, unlike the supraspinatus, is likely to be strongly activated in movements that are unusual and transient, it is much more likely to develop TrPs as the result of an acute overload than from tasks that impose a sustained overload.

Baker[2] found that between 20% and 30% of the infraspinatus muscles of patients experiencing their first motor vehicle accident had active TrPs following the accident regardless of the direction from which the impact came. This was slightly fewer than the number of TrPs that developed in the supraspinatus muscles of these patients.

8. PATIENT EXAMINATION (Fig. 22.3)

The Mouth Wrap-around Test is a useful screening test for TrP restriction of shoulder-girdle muscles and is illustrated and described in Chapter 18 (see Fig. 18.2). The Hand-to- shoulder Blade Test (Fig. 22.3) is more specific for identifying TrP restriction of the infraspinatus muscle.

The Hand-to-shoulder-blade Test requires full adduction and medial rotation

of the arm at the glenohumeral joint. The patient does this test by placing the hand behind the back and reaching as far up toward the opposite scapula as possible. Normally, the fingertips should reach at least to the spine of the scapula, farther than is shown in Figure 22.3. This test stretches the abductors and lateral rotators. When the range of these muscles (e.g., the infraspinatus) is stretch-limited because of TrP tautness and shortening of the fibers, the fingers may barely reach to the hip pocket. This limitation is similar when the movement is performed actively or passively. On the other hand, TrPs in the antagonist, the subscapularis muscle, may allow the fingers to reach the spinal column, or farther, if done passively without contracting the subscapularis muscle in the shortened position.

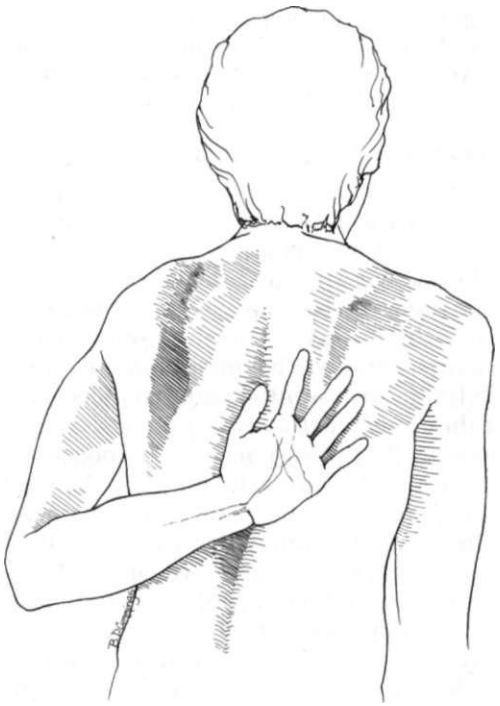


Figure 22.3. Hand-to-shoulder Blade Test, which requires adduction and medial rotation of the arm at the shoulder joint. This active movement places the infraspinatus muscle on stretch and contracts the subscapularis and latissimus dorsi muscles in the shortened position. This position of the hand would usually indicate some restriction of movement. In normal individuals, the fingertips ordinarily reach the spine of the scapula. However, this individual had short upper arms that limited this movement.

Palpation of the infraspinatus often reveals very painful TrPs even when its manual muscle test against resistance is painless.[31]

When there is restriction in range of motion of the shoulder complex and/or the elbow complex, these joints should all be examined for joint play.[32]

9. TRIGGER POINT EXAMINATION

The infraspinatus muscle frequently harbors myofascial TrPs. In 126 patients, referred pain to the shoulder region arose from the infraspinatus muscle in 31% of the cases, a frequency second only to that of the levator scapulae (55%).[40] Pace[33] made a similar observation. Among young, pain-free adults, the infraspinatus was third (18%) in the prevalence of latent TrPs, fewer than the levator scapulae (20%) and the upper trapezius (35%).[41]

The muscle may be examined for TrPs with the patient sitting, or lying on the pain-free side as for a TrP injection. When the patient is seated, slight tension is placed on the muscle by bringing the hand and arm across the front of the chest to grasp the far armrest of the chair. Flat palpation frequently discloses multiple spot tenderness in this muscle as indicated by the multiple Xs in Figure 22.1A. The most common TrP region is usually caudal to the junction of the most medial and adjacent quarter of the length of the scapular spine (upper medial X in Fig. 22.1A).

The next most common TrP (lateral upper X in Fig. 22.1A) is usually located caudal to the midpoint of the scapular spine, but may be as far lateral as the lateral border of the scapula. It, too, is found by flat palpation. Lange[26] illustrated the location of this infraspinatus trigger area.

A trigger area of spot tenderness that can refer pain like a TrP may be found along the vertebral border of the scapula (Fig. 22.1B). This same location was described previously[42,52] and most likely is a region of enthesopathy at the musculotendinous junction.

Firm bands in this superficial muscle may be more difficult to identify than one might expect. Local twitch responses

(LTRs) are moderately difficult to elicit by snapping palpation. The overlying skin is often thick and indurated by associated panniculosis. Referred pain can usually be evoked or aggravated by sustained pressure on an active infraspinatus TrP.

The reliability with which the physical features of TrPs could be determined was evaluated by four experienced physicians who, following a 3-hour training session immediately before the study, examined five pairs of muscles for five physical characteristics of TrPs in each of 10 subjects.[14] The muscles examined were the infraspinatus, latissimus dorsi, upper trapezius, extensor digitorum, and sternocleidomastoid. Agreement among examiners for TrP characteristics of the infraspinatus muscle was good for the detection of spot tenderness, a taut band, presence of referred pain, and reproduction of the subject's symptomatic pain. However, agreement on the presence or absence of an LTR was slight, and was poorer for the infraspinatus than for the other muscles tested. The LTR is difficult to elicit manually, particularly in the infraspinatus muscle. However, when it is observed, it is a strongly confirmatory finding, and it is especially valuable when needling TrPs therapeutically. Learning the skill necessary to palpate taut bands is an essential first step to the reliable recognition of TrPs by palpation.

10. ENTRAPMENT

No nerve entrapments are attributed to TrPs in this muscle.

11. DIFFERENTIAL DIAGNOSIS

Both *suprascapular nerve entrapment* and TrPs in the infraspinatus muscle can cause shoulder pain. However, prolonged nerve conduction latency, and/or muscular atrophy of the infraspinatus muscle indicate entrapment of the suprascapular nerve at the spinoglenoid notch where it passes from the supraspinatus to the infraspinatus muscle.[15,20] Entrapment of the nerve at the suprascapular notch involves both spinatus muscles. Additional confirmation of a surgical abnormality by magnetic resonance imaging or by ultrasound reinforces the previous findings.[44]

If a patient arrives with the diagnosis of *bicipital tendinitis* and has been treated unsuccessfully by several health care providers, it is likely that there are unidentified TrP sources in the infraspinatus or biceps brachii muscles which are responsible for the anterior shoulder pain.

The *scapulohumeral syndrome,* as defined by Long,[30] may be due to active infraspinatus TrPs. This syndrome also might include pain referred from TrPs in the pectoralis major and minor muscles and from the long head of the biceps brachii.

Infraspinatus TrPs refer pain in the distributions of the C_5, C_6, and C_7 spinal nerves, which may cause diagnostic confusion with *radiculopathy due to intervertebral disc disease*[33] unless neurological deficits and electromyographic findings are considered in addition to the distribution of pain.

Equally confusing is the fact that referred pain from TrPs in the infraspinatus muscle closely mimics that arising from *arthritis of the glenohumeral joint* itself.[38]

Associated Trigger Points

The *teres minor* lies in parallel with the infraspinatus and is its chief synergist. In addition, three families of muscles develop active TrPs in association with the infraspinatus, but any given patient usually exhibits involvement of only one of the three groups. The *anterior deltoid* muscle lies in the essential pain reference zone of the infraspinatus, and it often develops satellite TrPs in response to prolonged activation of the infraspinatus TrPs. Another family is the synergistic *supraspinatus-infraspinatus team,* which can be thought of as two traces of wagon wheels that raise the arm up and back, so that dual involvement is expected. The *biceps brachii* also may join this family. The third group includes the teres *major and latissimus dorsi,* which counter lateral rotation by the infraspinatus.

The antagonistic subscapularis and pectoralis major muscles also should be checked for associated TrPs.

Since the infraspinatus is one of the rotator cuff muscles, differential diagnosis should rule out rotator cuff lesions *(see* Chapter 21). With rotator cuff problems the

pain is severe and is usually exhibited through a limited *arc* of motion.

12. TRIGGER POINT RELEASE (Fig. 22.4)

If there is any suspicion of rotator cuff damage, the infraspinatus should not be stretched. Treatment can include nonstretching methods such as trigger point pressure release, deep massage to the taut hand, gentle hold-relax (no range of movement involved), indirect techniques, and/or injection. Application of vapocoolant or icing can precede any of these techniques.

When joint play is restricted in the shoulder complex (including the acromioclavicular and sternoclavicular articulations), as well as in the elbow articulations, it should be restored.

When muscle lengthening is needed and additional tension is not contraindicated, spray and stretch can be used. Three stretch positions may be employed effectively. First, the position of the Hand-to-shoulder Blade Test may be used with the patient seated (Fig. 22.4A). Second, with the patient in a relaxed position, the involved arm is placed across the front of the chest in full horizontal adduction (Fig. 22.4B). Third, the hand of the involved upper limb may be placed behind the ipsilateral pelvis with the patient lying on the pain-free side (Fig. 22.4C). Vapocoolant (Fig. 22.4A and C) or icing (Fig. 22.4B) is applied in slow parallel sweeps that follow the muscle fibers in a medial to lateral direction, cover the shoulder pain pattern, and continue down the arm to the fingertips and over the thumb. Finally, vapocoolant or icing sweeps are directed upward over the posterior cervical pain reference zone. *See the legend in Figure 22.4 for details of infraspinatus lengthening in each position.* Before finishing the treatment, stretch and spray are applied to the antagonistic anterior deltoid and pectoralis major muscles. This is important because these muscles can experience immediate or delayed shortening activation of latent trigger points (TrPs) in response to the unaccustomed shortening associated with the release of infraspinatus tension. The treatment ends with active range of motion of the involved muscles, followed by application of moist heat.

If TrP tenderness and LTRs remain in the infraspinatus muscle, trigger point pressure release or deep massage is usually helpful. Another effective technique is the application of postisometric relaxation of this medial rotator (progressive contract-relax) with respiratory augmentation while the patient is lying supine with the arm abducted and placed so that the elbow extends over the edge of the table and is flexed 90°. See Chapter 3 for more on these techniques. Since gravity alone tends to medially rotate the arm at the shoulder joint, this position can also be used for patient self-treatment.[27,29]

13. TRIGGER POINT INJECTION (Fig. 22.5)

While the patient lies on the pain-free side, the arm is abducted and flexed to about 45° and the elbow rests on a pillow that has been placed against the chest (Fig. 22.5). The trigger point (TrP) is located and pinned between the fingers against the scapula. The TrP is probed with a 3.8-cm (1.5-in) needle until the needle elicits an LTR, a local pain response, and usually the referred pain pattern of the TrP. As the procaine solution is injected, the clinician explores the area with the needle to reach any remaining TrPs. Rachlin[37] illustrates a similar injection technique in the midmuscle region. Instead of this injection method, the clinician may use dry needling with an acupuncture needle and rapidly pepper the region of the TrP until no more LTRs can be elicited;[16] this technique may be just as effective as injecting an anesthetic hut the dry needling results in more postinjection soreness.[17] Hemostasis is applied with the fingers of the palpating hand during and after injection. If residual tenderness and LTRs are still present, the remaining TrPs are localized by palpation and probed with the needle. A full passive stretch is carried out during the application of a few sweeps of vapocoolant spray, and then a moist hot pack is applied while the arm is supported in a comfortable neutral position.

Contrary to an early illustration,[45] the injection of TrPs is never done in a seated

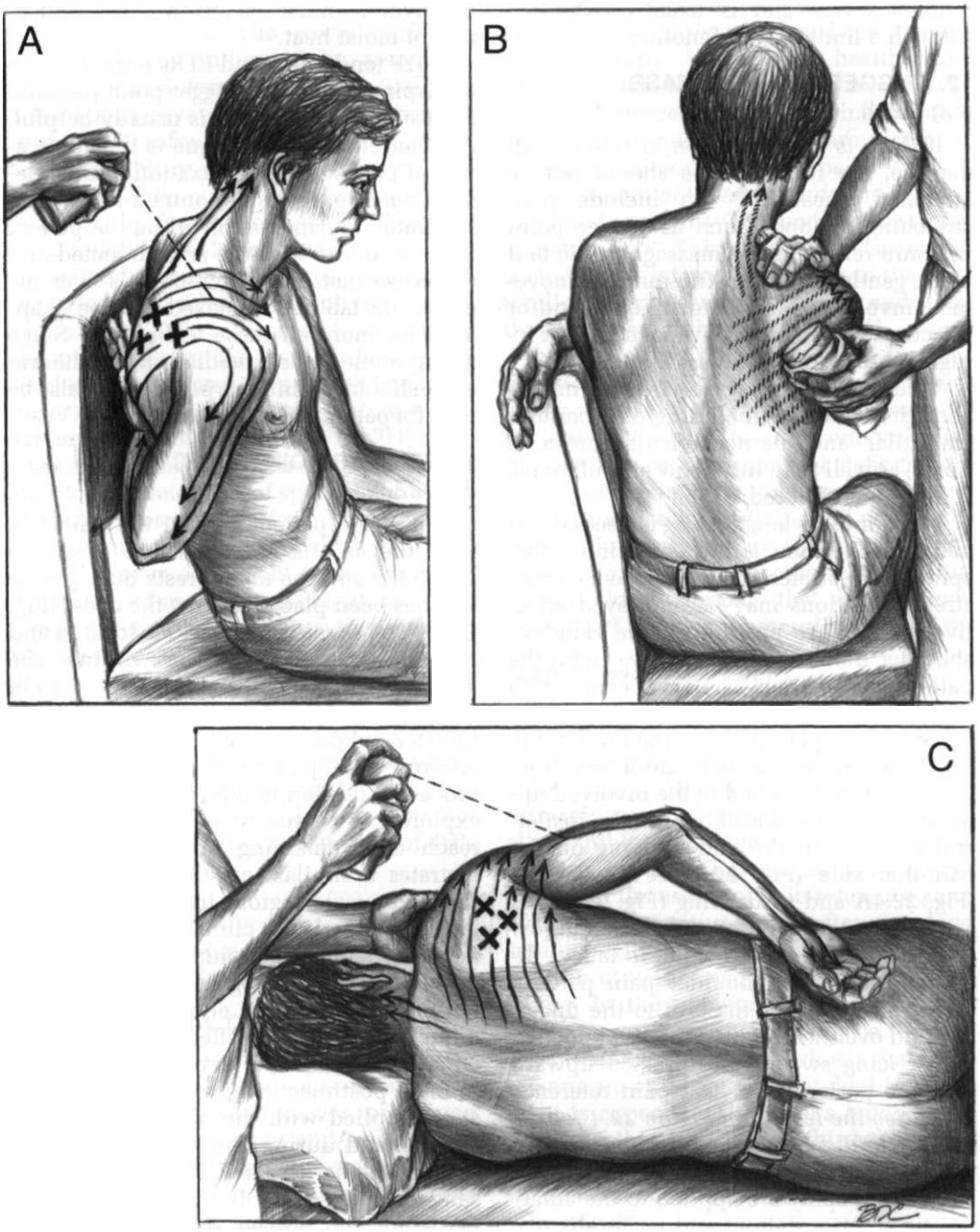


Figure 22.4. Stretch positions and spray patterns *(arrows)* for the infraspinatus muscle. **A,** application of vapocoolant spray, patient in the seated position with the hand behind the thorax. **B,** application of ice in the direction of the patterns for spray as shown in **A,** with the patient seated and the medially rotated arm in *front* of the chest to lengthen the muscle. When the patient tries to reach across the chest while the operator stabilizes the scapula, the active effort reciprocally inhibits the infraspinatus muscle and allows it to lengthen. A small cloth or paper towel can be used to blot the skin dry following application of the ice. **C,** Application of vapocoolant spray, patient lying on the pain-free side with the affected arm medially rotated by resting the back of the hand behind the pelvis. While the operator stabilizes the scapula, the patient takes up any slack that develops in the infraspinatus by letting the humerus and elbow drop forward, effectively moving the humerus toward the front of the chest, and increasing medial rotation of the arm.

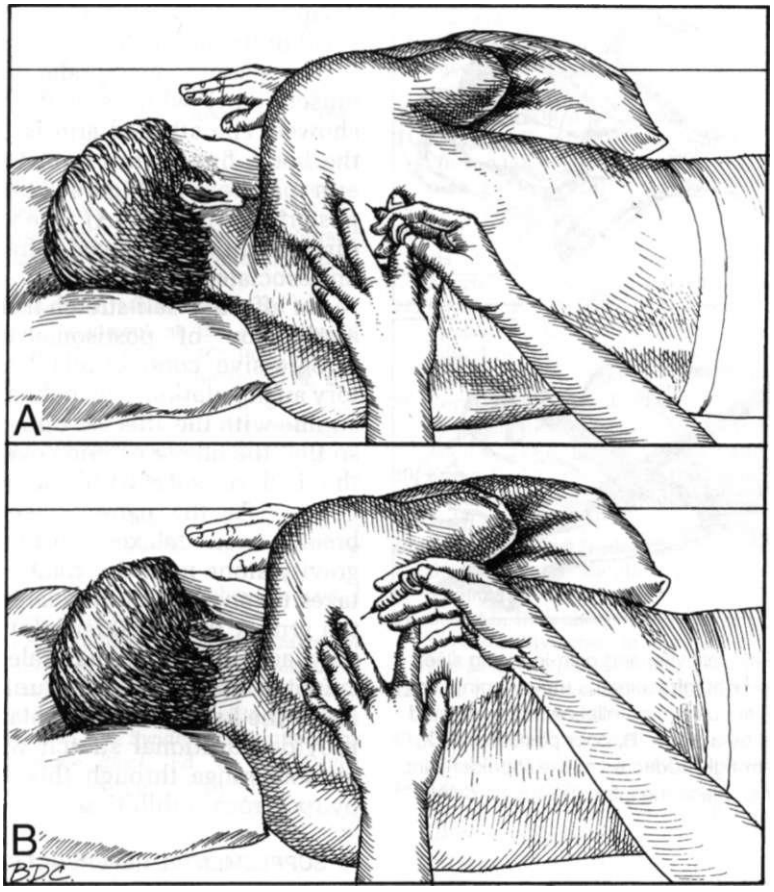


Figure 22.5. Technique for injecting two common trigger point regions in the infraspinatus muscle. **A,** the medial trigger point region. The operator's left middle finger presses against the lower (caudal) border of the scapular spine. **B,** a more lateral trigger point. The left ring finger presses against the lower border of the scapular spine.

patient; it is always performed with the patient in the recumbent position to minimize psychogenic syncope and the possible complications of falling, should the patient faint.

A physician described to Dr. Travell his experience of producing a pneumothorax while injecting an infraspinatus TrP. The needle penetrated the scapula through a fibrous membrane where he expected scapular bone. Portions of the infraspinous fossa can be paper-thin. One must be aware of this possibility, and be sensitive to the resistance encountered by the needle at that depth.

If, following injection therapy, the patient's range of motion in the Hand-to-shoulder Blade Test remains significantly restricted, the clinician should check for TrPs in the supinator muscle of the forearm, since this test can be restricted when the forearm does not pronate fully.

14. CORRECTIVE ACTIONS
(Fig. 22.6)

The patient should avoid habitual sustained or repetitive motions that overload the infraspinatus muscle, such as regularly rolling the hair up on night curlers, and reaching backward to objects on a bedside table. The table should be moved toward the foot of the bed, or the unaffected arm should be used to reach across. On retiring to bed, application of a hot pack to the

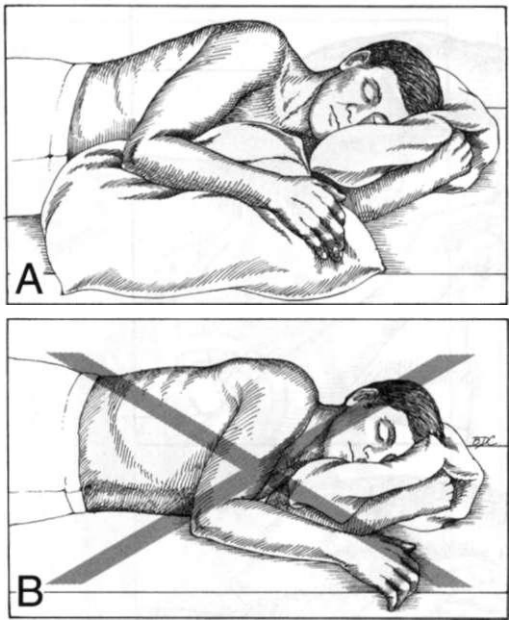


Figure 22.6. Pain-relieving and pain-inducing sleep positions when right infraspinatus trigger points are active. **A,** neutral position of relief, with the affected arm supported by a pillow. **B,** poor position (*red* **X)** with the arm strongly adducted at the shoulder joint. This position can place the infraspinatus on painful stretch.

muscle for 15-20 minutes can markedly reduce the irritability of its TrPs. A heating pad also can be used on the low setting. Precautions must be taken not to fall asleep with the heating pad on a high setting as severe burns can result.

When the patient lies on the uninvolved side, sleep is improved by supporting the uppermost elbow and forearm (painful limb) on a bed pillow (Fig. 22.6A) to avoid overstretching the affected infraspinatus muscle that can cause referred pain (Fig. 22.6B); a neutral position is best.

The patient may "press out" and inactivate an infraspinatus TrP with the application of TrP compression by lying on a tennis ball placed directly under a tender spot in the muscle; body weight is used to maintain increasing pressure for 1 or 2 minutes, as described and illustrated in Chapter 18, Figure 18.4. The tennis ball pressure treatment may be repeated daily or every second day, until TrP tenderness disappears. A Theracane® can be used instead of the tennis ball.

The patient may prefer to stretch the muscle daily while seated under a warm shower. The affected arm is pulled across the body, first in the front to achieve the arm position in Figure 22.4B, and then posteriorly (Fig. 22.4A). The warm water is directed on the involved infraspinatus and on associated muscles.

An effective self-stretch technique is the application of postisometric relaxation (progressive contract-relax) with respiratory augmentation. The patient learns to lie supine with the affected upper limb placed so that the elbow extends over the edge of the bed or sofa with the elbow flexed 90°.[27-29] As the patient takes slow deep breaths and relaxes during exhalation, gravity alone medially rotates the arm and takes up any slack in the lateral rotators of the arm at the shoulder joint. The patient may achieve additional release of infraspinatus tightness by voluntary effort to lower the hand (medially rotate the arm) to provide additional stretch within a comfortable range through this augmentation by reciprocal inhibition.

SUPPLEMENTAL REFERENCES, CASE REPORTS

Dr. Travell presented case reports illustrating the management of patients with infraspinatus TrPs.[45,50]

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991(pp. 386, 387 Figs. 6-40, 6-41).
2. Baker BA: The muscle trigger: evidence of overload injury. *J Neurol Orthop Med Surg* 7:35-44, 1986.
3. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 270. 273-276).
4. Bonica JJ, Sola AE: Other painful disorders of the upper limb. Chapter 52. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, et al. Lea & Febiger, 1990, pp. 947-958 (see p. 949).
5. Cailliet R: *Soft Tissue Pain and Disability.* F.A. Davis, Philadelphia, 1977 (pp. 149-152).
6. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 523, 524, Figs. 6-46).
7. *Ibid.* (p. 1209)
8. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 23).

9. *Ibid.* (Fig. 24).
10. *Ibid.* (Fig. 33).
11. *Ibid.* (Figs. 523, 524).
12. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 64).
13. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 31-35).
14. Gerwin RD, Shannon S, Hong CZ, *et al:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
15. Henlin JL, Rousselot JP, Monnier G, *et al:* [Suprascapular nerve entrapment at the spinoglenoid notch]. *Rev Neurol (Paris)* 348(5):362-367, 1992.
16. Hong CZ: Myofascial trigger point injection. *Crit Rev Phys Med Rehabil* 5:203-217, 1993.
17. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Rehabil* 73:256-263, 1994.
18. Inman VT, Saunders JB, Abbott LC: Observations on the function of the shoulder joint. *J Bone Joint Surg* 26:1-30, 1944 (Fig. 25, p. 23).
19. Ito N: Electromyographic study of shoulder joint. *J Jpn Orthop Assoc* 54:1529-1540, 1980.
20. Jerosch J, Hille E, Schulitz KP: [Selective paralysis of the infraspinatus muscle, caused by compression of the infraspinatus branch of the supraspinatus muscle (sic)]. *Sportverletz Sportschaden* 3(4):231-233,1987.
21. Judovich B, Bates W: *Pain Syndromes: Treatment by Paravertebral Nerve Block.* Ed. 3. F.A. Davis, Philadelphia, 1949 (Fig. 6, pp. 127, 128).
22. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3:175-190,1938 (pp. 179,184, Fig. 7).
23. Kelly M: The nature of fibrositis 1. The myalgic lesion and its secondary effects: a reflex theory. Ann *Rheum Dis* 5:1-7, 1945.
24. Kelly M: Some rules for the employment of local analgesia in the treatment of somatic pain. *Med J Aust* 3:235-239, 1947 (Table 1).
25. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 3. Williams & Wilkins, Baltimore, 1993 (p. 281).
26. Lange M: *Die Muskelharten (Myogelosen).* J.F. Lehmanns, Miinchen, 1931 (Fig. 40B, p. 129).
27. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 204, 205).
28. Lewit K: Role of manipulation in spinal rehabilitation. Chapter 11. In: *Rehabilitation of the Spine: A Practitioner's Guide.* Edited by Liebenson C. Williams & Wilkins, Baltimore, 1996 (p. 208).
29. Liebenson C: Manual resistance techniques and self-stretches for improving flexibility/mobility. Chapter 13. In: *Rehabilitation of the Spine: A Practitioner's Guide,* Edited by Liebenson C. Williams & Wilkins, Baltimore, 1996, pp. 253-292 (see pp. 282-283).
30. Long C, II: Myofascial Pain Syndromes: Part II—Syndromes of the head, neck and shoulder girdle. *Henry Ford Hosp Med Bull* 4:22-28, 1956 (p. 26).
31. Maigne R: *Diagnosis and Treatment of Pain of Vertebral Origin: A Manual Medicine Approach.* Williams & Wilkins, Baltimore, 1996 (p. 371).
32. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
33. Pace JB: Commonly overlooked pain syndromes responsive to simple therapy. *Postgrad Med* 58:107-113, 1975 (Fig. 3, p. 110).
34. Pernkopf E: Arias *of Topographical and Applied Human Anatomy,* Vol 2. W.B. Saunders, Philadelphia, 1964 (Fig. 28).
35. *Ibid.* (Figs. 44, 60).
36. Pink M, Jobe FW, Perry J: Electromyographic analysis of the shoulder during the golf swing. *Am J Sports Med* 38(2):137-140, 1990.
37. Rachlin ES: Injection of specific trigger points, Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994:197-360 (see pp. 322-325).
38. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114,1981 (Tables 1 and 2).
39. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Rehabil* 62:107-110, 1981.
40. Sola AE, Kuitert JH: Myofascial trigger point pain in the neck and shoulder girdle. *Northwest Med* 54:980-984, 1955.
41. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med* 34:585-590, 1955.
42. Sola AE, Williams RL: Myofascial pain syndromes. *J Neurol* 6:91-95, 1956 (pp. 93, 94, Fig. 2).
43. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 323).
44. Takagishi K, Maeda K, Ikeda T, *et al:* Ganglion causing paralysis of the suprascapular nerve. Diagnosis by MRI and ultrasonography. Acfa *Orthop Scand* 62(4):391-393, 1991.
45. Travell J: Basis for the multiple uses of local block of somatic trigger areas (procaine infiltration and ethyl chloride spray). *Miss Valley Med J 71:13-22,* 1949 (Figs. 2 and 3, Case 3, pp. 17 and 18).
46. Travell J: Ethyl chloride spray for painful muscle spasm. *Arch Phys Med Rehabil* 33:291- 298, 1952 (p. 293).
47. Travell J: Pain mechanisms in connective tissue. In: *Connective Tissues, Transactions of the Second Conference, 1951.* Edited by C. Ragan. Josiah Macy, Jr. Foundation, New York, 1952 (pp. 90, 91, 93).
48. Travell J, Berry C, Bigelow N: Effects of referred somatic pain on structures in the reference zone. *Fed Proc* 3:49, 1944.
49. Travell J, Rinzler S, Herman M: Pain and disability of the shoulder and arm: treatment by intramuscular infiltration with procaine hydrochloride. *JAMA* 320:417-422, 1942 (Fig. 2B).
50. Travell J, Rinzler SH: Pain syndromes of the chest muscles: Resemblance to effort angina and myocardial infarction, and relief by local block. *Can Med Assoc J* 59:333-338, 1948 (Fig. 1, Cases 1 and 3).
51. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 33:425-434, 1952.
52. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J Am Osteopath Assoc* 72:697-710, 1973 (Fig. 28).
53. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little, Brown & Company, Boston, 1988 (Fig. 12-2, p. 211).

CHAPTER 23
Teres Minor Muscle

HIGHLIGHTS: The teres minor functions as a "little brother" to the infraspinatus muscle. **REFERRED PAIN** from trigger points (TrPs) in the teres minor is often encountered as residual pain following inactivation of TrPs in the infraspinatus muscle. The pain focuses on an area localized near the region of the muscle's musculotendinous attachment. Referred dysesthesia of the fourth and fifth fingers may occur. **ANATOMY:** this muscle attaches immediately adjacent to, and just below, the attachments of the infraspinatus muscle. **INNERVATION** of the teres minor is through the axillary nerve, whereas that of the infraspinatus is through the suprascapular nerve. **FUNCTION** of this muscle is nearly identical to that of the infraspinatus: it assists in stabilization of the head of the humerus in the glenoid cavity during movement of the arm. It also acts to laterally rotate the arm at the shoulder joint. **SYMPTOMS** include chiefly posterior shoulder pain and may include dysesthesia of the fourth and fifth fingers. Reaching up and back may produce or aggravate symptoms. **ACTIVATION** of TrPs results from overloading the muscle while reaching up or while reaching out and behind the shoulder. **PATIENT EXAMINATION** reveals slight restriction of medial rotation at the glenohumeral joint on performance of the Hand-to-shoulder Blade Test. **DIFFERENTIAL DIAGNOSIS** includes the quadrilateral space syndrome, rotator cuff lesions, ulnar neuropathy, C_8 radiculopathy, and infraspinatus TrPs. **TRIGGER POINT RELEASE** using spray and stretch is performed by having the patient lie on the side opposite the involved muscle and bringing the involved arm over and behind the head. Meanwhile, the operator applies vapocoolant spray or icing in an upward direction over the muscle and its referred pain pattern. **TRIGGER POINT INJECTION** of this muscle involves precise localization of the TrP between the fingers. Injection is followed by active range of motion of the muscle. **CORRECTIVE ACTIONS** include elimination of mechanical stress on the muscle, attention to the sleeping position in bed, self-administration of TrP compression, and self-stretch exercises.

1. REFERRED PAIN
(Fig. 23.1)

A patient with active teres minor trigger points (TrPs) complains of a "painful bursa" about the size of a prune that seems to be deep in the posterior deltoid muscle close to the attachment of the teres minor on the humerus (Fig. 23.1). This concentrated area of pain lies proximal to the deltoid's attachment at the deltoid tubercle of the humerus. The spot of pain appears well below the subacromial bursa, but feels like "bursitis" to the patient because of its sharp localization and deep quality. If the patient complains of a broadly distributed aching pain in the arm and shoulder posteriorly, it is rarely due to TrPs in the teres minor alone. Bonica and Sola illustrate a broader distribution of pain in the region of the posterior deltoid muscle.[4]

One report of 4 patients[11] indicates that referred dysesthesia of tingling and numbness to the fourth and fifth fingers may be as common as pain referred to the shoulder by active TrPs in the teres minor muscle.

2. ANATOMY
(Fig. 23.2)

The teres minor muscle attaches ***medially*** to the upper two-thirds of the dorsal surface of the scapula near its axillary border and to the aponeuroses which separate this muscle from the infraspinatus and teres major muscles. It attaches ***laterally*** to the

lowermost impression (facet) on the greater tubercle of the humerus (Fig. 23.2).[5] The tendon is closely applied to the posterior part of the capsule of the shoulder joint.[13]

Supplemental References

Other authors have clearly illustrated the teres minor muscle as seen from behind,[1,5,6,81,7,22] from the side,[7] in cross section,[10,18] and in sagittal section.[2]

3. INNERVATION

The teres minor muscle is innervated by the axillary nerve through the posterior cord from the C_5 and C_6 spinal segments. This innervation differs from that of the infraspinatus muscle above (supplied by the suprascapular nerve), and from that of the teres major below (supplied by the lower subscapular nerve). All three muscles are supplied at least in part from cervical spinal segments C_5 and C_6.

4. FUNCTION

This muscle is a member of the rotator cuff team (*see* Fig. 21.2A). Many of the sources reviewed[3,9,13,14] equated the actions of the teres minor and the infraspinatus. Both muscles laterally rotate the arm at the glenohumeral joint regardless of whether the arm is abducted, flexed, or extended,[9] and help to stabilize the head of the humerus in the glenoid cavity during movement of the arm (*see* Chapter 22, Section 4). Supporting this concept, a study of these two muscles showed remarkably similar, almost linearly, increasing electrical activity as the arm was abducted at the shoulder joint and during flexion; the activity reached a peak at about 120° of flex-

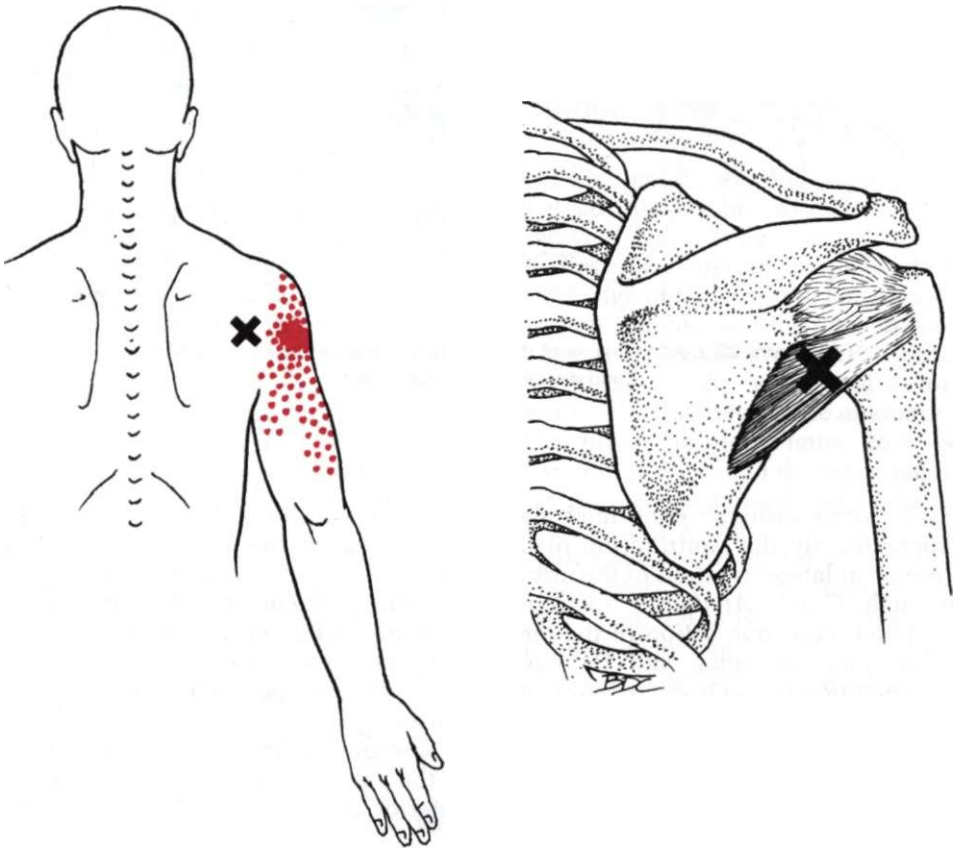


Figure 23.1. Referred pain pattern (essential zone *solid red,* spillover zone *stippled red)* of a trigger point **(X)** in the right teres minor muscle. It is common also to find TrPs slightly medial to the location of the **X** as described under **Injection.**

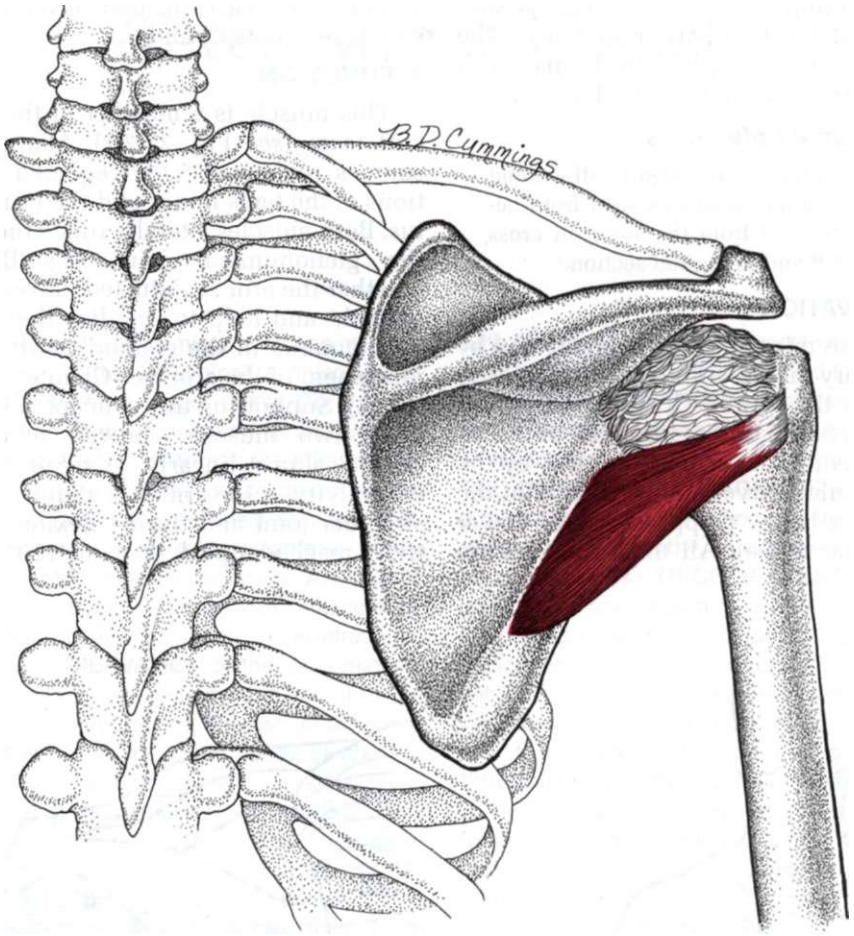


Figure 23.2. Attachments of the teres minor muscle showing location and direction of muscle fibers.

ion.[12] The same authors[12] confirmed electromyographically the contribution of the teres minor to lateral rotation of the arm.

Although *Gray's Anatomy*[5] identified weak adduction as one action of the teres minor muscle, Basmajian and De Luca[3] make no mention of electromyographic evidence, and Duchenne[9] no functional evidence, that adduction is a function of this muscle.

5. FUNCTIONAL UNIT

The teres minor muscle functions in parallel with the infraspinatus, to which it is a "little brother," having similar attachments but a different nerve supply. These muscles assist the other rotator cuff muscles, the supraspinatus and subscapularis, to stabilize the head of the humerus in the glenoid cavity during movements of the arm. The teres minor is also synergistic with the posterior fibers of the deltoid muscle.

The teres minor may act as an antagonist to the subscapularis, pectoralis major, and anterior deltoid muscles.

6. SYMPTOMS

Patients complain more of the posterior shoulder pain (Fig. 23.1) than of restricted

motion. When the patient presents with pain deep in the *anterior* shoulder, the symptom is likely to be due to active TrPs in the infraspinatus rather than the teres minor. After treatment, with relief of the anterior shoulder pain and restoration of the normal length of the infraspinatus, the patient then becomes aware of the pain that is referred to the back of the shoulder by the teres minor. Infraspinatus-referred pain apparently dominates, and release of the infraspinatus tension uncovers the pain pattern of the next-tightest line of parallel muscle fibers, the teres minor.

Escobar and Ballesteros[11] reported 4 patients with isolated active teres minor TrPs. All complained primarily of numbness and/or tingling of the fourth and fifth fingers aggravated by shoulder activity that required reaching above shoulder height or behind them. These movements also caused some pain in three of the patients.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

The teres minor muscle is usually not involved as a single-muscle syndrome. Its TrPs are activated by much the same overload stresses-reaching up or reaching out and behind the shoulder-that activate TrPs in the infraspinatus muscle (see Chapter 22). Patients have been known to activate acute teres minor TrPs in the following ways: as the result of a motor vehicle accident (particularly when holding on to something such as the steering wheel), by loss of balance while lifting a heavy object overhead, while working in cramped quarters with the arm reaching overhead, and while playing volleyball.[11]

Teres minor TrPs are perpetuated by continued overloading of the muscle when reaching up and back, and by systemic perpetuating factors (*see* Chapter 4).

8. PATIENT EXAMINATION

The teres minor is one of the less commonly involved muscles. About 7% of patients with myofascial pain complaints in the shoulder region were found to have TrPs in the teres minor.[20] Only 3% of healthy young adults had what we would identify as latent TrPs in the teres minor or teres major muscle.[21]

The shoulder joint complex should be examined for restricted range of motion. Where restricted range is found, the joints should be examined for restriction of accessory movements, or joint play.[16] Joints to be examined should include the glenohumeral, acromioclavicular, and sternoclavicular (the latter two especially following a motor vehicle accident). Movement of the scapula on the chest wall should also be examined for restriction.

Usually, the patient with obvious active TrPs in the teres minor muscle shows some restricted range of motion in the Hand-to-shoulder Blade Test (*see* Fig. 22.3) even after TrPs in the infraspinatus muscle have been inactivated by treatment. The Mouth Wrap-around Test (*see* Fig. 18.2) also may be restricted. The pain has shifted from the front of the shoulder (in the case of infraspinatus TrPs) to the back of the shoulder (pain distribution of teres minor TrPs), and palpation reveals evidence of TrP activity in the teres minor muscle.

9. TRIGGER POINT EXAMINATION (Fig. 23.3)

The patient lies on the side opposite the involved upper limb, with the uppermost (involved) arm resting on a pillow against the chest. The operator palpates along the lateral edge of the scapula, between the infraspinatus above and the teres major muscle below, to locate active TrPs in the parallel fibers of the teres minor muscle. Figure 23.3 illustrates these anatomical relationships; see also Figure 25.3, which shows the palpation of the teres major. The teres minor lies immediately superior to the teres major, but traverses posteriorly and attaches directly to the greater tubercle on the posterior side of the humerus, rather than joining the latissimus dorsi to attach on the front of the humerus, as the teres major does (Fig. 23.3). The long head of the triceps brachii muscle passes between them, and these muscles form three sides of the quadrangular space (Fig. 23.3).[1]

The teres minor can be identified by palpating the suspected muscle while the patient alternately attempts lateral and medial rotation of the arm against minimal resistance. It contracts during lateral rotation and relaxes during medial rotation.

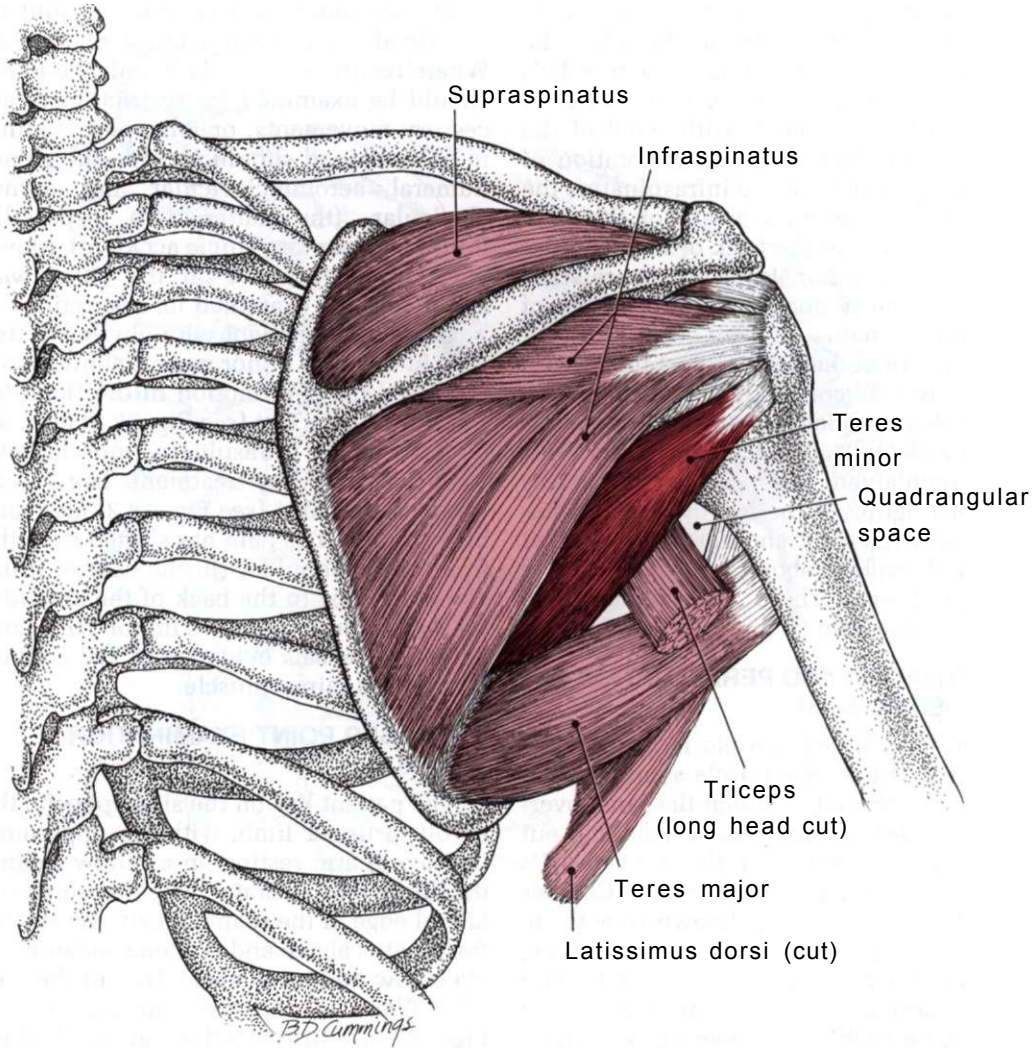


Figure 23.3. Anatomical relations of the teres minor *(dark red)* to other dorsal scapular muscles *(light red),* The lateral border of the scapula is usually palpable as an orienting landmark and can be located in the space between the teres minor and the teres major muscles when using pincer palpation. The long head of the triceps also passes through that space and, with the teres minor and teres major muscles and the humerus, helps to define the quadrangular (quadrilateral) space.

10. ENTRAPMENT

No nerve entrapments are attributed to TrP tension in this muscle.

11. DIFFERENTIAL DIAGNOSIS

Since the teres minor is one of the rotator cuff muscles, rotator cuff lesions should be ruled out (*see* Chapter 21). With teres minor TrPs, the patient usually does not describe a small *arc* of severe pain; rather, the pain is throughout the movement or at the end of the range of movement.

The **quadrilateral space syndrome** is characterized by shoulder pain and selective atrophy of the teres minor muscle due to compression of the axillary nerve by fibrous bands as the nerve passes through the quadrilateral space. This was demonstrated on three patients by magnetic resonance imaging.[15]

As the four case reports of Escobar and Ballesteros[11] so eloquently demonstrated, dysesthesia in the fourth and fifth fingers that is caused by active teres minor TrPs

can easily be mistaken for an **ulnar neuropathy** or **C_8 radiculopathy.** The neuropathy and radiculopathy can be ruled out by appropriate electrodiagnostic evaluation.

Because of the location of the pain and tenderness referred by these TrPs, one must not assume that these symptoms are caused by **subdeltoid bursitis,** but must also examine the teres minor for TrPs that can be causing the symptoms.

In cases of posttraumatic shoulder pain (particularly following an automobile accident in which the patient was holding on to the steering wheel or reaching the arm out for protection), one should rule out **acromioclavicular separation.**

The infraspinatus is the primary synergist of the teres minor and, in our experience, almost always becomes involved when there are TrPs in the teres minor. Additional muscles likely to be involved are those presented in Chapter 22, Section 11.

12. TRIGGER POINT RELEASE
(Fig. 23.4)

Scapular mobility and joint play[16] should be restored if indicated.

If a rotator cuff tear is suspected, nonstretch treatment techniques should be employed (*see* Chapter 21, Section 12). Also see Chapter 21, section 11 for a discussion of rotator cuff lesions.

For spray and release, when muscle lengthening is not contraindicated, the patient lies on the side opposite the involved muscle. The operator first applies a few sweeps of spray over the muscle while flexing the arm to bring it overhead, and far enough behind the head to take up the slack. With successive applications of spray, the operator gradually releases the muscle by allowing the arm to drop down *behind* the head and toward the floor (Fig. 23.4). Parallel sweeps of the vapocoolant spray are applied along the line of the muscle fibers and over the pain reference zone. The treatment is followed by hot packs over the muscle. Release is facilitated by postisometric relaxation and/or reciprocal inhibition (*see* Chapter 3). See Figure 22.4A and B for other stretch positions that can be utilized to lengthen the teres minor.

Instead of, or in addition to, this treatment, a member of the patient's family can be taught to apply gentle trigger point pressure release. The patient can apply self-treatment by lying on a tennis ball and applying gentle pressure to the trigger point (TrP) while taking up the slack that develops in the relaxed muscle during exhalation.

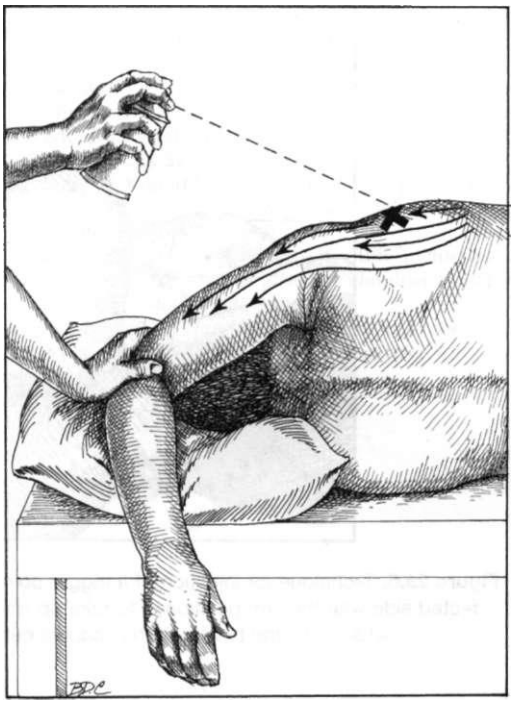


Figure 23.4. Stretch position and spray pattern *(arrows)* for a trigger point (X) in the teres minor muscle. The arm is slowly brought upward and then behind the head to take up any slack as it develops in the muscle. See Figure **22.4A** and **B** for other stretch positions that can be used for lengthening of the teres minor.

13. TRIGGER POINT INJECTION
(Fig. 23.5)

The patient lies on the unaffected side, with the involved arm in front, resting on a pillow. Alternatively, the patient may lie prone with the arm medially rotated (palm up) and abducted to approximately 45°, or less, to take up the slack in the muscle. The trigger points (TrPs) in the teres minor usually lie near the surface of the muscle and are located between the teres major and the infraspinatus, near the lateral border of the scapula. For injection, a TrP is fixed between the index and middle fingers

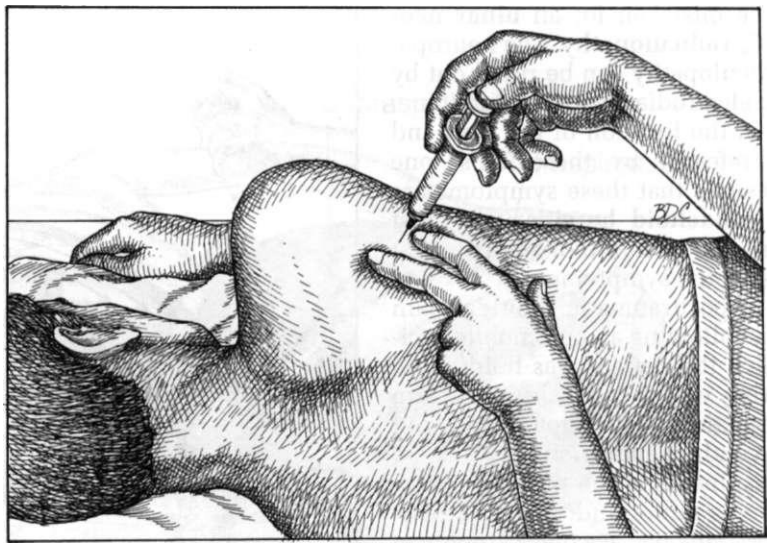


Figure 23.5. Technique for injection of a trigger point in the teres minor muscle. The patient lies on the unaffected side with the arm positioned to take up slack in the muscle. The tip of the operator's index finger marks the lateral border of the scapula between the teres major and teres minor muscles.

(Fig. 23.5), using one of the techniques described for the infraspinatus muscle *(see* Chapter 22). The needle is directed toward the scapula. Following injection, the patient makes the movement of the Hand-to-shoulder Blade Test to stretch the muscle while a few sweeps of vapocoolant are applied over it. A hot pack and active range of motion complete this treatment.

The anatomical relations of this muscle and the usual location of the TrPs for injection are also described and illustrated by Rachlin.[19]

14. CORRECTIVE ACTIONS

Corrective actions for the teres minor are essentially those described in detail in Chapter 22, Section 14. They include avoidance of excessive or repetitive load on the muscle, correct position of the arm to avoid full shortening during sleep, home application of hot packs and of TrP pressure release, and self-stretch exercises.

The patient or a member of the patient's family can be taught to inactivate the TrP by applying digital pressure to it (trigger point pressure release) daily for several days. Or, the patient can apply self-trigger point pressure release (and some deep massage) by lying on a tennis ball and rolling the TrP over it. A Theracane® can be used for this purpose, also. These are similar to the techniques recommended for the infraspinatus muscle.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (pp. 386, 387; Figs. 6-40, 6-41).
2. *Ibid.* (p. 395, Fig. 6-52).
3. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (p. 270).
4. Bonica JJ, Sola AE: Other painful disorders of the upper limb. Chapter 52. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, 1990 (pp. 947-958).
5. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 524, Fig. 6-46).
6. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 23).
7. *Ibid.* (Fig. 61).
8. *Ibid.* (Figs. 523, 524).
9. Duchenne GB: *Physiology of Motion,* Translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 64, 66).
10. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sect. 33).
11. Escobar PL, Ballesteros J: Teres minor: source of symptoms resembling ulnar neuropathy or C_8 radiculopathy. *Am J Phys Med Rehabil 67(3):120-*122, 1988.

12. Inman VT, Saunders JB, Abbott LC: Observations on the function of the shoulder joint. *J Bone Joint Surg* 26:1-30, 1944 (pp. 20, 22, 23, Figs. 26, 29).
13. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 84, 85).
14. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 281).
15. Linker CS, Helms CA, Fritz RC: Quadrilateral space syndrome: findings at MR imaging. *Radiology 188(3)675-676,* 1993.
16. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
17. Pernkopf E: Arias *of Topographical and Applied Human Anatomy.* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Figs. 27, 28, 57).
18. *Ibid.* (Fig. 60).
19. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994:197-360 (pp. 222-225).
20. Sola AE, Kuitert JH: Myofascial trigger point pain in the neck and shoulder girdle. *Northwest Med* 54:980-984, 1955 (p. 983).
21. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med* 34:585-590, 1955.
22. Spalteholz W: *Handatlas der Anatomie des Men-schen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 323).

CHAPTER 24
Latissimus Dorsi Muscle

HIGHLIGHTS: **REFERRED PAIN** from trigger points (TrPs) in the latissimus dorsi is readily misjudged as resulting from enigmatic intrathoracic disease. Pain usually concentrates in the area of the inferior angle of the scapula and may extend to the back of the shoulder and down the medial arm and forearm to the ulnar aspect of the hand, including the ring and little fingers. **ANATOMICAL** attachments to the trunk present a fan shape. The muscle connects, below, to the spinous processes of the lower six thoracic and all the lumbar vertebrae, the sacrum, the posterior crest of the ilium, and the last three or four ribs. Above, the muscle attaches to the intertubercular groove of the humerus jointly with the teres major. **FUNCTION** includes adduction, extension, and medial rotation of the arm at the shoulder joint and forceful depression of the shoulder girdle. **SYMPTOMS** are primarily pain, which is changed little by muscular activity or change of position. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** result from repetitively pulling down with the hands from overhead or pushing down with the hand beside the body. **PATIENT EXAMINATION** reveals minimal restriction of range of motion. **TRIGGER POINT EXAMINATION** requires pincer palpation of the latissimus dorsi in the posterior axillary fold at approximately the midscapular level with the arm in lateral rotation and abducted approximately 90° to take up the slack. **DIFFERENTIAL DIAGNOSIS** includes suprascapular nerve entrapment, C_7 radiculopathy, and bicipital tendinitis, as well as TrPs in the lower trapezius or rhomboid muscles. **TRIGGER POINT RELEASE** of this muscle is often effective, but one must be careful to get full stretch on the muscle and use augmentation techniques. The vapocoolant is applied upward from the pelvis over the entire muscle and continues over the referred pain pattern to the fingers. **TRIGGER POINT INJECTION** in this muscle is performed by grasping the muscle fibers within the posterior axillary fold in a pincer grip to inject them. Following injection, the patient fully flexes and extends the arm slowly three times through a *full* range of motion. **CORRECTIVE ACTIONS** focus on teaching the patient to avoid overloading the muscle and to perform release exercises regularly.

1. REFERRED PAIN (Fig. 24.1)

The latissimus dorsi is a frequently overlooked myofascial cause of midthoracic back pain. The most common myofascial trigger points (TrPs) responsible for that pain are usually located in the midportion of the most craniad group of fibers in the region of the posterior axillary fold (Fig. 24.1A). Bonica and Sola[8] illustrate this TrP location accurately in their Figure 58-10A. A constant aching pain is referred to the inferior angle of the scapula and the surrounding midthoracic region (Fig. 24.1A).[8,53] Referred pain also may extend to the back of the shoulder[37] and down the medial aspect of the arm, forearm and hand, including the ring and little fingers (Fig. 24.1A and B). In describing the center of this pain, the patient has difficulty reaching behind to the lower scapular region but, when asked to draw the pain, is apt to mark a solid circle centered on the inferior angle of the scapula.

This is a **key** TrP that can be responsible for satellite TrPs in muscles located in the referred pain zone of the latissimus, such as the triceps brachii and flexor carpi ulnaris,[29] the lower trapezius (see Chapter 6), and the iliocostalis thoracis (see Chapter 48).

Figure 24.1C and **D** shows a less common location of a latissimus dorsi TrP in

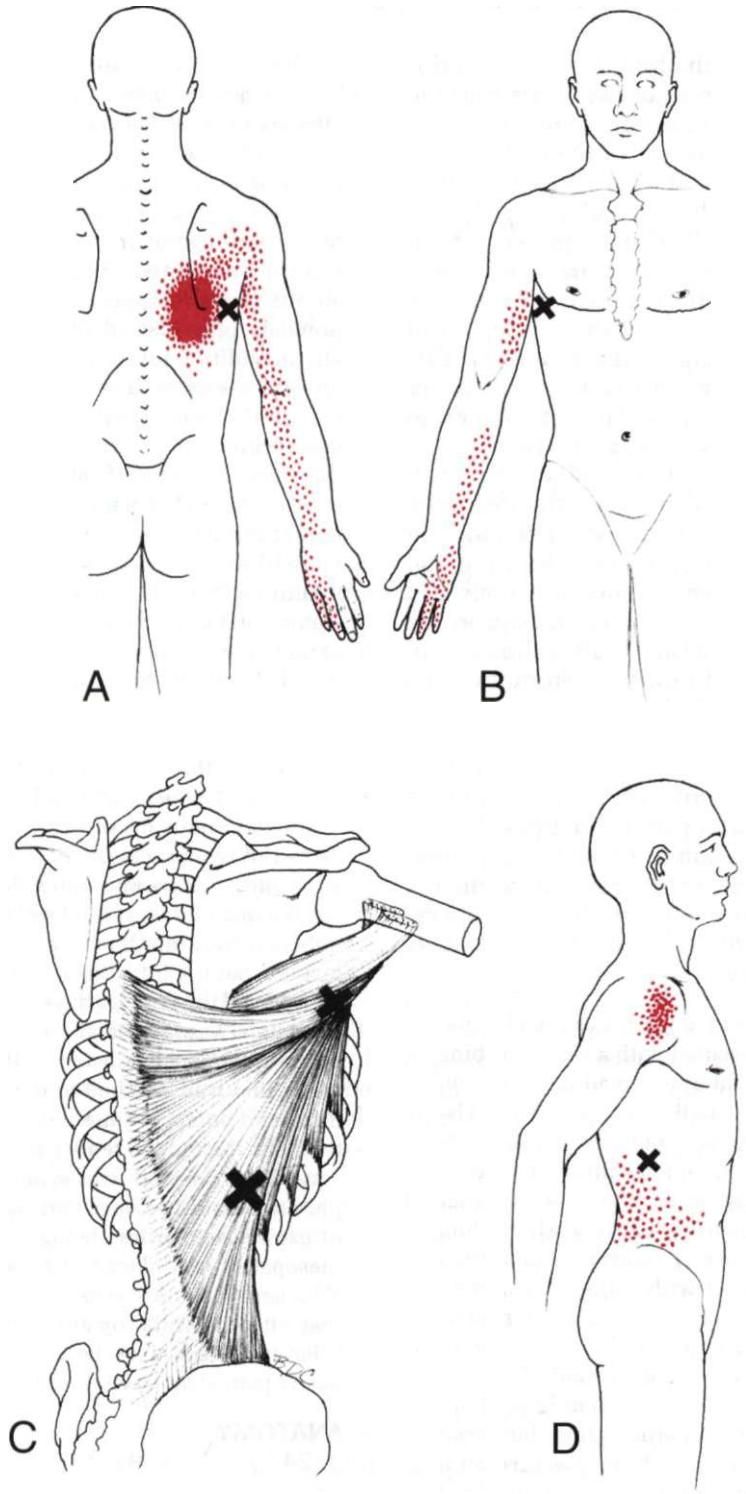


Figure 24.1. Referred pain patterns (essential portion is *solid red,* spillover portion is *stippled red*) referred from trigger points (Xs) in the right latissimus dorsi muscle. **A,** back view of the pain pattern from trigger points in their most common location within the axillary portion of the muscle. B, front view of same. **C,** anatomical view: most common, superior, location of trigger points (upper X) and inferior location (lower X). **D,** pain pattern of the inferior trigger point, which may also refer pain down the arm.

the midregion of the longest, more anterior fibers. This TrP location refers pain to the front of the shoulder and sometimes over the lower lateral aspect of the trunk above the iliac crest. The most anterior fibers form a series of shorter digitations that attach to the ribs. The pain patterns from TrPs in these digitations have not been identified and reported. An intermediate TrP (not shown here) in the midregion of the fibers attaching to the thoracolumbar region refers pain locally over the lower end of the posterior axillary fold, lateral to the scapula, as previously illustrated.[57]

Injection of hypertonic saline into a normal latissimus dorsi muscle in the area of the posterior axillary fold induces referred pain in various parts of the pattern shown in Figure 24.1A. Kellgren[34] reported that such an injection of 6% saline referred pain to the arm and forearm. Using 7.5% saline for injection, we found in a pilot study that the vertically oriented, deep fibers next to the teres major were more likely to refer pain to the back in the scapular area, whereas the superficial, more horizontally oriented fibers were likely to refer pain to the upper limb.[53]

Winter[60] attributed some cases of low back pain to TrPs in the fascial attachments of the latissimus dorsi in the lumbosacral area. These "TrPs" were likely trigger areas of enthesopathy.

Sandford and Barry[52] described a patient who presented with a sharp stabbing pain in the right upper quadrant of the abdomen, and it radiated to the back. The pain was of three months' duration starting after playing slot machines for 6 hours per day, was progressive, and was similar to pain experienced 9 years earlier before cholecystectomy. However, rather than being associated with eating, her current pain was associated with muscular activity. All gastrointestinal and laboratory studies were normal. Examination revealed a soft abdomen with mild right upper quadrant tenderness that increased with deep pressure. Firm pressure on a firm tender area over the latissimus dorsi muscle reproduced her abdominal pain. This was treated with the TrP therapeutic technique of spray and stretch for the latissimus dorsi muscle and with a home program of ice massage and self-stretch of the latissimus dorsi muscle. The patient became asymptomatic and remained so after six treatments over a 3-week period.

This case report illustrates an important characteristic of pain referred from TrPs. Where there is previous pain modification of central nervous system pathways, pain referred from TrPs in that region is prone to appear at the site of the previous pain instead of (and occasionally in addition to) its usual pattern of referral. Sometimes TrPs from several muscles in the region will refer to the same site of prior pain, a site that is not within their usual pattern of referral. The more intense the earlier pain and the more intense the emotions associated with it, the more likely this phenomenon becomes.

Dittrich,[16] in 1955, described the latissimus dorsi syndrome in four case reports as pain referred to the shoulder and shoulder-blade region, to the lower part of the chest posteriorly, and to the wrist and hand. Onset of pain was sudden in every case and in three cases related to muscular overload. One patient had tender spots in the latissimus dorsi, and all had some degree of tenderness in the region of the attachment of the latissimus dorsi to the lumbodorsal fascia at the level of L_2 or L_3. Subcutaneous injection of procaine to the level of, but not into, this fascia temporarily relieved the pain for days or weeks, but the pain always returned. Sustained relief followed surgical excision of the strip of fascia that was attached to the latissimus dorsi and contained the tender spot. Dittrich considered the tender site a TrP that caused the referred pain symptoms. This picture would be consistent with the site of excised tenderness being a site of enthesopathy secondary to latissimus dorsi TrPs, and it reinforces other observations that sites of enthesopathy secondary to TrPs can themselves be a source of referred pain characteristic of that muscle.

2. ANATOMY (Fig. 24.2)

This muscle attaches ***below*** to the spinous processes of the lower six thoracic vertebrae and of all the lumbar vertebrae, to the sacrum *via* the lumbar aponeurosis, and to the posterior part of the crest of the ilium. Bogduk and Twomey[7] describe and

illustrate in detail the fascial attachment of the latissimus dorsi to the thoracolumbar fascia. The caudal ends of the most vertical fibers of the muscle are anchored anteriorly to the last three or four ribs. ***Above,*** the latissimus dorsi curves around the lower border of the teres major and attaches to the floor of the intertubercular groove of the humerus (Fig. 24.2). The tendons of these two muscles are partially united near their attachments.[9]

The teres major attachment ends distal and dorsal to that of the latissimus dorsi (Fig. 24.2). There the two tendons are covered by the pectoralis major (*see* Chapter 42), which bridges the bicipital tendon to attach to the lateral lip of the intertubercular groove for the bicipital tendon. All latissimus fibers twist nearly 180° around the teres major muscle. The nearly vertical fibers of the latissimus dorsi, which attach to the ribs and the crest of the ilium, hug the teres major in the axillary fold and attach proximally on the humerus. The superior, most horizontal fibers of the latissimus usually pass over the inferior angle of the scapula and either directly or through a fascial extension are often adherent to the inferior angle.[5] These uppermost fibers form the free margin of the posterior axillary fold and attach more distally on the humerus. The anatomical relation of the latissimus dorsi muscle to other shoulder and pectoral girdle muscles appears in Figure 26.3.

An anatomical and functional analysis of 10 pigs[27] showed the latissimus dorsi to be divided into three compartments: the lateral, oblique and transverse segments of the muscle. Each compartment is supplied by its own neural branch with some overlap of innervation, especially in the oblique segment which is located between the other two.

Rarely, a variant called the axillary arch muscle crosses the lower axillary fossa between the humeral end of the latissimus dorsi and the costal end of the pectoralis major muscle.[9,19,28]

SUPPLEMENTAL REFERENCES

The latissimus dorsi muscle has been well-illustrated as seen from behind,[1,9,13,36,39,47,54] from the side,[19,20] and from the front.[11,40] Cross sections show the muscle at the thoracic level[3,46] and at the lumbar level.[4,12,55] Details of the relationship between the latissimus dorsi and the teres major muscles in the axilla and their attachment to the humerus are shown from behind,[14,48] from below,[56] and from in front.[2,10]

3. INNERVATION

The muscle is supplied by the thoracodorsal (long subscapular) nerve through the posterior cord and spinal nerves C_6, C_7 and C_8.[9] In pigs,[27] the thoracodorsal nerve divides into three branches at the latissimus dorsi muscle and separately innervates its three compartments.

4. FUNCTION

This extensive muscle (together with its fasciae) links the upper and lower body in a myriad of functions. Through its humeral and scapular attachments, the latissimus dorsi can influence the shoulder girdle and thus indirectly, neck posture. It can influence trunk and pelvic posture and movement by its attachment to the last six thoracic vertebrae and the last three or four ribs, and by its attachment through the lumbodorsal fascia to the lumbar vertebrae, the sacrum, and the iliac crest.

The latissimus dorsi primarily acts to extend the arm at the shoulder joint, as when swimming the crawl stroke or when chopping wood. It adducts and assists medial rotation of the arm,[36,51] and depresses the humerus.[6,22] The combination of humeral depression and extension by the latissimus, acting through the glenohumeral joint, adducts the scapula and draws the shoulder girdle downward and backward.[30] The vertical fibers of the latissimus dorsi, and to a lesser extent the lower fibers of the pectoralis major, lift and support the body weight when "chinning" oneself with the arms overhead and when walking with crutches.

An electromyographic (EMG) study using bipolar fine wire electrodes[32] showed marked activity of the latissimus dorsi in all 8 subjects when depression of the shoulder was resisted with 30 kilograms of weight. The muscle became moderately active during arm extension and during adduction in front of the body, but was strongly activated by arm adduction behind the body which added a degree of extension also. The muscle was silent during horizontal abduction and adduction.[32] Dur-

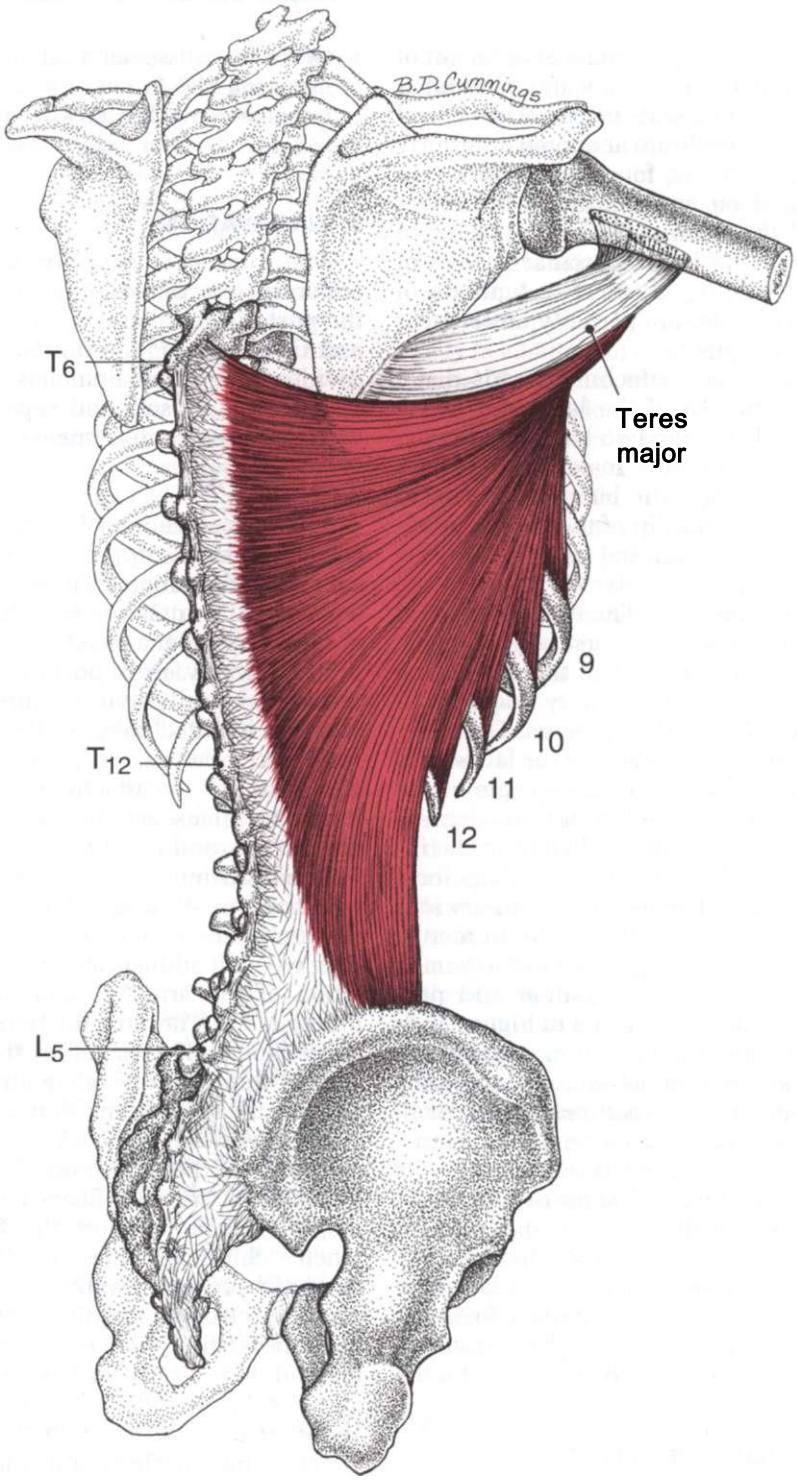


Figure 24.2. Attachments of the latissimus dorsi *(red),* and its relation to the teres major muscle, which arises from the edge of the scapula. The superior (horizontal) fibers of the latissimus dorsi swing around the teres major, and the tendons attach near each other (the teres major to the medial lip of the intertubercular groove of the humerus, and the latissimus dorsi to the floor of the groove). Both muscles are elongated by flexion and lateral rotation of the humerus.

ing conical movement of the arm[45] the latissimus dorsi became electromyographically active when the arm was moving obliquely downward away from the midline (toward extension).

Stimulation studies[18] showed that the upper one-third (nearly horizontal fibers) of the latissimus dorsi adducted and extended the arm while strongly retracting the scapula. When the muscles contracted bilaterally, this retraction strongly extended the thoracic spine. Stimulation of the lowest third of the muscle strongly depressed the shoulder and extended the arm. Medial rotation was produced only when the arm had been placed in abduction. The tendency of strong contraction of the latissimus dorsi to subluxate the glenohumeral joint was countered by the long head of the triceps brachii and the coracobrachialis muscles.[18]

Although the records of 12 subjects were highly variable, the average EMG activity of the latissimus dorsi during a golf swing began at minimal values that quickly increased to a maximum response (during forward swing) of about 50% of the maximum activity elicited by manual muscle strength testing. The activity slowly subsided throughout the rest of the swing to about 20% of the maximum test value. There was no marked bilateral difference.[49]

Fine wire EMG recordings of qualitative latissimus dorsi activity during aquatic swimming in 7 subjects[44] showed a predominantly propulsive function. Activity started at early pull-through when the arm was fully abducted and lateral rotation was at its maximum. Activity increased as the arm was medially rotated and adducted, progressing to the end of pull-through. This appeared during freestyle, butterfly and breaststroke swimming.

Monitoring the pitching of 4 professional baseball pitchers with fine wire EMG electrodes[31] revealed that latissimus dorsi activity during the cocking phase reach 168% of maximum activity recorded during manual muscle testing (MMT). During the acceleration phase the muscle activity continued to exceed 100% of MMT by 35% more activity. This excessive muscular activity during a coordinated, well-learned, meaningful activity is even more marked in some other muscles.[31] This illustrates an important principle: the EMG activation of a muscle can be remarkably different under test conditions as compared to EMG activation during meaningful, well-learned movements. This difference in activity during testing and during skilled activity can be very large in a muscle that is inhibited reflexly by active TrPs in a functionally related muscle.[26]

Comparison of 12 healthy uninjured athletes with 15 athletes skilled in throwing, who had chronic anterior instability of the shoulder that required operative intervention,[22] revealed marked muscle imbalance in the patients. The EMG activity in the latissimus dorsi muscles of the patients was nearly three times the normal level during late cocking phase and roughly one third of normal during acceleration. The authors postulated that this difference in neuromuscular control was a factor in producing or maintaining the anterior instability. This is the kind of incoordination that can be reflexly induced by active TrPs, but TrPs were not mentioned as part of the examination or as a consideration.

The latissimus dorsi showed minimal EMG activity during simulated automobile driving.[33] As would be expected, typing and various sitting postures caused little, if any, activation of the latissimus dorsi.[38]

In a study of pigs walking up a ramp, the three compartments of this muscle (the transverse, oblique, and lateral segments) showed progressive delay in onset of activity during each stride. Activity in the teres major muscle corresponded closely to the early onset of activity in the transverse segment.[27] This illustrates the functional compartmentalization of the latissimus dorsi muscle.

5. FUNCTIONAL UNIT

The functions of the teres major and the long head of the triceps brachii relate in a complex way to those of the latissimus dorsi; however, only the latissimus dorsi attaches to the trunk. With the arm at the side of the body, the latissimus dorsi and the long head of the triceps have antagonistic effects on displacement of the glenohumeral joint. In the abducted arm, their effect on this displacement is synergistic. With the scapula stabilized, the teres major

and the latissimus dorsi are strongly synergistic in humeral extension, adduction, and medial rotation because of their common attachment to the humerus. Through the glenohumeral joint, the latissimus dorsi can strongly influence positioning of the scapula/shoulder girdle.

The lower part of the pectoralis major is a synergist of the latissimus dorsi in shoulder girdle depression.

The thorax-elevation function of the scalene muscles and the shoulder girdle-elevation function of the upper trapezius are antagonistic to the depressor function of the latissimus dorsi.

Inferiorly, the anterior latissimus dorsi fibers interdigitate with the fibers of the lateral abdominal oblique muscle which synergistically fixes the lower ribs for these fibers of the latissimus to function.

6. SYMPTOMS

The pernicious infrascapular midthoracic backache projected from latissimus dorsi TrPs is distressingly unresponsive to stretching movements or change of position by the patient in an effort to obtain relief. The latissimus dorsi is a long slack muscle and, therefore, rarely causes pain on movements that stretch it only partially; however, it does refer pain with depressor movements that load it. Referred pain can occur when the patient stretches upward and far out in front of the body to handle something bulky. Patients with TrPs in the latissimus usually don't complain of referred pain from this muscle until the TrPs are sufficiently active to cause pain at rest. The fact that a patient is not aware of any particular activity that aggravates the mid-back pain immediately casts suspicion on the latissimus dorsi muscle.

The patient is likely to give a long history of negative diagnostic procedures, such as bronchoscopy, coronary angiogram, myelogram, or computerized tomography, and is likely to give a history of unsuccessful therapy (mistakenly) applied to the area of referred pain in the back rather than to its source.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Because of its long slack nature, the latissimus dorsi is unlikely to be activated by acute trauma or overload. Therefore, identifying the source of insidious activation requires a careful analysis of activities that require forceful shoulder-girdle depression (weight bearing) or repetitive extension, especially when combined with adduction. Symptoms are more likely to be experienced when the muscle is stretched by reaching forward and up rather than when its depressor and arm extension functions are overloaded.

A few common activities that can eventually activate these TrPs are reaching overhead to exercise by pulling heavy weights, throwing a baseball, hanging from a swing or rope, and pressing down to twist weeds out while gardening. The patient becomes aware of an enigmatic persistent back pain some time after the activity has ended.

The latissimus dorsi can develop satellite TrPs as a result of key TrPs in the serratus posterior superior muscle. Inactivation of the key serratus TrPs can inactivate the latissimus dorsi TrPs without further treatment of them.[29] Sometimes however, a satellite TrP does *not* clear up with inactivation of the key TrP and must be identified and inactivated as usual for that specific muscle. Just as frequently, the latissimus dorsi muscle itself can have key TrPs that are responsible for satellite TrPs in other muscles, such as the triceps brachii, the lower trapezius, and the iliocostalis thoracis.

The persistent compression of this muscle imposed by a tight brassiere around the chest can activate and perpetuate TrPs in it. If the excessive tension of the brassiere-as evidenced by deep indentation of the skin-is caused by tight elastic, its elasticity can be weakened by dampening the elastic part and heating it with a hot iron.

The body pressure of sleeping on the side of a latissimus TrP can activate it and seriously disturb sleep and disturb function the next day. Once activated, using the arm to assist getting up or down from a low seat can aggravate TrPs in the more vertical latissimus fibers.

An interesting case report[41] was that of a 68-year-old professional viola player who had developed multiple shoulder-girdle TrPs that prevented him from performing in concert. Initial examination re-

vealed involvement of the supraspinatus muscle including tenderness ("impingement") of its tendinous attachment, which cleared in 2 weeks with hold-relax treatment of the muscle and phonophoretic application of 10% hydrocortisone to the tendon attachment. The glenohumeral joint now tested normal during active and passive range of motion and exhibited minimal deficit in muscle strength. The patient noted greater ease in activities of daily living such as putting on his shirt and combing his hair, but pain still limited his viola playing and he could still only reach the T_{11} level with his hand behind his back. Further examination revealed active TrPs in the latissimus dorsi and teres major muscles and none in the pectoralis minor and serratus anterior muscles. Treatment of the two involved muscles with spray and stretch permitted him to resume playing his viola. Treatment included maintenance of strength and mobility of the shoulder-girdle muscular complex.[41] The sensitivity of the latissimus dorsi TrPs to the stretch position required for playing the viola seriously interfered with it. While slow stretch with augmentation techniques can release TrPs, sustained contraction to stabilize the scapula and repeated rapid movements can aggravate and perpetuate them.

8. PATIENT EXAMINATION

Because of this muscle's influence on the trunk and pelvis through its expansive attachments, patient examination should include an overall assessment of body symmetry and limb posture.

The patient with TrPs in the latissimus dorsi muscle is unaware of the slightly restricted range of motion that is demonstrated by the Mouth Wrap-around Test (see Fig. 18.2), by the Triceps Brachii Test (see Fig. 32.4), and by the Hand-to-shoulder Blade Test (see Fig. 22.3). To do the Triceps Test, the patient abducts the arm and, with the elbow held straight, brings the arm into firm contact with the ear and, if possible, behind the ear (see Fig. 32.4). Inability to hold the elbow straight in this test indicates additional involvement of the long head of the triceps.

Pain due to latissimus dorsi TrPs may be elicited by reaching far forward and upward with the arm laterally rotated (muscle stretched by arm flexion and being wrapped around the humerus), or by having the patient press down hard on the iliac crests (muscle activated in the shortened position, performing its shoulder depressor function).

In addition to range of motion testing, the glenohumeral joint should be examined for normal joint play.[42]

9. TRIGGER POINT EXAMINATION (Fig. 24.3)

With the patient lying supine, the latissimus dorsi is put on half stretch by placing the hand under the head or under the pillow with the arm laterally rotated and abducted to about 90°. The examiner grasps the latissimus dorsi muscle (Fig. 24.3) along the free border of the posterior axillary fold at the midscapular level where the latissimus dorsi wraps around the teres major muscle, as also shown by Lange.[37] While lifting the muscle off the chest wall, the firm bands and their points of maximal tenderness (TrPs) are rolled between the fingers and thumb to identify them. These TrPs usually lie a few centimeters (about an inch) below the top of the arch of the posterior axillary fold. Snapping palpation of one of these bands elicits a strong local twitch response, which is readily seen along the scapular margin or over the lower thoracic and lumbar regions, depending on which fibers are involved. A large twitch response of several bands contracting simultaneously may cause the arm to jerk in a movement that can be seen by the examiner.

The reliability with which the physical features of TrPs could be determined was evaluated by four experienced physicians, who, following a *three hour training session* immediately before the study, examined five pairs of muscles for five physical characteristics of TrPs in each of 10 subjects.[21] The muscles examined were the infraspinatus, latissimus dorsi, upper trapezius, extensor digitorum, and sternocleidomastoid. Agreement among examiners for TrP characteristics of the latissimus dorsi muscle was high ($P < 0.001$) for the detection of spot tenderness, detection of a taut band, presence of referred pain and reproduction of the subject's symptomatic

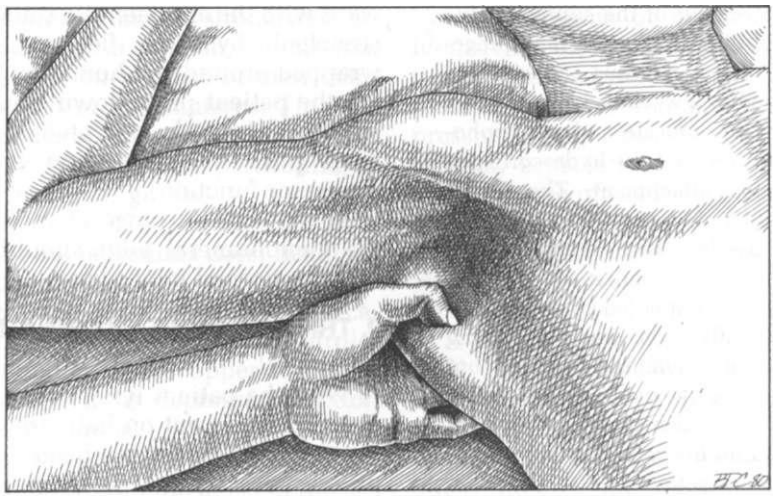


Figure 24.3. Pincer palpation of the right latissimus dorsi muscle to locate trigger points within the posterior axillary fold. The latissimus dorsi is distinguished from the teres major by palpating the axillary border of the scapula and including in the pincer grasp only those fibers that are inferior and not attached to the axillary border.

pain, and for the presence or absence of a local twitch response. Agreement was almost perfect for detection of spot tenderness and for reproduction of the subject's symptomatic pain. With a few hours of adequate training, experienced clinicians can learn to identify (with a high degree of reliability) TrPs in this easily examined muscle.

10. ENTRAPMENT

No nerve entrapment has been identified as due to TrP activity in this muscle.

11. DIFFERENTIAL DIAGNOSIS

Confusingly Similar Conditions

Differential diagnosis of conditions that produce pain similar to that of latissimus dorsi TrPs includes entrapment of the suprascapular nerve at the spine of the scapula, C_7 radiculopathy, ulnar neuropathy, and bicipital tendinitis. The nerve entrapments are distinguished by appropriate electrodiagnostic examinations, and bicipital tendinitis is distinguished by tenderness specifically of the biceps tendon, which is often associated with TrPs in the long head of that muscle.

Dittrich[17] attributed low back pain in many of his patients to tears and fibrous tissue pathology of the lumbodorsal fascia and subfascial fat, which he illustrated. Based on operative findings, he surmised that the damage was done by excessive tension of the latissimus dorsi.[15] Certainly, tense TrP bands would increase the tension on that part of the fascia to which the taut-band muscle fibers attach. However, he did not report looking for the corresponding taut bands and their midmuscle TrPs.

Articular Dysfunctions

Both latissimus dorsi and quadratus lumborum TrPs are associated with innominate dysfunction. In addition to being distinguished by distinctly different referred pain patterns, TrPs in the quadratus lumborum muscle are associated with sacroiliac dysfunction, whereas TrPs in only the latissimus dorsi muscle are associated with an upslip of the innominate. Therefore, the seated-flexion test would be positive in the case of quadratus lumborum involvement, but not in the case of latissimus dorsi involvement.

Additional articular dysfunctions typically associated with TrPs in the latissimus dorsi muscle are group dysfunctions spanning several segments from approximately T_7 or T_8 to L_3 or L_4. With these dysfunctions

there is sidebending of the vertebrae toward the involved latissimus dorsi and rotation away from the involved muscle. Rarely, one also may see an ipsilateral innominate upslip with a concurrent leg length discrepancy because of tension in latissimus dorsi fibers that attach to the iliac crest. With this ipsilateral innominate upslip, one finds a positive Standing-flexion Test[23] but a negative Seated-flexion Test[24], which rules out a primary sacroiliac dysfunction.

Latissimus dorsi TrPs may be associated with elevation (inhalation) lesions of the lower four ribs. This condition can be treated by using myofascial release[59] that is coordinated with respiration to augment relaxation. See also Section 12 in this chapter for latissimus dorsi release.

Uncompensated tension from the latissimus dorsi muscle tends to displace the head of the humerus anteriorly in the glenoid cavity. The joint should be tested for normal joint play.[42]

Associated Trigger Points

The latissimus dorsi is a member of the quadrad of muscles responsible for the myofascial pseudothoracic outlet syndrome (described in Chapter 18). The referred pain pattern of any one of these muscles can suggest the diagnosis of thoracic outlet syndrome. When at least three of these muscles—including the latissimus dorsi, pectoralis major, teres major, and subscapularis muscles—have active TrPs, the composite referred pain experienced by the patient is strongly suggestive of a thoracic outlet syndrome and often is misdiagnosed as such. These muscles cause no compression of structures in the thoracic outlet.

Eventually, the teres major muscle usually develops active TrPs in association with those in the latissimus dorsi, since these two muscles are anatomically and functionally closely related. The long head of the triceps brachii also tends to develop TrPs because of synergistic or antagonistic (depending on arm position) overload, especially in chronic cases.

One should consider other muscles that may refer pain to the midback, including the scaleni, upper rectus abdominis, subscapularis, iliocostalis, serratus anterior, serratus posterior superior and inferior, lower trapezius, and the rhomboids.

12. TRIGGER POINT RELEASE (Figs. 24.4 and 24.5)

Initially, the muscle may be stretched with the patient supine (Fig. 24.4). The vapocoolant spray is applied to the trunk in a cephalad direction, covering the length of the muscle and all of the posterior axillary fold, then down the posterior arm and forearm over the region of referred pain, including the fourth and fifth digits (Fig. 24.4). The operator continuously takes up any slack that develops. The full spray pattern is repeated as the muscle is passively elongated.

To ensure full stretch and complete coverage of the muscle posteriorly, the patient should next lie on the opposite side (Fig. 24.5), and the painful arm should be slowly brought overhead and then posteriorly until it reaches tightly behind the ear. To release the muscle tightness by elongation (Fig. 24.5), the sweeps of vapocoolant spray start in the area of the trigger points (TrPs) and cover all the posterior thoracic zones of referred pain. Next the spray is directed from the TrP area to the fingertips, covering the upper limb part of the referred pain pattern.

When the nearly horizontal latissimus fibers contain TrPs, the fibers can be stretched by placing the patient's arm anteriorly across the chest and pulling it into adduction to fully take up the slack. The spray again follows the lines in Figures 24.4 and 24.5. The spray covers the muscle fibers and the pain reference pattern, first toward the spine, and then to the hand.

See Figure 45.11 of this manual for a lower rib release technique that is effective also in releasing the latissimus dorsi muscle.

An effective latissimus dorsi release technique that also includes interscapular and other shoulder-girdle muscles is illustrated in Figure 18.3.

Because the latissimus dorsi is such a slack muscle to begin with, it is especially important to make full use of augmentation procedures when releasing it with stretch techniques. Slow exhalation, contract-relax, and voluntary assistance by having the patient contract antagonists (reciprocal inhibition) can be effective.

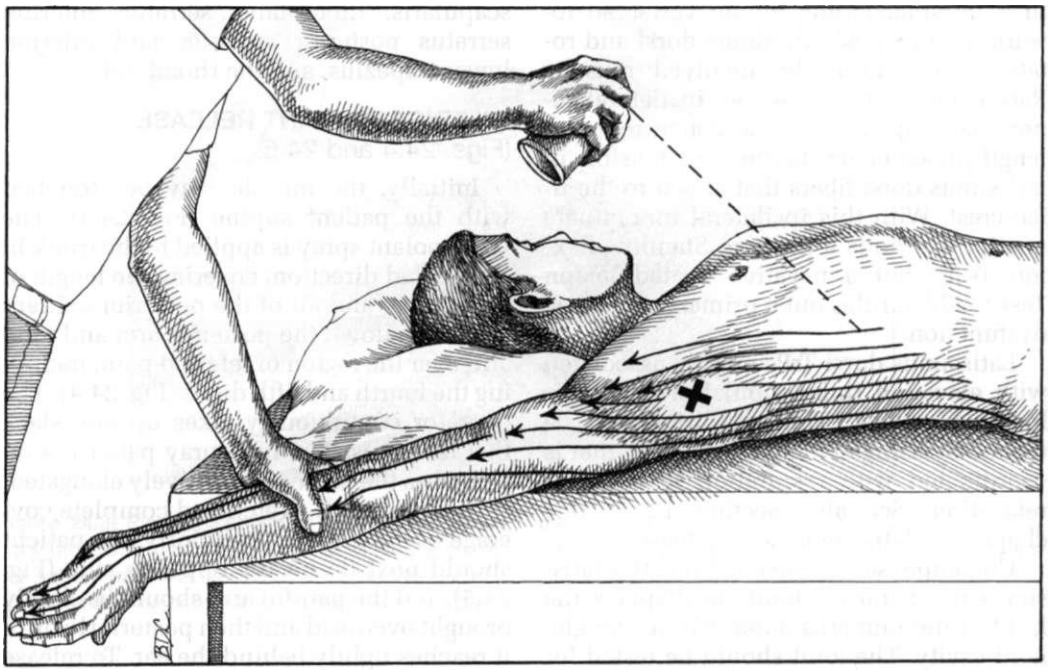


Figure 24.4. Supine stretch position, the most common location of trigger points (X), and vapocoolant spray pattern *(arrows)* for the latissimus dorsi muscle. Any added pressure by the operator should be applied at the distal humerus, *proximal* to the elbow. This position can be used also for spraying *prior* to a two-handed release of this muscle, stabilizing the pelvis with the right hand and using postisometric relaxation to release the muscle.

The vapocooling of this muscle is followed at once by moist heat, and then, most important, by active full range of motion. Several authors have reported individual cases of successful release of latissimus dorsi TrPs using the stretch-and-spray technique.[1643]

The TrPs in the posterior axillary fold are conveniently accessible to trigger point pressure release (using pincer palpation).

Release of key latissimus TrPs may release satellite TrPs in other muscles (e.g., the iliocostalis thoracis, lower trapezius, triceps brachii, flexor carpi ulnaris) without specific treatment to those muscles.

For those acquainted with osteopathic techniques, the integrated neuromusculoskeletal and myofascial release technique for the upper limb and shoulder joint in the supine position can be adapted for release of latissimus dorsi TrPs.[58]

13. TRIGGER POINT INJECTION (Figs. 24.6 and 24.7)

The latissimus dorsi trigger points (TrPs) within the posterior axillary fold are readily and effectively injected.[57] In the supine patient, they are located by pincer palpation, as described in Section 9. A TrP is fixed between the operator's digits for precise insertion of the needle and is then injected (Figs. 24.6 and 24.7); a strong local twitch response is usually both seen and felt when the needle penetrates a latissimus dorsi TrP. Both the superficial and deep axillary portions of the muscle should be probed for TrPs, which tend to occur in clusters.

The teres major often also harbors active TrPs which can be injected through the same skin puncture by sliding the skin into position, with the tip of the needle positioned subcutaneously. The teres major TrPs can be injected by repositioning the

pincer grasp used to inject latissimus dorsi TrPs.

Immediate hemostasis is maintained by the palpating hand both during needle probing and after the injection. Injections are followed by stretch and spray. The treatment is completed, first with hot packs to the axillary region, and then with full active range of motion of the muscle injected.

Rachlin[50] describes and illustrates injection of the midmuscle TrPs of the latissimus dorsi. The referred pain from trigger areas in the musculotendinous and aponeurotic portions of the lumbar-region attachment of the latissimus dorsi was temporarily relieved by injecting that region with procaine.[15-17] Dittrich did not locate and treat the primary TrPs in midmuscle; he treated recurrence of pain by excising the tender area surgically, which gave some patients relief.

14. CORRECTIVE ACTIONS

The patient is instructed, when pulling down on something, to keep the arm vertical (not forward of the abdomen) and the elbow beside the body. The patient also should be instructed to step up on a stool if needed to avoid reaching high for a heavy object, and at night to keep a pillow in the axilla between the elbow and the chest to prevent prolonged shortening of the muscle at rest (*see* Fig. 26.7).

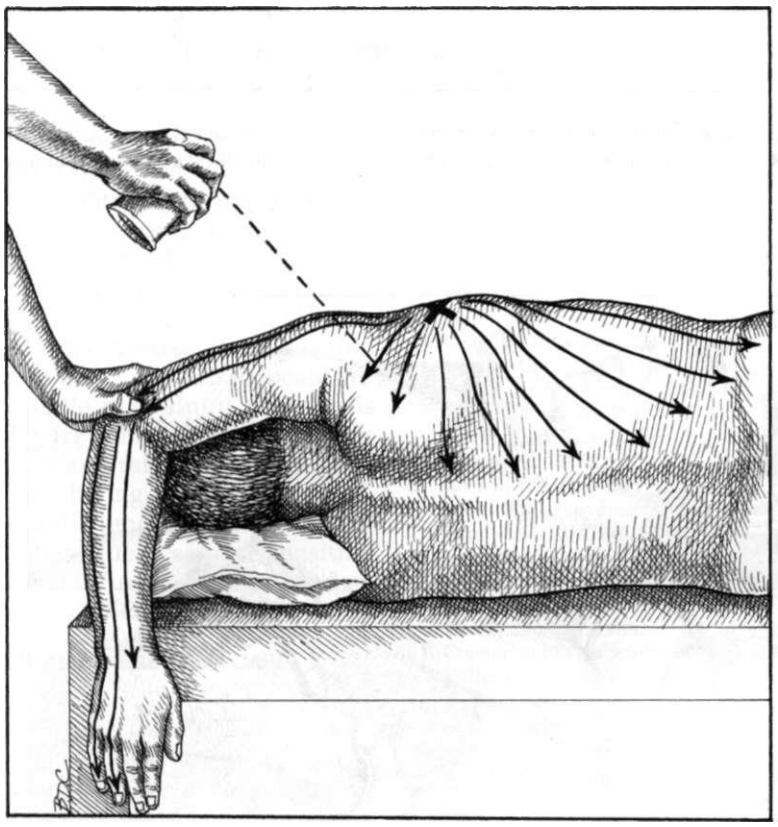


Figure 24.5. Sidelying stretch position, the most common location of trigger points (**X**), and vapocoolant spray pattern *(arrows)* for the latissimus dorsi muscle. Postisometric relaxation can release this muscle effectively by having the patient inhale and press the humerus *lightly* against the operator's hand, then exhale slowly and relax the muscle. For lengthening the more horizontal fibers of the muscle, the patient's arm is placed anteriorly across the chest; again, postisometric relaxation can be effective.

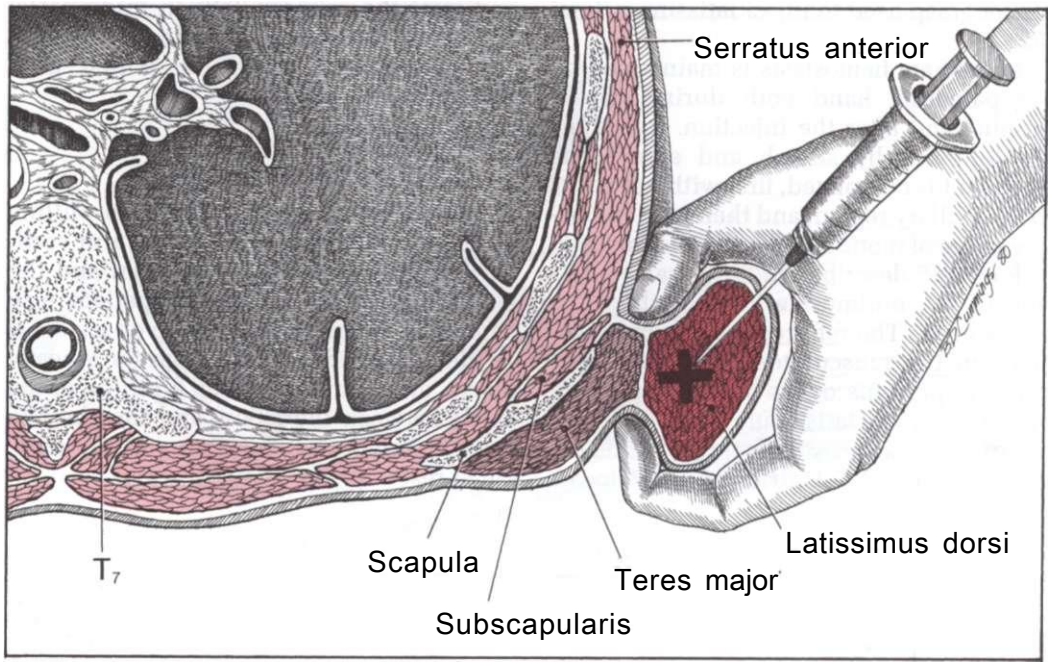


Figure 24.6. Cross section view of injection technique for the right latissimus dorsi muscle, using pincer palpation. The "**X**" locates a trigger point being injected. Section is at the level of the seventh thoracic vertebra.

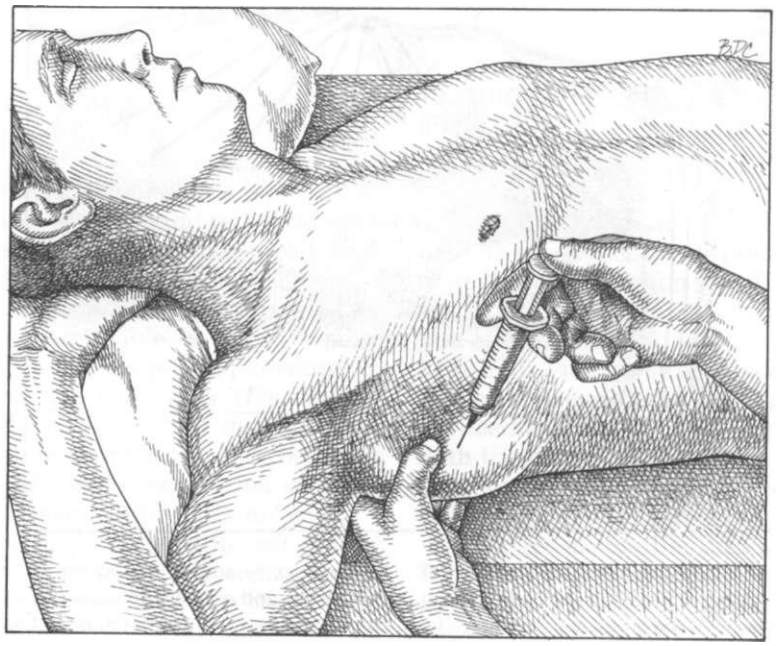


Figure 24.7. Injection of trigger points at their most common location in the latissimus dorsi muscle.

Home exercises to passively stretch the muscle include the Mouth Wrap-around Test (see Fig. 18.2) and the upper hand position of the In-doorway Stretch Exercise (see Fig. 42.9C). To effectively stretch the latissimus dorsi while doing the latter exercise, the low back must be arched (hyperextended) and the hips swung forward through the doorway; the patient should feel the tension in the latissimus dorsi muscle. As tension in the muscle releases with slow exhalation, the patient slides the hands a little higher in successive steps. Each stretch maneuver should be done smoothly, without jerking the muscle for a few repetitions daily until relief is obtained. The effectiveness of the exercise is increased by following it with moist heat, applied for 15-20 minutes, especially before retiring.

Greenman[25] illustrates an effective self-stretch technique for the latissimus dorsi with the patient in the quadruped position, utilizing upper limb reach and shifting of the pelvis to lengthen the muscle.

Effective self-treatment for latissimus dorsi TrPs can be obtained by applying trigger point pressure release with a tennis ball. The patient lies on the affected side with a pillow supporting the head and shoulders, and with the arm flexed to take up slack in the latissimus dorsi muscle. Body position is adjusted over the tennis ball to locate TrP tenderness. Gentle nonpainful pressure is applied to the TrP while the patient alternately contracts and relaxes the latissimus dorsi muscle, exhaling during each relaxation phase, until tenderness in that TrP fades. The patient then readjusts position on the tennis ball to find another TrP and repeats the pressure-release procedure.

SUPPLEMENTAL REFERENCES, CASE REPORTS

Dr Travell[57] reported in detail the management of a patient with latissimus dorsi TrPs. Kellgren[35] described a patient with involvement of the latissimus dorsi and other shoulder-girdle muscles. Meador,[41] reported successful treatment of a viola player and Nielsen[43] reported treatment of a dentist who had active latissimus dorsi TrPs.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991(p. 381, Fig. 6-32).
2. *Ibid.* (p. 376, Fig. 6-26).
3. *Ibid.* (p. 42, Fig. 1-45).
4. *Ibid.* (p. 239, Fig. 4-55).
5. Bardeen CR: The Musculature. Sect. 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (p. 402).
6. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 271, 426).
7. Bogduk N, Twomey LT: *Clinical Anatomy of the Lumbar Spine.* Churchill Livingstone, New York, 1987.
8. Bonica JJ, Sola AE: Chest pain caused by other disorders. Chapter 58. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990, pp. 1114-1145 (p. 1134).
9. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 513-515, Fig. 6-42).
10. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 49).
11. *Ibid.* (Fig. 233).
12. *Ibid.* (Fig- 522).
13. *Ibid.* (Fig. 523).
14. *Ibid.* (Fig. 524).
15. Dittrich RJ: Low back pain—referred pain from deep somatic structure of the back. *Lancet* 73:63-68, 1953.
16. Dittrich RJ: The latissimus dorsi syndrome. *Ohio State Med f 51(10)973-975,* 1955.
17. Dittrich RJ: Soft tissue lesions as cause of low back pain: anatomic study. *Am J Surg* 92:80-85, 1956.
18. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 38-39, 68-70).
19. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (pp. 357-368, Fig. 48).
20. Ferner H, Staubesand J: *Sobotta Atlas of Human Anatomy.* Ed. 10, Vol. 2, *Thorax, Abdomen, Pelvis, Lower Extremities, Skin.* Urban & Schwarzenberg, Baltimore, 1983 (p. 8).
21. Gerwin RD, Shannon S, Hong CZ, *et al:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
22. Glousman R, Jobe F, Tibone J, *et al:* Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg 70A(2)/220-226,* 1988.
23. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 24, 312).
24. *Ibid.* (pp. 26, 316).
25. *Ibid.* (p. 473).
26. Headley BJ: Evaluation and treatment of myofascial pain syndrome utilizing biofeedback. Chapter 5. In: *Clinical EMG for Surface Recordings,* Vol. 2. Edited by Cram JR. Clinical Resources, Nevada City, 1990.
27. Herring SW, Sola OM, Huang X, *et al.:* Compartmentalization in the pig latissimus dorsi muscle. *Acta Anat* 347:56-63, 1993.
28. Hollinshead WH: *Anatomy for Surgeons.* Ed. 3, Vol. 3, *The Back and Limbs.* Harper & Row, Hagerstown, 1982 (pp. 274, 281, Fig. 4-19).

29. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(2):29-59, 1994.
30. Jenkins DB: *HoIIinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W.B. Saunders, Philadelphia, 1991 (pp. 81-83).
31. Jobe FW, Moynes DR, Tibone JE, *et al:* An EMG analysis of the shoulder in pitching. *Am J Sport Med 12(3):218-220,* 1984.
32. Jonsson B, Olofsson BM, Steffner LC: Function of the teres major, latissimus dorsi and pectoralis major muscles: a preliminary study. *Acta Morpol Neerl-Scand* 9:275-280, 1972.
33. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving, IV. *Ergonomics* 18:643-649, 1975.
34. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3:175-190,1938 (p. 184, Fig. 7).
35. Kellgren JH: A preliminary account of referred pains arising from muscle. *Br Med J 1*:325-327,1938 (Case 3).
36. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 279).
37. Lange M: *Die Muskelharten (Myogelosen).* J.F. Lehmanns, Miinchen, 1931 (p. 93, Case 3, p. 129 Fig. 40).
38. Lundervold AJ: Electromyographic investigations of position and manner of working in typewriting. *Acta Physiol Scand 24{SuppI):84,* 1951 (pp. 66-68, 126).
39. McMinn RM, Hutchings RT, Pegington J, *et al: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (pp. 94, 119, 120).
40. *Ibid.* (p. 126).
41. Meador R: The treatment of shoulder pain and dysfunction in a professional viola player: implications of the latissimus dorsi and teres major muscles. *J Orthop Sport Phys Ther ll(2):52-55,* 1989.
42. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964 (pp. 80, 81).
43. Nielsen AJ: Case study: myofascial pain of the posterior shoulder relieved by spray and stretch. *J Orthop Sport Phys Ther* 3:21-26, 1981.
44. Nuber GW, Jobe FW, Perry J, *et al.:* Fine wire electromyography analysis of muscles of the shoulder during swimming. *Am J Sports Med 14(1):7-11,* 1986.
45. Pearl ML, Perry J, Torburn L, *et al.:* An electromyographic analysis of the shoulder during cones and planes of arm motion. *Clin Orthop* 284:116-127, 1992.
46. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 8).
47. *Ibid.* (Fig. 27).
48. *Ibid.* (Fig. 57).
49. Pink M, Jobe FW, Perry J: Electromyographic analysis of the shoulder during the golf swing. *Am J Sports Med 18(2):137-140,* 1990.
50. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994:97-360 (pp. 200-202).
51. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Lea & Febiger, Philadelphia, 1967 (pp. 166-167).
52. Sanford PR, Barry DT: Acute somatic pain can refer to sites of chronic abdominal pain. *Arch Phys Med Behabil* 69:532-533, 1988.
53. Simons DG, Travell JG: The latissimus dorsi syndrome: a source of mid-back pain. *Arch Phys Med Behabil* 57:561, 1976.
54. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 302).
55. *Ibid.* (p. 306).
56. *Ibid.* (p. 316).
57. Travell J, Rinzler SH: Pain syndromes of the chest muscles: resemblance to effort angina and myocardial infarction, and relief by local block. *Can Med Assoc J* 59:333-338,1948 (pp. 333, 334, Case 1, Fig. 2).
58. Ward RC: Integrated neuromusculoskeletal techniques for specific cases. Chapter 63. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997, p. 851-899 (see pp. 891-892).
59. *Ibid.* (pp. 870-874).
60. Winter Z: Referred pain in fibrositis. *Med Rec* 157:34-37, 1944 (pp. 4, 5).

CHAPTER 25
Teres Major Muscle

HIGHLIGHTS: **REFERRED PAIN** from trigger points (TrPs) in the teres major muscle penetrates deeply into the posterior deltoid region. **ANATOMY:** the teres major tendon merges with that of the latissimus dorsi for a short distance, and then attaches to the medial lip of the intertubercular groove of the humerus. Together, these two muscles form the posterior axillary fold. Medially, the teres major attaches to the scapula, whereas the latissimus dorsi attaches to the chest wall. **FUNCTION** of the teres major includes assistance of adduction, medial rotation, and extension of the arm from the flexed position, chiefly when these motions are resisted. It is strongly activated when adducting the arm across the back. **SYMPTOMS** include primarily pain when reaching forward and up, with little restriction of motion. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are likely to occur when driving a car that is hard to steer. **TRIGGER POINT EXAMINATION** of the midmuscle TrP is performed by pincer palpation with the fingers surrounding the latissimus dorsi. Examination of the posterior (medial) TrP region is done with flat palpation against the scapula. **TRIGGER POINT RELEASE** may be performed by a clinician or by the patient and may completely resolve acute symptoms. **TRIGGER POINT INJECTION** is often required to inactivate all TrPs located in the muscle. **CORRECTIVE ACTIONS** include avoidance of overload, self-stretch exercises, and pillow positioning to prevent muscle shortening at night. All corrections may be essential for sustained relief.

1. REFERRED PAIN
(Fig. 25.1)

Involvement of the teres major muscle is relatively uncommon. The tenderness of trigger points (TrPs) in this muscle was found in only 3% of the 256 latent TrPs observed in the shoulder-girdle muscles of 200 healthy young adult subjects,[31] and in 7% of the 126 active TrPs found in the shoulder-girdle musculature among 80 somewhat older patients treated for shoulder pain.[30]

Trigger points in the teres major muscle refer pain to the posterior deltoid region and over the long head of the triceps brachii (Fig. 25.1 A), as also observed by Kelly.[15] Teres major TrPs may refer pain into the shoulder joint posteriorly and occasionally to the dorsal forearm, but rarely, if ever, to the scapula or elbow. Trigger areas can occur in the teres major muscle in three locations. A midmuscle TrP is located in the posterior axillary fold, where the latissimus dorsi muscle wraps around the teres major (Fig. 25.1B). A more medial trigger area overlies the posterior surface of the scapula, and another trigger area is located near the lateral musculotendinous junction (Fig. 25.1C).

2. ANATOMY
(Fig. 25.2)

The teres major muscle attaches ***medially*** to an oval area on the dorsum of the scapula near its inferior angle, and to the fibrous septa shared with the teres minor and infraspinatus muscles (*see* Fig. 23.3); ***laterally*** it attaches to the medial lip of the intertubercular sulcus of the humerus (Fig. 25.2).[4] The borders of the teres major and latissimus dorsi tendons are joined for a short distance near their humeral attachments *[see* Fig. 24.2). The two tendons pass between the coracobrachialis muscle anterior to them and the long head of the triceps brachii muscle posterior to them (*see* Fig. 26.3).

Supplemental References

Other authors illustrate the teres major muscle from in front,[1,6,20] the muscle from

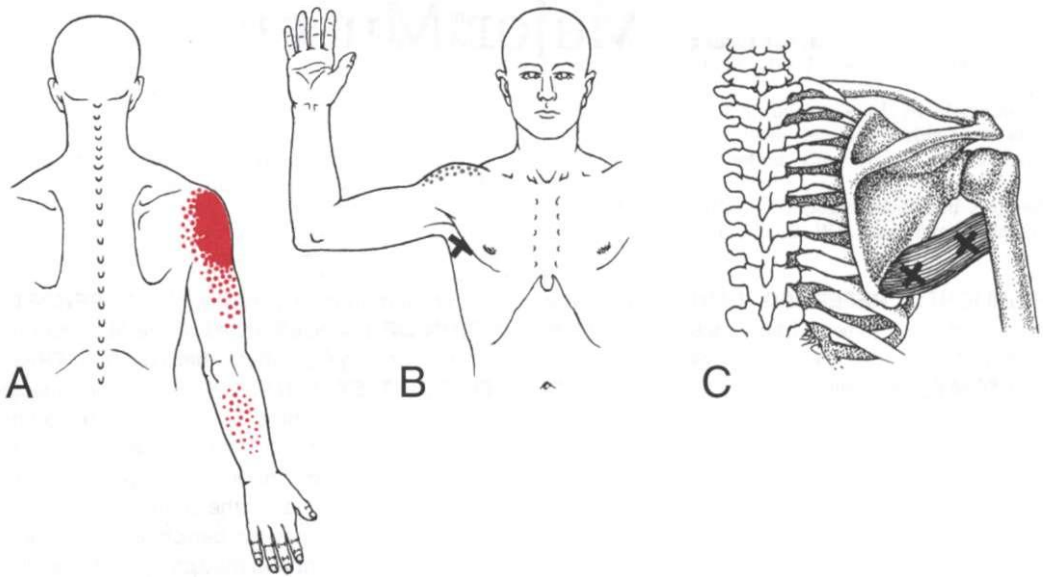


Figure 25.1. Three trigger areas (**X**s) in the right teres major muscle and their referred pain pattern. *Solid red* shows the essential portion; *stippled red* areas show the spillover portion of the pattern. **A,** rear view of referred pain pattern. **B,** front view showing midmuscle trigger point and part of the pain pattern. **C,** location of medial and lateral trigger areas near the regions of the medial and lateral musculotendinous junctions.

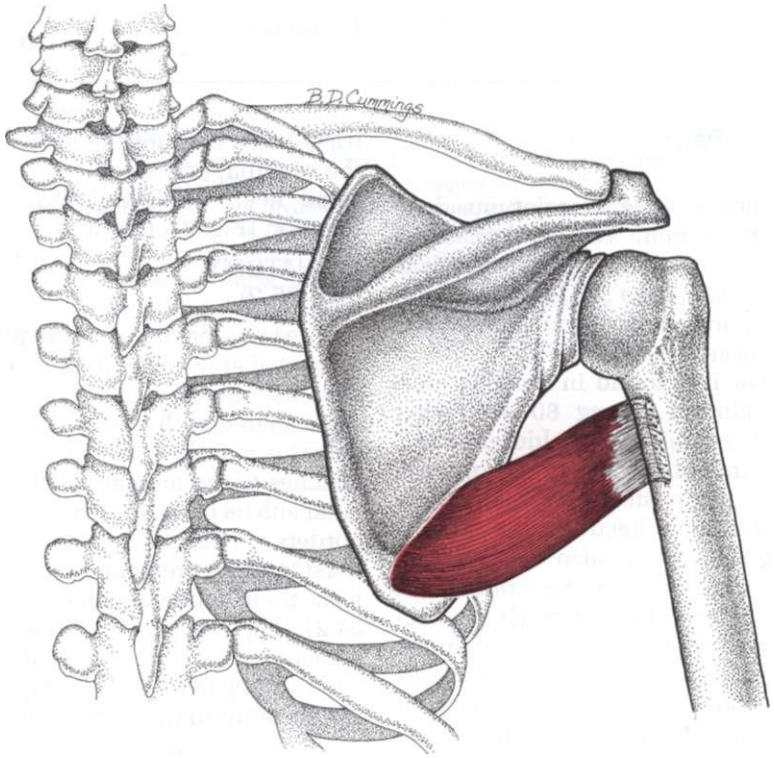


Figure 25.2. Attachments of the teres major muscle. See Figure 24.2 for its anatomical relation to the latissimus dorsi muscle, and Figure 26.3 for its relation to other shoulder-girdle muscles.

behind, [2,4,5,7,16,19,25] and its anatomic relations in transverse section.[26]

3. INNERVATION

The teres major muscle is innervated from spinal roots C_5 and C_6 *via* the posterior cord through branches of the lower subscapular nerve.[4]

4. FUNCTION

This muscle assists medial rotation (acting against resistance), is active during resisted adduction, and is active during extension of the arm from the flexed position;[3, 12, 16] it assists the latissimus dorsi in the wood-chopping movement. Stimulation studies[8] showed that, alone, the teres major only weakly adducted the arm to the side. However, when the scapula was stabilized by the levator scapulae and rhomboidei to fix the inferior angle, stimulation of the teres major muscle strongly adducted the arm.

An early report[11] categorically stated that the muscle never exhibited activity during movement of the arm, but came into action only when necessary to maintain a static position. This was accepted as fact[28] until Basmajian[3] demonstrated that the muscle is electromyographically active when the arm is medially rotated or extended, but only against resistance. The teres major also is activated during the backward swing of the arm in walking.[3]

Jonsson, ***et al.***[14] showed that the teres major was moderately activated by extension of the arm, and strongly activated by adducting the arm behind the back, but activated hardly at all when adducting the arm across the front of the body. During a rotary movement of the arm held in front of the body,[24] activity of the teres major muscle corresponded closely to the degree of extension of the arm as the arm was moving down, but showed only minor activity in this position when the circle was reversed and the arm was moving up in flexion.

An electromyographic (EMG) study of typing[17] revealed that striking a single typewriter key caused moderate activity of the teres major muscle in most subjects, and that with fatigue, the EMG activity increased markedly in amplitude. Writing long-hand caused moderate teres major activity. Elevation of the typewriter keyboard had little influence on EMG activity in this muscle.[17] The same should be true of a computer keyboard.

In a study of muscular activity when driving a car,[13] the teres major muscle was active when the hand moved the wheel downward on the same side as the muscle, which is the same finding as the rotary-movement experiment noted above.

5. FUNCTIONAL UNIT

The latissimus dorsi and the long head of the triceps brachii form a myotatic unit with the teres major for extension and medial rotation of the arm. These muscles commonly develop TrPs together. The teres major and latissimus dorsi entwine and attach side by side on the humerus.

6. SYMPTOMS

Pain (Section 1) on motion is the chief complaint, particularly while driving a heavy car without power steering. Occasionally shoulder pain occurs on reaching overhead and forward, as in serving a tennis ball. Pain at rest is usually mild. Patients compensate for the slight restriction in the overhead range of arm motion without being aware of it.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

A source of strain that repeatedly has been seen to activate teres major TrPs is driving a heavy car without power steering. Apparently, force exerted from the top of the steering wheel to turn toward the same side is most likely to overload the muscle and to activate its TrPs, especially on the weaker nondominant side. For example, one lady drove a large car without power steering for several years without shoulder trouble until, by error, over-sized steel-belted radial tires were placed on the front wheels. This made the car much harder to steer and the added stress activated left teres major TrPs, which resolved only with return to normal-sized front tires *and* with local injection of the TrPs.

8. PATIENT EXAMINATION

The patient with teres major TrPs has difficulty abducting the involved arm fully and cannot place it tightly against the homolateral ear (***see*** Triceps Test, Fig. 32.4). The Mouth Wrap-around Test (***see*** Fig.

18.2) is restricted by 3-5 cm (an inch or two) when only the teres major muscle is involved. Stretching the muscle by passively flexing and laterally rotating the arm causes pain, as does loading the muscle by resisting active extension and medial rotation of the arm at the glenohumeral joint.[18] Involvement of the teres major does not "freeze" the shoulder or seriously restrict its motion, but it does cause disabling pain near the full range.

When the patient reaches with the arm in a forward position, the scapula may evidence winging that is not apparent when the arm hangs down by the side. The increased tension of the shortened teres major produces this effect and is evidence of the overload imposed on the middle trapezius, rhomboid, and serratus anterior muscles.

The shoulder pain may also arise from dysfunction of the glenohumeral joint or the acromioclavicular joint, which can be identified by testing them for normal joint play.[22]

9. TRIGGER POINT EXAMINATION
(Fig. 25.3)

Although the teres major muscle was not one of the four muscles examined in a study by Gerwin, *et al.*,[9] those authors did include its sister muscle, the latissimus dorsi. For it they reported a high degree of agreement ($P < 0.001$) for detection of a taut band, the presence of spot tenderness, the presence of referred pain, reproduction of the patient's symptomatic pain, and for a local twitch response that is seen or felt at a distance from the point of stimulation. After the teres major has been correctly identified, the difficulty and reliability of determining the presence or absence of TrPs in it by palpation should be comparable to that of the latissimus dorsi muscle.

The TrPs in the axillary portion of the teres major muscle lie slightly cephalad to the most common location for latissimus dorsi TrPs, and they may be palpated by having the patient lie supine with the arm abducted nearly 90% and laterally rotated (Fig. 25.3B). First, the muscle mass of the latissimus dorsi is grasped between the thumb and fingers; this muscle forms the free border of the posterior axillary fold as it wraps around the teres major muscle (see Fig. 24.2). Deep pincer palpation of the axillary fold a few centimeters (about 1 in) below the arm locates the axillary border of the scapula (Fig. 25.3B). Since this location is above the attachment of the teres major to the scapula, a groove is palpable between the edge of the scapula and the teres major muscle. This groove lies just above the point where the teres major extends beyond the scapula and joins the latissimus dorsi (Fig. 25.3A). Axillary TrPs of the teres major are found in the muscle just inferior to the groove. Below this location, at the level of the inferior angle of the scapula, only the latissimus dorsi muscle forms the axillary fold; therefore, it is the only muscle within the pincer grasp when one palpates a groove between the lateral lower edge of the scapula and the axillary fold. At the level of the axillary TrP in the teres major, the axillary fold is formed by both muscles, which are separated by the palpable groove located between them.

The teres major is the deeper (medial) one of the two muscles. When taut bands are present in the teres major, they can be readily located and their local twitch responses felt and seen in all but the most obese patients. To confirm palpation of the teres major muscle, the clinician can instruct the patient to attempt to rotate the arm alternately medially and laterally against light resistance. The teres major tenses during medial rotation effort and relaxes with lateral rotation.

The posterior scapular (medial) trigger area is best examined with the patient lying on the uninvolved side and the uppermost arm resting on a pillow against the chest to ensure relaxation. The teres major is located in the axillary fold as described above, and the operator's fingers then follow the muscle fibers onto the scapula. Examination of the muscle by flat palpation reveals TrPs close to the lateral border of the lower third of the scapula.

10. ENTRAPMENT

No nerve entrapments by this muscle have been observed.

11. DIFFERENTIAL DIAGNOSIS

The symptoms produced by a number of commonly diagnosed causes of shoulder pain can be confusingly similar to those caused by teres major TrPs. These conditions include subacromial or subdeltoid bursitis, supraspinatus tendinitis, C_6-C_7 radiculopathy, and a thoracic outlet syndrome. One must be careful not to overlook one of these conditions, especially when active teres major TrPs are also present. Conversely, it can be a serious mistake in terms of cost and patient misery to ascribe symptoms caused by active TrPs to one of these other diagnoses and overlook the readily treatable TrP cause.

The teres major is one of the quadrad of muscles responsible for the myofascial pseudothoracic outlet syndrome which is described in Chapter 18.

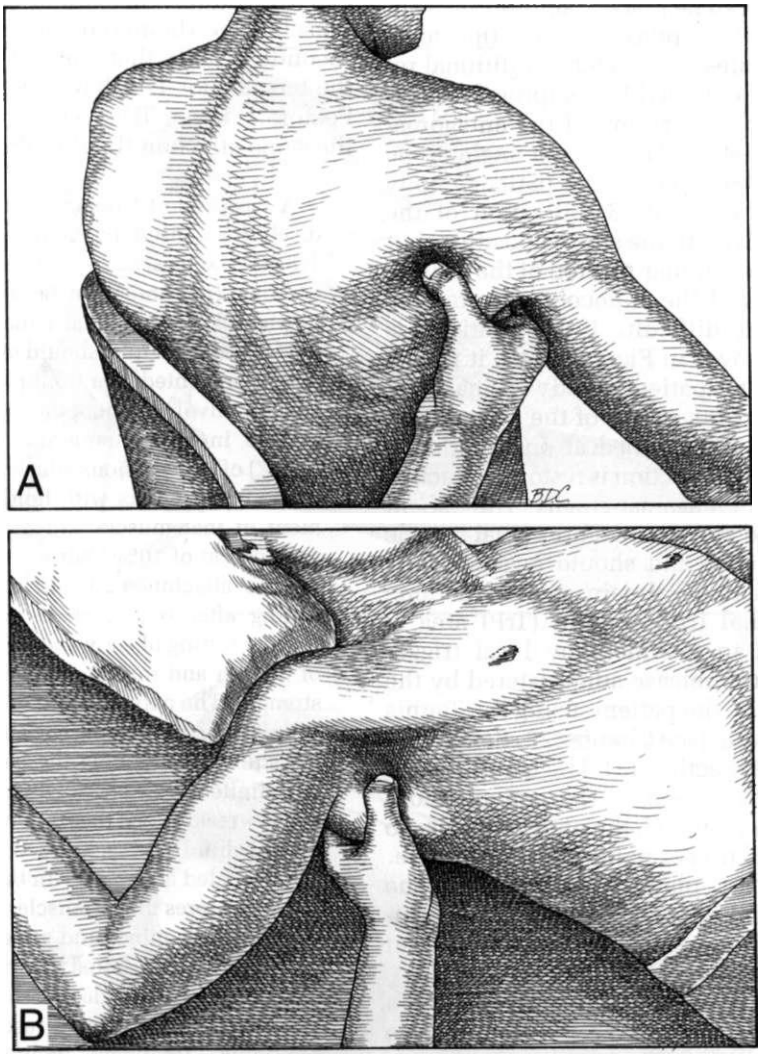


Figure 25.3. Examination of the midmuscle trigger point in the teres major muscle. In the axilla, the examiner's digits must fully encompass the latissimus dorsi muscle to reach the teres major. The groove between the teres major and the latissimus dorsi muscles is confirmed when the examiner's finger tips can identify the lateral border of the scapula between the two muscles. **A,** patient seated; **B,** patient supine.

12. TRIGGER POINT RELEASE
(Fig. 25.4)

The teres major may be released in the supine position (Fig. 25.4A), or with the patient lying partly on the uninvolved side (Fig. 25.4B); the affected arm is placed in abduction with the elbow bent to provide control of lateral rotation. After initial application of vapocoolant or icing, the operator takes up the slack, allowing the arm to move into full lateral rotation and abduction by small increments, until the patient's hand can be placed behind the head.

Postisometric relaxation of the teres major facilitates this stretch. Additional release can be obtained by reciprocal inhibition through contraction of the antagonistic lateral rotators. The inferior angle of the scapula is stabilized by the patient's body weight resting on it. Stabilization of the scapula is easier in the supine position, but reaching the scapular portion of the muscle with sweeps of the vapocoolant spray becomes more difficult. If the patient is turned as shown in Figure 25.4B, it is best to support the patient's body to maintain full muscular relaxation of the patient.

The skin is rewarmed at once with hot packs, and then function is restored by active range of motion against gravity. The patient should avoid strenuous effort of that muscle for several days and should begin a daily home program to maintain range of motion.

The medial trigger point (TrP) area is well suited to treatment by local trigger point pressure release administered by the operator or by the patient, using the Tennis Ball Technique (see Chapter 22, Section 14 or Chapter 3, Section 12). The lateral trigger area is within reach of the patient's contralateral hand, which the patient can use to apply digital trigger point pressure release.

When joint play is restricted in the glenohumeral joint or the acromioclavicular joint, it should be restored to normal function as described by Mennell.[22]

Nielsen[23] presented a case study of a dentist that demonstrated the effective use of spray and stretch to inactivate teres major and associated TrPs.

Related Trigger Points

The latissimus dorsi muscle and the long head of the triceps brachii muscle commonly become involved with the teres major muscle. Eventually, the posterior deltoid, teres minor, and subscapularis also may develop associated TrPs, causing greatly impaired function and much pain in the shoulder region, a condition often diagnosed as "frozen shoulder."

Following successful treatment of teres major TrPs, the patient may now be relieved of interscapular pain that had been due to sustained tension and stretching of the rhomboid muscles caused by the abnormal TrP-induced tension of the teres major muscle. The rhomboids also may develop secondary TrPs that defy treatment until the teres major TrPs have been inactivated. Pectoralis major TrPs very commonly have the same effect on the rhomboid muscles.

A case report[21] demonstrates how the disability caused by active teres major TrPs can be masked by more common and obvious TrPs and must be unmasked. A 68-year-old professional viola player had developed multiple shoulder-girdle TrPs which prevented him from performing in concert. Involvement of the supraspinatus muscle included tenderness ("impingement") of its tendinous attachment, which cleared in 2 weeks with hold-relax treatment of that muscle and phonophoretic application of 10% hydrocortisone to the tendon attachment. Glenohumeral joint testing after treatment revealed normal routine testing of active and passive range of motion and minimal deficit in muscle strength. The patient noted greater ease in activities of daily living such as putting on his shirt and combing his hair, but pain still limited his viola playing, and he was able to reach only the T_{11} level with his hand behind his back. Further examination revealed active TrPs in the latissimus dorsi and teres major muscles but none in the pectoralis minor and serratus anterior muscles. The additional treatment of these two involved muscles with spray and stretch permitted him to resume playing his viola. Treatment included maintenance of strength and mobility of the shoulder-girdle muscular complex.[21] It is unlikely that the teres major TrPs were activated by the viola playing, but their sensitivity to the stretch position required for

that activity seriously interfered with it. While slow stretch with augmentation techniques can release TrPs, stretch-irritation of them with repeated rapid movements can aggravate and perpetuate them.

13. TRIGGER POINT INJECTION (Fig. 25.5)

The medial trigger area in the teres major is injected over the posterior aspect of the scapula as with trigger points (TrPs) in the infraspinatus muscle, but more caudally (Fig. 25.5A).

The midmuscle TrPs are injected with the patient supine and the arm abducted to 90°; they are approached from the inside, or anterior face, of the posterior axillary fold (Fig. 25.5B). The TrPs are identified within the posterior axillary fold and localized between the thumb and fingers, by pincer grasp. Local twitch responses are clearly felt when the needle impales a TrP, and these twitch responses confirm accurate placement of the needle in the TrP.[10] The area is peppered with the needle, since a cluster of TrPs is usually present. It

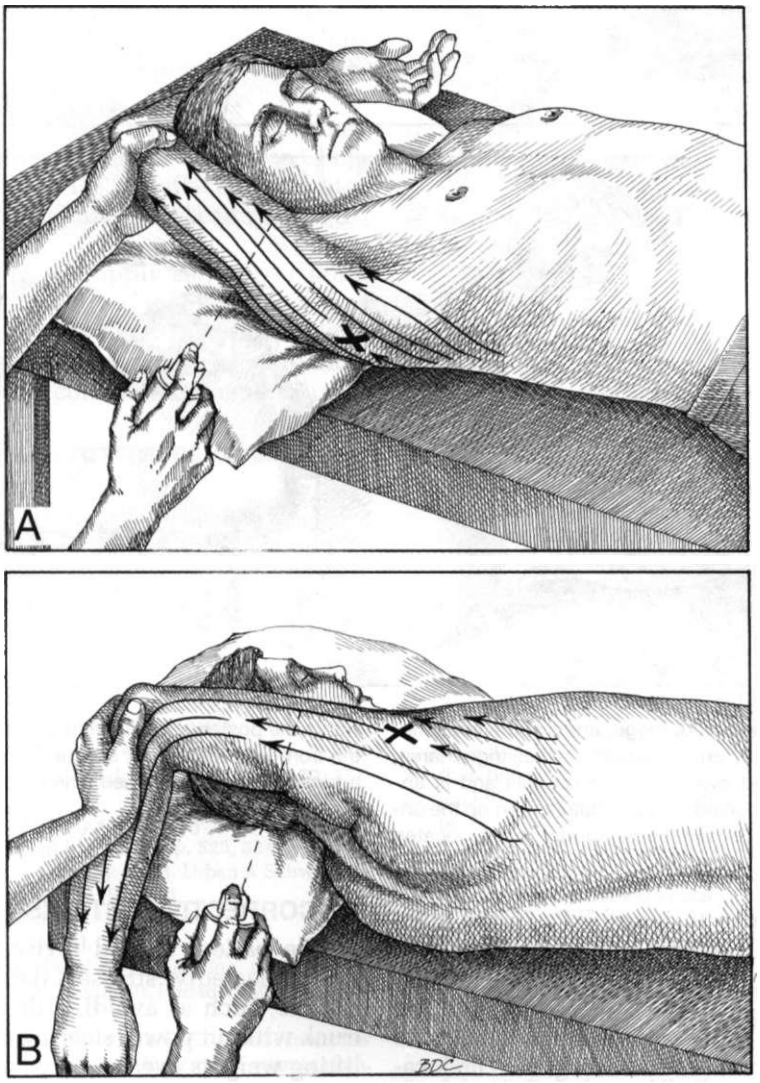


Figure 25.4. Stretch position and spray pattern *(arrows)* for a trigger point **(X)** in the right teres major muscle. **A,** patient supine; **B,** patient semisupine, turned partly on the uninvolved side.

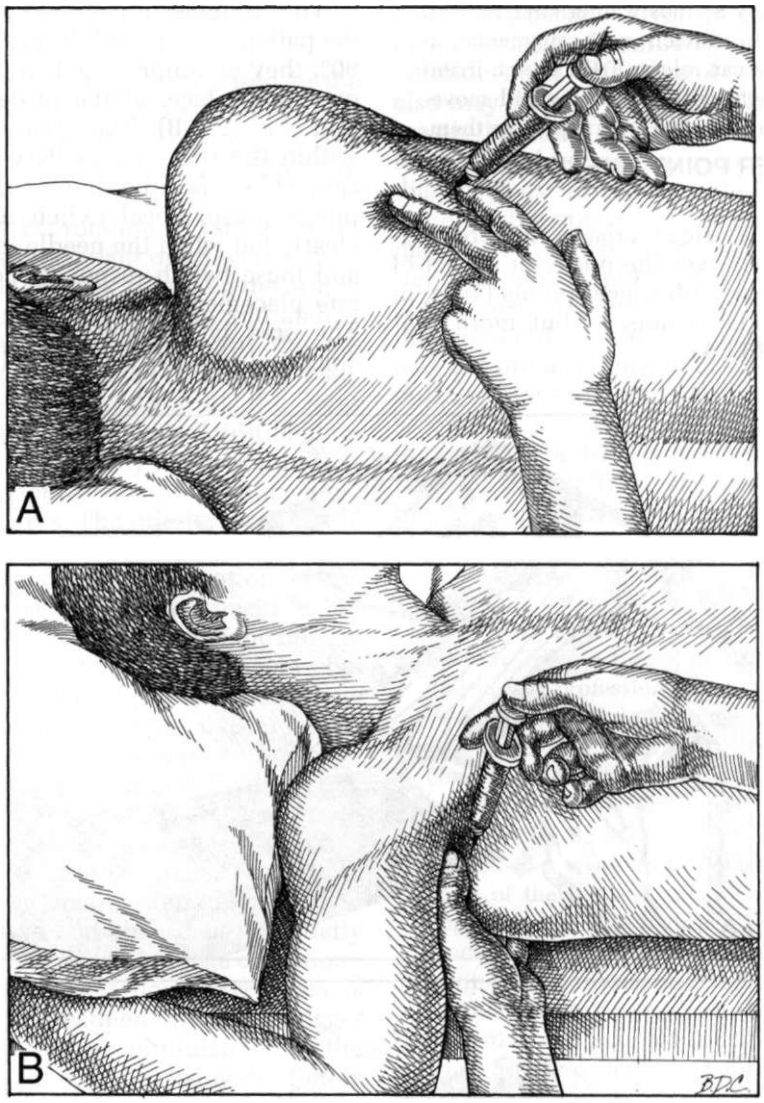


Figure 25.5. Injection of trigger areas in the teres major muscle. **A,** posterior scapular (medial) trigger area, which is located over the lower scapula and is approached from behind with the patient lying on the uninvolved side. **B,** midmuscle trigger point, located within the posterior axillary fold and approached from the front with the patient supine. Effective location of a trigger point is confirmed when the needle elicits a local twitch response.

is possible also to inject TrPs in the adjacent latissimus dorsi through the same skin puncture by sliding the skin and needle laterally.

Rachlin[27] illustrates injection of teres major TrPs in the midfiber region. The location of central TrPs depends on the location of the attachments of the involved fibers as to where the midfiber region will be.

14. CORRECTIVE ACTIONS

The patient should revise any activity that repeatedly stresses the teres major muscle, such as avoiding driving a car or truck without power steering, and avoiding lifting weights overhead.

The patient learns to stretch the muscle gently but firmly, first by placing the painful arm behind the head (start of Mouth

Wrap-around Test), and then by holding the arm with the other hand to release the teres major by using the contract-relax technique. Additional release may be obtained by using reciprocal inhibition through self-resisted contraction of the antagonistic lateral rotators. The patient should do this while seated under a warm shower, with the water beating on the skin overlying the region of the teres major muscle.

To prevent full shortening of this muscle while sleeping on the affected side, a small pillow is placed between the elbow and the lateral aspect of the trunk to maintain a neutral position of the muscle (see Fig. 26.7). A pillow support can be used for this purpose also when sleeping on the uninvolved side (see Fig. 22.6A).

Self-stretch of this muscle is performed like that of the latissimus dorsi (see Chapter 24); however, for full effectiveness, it is important to stabilize the scapula against abduction.

The patient can apply self-trigger point pressure release to the medial trigger area using a tennis ball under the body weight, and to the lateral trigger area using manual pressure by the contralateral hand.

SUPPLEMENTAL REFERENCE, CASE REPORT

Rinzler and Travell reported management of a patient with teres major TrPs.[29]

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 376, Fig. 6-26).
2. *Ibid.* (pp. 386, 387; Figs. 6-40, 6-41).
3. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 270, 271, 385).
4. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 523, 524).
5. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 23).
6. *Ibid.* (Figs. 49, 53).
7. *Ibid.* (Figs. 523, 524).
8. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 81-83).
9. Gerwin RD, Shannon S, Hong CZ, *et al:* Interrater reliability in myofascial trigger point examination. Pain 69:65-73, 1997.
10. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Behabil* 73:256-263, 1994.
11. Inman VT, Saunders JB, Abbott LC: Observations on the function of the shoulder joint, *J Bone Joint Surg* 26:1-30, 1944 (pp. 24-26, Fig. 30).
12. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 85).
13. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving. Part V. The supraspinatus, infraspinatus, teres minor and teres major muscles. *Ergonomics* 39.711-717, 1976.
14. Jonsson B, Olofsson BM, Steffner LC: Function of the teres major, latissimus dorsi and pectoralis major muscles: a preliminary study. *Acta Morpol Neerl-Scand* 9:275-280, 1972.
15. Kelly M: Some rules for the employment of local analgaesics in the treatment of somatic pain. *Med J Aust* 3:235-239, 1947 (p. 236).
16. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 276, 294).
17. Lundervold AJ: Electromyographic investigations of position and manner of working in typewriting. *Acta Physiol Scand* 24:Suppl 84, 1951. (pp. 66-68, 80-81, 94-95, 101, 157).
18. Macdonald AJ: Abnormally tender muscle regions and associated painful movements. *Pain* 8:197-205, 1980.
19. McMinn RM, Hutchings RT, Pegington J, *et al: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (pp. 119, 120).
20. *Ibid.* (p. 126).
21. Meador R: The treatment of shoulder pain and dysfunction in a professional viola player: implications of the latissimus dorsi and teres major muscles. *J Orthop Sport Phys Ther ll(2):52-55,* 1989.
22. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
23. Nielsen AJ: Case study: myofascial pain of the posterior shoulder relieved by spray and stretch. *J Orthop Sport Phys Ther* 3:21-26, 1981.
24. Pearl ML, Perry J, Torburn L, *et al:* An electromyographic analysis of the shoulder during cones and planes of arm motion. *Clin Orthop* 284:116-127,1992.
25. Pernkopf E: Arias *of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 28).
26. *Ibid.* (Figs. 44, 57).
27. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, (pp. 200-202).
28. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (p. 167).
29. Rinzler SH, Travell J: Therapy directed at the somatic component of cardiac pain. *Am Heart J* 35:248-268, 1948 (pp. 261-263, Case 3).
30. Sola AE, Kuitert JH: Myofascial trigger point pain in the neck and shoulder girdle. *Northwest Med* 54:980-984, 1955.
31. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med* 34:585-590, 1955.

CHAPTER 26
Subscapularis Muscle

HIGHLIGHTS: Subscapularis trigger points (TrPs) are often the key to a "frozen shoulder" syndrome. **REFERRED PAIN** from TrPs in the subscapularis muscle concentrates in the posterior deltoid area and may extend medially over the scapula, down the posterior aspect of the arm, and then skip to a band around the wrist. This referred pain produces a distinctive, easily recognized pattern. **ANATOMY:** medially, the subscapularis muscle attaches to the inner surface of the scapula and, laterally, to the lesser tubercle on the anterior aspect of the humerus. **FUNCTION** of the subscapularis is chiefly to help secure the head of the humerus in the glenoid fossa during arm movements, particularly abduction. It is active in medial rotation and adduction of the arm at the shoulder joint. The **SYMPTOMS** caused by subscapularis TrPs are primarily posterior shoulder pain with progressive painful restriction of abduction and lateral rotation of the arm. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in this muscle are often caused by chronic muscular strain or sudden trauma to the shoulder. **PATIENT EXAMINATION** identifies involvement of this muscle by a marked reciprocal limitation of either abduction or lateral rotation of the arm at the glenohumeral joint and an even greater restriction of the combined movement. The humeral attachment of the muscle is often tender to palpation. **TRIGGER POINT EXAMINATION** is exacting in its technique, but rewarding. Abduction of the scapula is necessary to reach many of the TrPs in this muscle. **DIFFERENTIAL DIAGNOSIS** of subscapularis TrPs includes C_7 radiculopathy, thoracic outlet syndrome, adhesive capsulitis, and "impingement" syndrome. The pain and restricted range of motion of a "frozen shoulder" and of the shoulder of a patient with hemiplegia are frequently caused by subscapularis TrPs that have been overlooked. **TRIGGER POINT RELEASE** of this muscle requires that the patient's arm be gradually abducted and laterally rotated while the vapocoolant or ice is applied over the lateral chest wall, over the scapula, and over the pain pattern on the back of the arm and wrist. **TRIGGER POINT INJECTION** requires identification of the TrPs for injection by palpation of the subscapularis fibers against the scapula and requires a longer needle than usual. With proper positioning of the patient, careful technique, and follow-up stretch, injection of the TrPs is safe and effective. The presence of spasticity and TrPs in patients with hemiplegia deserves special consideration. **CORRECTIVE ACTIONS** include avoidance of prolonged shortening of the muscle both at night and during the daytime, avoidance of a "slumped" forward posture, and regular use of the In-doorway Stretch Exercise at home.

1. REFERRED PAIN
(Fig. 26.1)

Subscapularis trigger points (TrPs) cause severe pain both at rest and on motion of the upper limb. The essential zone of the referred pain pattern lies over the posterior aspect of the shoulder (Fig. 26.1). Spillover reference zones cover the scapula and extend down the posterior aspect of the arm to the elbow. A diagnostically useful accent, when present, is a strap-like area of referred pain and tenderness around the wrist.[55,61] The dorsum of the wrist is usually more painful and tender than the volar surface.

2. ANATOMY
(Fig. 26.2)

The connection of the subscapularis to the humerus is the most anterior attachment of the four muscles that form the rotator cuff; the others are the supraspinatus, infraspinatus and teres minor muscles.[4]

Medially the subscapularis attaches to most of the inner (anterior) surface of the scapula, filling the subscapular fossa from the vertebral to the axillary border of the scapula (Fig. 26.2). ***Laterally*** it passes across the front of the shoulder joint *via* a tendon that attaches to the lesser tubercle on the anterior (ventral) aspect of the humerus and to the lower half of the capsule of the shoulder joint, blending with the capsule.[14] The location of this attachment to the humerus in relation to the attachment of other shoulder-girdle muscles is illustrated in this volume (see Fig. 29.4) and elsewhere.[17] The large subscapular bursa, which usually communicates with the cavity of the shoulder joint, separates the tendon of the subscapularis muscle and the underlying joint capsule medially.[13]

Supplemental References

Other authors illustrate the subscapularis muscle as seen from the front, but partially covered by overlying structures.[2,16,18,34] from the front with an unobstructed view,[52,54] from below,[1] from the side,[53] and in cross section.[3,43]

3. INNERVATION

The muscle is innervated by the superior and inferior subscapular nerves, through the posterior cord of the brachial plexus from spinal nerves C_5 and C_6.[15,26,29] The superior subscapular nerves (usually two of them) enter the more horizontal, superior part of the subscapularis muscle. The inferior subscapular nerve enters the more distal part of the subscapularis muscle and ends in the teres major muscle. This innervation pattern suggests that the subscapularis muscle is composed of at least two compartments, each of which would have its individual endplate zone-an important point when performing motor point blocks[24] or injecting TrPs.

4. FUNCTION

The subscapularis muscle adds to stability of the glenohumeral joint by helping to maintain the head of the humerus in the glenoid fossa. It helps to prevent anterior displacement of the humerus.

Acting alone, the subscapularis medially rotates and adducts the arm,[7,29] and helps to hold the head of the humerus in the glenoid fossa.[7,14] Because the deltoid muscle attaches to the proximal portion of the humerus, during abduction the vertical vector tends to pull the head of the humerus upward out of the glenoid fossa and against the acromion. During abduction, the depressor action of the subscapularis contributes a major force to counteract this upward displacement caused by the deltoid.[25] This stabilizing function of the subscapularis was substantiated by electromyographic (EMG) activity of the subscapularis that increases during abduction from 0° to 90°, plateaus from 90° to 130°, and rapidly diminishes from there to 180° as the deltoid no longer exerts an upward displacement force.[25] The subscapularis is active in forward swing of the arm during walking.[4]

Electrical stimulation of the subscapularis elicits strong medial rotation of the arm at the shoulder.[19] When a strongly shortened subscapularis muscle maintains medial rotation of the arm, it is not possible to fully supinate the hand of the outstretched upper limb because of the restricted lateral rotation at the shoulder.[19] In this way, subscapularis TrPs can indirectly impair function at the hand.

Although the records of 12 subjects throughout a right dominant **golf swing** were highly variable, the mean EMG activity of the right subscapularis muscle began at takeaway with only 15% of the maximum activity elicited by manual muscle strength testing. The activity increased to 65% during acceleration, and subsided slightly thereafter. The left subscapularis muscle maintained a moderate amount of activity during the swing, ranging around 30% of the maximum test activity.[44]

A similar study of men and women professional golfers[27] reported a very similar pattern bilaterally for women golfers; however, the male subjects showed activity on the right side that started with mean takeaway activity at only 12% of maximum test activity, increased to 80% by the time of the acceleration phase, and maintained that level of EMG activity throughout the remainder of the swing. The left subscapularis muscle in men, like both sides in women, maintained a mean of approximately 45% throughout all 5 phases of the golf swing.[27]

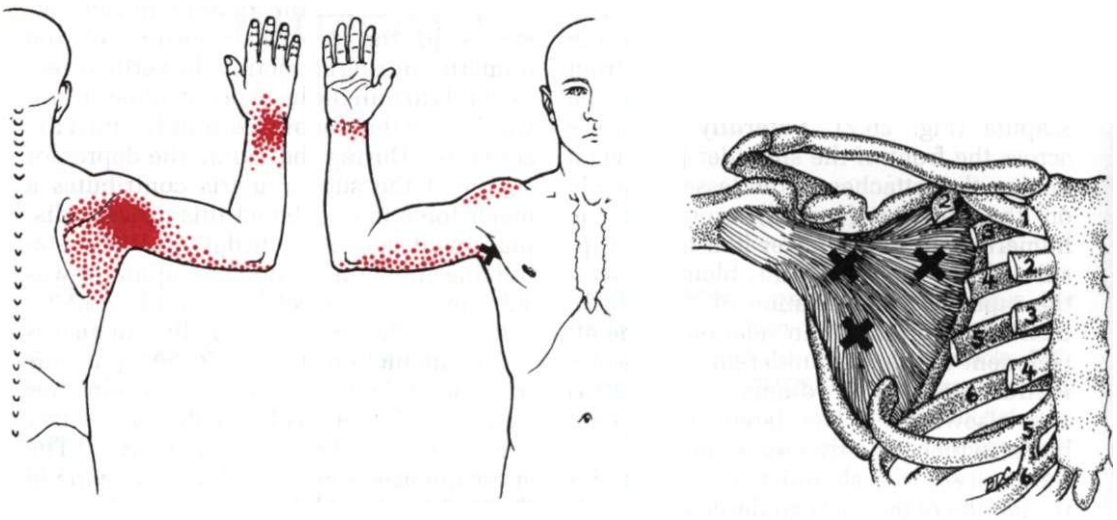


Figure 26.1. Referred pain pattern projected from two lateral trigger points and a more medial trigger area (**X**s) in the right subscapularis muscle. The essential referred pain zone is *solid red;* the spillover zone is *stippled red.* Portions of the second through the fifth ribs have been removed for clarity.

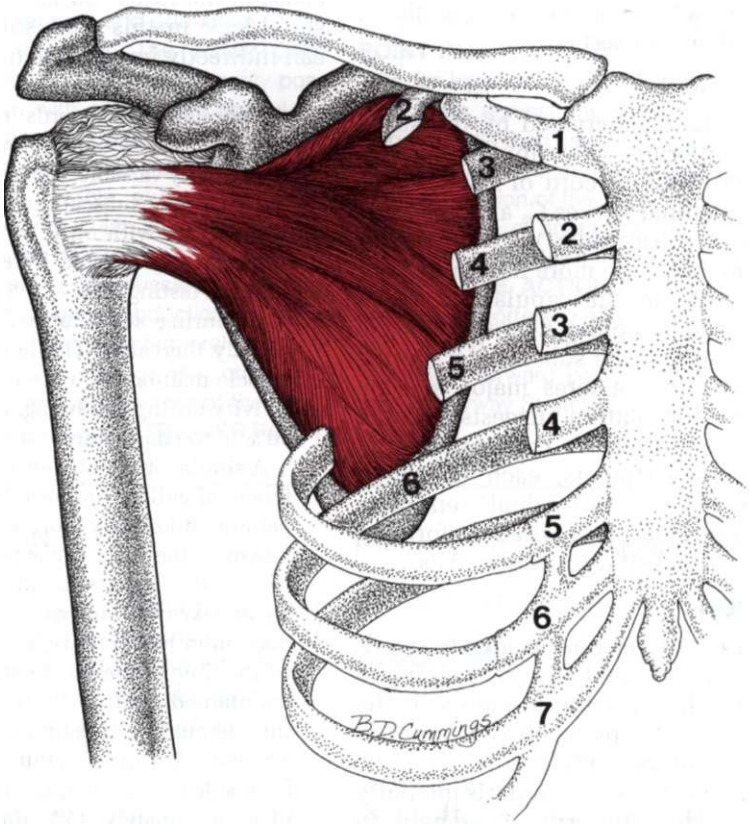


Figure 26.2. Attachments of the right subscapularis muscle, as seen from the front with the arm laterally rotated. Parts of ribs two through five have been removed for clarity.

Fine wire EMG recordings of qualitative subscapularis muscle activity during freestyle swimming in 14 subjects, with a painful shoulder,[51] were compared with a previous study of 12 pain-free shoulders. Subjects with painful shoulders showed a pattern similar to normal subjects until the recovery phase, when average activity was only half that of normal values. In this phase, the subscapularis puts the shoulder in the painful position of medial rotation (which is when strong contraction of that muscle would be painful if it had active TrPs). The swimmer may be avoiding that pain.

The EMG activity of the subscapularis muscle in 15 skilled throwers with shoulder girdle symptoms and chronic anterior instability of the shoulder was compared to that of 12 healthy, skilled throwers.[22] In healthy subjects, during wind up, the subscapularis muscle exhibited only 5% of the amount of EMG activity recorded during manual muscle testing, but during late cocking it reached a mean value of 147% of the test value. Subscapularis activity increased to 185% of test value during acceleration, and still averaged 97% during follow-through. The athletes with painful shoulders started out with normal values, but reached only one-third of normal values during late cocking and half of normal values during acceleration and follow-through. The authors considered this marked difference in neuromuscular control to be a factor in producing or maintaining chronic anterior instability. However, they offered no satisfactory explanation for the marked inhibition of subscapularis activity and apparently had not considered treatable myofascial TrPs as a possible major contributor to the problem.

These studies illustrate an important principle: The EMG activation of a muscle can be remarkably different under test conditions compared to meaningful, well-learned activity. This effect can be very strong in a muscle inhibited reflexly by active TrPs in a functionally related muscle.[23]

5. FUNCTIONAL UNIT

The teres major most nearly matches the functions of the subscapularis muscle and is strongly synergistic with it. Both the latissimus dorsi and pectoralis major also adduct and medially rotate the arm, and thus can act synergistically with the subscapularis, but these muscles attach to the trunk rather than to the scapula.

The arm-rotation function of the subscapularis is opposed primarily by the infraspinatus and teres minor muscles. However, these three muscles work together to hold the head of the humerus in the glenoid fossa during elevation movements of the arm.

6. SYMPTOMS

In the early stage of myofascial involvement of the subscapularis, patients can reach up and forward, but are unable to reach backward with the arm held at shoulder level, as when starting to throw a ball. With progression of TrP activity, abduction at the shoulder becomes severely restricted to 45° or less. These patients complain of pain both at rest and on motion, and of inability to reach across to the opposite armpit. The patient has often been told that he or she has a "frozen shoulder," adhesive capsulitis or "pitcher's arm." When asked about the wrist, the patient often says that it is sore and painful in a strap-like area, especially on the dorsum. Because of this referred tenderness, the patient may move the wristwatch to the opposite wrist.

Active TrPs are a major source of the pain and limited shoulder motion, especially abduction and lateral rotation, in patients with hemiplegia. The TrP shortening also contributes to subluxation of the head of the humerus.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Subscapularis TrPs are **activated** in the following ways:

1. By unusual repetitive exertion requiring forceful medial rotation when the subject is out of condition, as in the overhead stroke of the crawl during swimming, or pitching a baseball
2. Due to repeated forceful overhead lifting while exerting strong adduction, as when swinging a small child back and forth, from between an adult's legs, up overhead, and down again

3. By the sudden stress overload of reaching back at the shoulder level to arrest a fall
4. When the muscles are stressed by dislocation of the shoulder joint
5. At the time of fracture of the proximal humerus, or tear of the shoulder joint capsule
6. By prolonged immobilization of the shoulder joint in the adducted and medially rotated position.

These TrPs are **perpetuated** by repetitive movements requiring medial rotation of the humerus. A "slumped" forward-head, abducted-scapulae posture can perpetuate these TrPs by fostering sustained medial rotation of the humerus. Refer to Chapter 4 of this volume for specific systemic and mechanical perpetuating factors.

8. PATIENT EXAMINATION

Lateral rotation is a prerequisite for full elevation (abduction and flexion) of the humerus; TrPs in the subscapularis muscle restrict lateral rotation.

When examining a shoulder with restricted abduction, one of the first questions that should be answered is the freedom of scapular mobility as distinguished from glenohumeral movement. This difference can be detected by placing the hand on the scapula to note its movement as the arm is abducted. Involvement of only the subscapularis muscle restricts glenohumeral movement, but does not restrict scapular movement on the chest. Restriction also of scapular mobility makes one think of additional TrPs in the pectoralis minor, serratus anterior, trapezius, and rhomboid muscles.

When only the subscapularis muscle is shortened and taut, abduction and lateral rotation at the shoulder are reciprocally limited; one movement can be traded for the other, which is easily demonstrated. If the patient has moderately active subscapularis TrPs, abduction of the arm at the glenohumeral joint is limited to about 90°, and when the forearm hangs down, the shortened muscle tends to medially rotate the arm. No lateral rotation of the arm at the shoulder joint is possible in the abducted position. However, with the arm adducted by placing the elbow at the side and with the elbow bent at 90° to show shoulder joint rotation, the forearm can swing outward to nearly 90° of lateral rotation of the arm. The arm is medially rotated when the hand touches the abdomen and performs 90° of lateral rotation of the arm at the glenohumeral joint when the hand points laterally away from the body. Involvement of the teres major, anterior deltoid, and lower fibers of the pectoralis major also can produce some of this limitation of lateral rotation, but not as severely or consistently as the subscapularis. A lesser degree of subscapularis involvement can be detected if the muscle refers pain in its characteristic pattern to the back when the arm is fully flexed in lateral rotation at the shoulder. This referred pain from subscapularis TrPs may be encountered when the arm is placed in this position to stretch and spray the long head of the triceps.[47]

The humeral attachment of the subscapularis (Fig. 26.2) is often very tender to palpation due to secondary enthesopathy when there is chronic TrP involvement of the muscle. To examine this attachment, the arm is placed by the side and laterally rotated as the patient tries to bring the elbow behind the plane of the back. This rotates the humeral attachment to the front of the shoulder, where it can be more readily palpated (Fig. 26.2).

To eliminate articular dysfunction as a contributing cause of the patient's pain, the glenohumeral and acromioclavicular joints should be examined for restriction of normal joint play,[36] and also the wrist articulations, if the patient's pain includes that region. If this type of movement restriction is present, it should be released. Unrestricted range of motion of the arm also requires normal mobility of the sternoclavicular joint.

9. TRIGGER POINT EXAMINATION (Figs. 26.3 and 26.4)

To determine the most useful diagnostic criteria, Gerwin, *et al.*[21] tested the reliability with which four experienced and trained examiners could identify five characteristics of TrPs. The four consistently reliable characteristics were the presence of a taut band, the presence of spot tenderness, the presence of referred pain that is felt at a distance from the point of stimulation, and reproduction of the subject's symptomatic pain. Determination of the presence or absence of a local twitch response (LTR), although very helpful diagnostically when

observed, was reliably identified only in the most accessible and readily palpated muscles. The subscapularis is one of the more difficult muscles to examine reliably for LTRs.

There are two common lateral TrP locations and a medial trigger area in the subscapularis muscle (Fig. 26.1) The most accessible lateral TrPs are found in the relatively vertical fibers which lie inside the lateral border of the scapula on the ventral aspect. Lange[31] identified only this more accessible site. The other lateral TrP region lies superior to the first and is more difficult to reach. It lies in the nearly horizontal bundle of fibers that extend across the scapula (Fig. 26.2). The third location is the trigger area along the vertebral border of the scapula where the subscapularis muscle attaches to the vertebral half of the inner (ventral) surface of that bone. A tender spot that refers pain from this part of the muscle may represent enthesopathy secondary to primary midfiber TrPs.

When the patient has become fully relaxed, the examiner first abducts the arm of the supine patient away from the chest wall to the onset of tissue resistance, to 90° if possible. Patients with marked shortening of the subscapularis muscle due to very active TrPs may not tolerate abduction of the arm beyond 20° or 30°. Figure 26.3 shows the relationship of the subscapularis muscle to the scapula, the latissimus dorsi, teres major and to other adjacent muscles. If the arm cannot be abducted sufficiently for examination, sufficient release of the subscapularis may be achieved by using the hold-relax or the contract-relax technique (see Chapter 3, Section 12). Adequate abduction (lateral displacement) of the scapula is necessary to bring the ventral (inner) surface of the scapula and its subscapularis muscle within reach for palpation.

Next, the examiner grasps the latissimus dorsi and teres major muscles (Fig. 26.3) in a pincer grip (Fig. 26.4A and B) and locates the hard edge of the scapula with the tips of the digits. Traction must be maintained on the humerus to abduct the scapula adequately (*arrow* in Fig. 26.4B shows direction of pull). The phantom finger *"C"* in Figure 26.4B locates the same portion of the subscapularis as is being palpated in Figure 26.4C, illustrating the increased accessibility of the subscapularis by abducting the scapula.

To reach the TrPs frequently located along and superior to the lateral margin of the muscle, the palpating finger slides into the space between the serratus anterior, which lies against the chest wall along the back of the finger, and the subscapularis muscle, beneath the finger on the underside of the scapula (Fig. 26.3). To reach the superior TrP area, the finger is directed cephalad and toward the coracoid process of the scapula to locate a large firm band of muscle fibers in the TrP area. Sustained, light-to-moderate pressure on an active subscapularis TrP will reproduce the patient's posterior shoulder and scapular pain, occasionally with a referred twinge in the wrist. Local twitch responses are sometimes seen. When detected, LTRs are more likely to be felt with the palpating finger than seen, and they are strongly confirmatory of (but not essential for) a TrP diagnosis in this muscle.

In thin supple patients, more direct control of the scapula is obtained if the examiner hooks the fingers of the nonpalpating hand directly around the vertebral border of the scapula and pulls the scapula laterally, away from the midline of the body.

In patients with severe subscapularis involvement, deep tenderness in the muscle is usually so exquisite that the patients can tolerate only very light digital pressure on the muscle. Normal subscapularis muscles palpated in this way are not tender. However, an inadequately trimmed fingernail on the palpating finger will cause confusing severe skin pain. The skin should show no fingernail marks following palpation.

Palpation for tenderness in the subscapularis trigger area of enthesopathy on the ventral aspect of the vertebral border of the scapula is complicated by two facts. For most examiners and subjects it is unreachable anterior to the scapula when approached from the lateral border of that bone. It is also very unlikely that one can palpate the subscapularis muscle along the vertebral border of the scapula. The examiner must palpate through a relatively thick trapezius muscle, the rhomboid muscle layer, and the serratus anterior muscle, which all attach along that border of the scapula and are also subject to enthesopa-

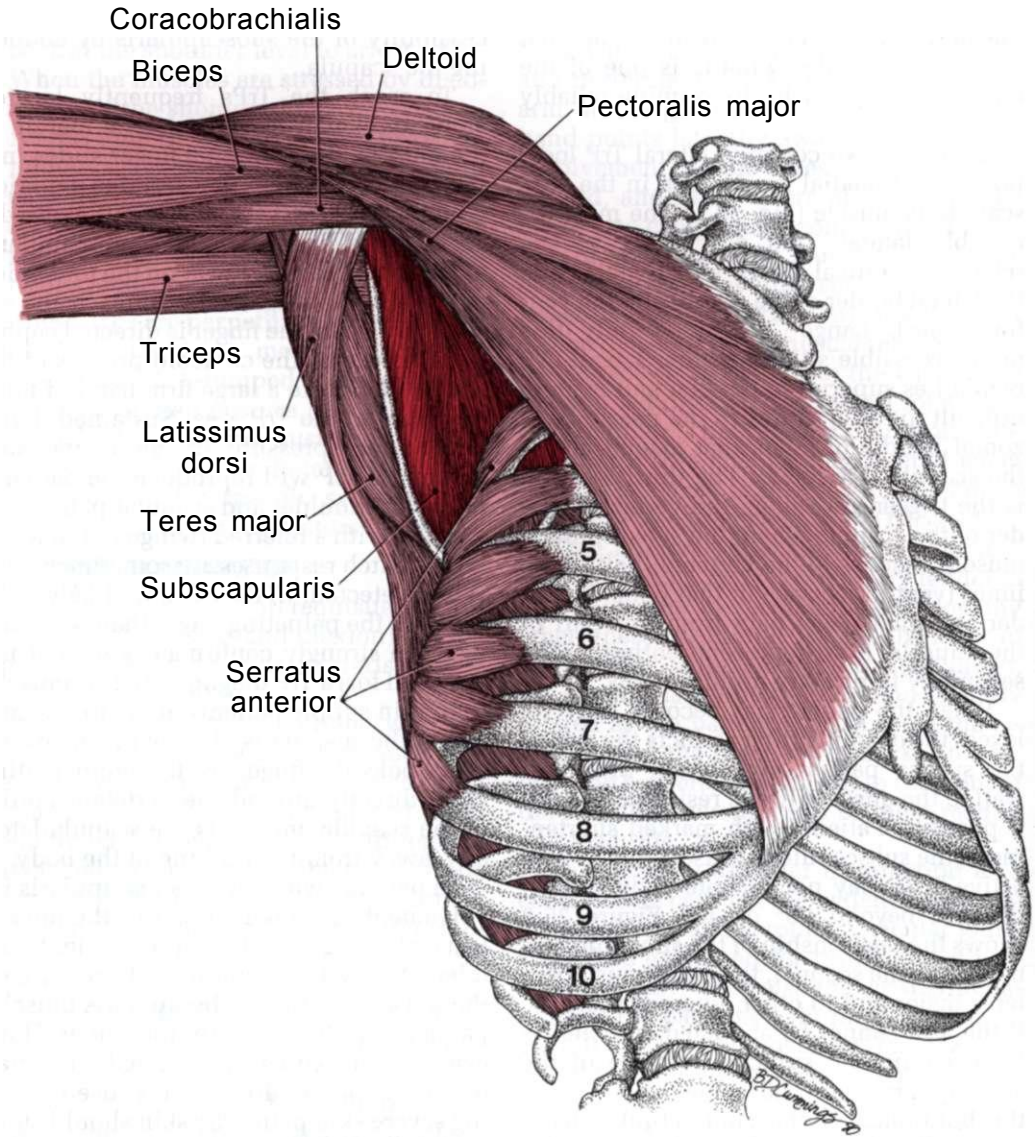


Figure 26.3. Relation of the subscapularis muscle *(dark red)* to the surrounding muscles *(lighter red)* when the scapula (shown as a vertical white line) has been pulled away from the chest wall by the examiner (compare with Fig. 26.2).

thy. In this region, tenderness to palpation alone does not identify which muscle is responsible for it.

Related Trigger Points

When there is moderate TrP involvement of the subscapularis muscle, the patient's arm movement may be restricted by this muscle alone without associated TrP activity in other shoulder-girdle muscles. When the subscapularis TrPs become sufficiently active, the pain-induced restriction of motion at the shoulder joint becomes severe. Then, functionally related muscles quickly become involved (Section 5), so that many, or most, of these muscles develop active TrPs. Motion at the shoulder is then "frozen." Autonomic trophic changes are likely to follow.

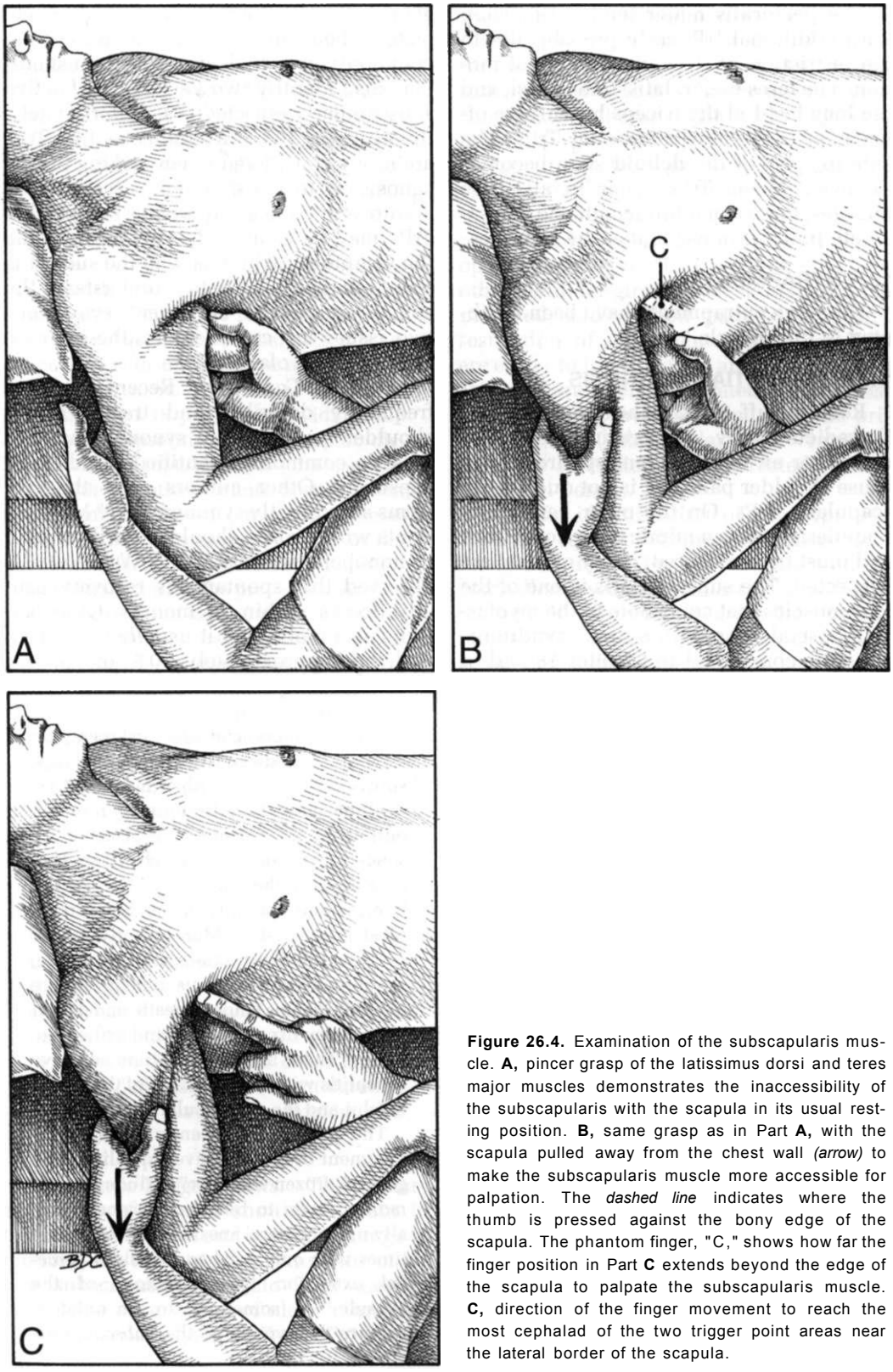


Figure 26.4. Examination of the subscapularis muscle. **A,** pincer grasp of the latissimus dorsi and teres major muscles demonstrates the inaccessibility of the subscapularis with the scapula in its usual resting position. **B,** same grasp as in Part **A,** with the scapula pulled away from the chest wall *(arrow)* to make the subscapularis muscle more accessible for palpation. The *dashed line* indicates where the thumb is pressed against the bony edge of the scapula. The phantom finger, "C," shows how far the finger position in Part **C** extends beyond the edge of the scapula to palpate the subscapularis muscle. **C,** direction of the finger movement to reach the most cephalad of the two trigger point areas near the lateral border of the scapula.

The **pectoralis major** tends to develop these additional TrPs early, probably due to the restriction of its normal range of motion. The **teres major, latissimus dorsi,** and the **long head of the triceps brachii** are often next to develop additional TrPs. The anterior part of the **deltoid** soon becomes involved. When TrPs occur in all these muscles, none reach full length and can severely limit all movement at the shoulder.

10. ENTRAPMENT

No nerve entrapments have been attributed to this muscle.

11. DIFFERENTIAL DIAGNOSIS

Rotator cuff tears, adhesive capsulitis, C_7 radiculopathy, a thoracic outlet syndrome, or an impingement syndrome can cause shoulder pain that is not due to subscapularis TrPs. On the other hand, subscapularis TrPs can mimic these conditions and must be considered, if any of them are suspected. The subscapularis is one of the four muscles that contribute to the myofascial pseudothoracic outlet syndrome, which is considered in Chapter 18 and in Chapter 20, Section 11. The major contribution of subscapularis TrPs to the pain and restricted range of motion of the shoulder in hemiplegia is often overlooked and is discussed in this section. The "frozen shoulder" is covered in detail below.

"Frozen Shoulder"

The descriptive term "frozen shoulder" is not a specific diagnosis and frequently is based only on the presence of a painful shoulder that exhibits restricted range of motion. The term has been identified with several categories of disease: neurologic (hemiplegia,[49] shoulder-hand syndrome[57]), idiopathic (an idiopathic capsulitis[20,57,59]), rheumatologic (periarthritis or periarticular arthritis,[10,30] acromioclavicular arthritis[5,38]), and adhesive capsulitis, which has characteristic objective findings[5,49] of unknown etiology.[57] The label "frozen shoulder," when presented as the diagnosis that accounts for the patient's symptoms, serves as a warning that the patient is in need of a more specific diagnosis.

When so many authors agree that the cause of a disease is enigmatic, there is good reason to expect that a major etiologic factor is being overlooked. The two criteria commonly used to diagnose "frozen shoulder" also identify two key effects of active subscapularis muscle TrPs. Unfortunately, the available literature indicates that TrPs are rarely considered when making the diagnosis of "frozen shoulder." The same literature and clinical experience suggest that TrPs may be a major factor in producing the symptoms, which is why the subject is reviewed here. To better understand the source of "frozen shoulder" symptoms, two issues are considered: adhesive capsulitis and myofascial TrPs.

Adhesive Capsulitis. Recent literature frequently describes and treats "frozen shoulder" as if it were synonymous with what is commonly identified as adhesive capsulitis. Other authors treat the two terms as explicitly synonymous.[49] Most patients with "frozen shoulder" will respond to nonoperative treatment.[45] Weber *et al.*[60] observed that spontaneous recovery usually occurs within 30 months. Other authors also found that it *usually* is self-limited, but approximately 10% of patients had long-term problems.[40]

Some authors consider arthrographic findings to be diagnostic of adhesive capsulitis.[33, 37, 38] The arthrogram contrast medium shows that the normally rounded outline of the capsule is replaced by a squat, square contracted patch. The redundant fold at the inferior portion of the joint, which normally hangs down like a pleat, is obliterated.[5] More recently, Rizk *et al.*[49] identified restrictions of joint volume, serration of the bursal attachments, failure to fill the biceps tendon sheath, and partial obliteration of subscapular and axillary recesses. These findings associate adhesive capsulitis with the long head of the biceps brachii and the subscapularis muscles.

The procedures recently reported for treatment of the adhesive capsulitis category of "frozen shoulder" include *forceful manipulation* to release adhesions (usually under general anesthesia,[35,45,60] sometimes with only local anesthesia[20]), *forceful extension* (pressurization) of the capsule,[20,40] (sometimes to the point of rupture[49]), division of the *subscapularis*

tendon,[40] ***resection of inflammatory synovium*** between the supraspinatus and subscapularis attachments,[40] excision of the ***coracohumeral ligament,***[9] arthroscopic excision of the ***rotator interval*** of the capsule[9] or release of the ***anterior capsule.***[59] The reports by these authors indicate that, to them, the source of the irritation that caused the adhesions being treated remains enigmatic.

Many of the above-listed procedures implicitly or explicitly identify the bursae and or tendons of the supraspinatus and subscapularis muscles as being closely associated with the adhesive restriction of joint movement. Rizk, ***et al.***[49] reported treatment of 16 patients with idiopathic adhesive capsulitis by arthrographic distention and rupture of the joint capsule. Only those patients whose posttreatment arthrograms showed iatrogenic capsular tears at the subscapular bursa or at the subacromial bursa experienced sudden relief of pain during the procedure. Among the 3 patients not experiencing sudden relief of pain, two had a distal bicipital sheath rupture, and one had subscapular rupture. The coracohumeral ligament also has a muscular relationship because it attaches to the rotator cuff in conjunction with the supraspinatus tendon.[13]

When restriction persists after the inactivation of TrPs in muscles that could be responsible for the restricted range of motion, or if there is arthrographic evidence of adhesive capsulitis, the antifibrotic medication, Potaba® (aminobenzoate potassium-a member of the B vitamin complex) manufactured by Glenwood, Inc. may be administered. Its effectiveness is related to adequate dosage (12 g/day taken as 0.5 g capsules or tablets in divided doses either four or six times daily) and to sufficient duration of medication (usually a minimum of 3 months).

Relation to Trigger Points. The primary symptoms of "frozen shoulder"—pain in the shoulder region and restricted range of motion—are also primary symptoms of active **subscapularis muscle** TrPs. Lewit[32] voiced the observation of many clinicians skilled at identifying TrPs that "painful spasm of the subscapularis, with trigger points, accompanies frozen shoulder from the outset." The "frozen shoulder" literature often refers to the importance of trying conservative therapy first and frequently identifies physical therapy or physical therapeutic techniques as an essential part of that conservative therapy.[35,45,57,59]

The reason the shoulder becomes so painful and "frozen" when a patient develops subscapularis TrPs is that so many other shoulder-girdle muscles also become involved, adding their pain patterns and restriction of movement. The other TrPs are easier to identify than are subscapularis TrPs and are often inactivated with at least temporary improvement; but until the primary cause (subscapularis TrP involvement) is identified and corrected, symptoms will persist.

Specific identification of subscapularis TrPs as a focus of therapeutic attention is rarely mentioned in the literature, and no controlled research studies could be found that specifically addressed the TrP component of "frozen shoulder." Many clinicians agree that subscapularis TrPs can be responsible for the symptoms of "frozen shoulder" and can be simply and effectively treated.[8,32] However, in the current climate of managed health care, clinical success is not sufficient; competent research substantiation is essential.

In addition, it is quite likely that TrPs in the **supraspinatus** or **subscapularis** muscles can be a major factor in the development of adhesive capsulitis. The supraspinatus muscle, as noted in Chapter 21, is prone to develop enthesopathy or enthesitis. Since the humeral tendinous attachment region of the subscapularis is not so accessible to direct palpation, its tendency to develop enthesitis is not so well recognized. The humeral attachment of the subscapularis tendon lies in close approximation to the subscapular bursa. As noted above, adhesions within the subscapular bursa were identified as a major component of adhesive capsulitis. It is possible that a chronic enthesitis of the subscapularis muscle adjacent to its bursa could induce an inflammatory reaction that could then induce fibrosis of the bursa which requires forceful manipulation, inflation of

the bursa, or arthroscopic surgery to release it. In this case, this stage of fibrosis could be prevented by prompt recognition of the subscapularis TrPs when they first develop. Treating these acute TrPs promptly and effectively would prevent much subsequent pain, disability, and expense.

Similar considerations apply to supraspinatus TrPs and enthesitis of the supraspinatus tendon in the region where it blends with the joint capsule. Both the subacromial bursa and the coracohumeral ligament lie in close approximation to this region of supraspinatus attachment.

Well-designed research studies exploring the TrP component of "frozen shoulder" should help improve recognition of one important etiology of this condition and help to resolve much of the enigma associated with it.

The Subscapularis Muscle in Hemiplegia

A very common and distressing problem of patients with hemiplegia is pain and loss of range of motion at the shoulder, which are usually attributed to spasticity, but which also are cardinal features of subscapularis muscle TrPs.

One study[28] reported an attempt to identify the source of shoulder pain in patients with hemiplegia by testing the degree of association of variables and by injecting 28 patients with a local anesthetic in the subacromial area where they complained of pain. The author made no mention of TrPs, but reported that patients with better sensation tended to have lateral shoulder pain with radiation to the arm (Fig. 26.1). The pain was related most to loss of motion and NOT to spasticity, subluxation, loss of strength, or sensation. The subacromial injection resulted in moderate to marked relief in nearly 50% of cases ("dramatic" relief in some cases), suggesting that in those cases, the source of pain had been addressed.[28] Dramatic relief may have been the result of injecting a region of supraspinatus enthesopathy, and the failure of relief in other cases may have been the result of having overlooked a contributing TrP in the subscapularis muscle (or in the supraspinatus muscle).

A pair of studies[12,24] reported the successful use of phenol for motor point block of the subscapularis muscle in patients with painful hemiplegic shoulders. Range of motion improved markedly: 42% for lateral rotation and 22% for flexion of the arm at the shoulder. All patients indicated less pain in the original arc of motion but still had pain at the new extremes of range of motion. The effect of block lasted 3-5 months. This treatment would also serendipitously inactivate the active loci of TrPs in motor endplates that were injected.

Botulinum A toxin has been successfully used for treating spasticity in upper limb muscles of patients with strokes.[6] It has several potential advantages over phenol. Its toxicity is specific to motor endplates, it has no effect on sensory nerves so it is not prone to painful sequelae, and it should be equally effective for treatment of spasticity and TrPs in the subscapularis muscle. Most patients with hemiplegia who have shoulder pain and restricted range of motion suffer spasticity, TrPs, or both conditions in the subscapularis muscle. Both conditions need therapeutic attention and both respond to the same treatment. Botulinum A toxin is administered by much the same technique as that used for phenol block when primarily concerned about spasticity, and administered by looking for active loci to inject when primarily concerned with TrPs. For either condition, this toxin is effective only when it is injected where endplates are located.

Although clinicians skilled in the identification of TrPs are impressed with how commonly subscapularis TrPs in hemiplegic patients are a major contributor to both their pain and loss of shoulder range of motion, no controlled research studies of the clinical effectiveness of this therapeutic approach were found. Research studies conducted by experienced clinicians who are trained in how to identify and treat TrPs are urgently needed.

12. TRIGGER POINT RELEASE (Fig. 26.5)

Joint play should be restored if it is restricted in the glenohumeral, acromioclavicular, and sternoclavicular joints.[36]

To release subscapularis trigger points (TrPs) using spray and release, the patient lies relaxed in the supine position. The op-

erator first applies a few initial sweeps of spray (Fig. 26.5A) and then abducts the arm by taking up slack as it develops, holding the arm in the neutral position between medial and lateral rotation. This provides an opening in the axilla for entry of the spray. The operator continues to laterally rotate the arm to the position of Figure 26.5A as slack develops and then abducts it to the position of Figure 26.5B; the vapocoolant spray is again swept upward over the fold of the axilla (Fig. 26.5B). The patient's body weight helps to fix the scapula. Further subscapularis range of motion is gradually obtained by additional abduction and lateral rotation of the arm. The operator places the patient's hand successively under the head, then under the pillow, and finally over the head of the bed (Fig. 26.5C). To achieve the full effectiveness of the spray in this position, the patient's body is turned and supported sufficiently in a relaxed position to sweep the vapocoolant over the dorsal surface of the scapula, including its vertebral border.

In cases of severe involvement and great sensitivity to muscle activity and stretch, it may be necessary to begin release with the shoulder submerged in tepid water where the load of gravity is removed and small movements are well tolerated.

Other noninvasive techniques for release of taut bands in the subscapularis muscle include trigger point pressure release, deep massage to taut bands that can be accessed, hold-relax and contract-relax,[56] and other methods of myofascial manipulation as described by Cantu and Grodin.[11] Application of vapocoolant or icing can precede any of these techniques.

Nielsen[39] described treatment of subscapularis TrPs by using stretch and spray, and Lewit[32] described release using gravity-assisted postisometric relaxation.

Chironna and Hecht[12] reported two cases of shoulder pain with restricted range of motion that they ascribed only to spasticity which they successfully treated with motor point block of the subscapularis muscle using phenol. They noted that their treatment (which incidentally would effectively inactivate TrPs in that muscle) inexplicably resulted in immediate increase in volitional range of motion, but they were apparently unaware of TrPs.

Osteopathic techniques are often applied to release tight muscles in a general sense, but are rarely identified for the purpose of releasing TrPs in a specific muscle. Two techniques could be helpful for releasing TrP tension in the subscapularis muscle, but likely would be more beneficial if they were modified to more effectively release the subscapularis muscle. One is the Spencer technique as illustrated with abduction and lateral rotation,[42] and the other is the integrated neuromusculoskeletal technique for the upper limb and shoulder, subject prone.[58]

When other shoulder muscles also are involved—especially the teres major, latissimus dorsi, pectoralis major, and anterior deltoid—the full range of abduction and lateral rotation at the shoulder may be blocked until these other muscles are released. When full lateral rotation is approached during abduction, the unaccustomed shortening may cause shortening activation (reactive cramping) of the supraspinatus muscle, an antagonist of the subscapularis. This activation of the supraspinatus latent TrPs may cause sudden, severe pain referred to the shoulder, but can be prevented or relieved if the supraspinatus muscle is promptly lengthened and sprayed.

One may think of the release of these successively activated muscles as unravelling the history of the condition, much as one unwinds layers of a bandage, with the subscapularis as the initial layer.

In hemiplegic patients, spray and release are likely to provide only temporary benefit in the acute phase, or if there is resting spasticity. There is no contraindication to the application of spray and release several times a day, and it can provide much relief of pain. After several months, and with no resting spasticity, TrP release can lead to lasting relief of pain and to permanent improvement in the range of shoulder motion.

Spray and release are followed at once by hot packs, then by active range of motion exercises, and finally by the middle hand-position of the In-doorway Stretch Exercise (see Fig. 42.9).

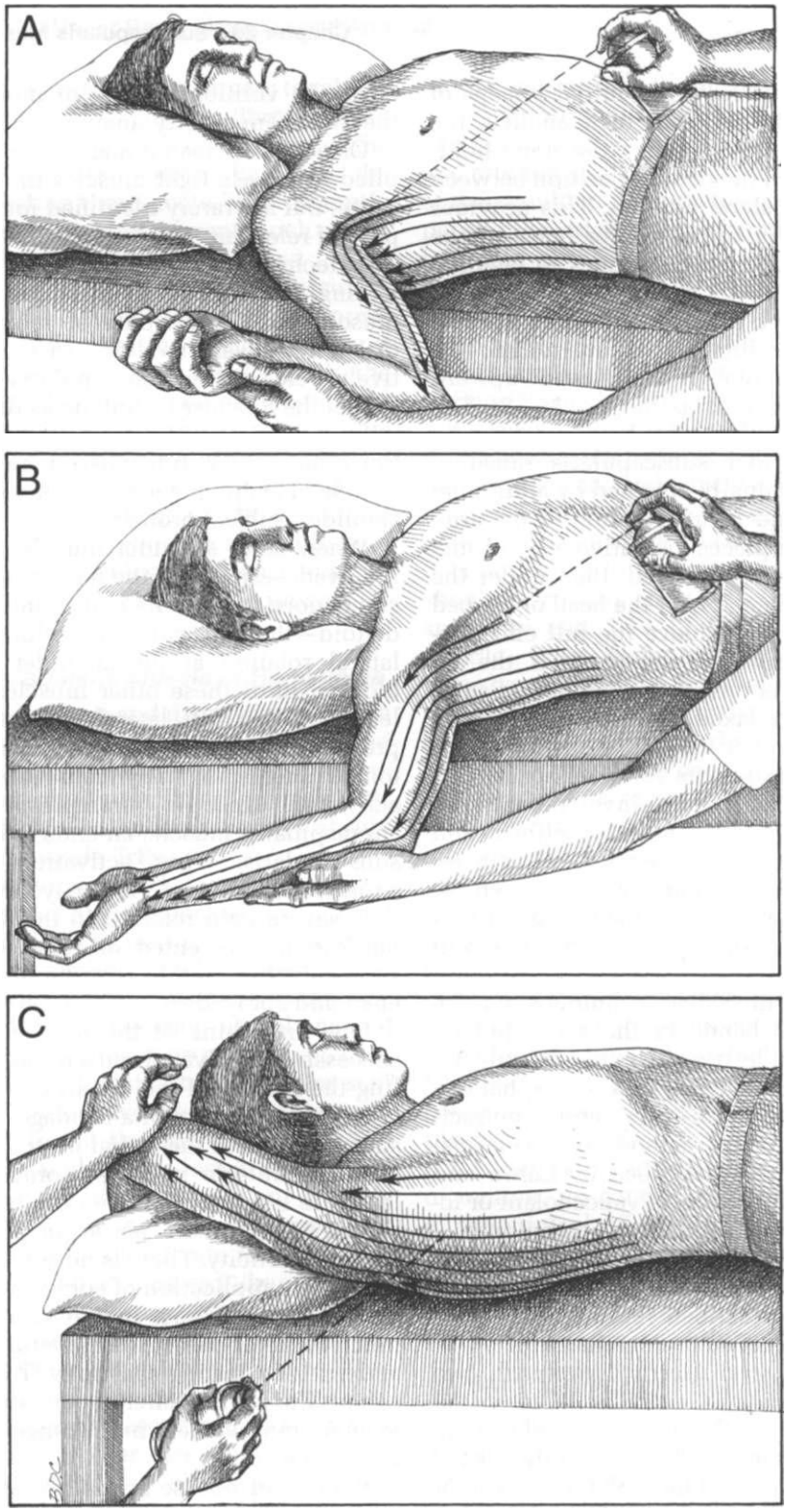


Figure 26.5. Stretch position and spray pattern *(arrows)* for trigger points in the subscapularis muscle. **A,** initial stretch position. **B,** intermediate stretch position that is reached as the taut bands of the TrPs partially release. **C,** full stretch of the subscapularis muscle. The involved side of the chest can be turned up, away from the table, sufficiently for the spray to cover *all* of the skin that overlies the subscapularis posteriorly, but with the body supported in a way that does not lose full relaxation of the patient.

13. TRIGGER POINT INJECTION (Fig. 26.6)

If trigger point (TrP) tenderness, pain, and restriction of movement remain after noninvasive treatment by spray and release, precise injection of the active TrPs may be effective. The patient lies supine in the same position as that used for vapocooling, with the arm abducted. If sufficient abduction is not available to provide room for performing the injection, TrP release techniques should be applied to provide it. The patient's hand is placed under the pillow, or with the wrist at shoulder level (Fig. 26.5A), if that is as high as it will go. The patient's body weight holds the scapula in position after it is pulled laterally (Fig. 26.4B and C). The active TrP site to be injected is located and fixed between the fingers. A 6- or 7.5-cm (2 1/2- or 3-inch), 22-gauge needle is inserted *between the examiner's fingers* into the depth of the axillary fossa (Fig. 26.6). The needle is directed parallel to the rib cage and cephalad, toward the face of the scapula, directly into the TrPs identified by palpation. The needle is always inserted through the skin caudal to the TrPs being injected and directed cephalad to avoid encountering the rib cage, which can easily happen in this location. A similar injection technique is described and illustrated by Rachlin.[46]

If pain remains after the inferior TrPs along the lateral scapular border have been inactivated, the lateral TrP in the superior region shown in Figure 26.1 may be responsible. These TrPs lie in the thick band of fibers that arch across the middle of the muscle and attach to the vertebral half of the scapula. These fibers are shown between the posterior cut ends of ribs four and five in Figure 26.2.

The TrP injection is followed immediately by spray and release, and then a hot pack to warm the skin over the subscapularis.

When a patient with hemiplegia has a subscapularis muscle with both spasticity and active TrPs, this is one valid indication for injection of the motor endplate zone with botulinum A toxin while looking specifically for TrPs (identified by LTRs and/or EMG activity characteristic of active loci of TrPs as described in Chapter 2). This injection should be done under EMG guidance with a Teflon-coated hypodermic needle specifically made for botulinum A injections.

The **medial trigger area** requires special consideration for injection. Unequivocal determination that subscapular tenderness along the vertebral border is caused by enthesopathy of the subscapularis muscle is difficult. The tenderness also could be in the middle trapezius, lower trapezius, rhomboid, and/or serratus anterior muscles through which one must perform the

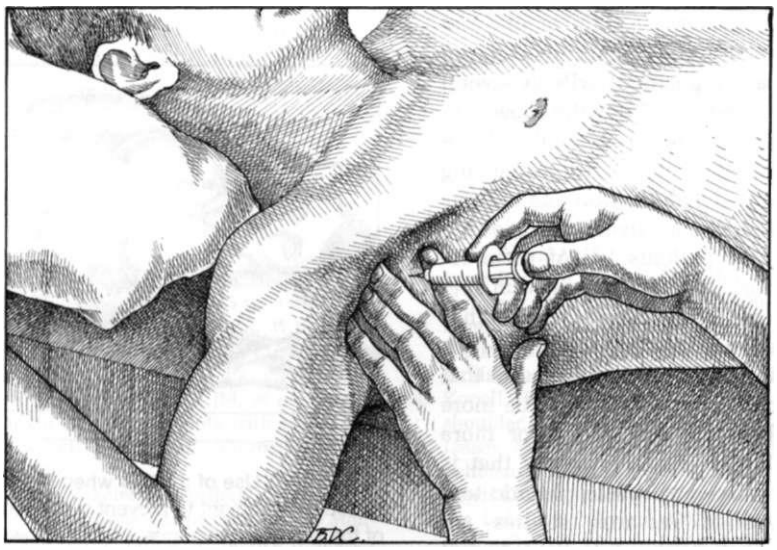


Figure 26.6. Injection of trigger points in the subscapularis muscle along the axillary border of the scapula.

palpation. Since enthesopathy in each of these muscles would most likely be secondary to TrPs in their respective muscle bellies, each muscle should be examined for TrPs, and if found, they should be inactivated. Since it may take some time before the attachment area can recover sufficiently from the sustained overload to become symptom-free, injection of 0.5% procaine or lidocaine into the tender attachment area expedites recovery. Injection of steroid that could reach the weakness-prone lower trapezius and rhomboid muscles is not recommended. If one wishes to inject the subscapularis trigger area along its vertebral margin, one technique[41] has the patient forcefully abduct the scapula by placing the hand of the involved side across the front of the body, reaching far back on the uninvolved shoulder. This causes a degree of winging which usually makes it possible to reach the subscapularis muscle beneath the scapula. At least a 1.5-inch needle is required, but care must be taken to stay clear of the rib cage.

Ormandy[41] described this technique as the treatment for the scapulocostal syndrome by infiltrating a TrP in the subscapularis region of the medial aspect of the scapular spine. The syndrome was diagnosed by pain deep in the shoulder region and upper back that often radiated into the neck and down the posterior aspect of the upper extremity to the fingers, with marked tenderness at the medial end of the scapular spine (a pain distribution suggesting a composite of TrPs in several regional muscles, including the subscapularis). The syndrome was generally attributed to altered posture. After injecting this subscapular TrP location one to three times with lidocaine hydrochloride and steroid in 440 patients, all of them returned to work. It was not clear what specific structure or structures the author thought he had injected with a 1-inch needle. Clinical experience suggests that steroid and local analgesic may be more effective than analgesic alone for more rapid resolution of enthesopathy that is no longer being exposed to chronic tension. Controlled research studies are needed to confirm or refute this clinical impression.

14. CORRECTIVE ACTIONS (Fig. 26.7)

Sleep Position

When sleeping on the painful side or on the back, the patient should keep a small pillow between the elbow and side of the chest (Fig. 26.7), thus maintaining some arm abduction and preventing prolonged positioning of the subscapularis muscle in a shortened position. When sleeping on the pain-free side, the pillow is moved to support the painful arm in front of the body (*see* Fig. 22.6). This prevents folding the arm across the chest in the fully adducted and medially rotated position, which is the fully shortened position.

Correction of Posture Stress

The patient must learn to avoid a "slumped" forward-head, abducted-scapulae posture (avoiding sustained medial rotation of the arm). *See* Chapter 41.

The patient should hook the thumb in the belt or on the hip when standing for a long period of time to prevent the arm from remaining close to the side. Also, when sitting, the patient should move the arm frequently to stretch the muscle. As a passenger in a car, the patient can stretch by resting the arm across the back of the seat, or by reaching the arm up and back behind the head, or reaching upward toward the ceiling. When a patient drives long distances, the subscapu-

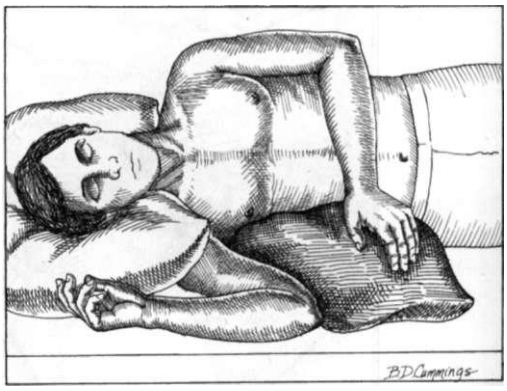


Figure 26.7. Use of a pillow when lying on the affected side at night to prevent sustained shortening of the subscapularis muscle. The pillow should be placed between the affected (right) arm and the body.

laris muscle generates much referred pain if it remains in the shortened position without movement; a nondominant left subscapularis muscle is more vulnerable, since a dominant right arm is more active. Use of an armrest helps to hold the arm in some abduction and avoid the completely shortened position.

Home Exercise

The patient learns to passively lengthen the muscle by using the middle and lower hand-positions of the In-doorway Stretch Exercise *[see* Fig. 42.9). Three cycles of each of these hand positions should be performed at least twice daily, preferably after a moist hot pack, warm shower, or warm bath.

Circumduction, or an arm-swinging exercise with the person leaning over and the arm hanging down (Codman's exercise), is very helpful. A weight may be hung from the fingers or wrist to provide slight traction. An attempt should be made to laterally rotate the arm and make a wide swing.

Rhythmic stabilization of the subscapularis muscle (cyclic resisted abduction and lateral rotation at the shoulder to the limit of pain) increases the tolerance of the muscle to stretch by reflex reciprocal inhibition, thus improving its range of motion.[50]

SUPPLEMENTAL REFERENCE, CASE REPORTS

Rinzler and Travell described the management of a patient with TrPs in multiple muscles, including the subscapularis.[48]

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:370 (Fig. 6-17).
2. *Ibid.* p. 376 (Fig. 6-26).
3. *Ibid.* p. 371 (Fig. 6-19).
4. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (p. 385).
5. Bateman JE: *The Shoulder and Neck.* W.B. Saunders, Philadelphia, 1972 (pp. 134, 145- 146, 149, 284-290).
6. Bhakta BB, Cozens JA, Bamford JM, *et al.:* Use of botulinum toxin in stroke patients with severe upper limb spasticity. *J Neurol Neurosurg Psych* 61 1):30-35, 1996.
7. Bonica JJ: Musculoskeletal disorders of the upper limb: basic considerations. Chapter 49. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990 (pp. 882-905).
8. Bonica JJ, Sola AE: Other painful disorders of the upper limb. Chapter 52. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990 (p. 951).
9. Bunker TD, Anthony PP: The pathology of frozen shoulder. A Dupuytren-like disease. *J Bone Joint Surg* 77B(5):677-683, 1995.
10. Cailliet R: *Soft Tissue Pain and Disability,* F.A. Davis, Philadelphia, 1977 (pp. 161, 162).
11. Cantu RI, Grodin AJ: *Myofascial Manipulation: Theory and Clinical Application.* Aspen, Gaithersburg, 1992 (pp. 154-155).
12. Chironna RL, Hecht JS: Subscapularis motor point block for the painful hemiplegic shoulder. *Arch Phys Med Rehabil* 72:428-429, 1990.
13. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 369, 373).
14. *Ibid.* (pp. 522-523).
15. *Ibid.* (p. 1209).
16. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 21, 49).
17. *Ibid.* (Fig. 50).
18. *Ibid.* (Fig. 233).
19. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 64, 66).
20. Esposito S, Ragozzino A, Russo R, et *al:* [Arthrography in the diagnosis and treatment of idiopathic adhesive capsulitis]. *Radiologia Medica 85(5):583-*587, 1993.
21. Gerwin RD, Shannon S, Hong CZ, *et al.:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
22. Glousman R, Jobe F, Tibone J, *et al.:* Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg 70A(2):220-226,* 1988.
23. Headley BJ: Evaluation and treatment of myofascial pain syndrome utilizing biofeedback. Chapter 5. In: *Clinical EMG for Surface Recordings,* Vol. 2, Edited by Cram JR. Clinical Resources, Nevada City, 1990.
24. Hecht JS: Subscapular nerve block in the painful hemiplegic shoulder. *Arch Phys Med Rehabil* 73.1036-1039, 1992.
25. Inman VT, Saunders JB, Abbott LC: Observations on the function of the shoulder joint. *J Bone Joint Surg* 26.1-30, 1944 (pp. 14, 15, 21-24).
26. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 73-74).
27. Jobe FW, Perry J, Pink M: Electromyographic shoulder activity in men and women professional golfers. *Am J Sports Med 17(6):7S2-787,* 1989.
28. Joynt RL: The source of shoulder pain in hemiplegia. *Arch Phys Med Rehabil* 73:409- 413, 1992.
29. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 294).
30. Kopell HP, Thompson WA: Pain and the frozen shoulder. *Surg Gynecol Obstet 109.*-92- 96, 1959.
31. Lange M: *Die Muskelharten (Myogelosen).* J.F. Lehmanns, Miinchen, 1931 (p. 129, Fig. 40A).
32. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 204, 205).
33. Marmor LC: The painful shoulder. *Am Fam Phys* 3:75-82, 1970 (pp. 78-79).

34. McMinn RM, Hutchings RT, Pegington J, *et al: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 126).
35. Melzer C, Wallny T, Wirth CJ, *et al:* Frozen shoulder—treatment and results. *Arch Orthop Trauma Surg 114(2):S7-91,* 1995.
36. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown and Company, Boston, 1964 (pp. 78-90).
37. Mikasa M: Subacromial bursography. *J Jpn Orthop Assoc* 53:225-231, 1979.
38. Neviaser JS: Musculoskeletal disorders of the shoulder region causing cervicobrachial pain; differential diagnosis and treatment. *Surg Clin North Am* 43:1703-1714, 1963 (pp. 1708-1713).
39. Nielsen AJ: Case study: myofascial pain of the posterior shoulder relieved by spray and stretch. *J Orthop Sport Phys Ther* 3:21-26, 1981.
40. Ogilvie-Harris DJ, Biggs DJ, Fitsialos DP, *et al:* The resistant frozen shoulder. Manipulation versus arthroscopic release. *Clin Orthop* 329:238-248, 1995.
41. Ormandy L: Scapulocostal syndrome. *VA Med Q 121(2):105-108,* 1994.
42. Patriquin DA, Jones JM III: Articulatory techniques. Chapter 55. In: *Foundations of Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997 (pp. 778, 779, Fig. 55.26).
43. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 60).
44. Pink M, Jobe FW, Perry J: Electromyographic analysis of the shoulder during the golf swing. *Am J Sports Med* 28(2):137-140, 1990.
45. Pollock RG, Duralde XA, Flatow EL, et *al.:* The use of arthroscopy in the treatment of resistant frozen shoulder. *Clin Orthop* 304:30-36, 1994.
46. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 200-202).
47. Reynolds MD: Personal Communication, 1980.
48. Rinzler SH, Travell J: Therapy directed at the somatic component of cardiac pain. *Am Heart J* 35:248-268, 1948 (Case 3, pp. 261-263).
49. Rizk TE, Gavant ML, Pinals RS: Treatment of adhesive capsulitis (frozen shoulder) with arthrographic capsular distension and rupture. *Arch Phys Med Rehabil* 75(7):803-807, 1994.
50. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Rehabil* 62:107-110, 1981.
51. Scovazzo ML, Browne A, Pink M, *et al.:* The painful shoulder during freestyle swimming. *Am J Sports Med* 29(6):577-582, 1991.
52. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 318).
53. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1, Macmillan, New York, 1919 (p. 277).
54. *Ibid.* (p. 313).
55. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 2 2:425-434, 1952.
56. Voss DE, Ionta MK, Myers BJ: *Proprioceptive Neuromuscular Facilitation.* Ed. 3. Harper and Row, Philadelphia, 1985.
57. Waldburger M, Meier JL, Gobelet C: The frozen shoulder: diagnosis and treatment. Prospective study of 50 cases of adhesive capsulitis. *Clin Rheumatol* 22(3):364-368, 1992.
58. Ward RC: Integrated neuromusculoskeletal techniques for specific cases. Chapter 63. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997, p. 851-899 (pp. 887, 890, 891, Figs. 63.80 and 63.81).
59. Warner JJ, Allen A, Marks PH, *et al:* Arthroscopic release for chronic, refractory adhesive capsulitis of the shoulder. *J Bone Joint Surg* 78A(12).1808-1816,1996.
60. Weber M, Prim J, Bugglin R, *et al:* Long-term follow up to patients with frozen shoulder after mobilization under anesthesia, with special reference to the rotator cuff. *Clin Rheumatol 14 (6)*:686-691, 1995.
61. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little, Brown & Company, Boston, 1988 (Fig. 12-2, p. 211).

CHAPTER 27
Rhomboid Major and Minor Muscles

HIGHLIGHTS: Both of the rhomboid muscles often "complain" because of remaining in the stretched position for long periods due to latent or active myofascial trigger points (TrPs) in the powerful pectoralis major muscles. These tense pectoral muscles commonly shorten and pull the shoulders forward into a round-shouldered posture, overloading the weaker interscapular muscles. **REFERRED PAIN** from the rhomboid muscles concentrates medially along the vertebral border of the scapula, and between that border and the vertebrae. Some of this pain may be enthesopathy from sustained stretch due to pectoral muscle tension. **ANATOMY:** these muscles arise from the spinous processes of vertebrae C_7 through T_5. They attach below and laterally to the vertebral border of the scapula. These scapular stabilizers **FUNCTION** primarily to adduct the scapula and rotate it medially, turning the glenoid fossa down. The **FUNCTIONAL UNIT** includes the trapezius muscle as the chief synergist, and the pectoral muscles as antagonists. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are usually caused by poor posture, which often occurs with active or latent TrPs in the pectoral muscles. **PATIENT EXAMINATION** reveals little or no restriction in the range of motion of the arm or in scapular mobility, but often discloses a critically important round-shouldered posture. **TRIGGER POINT EXAMINATION** by palpation usually discloses multiple TrPs between the vertebral column and the vertebral border of the scapula. **TRIGGER POINT RELEASE:** This muscle responds well to trigger point pressure release and myofascial release techniques. Nonstretching techniques are used when the rhomboids are weak. Full stretch, when using spray and stretch, requires abduction of the scapula with upward rotation of the glenoid fossa. Vapocoolant spray or icing is applied in a caudal direction, parallel to the muscle fibers. **TRIGGER POINT INJECTION** is effective and avoids stretching the muscle, but must be done with care to avoid intrapleural penetration. **CORRECTIVE ACTIONS** include inactivation of pectoral muscle TrPs with full release of tight pectoral musculature, correction of round-shouldered posture, correction of functional scoliosis, self-administration of TrP pressure release, and home use of the In-doorway Stretch Exercises to maintain pectoral muscle range of motion.

1. REFERRED PAIN
(Fig. 27.1)

Pain referred from trigger points (TrPs) in the rhomboid muscles concentrates along the vertebral border of the scapula between the scapula and the paraspinal muscles.[3,21] It also may spread upward over the supraspinous portion of the scapula (Fig. 27.1). The pain pattern somewhat resembles that of the levator scapulae, but without the neck component and without restriction of neck rotation. Referred pain extending to the arm has not been reported.

Experimental injection of hypertonic saline into normal rhomboid muscles caused referred pain felt over the upper lateral part of the scapula and extending over the acromion.[19]

Tenderness in the region of these muscles may be local tenderness of their TrPs, referred tenderness in the referred pain zone of other muscles like the scaleni, and/or enthesopathy of these muscles caused by sustained muscle tension.

2. ANATOMY
(Fig. 27.2)

The more cephalad and smaller of the two rhomboid muscles, the rhomboid minor attaches ***above*** to the ligamentum nuchae and to the spinous processes of the C_7 and T_1 vertebrae, and ***below*** to the vertebral (me-

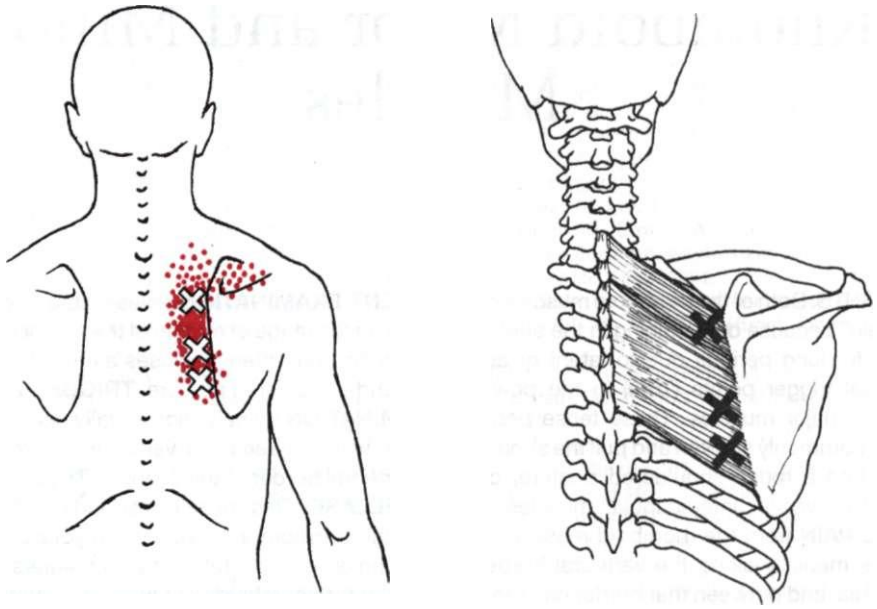


Figure 27.1. Composite referred pain pattern (essential zone *solid red,* spillover zone *stippled red)* caused by midmuscle trigger points (*white* **X**s) and trigger areas (enthesopathy) of the right rhomboid muscles.

dial) border of the scapula at the root of its spine (Fig. 27.2). The rhomboid major attaches ***above*** to the spinous processes of the T_2 through T_5 vertebrae, and ***below*** to the medial border of the scapula between its spine and inferior angle.

Supplemental References

Other authors have illustrated these muscles from behind,[1,4,5,7,20,23,25,32] from the side,[8] and in cross section.[9,10,26]

3. INNERVATION

The rhomboid muscles are innervated by the dorsal scapular nerve *via* the upper trunk of the brachial plexus from the C_5 (and occasionally also from the C_4) root.

4. FUNCTION

Based on anatomical considerations, the rhomboid muscles adduct (draw medially) and elevate the scapula.[4,17,20] The attachment of the rhomboid major fibers to the lower vertebral border of the scapula tends to rotate the scapula medially, turning the glenoid fossa down.[2,4,17,20,28] These muscles assist forceful adduction and extension of the arm by stabilizing the scapula in the retracted position.[28]

Electromyographically, these muscles were more active during abduction than during flexion of the arm at the shoulder joint, like the fibers of the middle trapezius.[2] In a similar study, Ito[16] showed that the rhomboid muscles exhibited steadily increasing activity throughout abduction and similarly during flexion, but in the latter case the electromyographic (EMG) activity reached only about two-thirds of the amplitude seen with abduction. In another study, the electrical activity of the rhomboidei rapidly increased in intensity between 160° and 180° of either movement.[15] This activity is not predicted by any of the anatomically-based actions listed above. The stabilization function during lightly loaded abduction is apparently an additional action that fixes the scapula firmly against the paraspinal soft tissues. The rhomboid muscles are active in both forward and backward swings of the arm during walking,[2] probably also to stabilize the scapula. Although the strength of adduction and extension of the humerus is diminished by loss of rhomboid fixation of the scapula, ordinary function of the arm is affected less by loss of rhomboid fixation of the scapula than by

loss of either the trapezius or the serratus anterior.[20]

No distinction was drawn between the functions of the rhomboid major and rhomboid minor by the authors quoted above. Because of the differences in attachments of these two muscles to the scapula, the rotator effect of the major may be much greater than that of the minor.

Fine wire EMG recordings of qualitative rhomboid muscle activity during aquatic swimming in 14 subjects with a painful shoulder[29] were compared with a

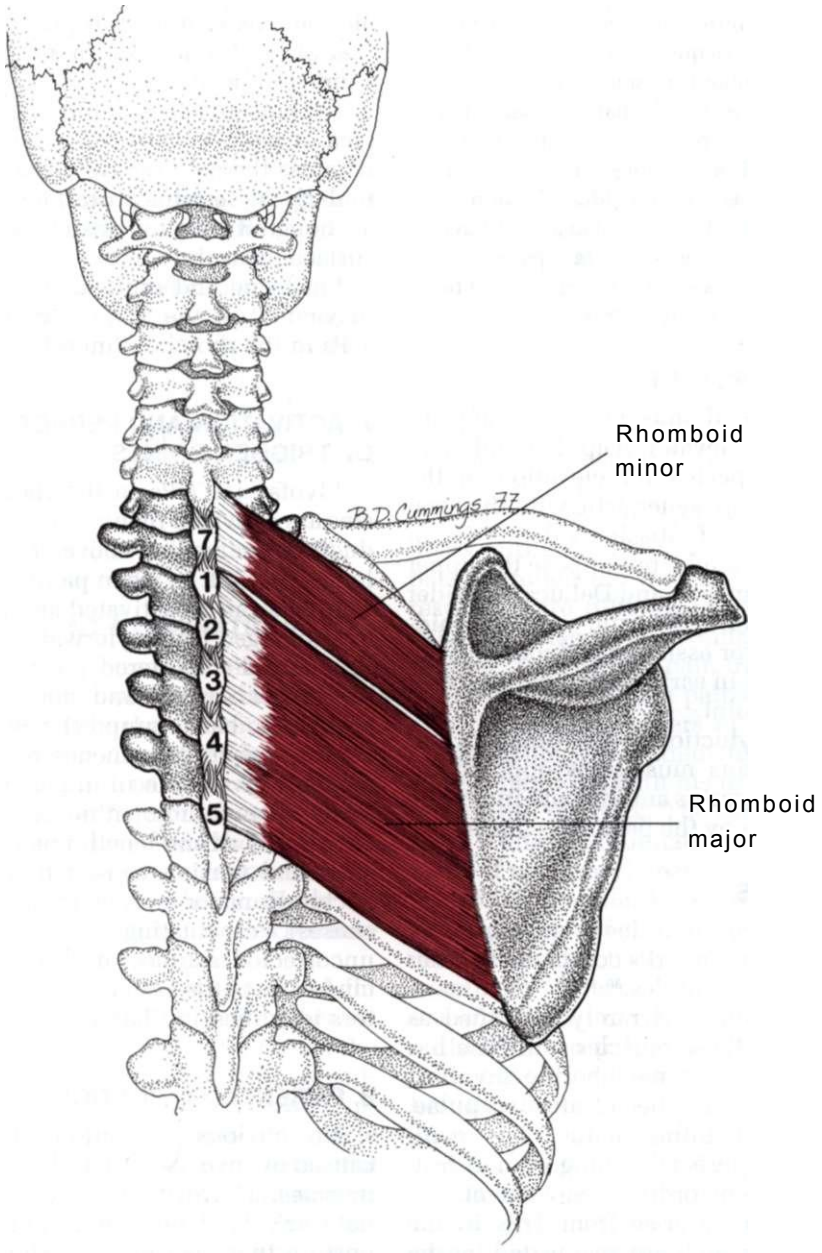


Figure 27.2. Attachments of the rhomboid major and rhomboid minor muscles to the vertebral spinous processes and to the medial border of the scapula, showing the direction and extent of the muscle fibers.

previous study of 12 pain-free shoulders. At hand entry EMG activity in painful shoulders was only one-fourth that in normal subjects, but during middle pull-through it was four times that of normal and then fell back to less than normal throughout early recovery. The initial pattern of inhibition could be expected in this muscle which is considered to be prone to inhibition and weakness.[22] However, the subsequent abnormally high level of rhomboid muscle activity is surprising and is more characteristic of a muscle that is strongly compensating for the dysfunction of another muscle, such as the serratus anterior. Identification of which muscles had TrPs that might have been causing the shoulder pain and which muscles were free of TrPs would be invaluable in a study of this type.

5. FUNCTIONAL UNIT

The rhomboid muscles act synergistically with the levator scapulae and with the upper trapezius for elevation of the scapula. They are synergistic with the levator scapulae[17] and latissimus dorsi[28] in rotation of the scapula, but oppose the upper trapezius. Basmajian and DeLuca[2] consider the rhomboidei synergistic with the middle trapezius for assisting abduction of the arm to 90° and in early flexion of the arm at the shoulder joint.

Scapular adduction by the rhomboid and middle trapezius muscles is opposed directly by the serratus anterior and indirectly but powerfully by the pectoralis major.

6. SYMPTOMS

The rhomboid muscles relatively rarely develop myofascial TrPs compared to other shoulder-girdle muscles.[30,31]

Pain (Section 1) is rarely identified as originating in these muscles until one has inactivated TrPs in neighboring involved muscles, such as the levator scapulae, trapezius, and infraspinatus. The complaint is of superficial aching pain at rest, not influenced by ordinary movement.

This pain may arise from TrPs in the muscle belly which are aggravated by the muscle being placed in the shortened position for a period of time by lying on the side of pain in a way that adducts the scapula. The pain also may arise from enthesopathy aggravated by the sustained stretch position imposed by round-shouldered posture, by tense, shortened pectoralis major muscles, or by reaching forward or stretching down to reach something. The enthesopathy pain, like stretch weakness,[20] gradually subsides if the muscle remains in a neutral position that neither places it under strain, nor holds it in a shortened position.

Patients reach for, and try to rub, the area of pain referred from rhomboid TrPs, whereas the pain referred from TrPs in the underlying serratus posterior superior feels as though it were too deep to be reached by surface pressure.

Snapping and crunching noises during movement of the scapula may be due to TrPs in the rhomboid muscles.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Myofascial TrPs in the rhomboid muscles are activated by holding the arm in abduction or flexion above 90° for a prolonged period, as when painting overhead. The TrPs can be activated and perpetuated by prolonged leaning forward and working in a round-shouldered position (as when writing or sewing and not leaning back against a back support), by prolonged stretch due to prominence of the scapula on the convex side in upper thoracic scoliosis (due to idiopathic scoliosis, chest surgery, or a limb-length inequality), or by sustained tension caused by a shortened pectoralis major muscle (from TrPs or other causes). Perpetuating factors in addition to uncorrected sources of chronic strain include any of the systemic perpetuating factors identified in Chapter 4 of this volume.

8. PATIENT EXAMINATION

No obvious restriction of motion is caused by myofascial TrPs in the rhomboid muscles. However, the examiner should note any tendency for round-shouldered posture that may indicate shortening and tightness of the pectoralis major muscle and can put sustained tension on the rhom-

boid and middle trapezius fibers. Such overstretching has given the rhomboid muscles the reputation of being prone to weakness and inhibition. There is a lack of published EMG data to establish whether the muscle is inhibited or whether it is contracting, but overpowered. Whether it or other muscles of its functional unit have developed TrPs could greatly influence its response. Correction of forward slump or round-shouldered posture is absolutely necessary to resolve fully an enthesopathic component of rhomboid-generated pain. Testing the rhomboid muscles for weakness in the presence of remaining shoulder-girdle muscles of near-normal strength is difficult since each of its functions is provided by more powerful muscles. The most reliable clinical indication of rhomboid major weakness is obtained by palpation of the rhomboid major during adduction and elevation of the scapula with *downward (medial) rotation* (see the following section). Otherwise, contraction of the overlying trapezius can obscure rhomboid activation.

The examiner should test the scapula for normal mobility over the chest wall.[24]

9. TRIGGER POINT EXAMINATION

Gerwin *et al.*[12] found the most reliable examinations for making the diagnosis of myofascial TrPs to be the detection of a taut band, the presence of spot tenderness, the presence of referred pain, and reproduction of the patient's symptomatic pain. Their study did not include the rhomboid muscles. The purely objective local twitch response is difficult to elicit reliably by manual palpation in these muscles because of the overlying trapezius muscle.

The rhomboidei are best examined for myofascial TrPs with the patient seated and the arms hanging forward to relax the muscle and abduct the scapulae, spreading them away from the vertebral column. A taut band in a rhomboid muscle can be clearly distinguished from the overlying trapezius by the direction of its fibers. The rhomboid muscle fibers are directed obliquely downward and laterally, away from the vertebrae, and the lower trapezius fibers are angled upward and laterally. Middle trapezius fibers lie in a relatively horizontal direction. Medial rotation and adduction of the humerus places the scapula in downward rotation (medial rotation of the inferior angle) and accentuates the rhomboid fibers as scapular adduction is attempted. The difference in fiber direction of the trapezius is accentuated by abducting the arm to rotate the scapula upward (assuming functioning humeral abductors and rotators).

The rhomboid muscles are palpated for taut bands and the midbelly region of each taut band is examined for TrP tenderness (indicated by Xs in the pain pattern drawing on the left side of Fig. 27.1). In addition, palpation along the vertebral (medial) border of the scapula may reveal trigger areas near the attachment (indicated by Xs in anatomy drawing on the right side of Fig. 27.1). This tenderness adjacent to the scapula often represents enthesopathy secondary to tension from rhomboid TrPs and/or from overload produced by tight pectoral muscles.

All but the caudal ends of the lowermost fibers of the rhomboid major must be palpated through the trapezius. Local twitch responses are difficult to elicit, but are a valuable diagnostic confirmation when present. The referred pain from active TrPs is reproduced by deep palpation.

If the precise borders of these muscles are in doubt, the patient to be examined should lie prone with his or her hand resting behind the back. The examiner tries to place a finger (reinforced with the opposite hand, if necessary) deep to the medial border of the scapula. When the patient lifts the hand up off the back, the rhomboid muscles contract vigorously, pushing the examiner's finger out from under the scapula. Once the rhomboidei have been outlined, deep palpation *across* the direction of the rhomboid muscle fibers identifies the firm "ropy" bands that contain TrPs.

Active TrPs in the upper trapezius muscle can act as key TrPs that induce satellite TrPs in the rhomboid minor muscle. In that case, inactivating the trapezius TrP also usually inactivates the satellite rhomboid TrP.[13]

10. ENTRAPMENT

No nerve entrapments have been attributed to these muscles.

11. DIFFERENTIAL DIAGNOSIS

Patients with chronic axial pain and additional regional complaints that fit the diagnosis of **fibromyalgia** should be examined for the tender point count and other requirements for the diagnosis of fibromyalgia.[34] In one study of 96 subjects,[11] 20% of the patients with myofascial pain caused by TrPs also had fibromyalgia. Treatment of patients with both conditions requires special consideration.

The pain caused by rhomboid TrPs may be erroneously diagnosed as **scapulocostal syndrome** if the TrP examination was overlooked or improperly performed.

Articular Dysfunction

Articular dysfunction associated with rhomboid TrPs can involve any of the spinal segments from C_7 to T_5. Usually two or more segments are involved. Typically one observes a multiple level dysfunction with vertebral sidebending in the direction of the muscle harboring TrPs and rotation away from the involved muscle. Occasionally one finds a central dysfunction, usually at approximately T_3, which includes an element of single segment vertebral extension as well as sidebending and rotation in the same direction. This usually appears as a flattened upper thoracic spine that does not flex forward on attempted flexion, and there is a concurrent scapular adduction with rhomboid muscle involvement. This central segmental dysfunction must be recognized and treated. When this articular dysfunction has been corrected, one often finds that the rhomboid TrP was inactivated at the same time.

Related Trigger Points

Several muscles that refer pain in a similar pattern to that of the rhomboids are the scalene, levator scapulae, middle trapezius, infraspinatus and latissimus dorsi muscles. These muscles should also be examined for TrPs, especially if the therapeutic response to rhomboid treatment is incomplete.

Active rhomboid TrPs usually become obvious only after elimination of TrPs in the levator scapulae, trapezius, and infraspinatus muscles. Patients with rhomboid TrPs complain of upper back and scapular pain. They are frequently stooped and round-shouldered, appear flat-chested, and are unable to stand up straight because of TrP-induced tautness in either or both the pectoralis major and minor. The rhomboid and middle trapezius muscles bilaterally are then overloaded by having to oppose the stronger, shortened pectoral muscles. The pectoralis TrPs may be latent and not signaling trouble by pain, but they are nevertheless overloading their dorsal antagonists, which do the complaining. Serratus anterior TrPs also can contribute to rhomboid muscle overload.

12. TRIGGER POINT RELEASE
(Figs. 27.3 and 27.4)

Since this muscle is prone to inhibition and stretch weakness,[20] one must be careful not to aggravate its problems with additional overstretching. Relief of the stretch weakness depends on relieving the persistent tension on the muscle and restoration of normal rhomboid muscle function and muscle balance.[20]

The clinician must clearly distinguish myofascial trigger point (TrP) tenderness and taut bands from tenderness of enthesopathy caused by overstretch. Treatment should start anteriorly with inactivation of any pectoral muscle TrPs and restoration of normal resting length to the pectoralis muscles. The serratus anterior muscle (particularly the lower half) also should be checked and any TrPs inactivated. The rhomboid attachments at the scapular border should be carefully examined for tenderness indicating enthesopathy secondary to TrP tension. If TrP and enthesopathic trigger area tenderness are both present, it is better to start with trigger point pressure release and deep massage of the taut bands to reduce tension on the irritated attachments. Correction of slumped round-shouldered posture (Chapter 41) is essential for lasting relief of rhomboid TrPs or an enthesopathy component of rhomboid-generated pain.

If the clinician determines that muscle tension needs to be released, a spray and

release technique can be applied to the rhomboid TrPs with the patient seated and relaxed, the upper thoracic spine flexed, and the arms hanging between the knees (Fig. 27.3A), or crossed in front of the chest (Fig. 27.3B). The patient should "hump" the back (flex the thoracic spine) and let the weight of the arms pull the shoulder blades forward and laterally. The spray is applied downward in slow parallel sweeps over the rhomboid muscles, in the direction of their muscle fibers (Fig. 27.3B). The uppermost sweeps of spray are continued across the scapula to cover the lateral extension of the referred pain pattern. Following application of the vapocoolant, the patient inhales deeply, and then exhales slowly to relax the muscles, reaching farther across the front of the body to take up slack in the rhomboids.

Several alternative techniques are available that minimize the danger of overstretching this muscle. Some of the nonstretching methods are trigger point pressure release, deep massage to the taut band, hold-relax[33] applied to the tense muscle, and indirect techniques.[18] If TrPs and attachment tenderness are present, it is important to release the TrPs without aggravating the enthesopathy. Figure 27.4 illustrates the postisometric relaxation (myofascial) release of the right rhomboid muscles. The operator lightly resists scapular adduction (retraction) while the patient slowly breathes in and looks upward to the right. The patient looks downward to the left and slowly breathes out, relaxing fully. As the muscle releases, the operator's hand follows the movement of the scapula into abduction *[arrow],* taking up the slack as the patient exhales and concentrates on relaxing all muscles.

An alternate position is with the patient prone and placing the arm and shoulder over the edge of the treatment table.

The interscapular muscle release and scapular mobilization illustrated in Figure 18.3 is well suited to release of rhomboid TrPs. A soft tissue technique for the rhomboid muscles also is described and illustrated by Ehrenfeuchter.[6]

In a variation of this, the patient is sidelying with the affected side uppermost. The therapist faces the front of the patient,

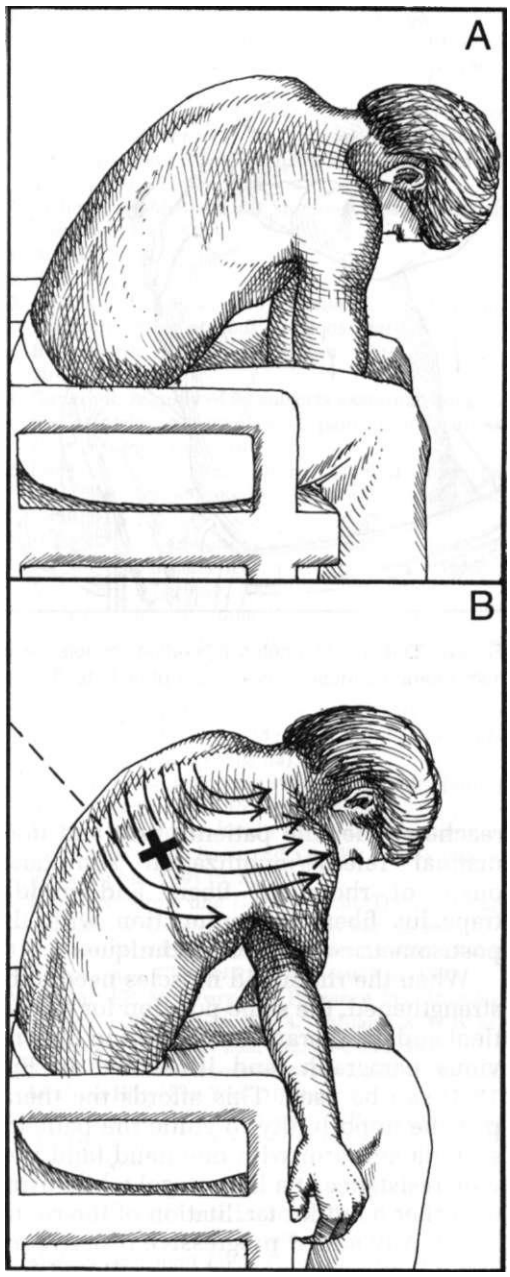


Figure 27.3. Stretch position and spray pattern *(thin arrows)* for a trigger point (**X**) in the right rhomboid major muscle, position to be used only if the muscle shows increased tension. **A,** optimum position for gravity-assisted release of the rhomboid minor. **B,** optimum position for gravity-assisted release of the rhomboid major and spray pattern for both muscles. The operator should not apply additional manual pressure against the scapula to increase the stretch because this muscle is readily overstretched.

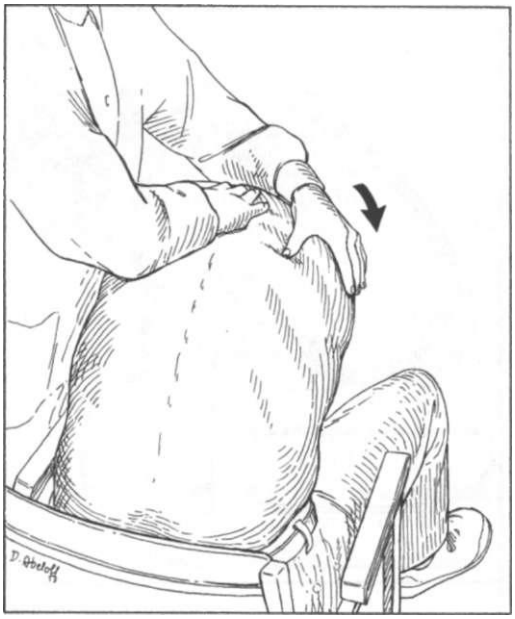


Figure. 27.4. Postisometric relaxation for release of right rhomboid muscles (see description in text).

reaches under the patient's arm and does manual release/mobilization simultaneously of rhomboid fibers and middle trapezius fibers in conjunction with the postisometric-relaxation technique.

When the rhomboid muscles need to be strengthened, the same position for the patient and the therapist described in the previous paragraph (and illustrated in Fig. 18.3) can be used. This affords the therapist the opportunity to guide the patient's scapula and arm with one hand (and provide resistance at a later stage) while using the other hand for facilitation of the rhomboids. Any added progressive resistive exercise program should be monitored and guided by the therapist. A biofeedback system for home use could enable the patient to follow a home program without overstretching a weak muscle.

Use of spray and release is followed by moist heat, and all release techniques are followed by having the patient move the scapula through two or three repetitions of full active range of motion.

13. TRIGGER POINT INJECTION

The trigger point (TrP) must be accurately located in the midfiber portion of the muscle and fixed against the chest wall between the fingers of the palpating hand. For injection of the TrP, a 3.8-cm (1 1/2-in) needle is directed almost tangential to the surface. Injection of 0.5% procaine or 1% lidocaine reduces postinjection soreness compared to dry needling.[14] The needle is aimed toward a rib to avoid penetrating an intercostal space. The risk of pleural penetration by the needle can be essentially eliminated by placing the 2nd and 3rd fingers into the intercostal spaces above and below the site of the TrP injection.[3] A local twitch response (LTR) confirms accurate penetration of an active locus of a TrP by the needle. Injection is much less likely to be successful in the absence of an LTR.[14]

Moist heat is applied to reduce postinjection soreness, and then three cycles of full active range of motion are performed to restore normal muscle function and coordination.

Rachlin describes and illustrates a similar technique for injecting rhomboid TrPs.[27]

Myofascial TrPs in the rhomboid minor may be *satellites* of a key TrP in the upper trapezius muscle. In that case, inactivating the trapezius TrP usually also inactivates the satellite rhomboid minor TrP.[13]

14. CORRECTIVE ACTIONS

Since the rhomboid muscles are prone to inhibition and stretch weakness,[20,22] corrective actions concentrate on self-administered manual release techniques and correction of faulty posture rather than stretch exercises. The enthesopathy pain from rhomboid TrPs, like stretch weakness, gradually subsides if the muscle remains in a neutral position so that the muscle is neither under strain nor shortened. If the patient has involvement of the pectoral muscles, daily use of the In-doorway Stretch (*see* Fig. 42.9) can be effective following a warm shower, bath, or application of moist heat.

The patient should learn how to apply trigger point pressure release to the rhomboid TrPs by lying on a tennis ball placed on the floor (or on a large thin book on the bed), or by using a Theracane®. The patient

can "press out" the spot tenderness due to each rhomboid TrP by rolling a single tennis ball along the medial border of the scapula. If there is bilateral involvement, a cold *pair* of tennis balls can be used (see Fig. 18.4). The pressure is centered on a tender spot until the tenderness fades, usually in 20 or 30 seconds but sometimes as long as a minute; then the patient can roll the ball on to the next tender spot.

The use of a lumbar pillow or a thoracolumbar support helps to correct a round-shouldered posture, especially while working at a desk or driving a car. One should avoid any chair that pushes the upper torso and shoulders forward. Some backward slope of the backrest with lumbar support is needed for a comfortable desirable seated posture.

For a patient who becomes preoccupied at a desk and forgets to change position and thus relieve the strain on the muscles from time to time, an interval timer can be placed across the room and set to ring. Then, the patient must get up at regular intervals of 20-30 minutes to turn it off and reset it. This need not interrupt the train of thought.

Any protrusion of the scapula due to *functional* scoliosis that is caused by a limb-length inequality or an asymmetrical pelvis can be corrected by leveling the pelvis and straightening the spine with appropriate lifts (see Volume 2, Chapter 4).

When the clinician determines that the muscle has recovered normal function sufficiently to be able to tolerate a gentle, augmented self-stretch program safely, the patient should be taught how to perform the Middle-trapezius Stretch Exercise (see Fig. 6.15), in conjunction with a postisometric relaxation technique. This also releases any rhomboid tightness.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:234,381 (Figs. 4-48,6-32).
2. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 268, 385, Fig. 12-1).
3. Bonica JJ, Sola AE: Chest pain caused by other disorders. Chapter 58. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990, pp. 1114-1145 (p. 1135).
4. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 515, 516, Fig. 6-42).
5. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 523).
6. Ehrenfeuchter WC: Soft tissue techniques. Chapter 56. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997, pp.781-794 (p.792).
7. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (Fig. 51).
8. *Ibid.* (Fig. 52).
9. *Ibid.* (Fig. 68).
10. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 28, 30-35).
11. Gerwin R: A study of 96 subjects examined both for fibromyalgia and myofascial pain. *J Musculoske Pain 3(SuppI 1):\21,* 1995.
12. Gerwin RD, Shannon S, Hong CZ, *et al:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
13. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(1):29-59, 1994.
14. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Rehabil* 73:256-263, 1994.
15. Inman VT, Saunders JB, Abbott LC: Observations on the function of the shoulder joint. *J Bone Joint Surg* 26:1-30, 1944 (p. 27, Fig. 33).
16. Ito N: Electromyographic study of shoulder joint. *J Jpn Orthop Assoc* 54:1529-1540, 1980.
17. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 83).
18. Jones LH: *Strain and Counterstrain.* The American Academy of Osteopathy, Colorado Spring, 1981.
19. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3:175-190, 1938 (p. 183).
20. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 282, 283, 294, 334, 335).
21. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (p. 98).
22. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991.
23. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 120).
24. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown and Company, Boston, 1964 (pp. 78-89).
25. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 28).
26. *Ibid.* (Fig. 44).
27. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (pp. 312-314).
28. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Lea & Febiger, Philadelphia, 1967 (p. 151).

29. Scovazzo ML, Browne A, Pink M, *et al.*: The painful shoulder during freestyle swimming. *Am J Sports Med* 19(6):577-582, 1991.
30. Sola AE, Kuitert JH: Myofascial trigger point pain in the neck and shoulder girdle. *Northwest Med* 54:980-984, 1955 (p. 983).
31. Sola AE, Rodenberger ML, Gettys BB: Incidence of hypersensitive areas in posterior shoulder muscles. *Am J Phys Med* 34:585-590, 1955.
32. Spalteholz W: *Handatlas der Anatomie des Men-schen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 303).
33. Voss DE, Ionta MK, Myers BJ: *Proprioceptive Neuromuscular Facilitation.* Ed. 3. Harper & Row, Philadelphia, 1985.
34. Wolfe F, Smythe HA, Yunus MB, *et al.*: American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia: Report of the Multicenter Criteria Committee. *Arthritis Rheum* 33:160-172, 1990.

CHAPTER 28
Deltoid Muscle

HIGHLIGHTS: **REFERRED PAIN** from active trigger points (TrPs) in the deltoid muscle is generally not referred to a distance as in most muscles, but spreads locally in the region of the affected (anterior, middle, or posterior) part of the muscle. The location of the TrPs corresponds to the location of each endplate zone. **ANATOMY:** proximally, the fibers of the anterior, middle, and posterior parts attach to the clavicle, acromion, and spine of the scapula, respectively. Distally, they all attach to the deltoid prominence of the humerus. Different fiber arrangements of the middle part of the muscle as compared to the anterior and posterior parts result in different arrangements of the endplate zones. **FUNCTION:** the anterior part of this superficial muscle, which covers the head of the humerus, is primarily a flexor of the arm, the middle part is primarily an abductor, and the posterior part primarily an extensor of the arm. All three parts assist abduction. The anterior part is antagonistic to the posterior part during flexion and extension. Working synergistically, the middle, anterior, and posterior parts help the supraspinatus muscle abduct the arm at the glenohumeral joint. **FUNCTIONAL UNIT:** The anterior part of the deltoid functions synergistically with the clavicular section of the pectoralis major, the biceps brachii (long head), and the coracobrachialis muscles. The posterior part acts synergistically with the latissimus dorsi, teres major, and the triceps brachii (long head) muscles. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** may result from impact trauma in sports or other activities, from overexertion, or from the hypodermic injection of irritant medication where latent TrPs are located. The deltoid muscle also may develop satellite TrPs from key TrPs in another muscle, especially the infraspinatus. **PATIENT EXAMINATION:** active TrPs in the anterior deltoid cause painful restriction of the Back-rub Test and painfully weakened abduction of the laterally rotated arm. Posterior deltoid TrPs cause painfully weakened abduction of the medially rotated arm. **DIFFERENTIAL DIAGNOSIS** includes rotator cuff tears, subdeltoid bursitis, impingement syndrome, C_5 radiculopathy, and TrPs in the supraspinatus and infraspinatus muscles, and in the clavicular head of the pectoralis major. **TRIGGER POINT RELEASE** by spray and stretch requires specific positioning for anterior, middle, and posterior deltoid TrPs and employs a proximal-to-distal spray pattern. This muscle responds well to augmentation of postisometric relaxation. One who does **TRIGGER POINT INJECTION** should take into consideration the cephalic vein when injecting TrPs in the anterior portion of this muscle. **CORRECTIVE ACTIONS** include eliminating perpetuating mechanical stresses, identifying and correcting systemic perpetuating factors, and doing a program of daily stretching exercises to prevent TrPs reactivation.

1. REFERRED PAIN
(Fig. 28.1)

The deltoid is one of the muscles that often develops myofascial trigger points (TrPs).[20] When these hyperirritable foci appear in the anterior part of the deltoid (Fig. 28.1 A), they refer pain to the anterior and middle deltoid regions.[10,31,52,55,57] Active TrPs in the posterior part of the deltoid (Fig. 28.1B) refer pain that concentrates over the posterior shoulder, sometimes spilling into adjacent areas of the arm.[10] Trigger points in the middle deltoid produce pain centered in that region of the muscle with some spillover pain to adjacent areas (Fig. 28.1C). The deltoid muscle lacks any distant projection of referred pain. Referred pain from this muscle was demonstrated experimentally by the injection of hypertonic saline.[51]

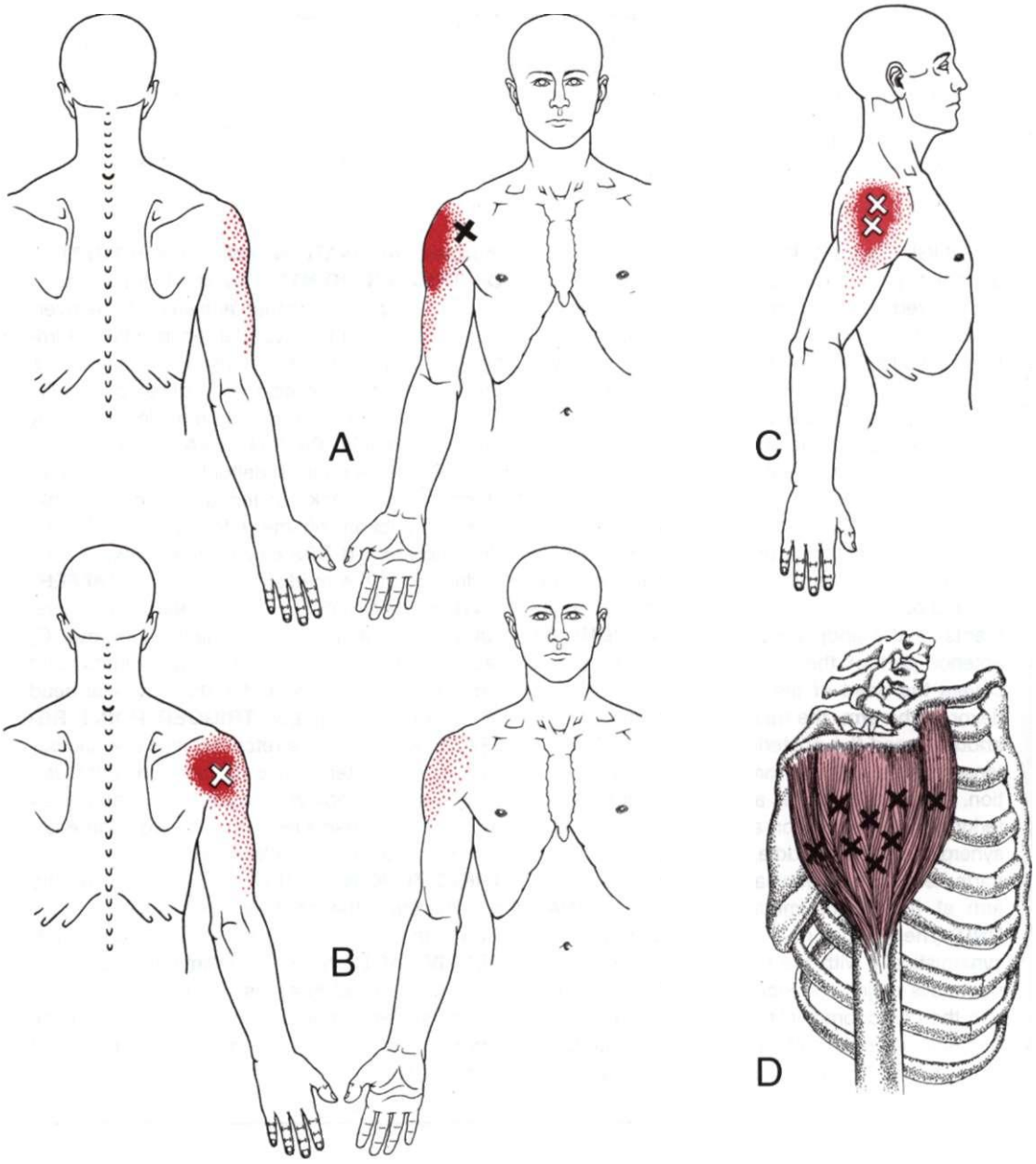


Figure 28.1. Referred pain patterns (*dark red)* from trigger points (**X**s) in the right deltoid muscle *(light red)*. **A,** pain pattern from trigger points in anterior part of the muscle. **B,** pain pattern from the posterior part. **C,** pain pattern of trigger points in the middle part of the muscle. **D,** usual location of trigger points in the muscle, lateral view. The distribution of trigger points in the anterior and posterior parts of the deltoid muscle has a different pattern than the distribution of trigger points in the middle deltoid. Figure 28.3 shows why.

Figure 28.1D illustrates where one is most likely to find TrPs in deltoid musculature. This figure relates closely to Figure 28.3 which shows schematically the difference in the location of endplate zones (black dots) in the three parts of the deltoid muscle.

2. ANATOMY
(Figs. 28.2 and 28.3)

Proximally the anterior part of the deltoid muscle attaches to the lateral one-third of the clavicle (Fig. 28.2); the middle part, to the acromion; and the posterior part, to the lateral portion of the spine of the scapula. ***Distally*** all fibers converge near the midpoint of the lateral aspect of the humerus and attach to its deltoid prominence. This point appears, in most patients, as a dimple in the skin at the base of the "V" formed by the belly of the muscle.

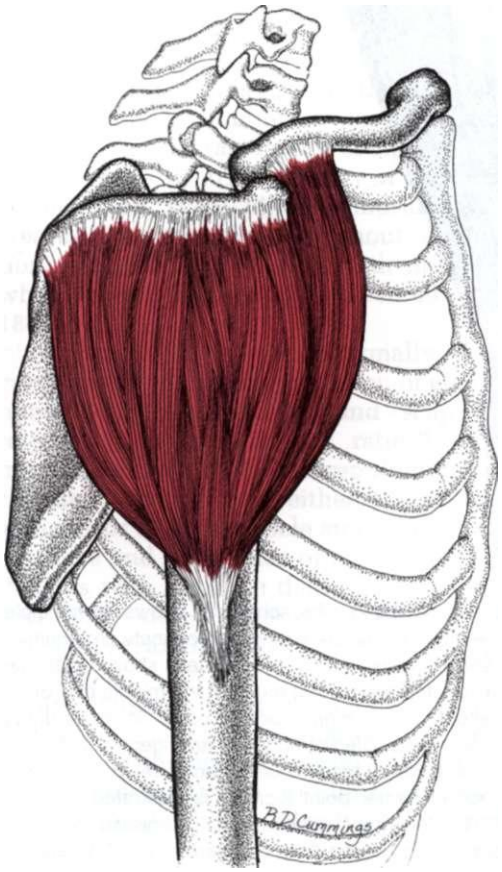


Figure 28.2. Attachments of the right deltoid muscle *(dark red)*. Compare the diagonal and complexly interwoven fibers of the middle part with the simple fusiform arrangement in the anterior and posterior parts. The schematic of Figure 28.3 shows how, in principle, these fiber arrangements affect endplate distribution.

The anterior and posterior parts of the deltoid have a fusiform arrangement of long fiber bundles which extend directly from one attachment to the other. The middle part is multipennate. Its fibers slant obliquely between proximal tendons (usually four) that extend downward from the acromion into the substance of the muscle. Three interdigitating tendons extend upward from the deltoid prominence, as previously described[15] and clearly drawn schematically.[5] Thus, the middle part of the muscle, by design, produces more force through a shorter distance than do the anterior and posterior parts of the muscle.

The true complexity of the middle part of the deltoid is more realistically portrayed in *Grant's Atlas of Anatomy*[2] and in Figure 28.2 than in the schematic simplification of Figure 28.3. This schematic representation of the location of endplate zones in Figure 28.3 reflects the difference in endplate distribution within each part of the muscle. The location of the TrPs in Figure 28.1 corresponds generally to the location of the endplate zones in Figure 28.3.

The location of endplates in the deltoid muscle is illustrated schematically in Figure 28.3. An endplate is normally located close to the midportion of the muscle fiber that it supplies. Thus, the endplate zone in a fusiform muscle like the anterior and posterior parts of the deltoid (also the biceps brachii) is a single (sometimes irregular) band of motor endplates extending across the midportion of the muscle. However, the endplates in the angulated fibers of the middle deltoid are more widely distributed throughout the muscle.[14] That explains why the TrPs of the anterior and posterior parts of the deltoid muscle are found close to midmuscle, but TrPs of the middle part can be widely distributed.

A histological study of 24 bilateral deltoid muscles[30] showed that 60% of the fibers were type 1 (slow twitch, fatigue resistant) without regard to hand domi-

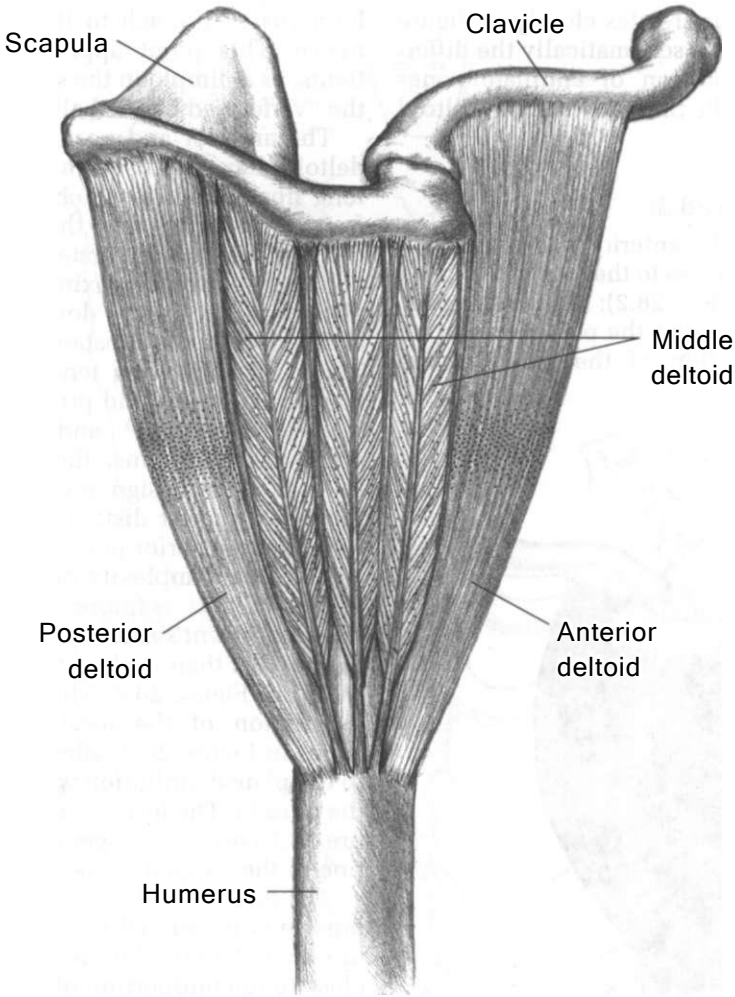


Figure 28.3. Schematic of the fiber arrangements and the corresponding distribution of endplates (*red dots)* in the three parts of the deltoid muscle. An endplate normally is located close to the middle of the muscle fiber that it innervates. The anterior and posterior parts of the muscle have a fusiform arrangement and their fibers are nearly parallel to the long axis of the muscle, an arrangement that provides speed at the expense of strength and results in a band of endplates across the middle of the muscle. The schematic for the middle part of the deltoid shows a multipennate fiber arrangement, which provides strength at the expense of speed. The schematic shows, in principle, the effect of the alternating multipennate arrangement seen in Figure 28.2. This schematic shows endplates distributed throughout much of the middle part of the deltoid. Since trigger points occur in an endplate zone, these different endplate arrangements also determine where trigger points can develop. This difference in trigger point location is illustrated in Figure 28.1. (Schematic adapted with permission from Anderson JE: *Grant's Atlas of Anatomy.* Ed. 7. Williams & Wilkins, Baltimore, 1978.)

nance, age, sex, or occupation of the subject. Although not stated, these samples were probably taken from the middle and not the anterior or posterior parts of the muscle.

Supplemental References

The deltoid muscle has been well illustrated from the anterior view,[1,37,42,48] the side view,[2,16,38,49] the posterior view,[3,17,39,43,50] and in cross section.[4,44]

3. INNERVATION

This muscle is supplied through the C_5 and C_6 spinal roots *via* a branch of the posterior cord known as the axillary nerve.[15]

4. FUNCTION

At one time, it was thought that the deltoid initiated abduction at the shoulder and that the supraspinatus completed it. However, the electrical activity of both the deltoid and supraspinatus muscles increases progressively throughout abduction. Activity is greatest in both muscles when the arm is elevated between 90° and 180°.[25]

Abduction of the arm normally progresses with smooth coordination of glenohumeral joint movement and scapular rotation in a constant 2:1 ratio.[25] This mechanism is called the scapulohumeral rhythm.[13] Paralysis of either the supraspinatus or deltoid muscle simply reduces the force and endurance of abduction.[18,19] Patients with multiple deltoid TrPs, however, may show serious impairment of strength, or total inability to reach 90° of abduction.

Simultaneous contraction of the anterior, middle, and posterior parts of the deltoid abduct the arm.[6,15] Abduction is performed chiefly by the middle fibers with glenohumeral joint stabilization provided by the anterior and posterior fibers.[32] Only the most peripheral fibers of both the anterior and posterior parts adduct the arm.[27] Otherwise, the anterior and posterior parts oppose each other.

The **anterior part** of the deltoid flexes the arm forward,[15,27,45] as confirmed by electromyographic (EMG)[6] and electrical stimulation[19] studies; it also acts in horizontal adduction of the arm across the chest.[45] One study showed that the anterior part was recruited most strongly when these two movements were combined to move the arm obliquely upward inclined toward the midline.[41] The attachments look as if this part of the muscle should medially rotate the arm,[15,27,32] but use of this action is questioned by electromyographers.[6] Movement of the hand to the face requires adequate function of the anterior deltoid and serratus anterior muscles,[19] both of which contribute to the scapulohumeral rhythm.

The **middle part** of the deltoid muscle is designed structurally for abduction, during which it shows strong EMG response.[6] During a cone-shaped hand movement, maximum recruitment of the middle part appeared when the arm moved obliquely upward inclined away from the midline.[41] The linear increase in EMG activity during abduction of the arm indicates a primary abduction function of the middle part of this muscle. However, during flexion, a nonlinear increasing activity of the middle part above 60° of arm elevation indicates that its flexor action is enhanced as arm elevation increases.[26]

The **posterior part** of the deltoid extends the arm;[6,15,27,45] this function is essential in order to reach behind the body to the gluteal area and beyond.[19] During arm movement in cones and planes, the posterior fibers were recruited during lateral movement along the horizontal.[41] Anatomically, the posterior part should assist lateral rotation,[15,27] but that function has not been substantiated electromyographically.[6] A finding of marked increase in the electrical activity of the more horizontal, posterior-margin fibers of the deltoid, when the dependent upper limb was carrying weight, confirmed the important role of these horizontal fibers in helping to secure the head of the humerus in the vertically oriented, capsule-rimmed glenoid cavity. Contraction of these deltoid fibers and the supraspinatus muscle helps to wedge the head of the humerus solidly into the glenoid fossa to prevent downward dislocation.[6]

During sports activities that require underhand, overhand, and sidearm movements, the amplitude of motor unit activity in the anterior part of the deltoid was consistently greater than in the other parts of the muscle, with one exception: during the tennis serve, the middle part of the muscle showed a strong double peak of maximum activity.[11]

Raising the typewriter keyboard clearly increased the continuous electrical activity (strain) present in the deltoid muscle.[36] In a subsequent methodological study, Hagberg and Jonsson[22] showed that the activity load on the deltoid is increased if the work height is either too low or too high, and that activity was lowest when the elbow was held approximately at a right angle.[28]

In a study of tolerance for holding the arm in two elevated positions (one at 90° of forward flexion and the other at 90° of abduction),[21] few subjects showed EMG signs of fatigue in the middle part of the deltoid after 5 minutes in abduction (or flexion) as compared to the fatigue shown in the upper trapezius, supraspinatus, infraspinatus, and the anterior part of the deltoid muscle. Although the deltoid is recruited as a prime mover, the middle part is apparently more tolerant of sustained contraction than the other muscles, either because it has more type 1 fibers or because its multipennate structure makes it more resistant to fatigue.

Driving a car with the hands on top of the steering wheel activated chiefly the anterior, and to a lesser extent the middle, part of the muscle. Activation occurred when the driver pushed the steering wheel toward the side opposite the moving arm, a movement of horizontal adduction. The posterior deltoid was rarely activated.[29]

During freestyle swimming, the normally marked increase in middle deltoid activity during the beginning and end of pull-through was significantly reduced (inhibited) in subjects with painful shoulders. In the anterior deltoid, only the marked activity of the early pull-through phase was significantly and strongly inhibited. The late pull-through and early recovery phase showed only moderate and inconsistent decrement in the swimmers with a painful shoulder.[47] Unfortunately, the structures which were responsible for the pain were not identified in this study. Myofascial TrPs can cause this kind of muscle inhibition when a person performs a well-learned activity.

5. FUNCTIONAL UNIT

The anterior deltoid, coracobrachialis, clavicular section of the pectoralis major, and the long head of the biceps brachii are commonly involved together as a functional unit. The pectoralis minor muscle also may develop active TrPs in association with those in the anterior deltoid.

Throughout abduction of the arm, the upper trapezius, supraspinatus, and rhomboid muscles show increasing EMG activity synergistically with the middle part of the deltoid. During flexion a similar response occurs, except that the deltoid and supraspinatus activity develops more slowly (nonlinearly), and the rhomboid muscle is less active than during abduction.[26]

The posterior deltoid forms a myotatic unit with the long head of the triceps brachii, the latissimus dorsi and teres *major* muscles. The teres *minor* fibers, which are aligned with the posterior deltoid only in full abduction, are less likely to develop associated TrPs.

Since, as noted in Section 4, the anterior and posterior deltoid fibers commonly function as antagonists, they may develop active TrPs together.

6. SYMPTOMS

The patient history may reveal onset of symptoms after impact trauma to the deltoid muscle during sports or other activities.

The patient complains of pain on shoulder motion and, less frequently, of pain at rest deep in the deltoid area (Fig. 28.1). The patient with active anterior deltoid TrPs has difficulty in raising the arm to the horizontal, as in bringing the hand to the mouth, and in reaching back at shoulder level. Patients with multiple deltoid TrPs may show serious impairment of strength, or total inability to reach 90° of abduction.

When the patient complains of an acutely painful "catch" at approximately 15° of elevation in the anterolateral direc-

tion, it can be due to the *combination* of an area of severely tender enthesopathy at the attachment of the supraspinatus tendon (secondary to supraspinatus TrPs) and a taut band and TrP in the anterior deltoid muscle. The increased anterior deltoid TrP tension compresses the tender region against the acromion, producing an "impingement" syndrome which is greatly relieved by releasing anterior deltoid TrPs. Full function returns as soon as the enthesopathy clears following inactivation of the supraspinatus TrPs.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Few other muscles are so likely to receive forceful impacts directly against underlying bone. Impact trauma may occur from a hit by a tennis or golf ball, or from falling directly on the muscle.

The **anterior deltoid** can be traumatized by the repeated recoil of a gun when shooting. Trauma by sudden overload often occurs during a loss of balance when going down steps and reaching out to a bannister or railing to "catch a fall." Overload activation of TrPs by repetitive strain develops during prolonged lifting (holding a power tool at shoulder height), or episodic overexertion (unaccustomed deep sea fishing). Sorting mail into shoulder-height mailboxes for hours at a time or a similar work task can activate TrPs and can perpetuate them if the task is continued as before.

Overexercise may activate TrPs in the **posterior deltoid,** such as by excessive poling when skiing. This part of the deltoid muscle rarely develops TrPs alone as the result of activity, but usually in association with TrPs in other muscles.

Intramuscular injection of locally irritant solutions (e.g., B vitamins, penicillin, tetanus toxoid, diphtheria or influenza vaccine) into a latent TrP is likely to activate it and to cause a persistently painful shoulder.[53] This disability can be avoided (1) by preliminary palpation of the injection site for tender spots (latent TrPs) so as to avoid them, (2) by adding enough 2% procaine solution in the syringe before injection to bring it to a 0.5% solution of procaine, (3) by peppering the site with 1 ml of 0.5% (or stronger) procaine solution immediately after the injection, if continuing pain at the injection site indicates that a TrP was activated, (4) by routine stretch and spray of the muscle following any intramuscular injection, or (5) by selecting another injection site, such as the lateral thigh.

The **middle part** of the deltoid is less vulnerable to sustained overload than are the anterior and posterior parts of the muscle; however, TrPs in the middle part can be activated by vigorous (jerky) movements into abduction, particularly when repetitive.

The deltoid muscle may develop satellite TrPs from key TrPs in another muscle, especially the infraspinatus.

Perpetuating factors described in detail in Chapter 4 can cause poor response to treatment and persistence of deltoid TrPs.

8. PATIENT EXAMINATION

After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram representing the pain distribution described by the patient. The drawing should be in the style of the pain patterns in this volume using a copy of an appropriate body form found in Figures 3.2-3.4.

In order to identify active or latent TrPs that may be limiting range of motion and thus influencing dysfunction, the examiner should: (1) identify limited range of motion by performing specific range of motion testing for all parts of the deltoid, (2) take up slack to the point of tension before changing the position, (3) ask the patient *where* he or she feels the tension or where it hurts, and (4) search (palpate) *there* for a taut band and TrP. A TrP that is limiting range of motion and producing dysfunction has been called a **relevant** TrP.[9] For the deltoid specifically, motion should be tested by moving the humerus into horizontal adduction across the front of the chest (and while the arm is *in* that position, medially rotate the humerus and then laterally rotate it), by moving the humerus into horizontal abduction and lateral rotation, and finally by moving the humerus into extension with adduction behind the back.

In addition, testing of voluntary contraction to increase muscle tension can be performed by asking the patient to straighten the elbow and try to abduct the arm to 90°, first with the thumb up (palm forward) and then with the thumb down (palm backward). The thumb-up position is painful when fibers in the anterior part of the deltoid muscle harbor active TrPs; the thumb-down position is painful when it loads TrPs in the posterior part of the muscle.

Involvement of the anterior deltoid impairs performance of the Back-rub Test (see Fig. 29.3). When the patient with active TrPs in the posterior deltoid attempts the Mouth Wrap-around Test *[see* Fig. 18.2), the arm can reach over the head, but not behind it, because of pain induced by forceful contraction of the affected posterior deltoid fibers in the shortened position.

The deltoid muscle is tested for weakness as described by Kendall, *et al.*[32] The deltoid, without distinguishing its parts, has been identified as being prone to inhibition and weakness rather than hyperactivity and tenseness.[35] In our experience this is more likely to be characteristic of the anterior and posterior parts of the muscle than of the middle part. Trigger points in either the deltoid or in functionally related muscles can cause inhibition of a part of the deltoid muscle during customary activities but may not cause inhibition during an isolated test contraction of that part of the deltoid. Each part of the deltoid needs to be tested electromyographically in research studies of this kind of inhibition.

When the patient has shoulder pain and restricted motion suggestive of deltoid TrPs, the examiner should test the glenohumeral joint for normal joint play,[40] and if it is restricted, restore it.

9. TRIGGER POINT EXAMINATION

The deltoid is a superficial muscle, which simplifies detection of its palpable bands and vigorous local twitch responses. The relaxed muscle is examined by snapping palpation across the TrPs with the arm positioned in 30° of abduction. If the arm is abducted to 90° or more, as is sometimes recommended,[33 49] the taut bands and their twitch responses are less evident, if detectable at all.

The palpable bands associated with anterior deltoid TrPs (Fig. 28.1D) are readily palpable. The TrPs are usually located close to the cephalic vein, which separates the deltoid muscle and the clavicular portion of the pectoralis major muscle.

Trigger points in the middle part of the deltoid may develop almost anywhere since this middle part of the muscle is multipennate and its motor endplates are widely distributed (Figs. 28.1D and 28.3). The tenderness of enthesopathy of the supraspinatus attachment at the rotator cuff (which may be in response to chronic tension from TrPs and taut bands in the supraspinatus muscle) can be confused with tenderness of deltoid TrPs. When the arm is passively abducted to 90° the supraspinatus attachment is protected from digital pressure beneath the acromion while deltoid TrPs remain tender to palpation. With supraspinatus attachment enthesopathy, active abduction of the arm to 90° or more usually causes shoulder pain.

The posterior deltoid TrPs (Fig. 28.1D) are located along the posterior margin of the muscle, slightly more distally than those of the anterior part.[56]

One rarely finds TrP involvement of the deltoid muscle alone.

10. ENTRAPMENT

Entrapment of the axillary nerve due to TrPs in this muscle has not been observed.

11. DIFFERENTIAL DIAGNOSIS

Deltoid TrPs are commonly misdiagnosed as rotator cuff tears, bicipital tendinitis, subdeltoid bursitis, glenohumeral joint arthritis, impingement syndrome, or C_5 radiculopathy. These conditions need to be considered in diagnosis. They may cause deep shoulder pain and tenderness similar to deltoid TrP tenderness and referred pain, but they lack the specific physical signs of palpable bands and local

twitch responses in the muscle. Occasionally, one of these conditions coexists with deltoid TrPs; then both conditions must be treated.

Referred pain from any part of the deltoid muscle can mimic **pain arising in the glenohumeral joint,**[46] and thus, easily can be misdiagnosed as arthritis of that joint. A misdiagnosis can lead to the erroneous assumption that the joint is the source of pain and needs to be injected. Since TrPs in the anterior part of the deltoid can lie in the path of the anterior approach to the joint, these TrPs may be penetrated unintentionally, and thereby unknowingly inactivated, during the joint injection. The relief of pain thus obtained would further reinforce an incorrect conclusion that inflammation of the joint had been responsible for the pain. Any myofascial TrPs in the deltoid muscle should be inactivated and the response observed before deciding to inject the shoulder joint. Sometimes both the muscle and the joint must be treated.

When attention is directed only to the subacromial area of referred pain and tenderness, and active TrPs in any or all three parts of the deltoid muscle are overlooked, a diagnosis of "**subdeltoid bursitis**" is often rendered. A normal bursa may then be injected, to the neglect of the active deltoid TrPs, often resulting in a poor therapeutic result.

The **acromioclavicular joint** underlies the proximal attachment of the anterior deltoid muscle. Pain due to sprain, subluxation, or complete dislocation or separation of this joint mimics the pain pattern of anterior deltoid TrPs, or *vice versa. A* sprain of the acromioclavicular joint produces localized tenderness over the joint, rather than TrP tenderness in the deltoid muscle, and causes pain on passive mobilization of the joint by arm motion which rotates or elevates the scapula. Acromioclavicular subluxation and dislocation are more likely during sports activities and following an automobile accident in which the patient was holding on to the steering wheel or stretched the arm out for protection. Subluxation and dislocation are identified by increasing loss of mobility.[7] Bilateral standing X-ray examination with a weight held in each hand for comparison, or physical examination of the joint under local anesthesia, help to identify the depression and forward displacement of the clavicle in relation to the acromion.[12] For this joint problem, either conservative[12] or surgical[7] treatment is recommended. The acromioclavicular joint and the deltoid muscle may be involved simultaneously, both needing attention for complete pain relief and return of function.

Related Trigger Points

Active TrPs in the anterior part of the deltoid muscle are often associated with TrPs: (1) in the clavicular section of the pectoralis major (adjacent to the anterior deltoid); (2) in the biceps brachii; and (3) in the antagonistic, posterior part of the deltoid.

When an active TrP is found in the posterior deltoid, one should check the proximal third of the long head of the triceps brachii, the latissimus dorsi, and the teres major muscles for associated TrPs. The posterior deltoid is unlikely to be the only muscle affected with active TrPs, unless latent TrPs were activated by local injection of an irritant solution into the muscle, after which the TrP activity tends to be self-sustaining.

Because the deltoid muscle lies in the essential pain reference zones of both the infraspinatus and supraspinatus muscles, it rarely escapes the development of satellite TrPs when these two scapular muscles harbor active TrPs. Hong[23] reported that key TrPs in the scaleni or in the supraspinatus muscle can induce satellite TrPs in the deltoid muscle. The increased irritability of motor units in the reference zone was demonstrated experimentally by motor unit activity (referred spasm) in the anterior deltoid in response to pressure on an active TrP in the infraspinatus muscle that caused referred pain over the front of the shoulder. At the same time, recording needles in the biceps and triceps brachii showed electrical silence.[54]

If inactivation of deltoid TrPs restores abduction of the arm only to about 90°,

then any active supraspinatus TrPs should be located and eliminated. This usually restores the full range of arm motion in the overhead position, unless antagonists to abduction are also involved.

12. TRIGGER POINT RELEASE (Figs. 28.4 and 28.5)

Postisometric relaxation and reciprocal inhibition may be applied separately, as described in Chapter 3, Section 12, or in conjunction with spray and stretch. Lange[33] described deep massage for myogelosis (trigger points [TrPs]). We find the more gentle trigger point pressure release against the bony humerus to be effective for inactivating deltoid TrPs. Trigger point pressure applied with the deltoid relaxed in a position of ease (supported at about 45° of abduction) can be particularly effective.

For **spray and stretch** of the *anterior* part of the deltoid, the patient is seated, and the muscle is lengthened to take up its slack by horizontally abducting (horizontally extending) the arm and laterally rotating it at the shoulder joint (Fig. 28.4A). The vapocoolant spray pattern slowly traces the course of the muscle fibers distally and then covers the area of referred pain as illustrated. The operator takes up the slack by applying *gentle* stretch tension (Fig. 28.4A).

The *posterior* part of the deltoid is stretched by medially rotating the arm and moving it across the chest of the seated patient (Fig. 28.4B). Sweeps of the spray are directed over the posterior deltoid fibers in a distal direction to cover the muscle and include the pain reference zone in the pattern of Figure 28.4B. This position also stretches the supraspinatus and infraspinatus muscles. Both of these muscles should be included in the spray pattern, particularly if they are tender, or if a full range of shoulder motion is not achieved after release of the posterior part of the deltoid muscle by the spray-and-stretch procedure.

Two stretch positions and the spray pattern for the *middle* deltoid are shown and described in detail in Figure 28.5. In the anterior arm position of Figure 28.5A, the arm is laterally rotated but still includes considerable stretch of the posterior part of the deltoid muscle as well as the middle part. When stretching the middle deltoid in the posterior arm position of Figure 28.5B, one also may inactivate TrPs in the anterior part of the muscle. Since all parts of the muscle are stretched to some degree in these positions, the spray pattern should include the entire muscle.

Following any deltoid stretch procedure, the patient should move the arm slowly through three cycles of full active range of motion.

13. TRIGGER POINT INJECTION (Fig. 28.6)

The trigger points (TrPs) in the anterior, middle, and posterior parts of the deltoid muscle are readily identified by flat palpation, and then localized between the fingers and injected as in Figure 28.6. Active deltoid TrPs give readily visible or palpable local twitch responses, and they usually produce transient local aching and nearby referred pain when impaled by the needle. These phenomena indicate that the needle has effectively encountered at least one active locus of the TrP.[24]

Myofascial TrPs in the **anterior deltoid** lie near the midportion of this part of the muscle (Fig. 28.1) and often are close to the anterior border of the muscle where the cephalic vein lies subcutaneously between the deltoid and pectoralis major muscles. This landmark establishes which muscle has the TrP, which otherwise is not easy to identify because their fibers have adjacent attachments. When injecting these TrPs (Fig. 28.6A), one can avoid the vein by placing one finger of the palpating hand on it, penetrating the skin with the needle close to it, and directing the needle away from the vein and into the TrP.

The details of the basic injection technique are presented in Chapter 3, section 13.

Since the **middle deltoid** has multiple interlaced digitations, its taut bands are shorter than in the anterior and posterior parts of the muscle, and its TrPs are more scattered throughout the muscle.

Trigger points in the **posterior deltoid** are nearly always found in the midbelly region of the muscle, and those in the longer

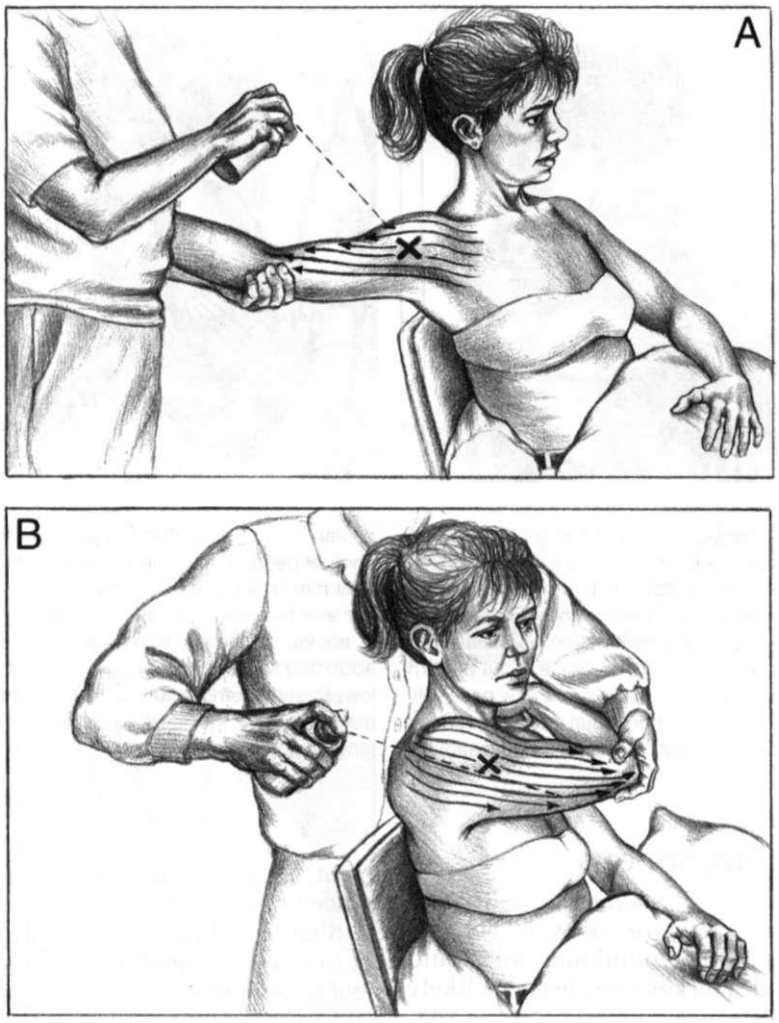


Figure 28.4. Stretch positions and patterns for application of intermittent cold *(arrows)* for trigger points (**Xs**) in the anterior and posterior parts of the right deltoid muscle, patient seated. **A,** anterior deltoid. While the patient leans back in a relaxed position and slowly breathes out, the operator applies vapocoolant as indicated and picks up slack in the muscle by passively moving the arm into horizontal abduction. The patient's elbow is flexed slightly to avoid excess stretch on the long head of the biceps. **B,** posterior deltoid. Rhythm is similar to **A,** above, except that the arm moves into horizontal adduction high across the chest. Postisometric relaxation facilitates muscle lengthening in these procedures.

fibers of the posterior margin can sometimes be localized for injection using bimanual palpation.

Upon completion of injection, counterpressure is applied for at least 1 minute to ensure hemostasis. Then the patient performs three cycles of active full range of motion specifically for the part(s) of the muscle injected.

A case report[8] described a patient with shoulder pain of 3-months' duration and unidentified onset that improved only slightly with treatment for supraspinatus tendonitis. The shoulder pain resolved 10 days later, following identification and injection of a deltoid TrP that responded to the injection with a huge local twitch.

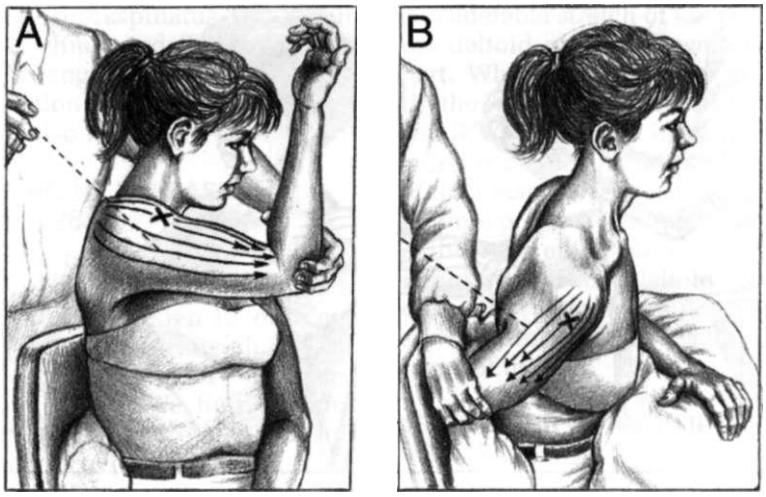


Figure 28.5. Stretch positions and pattern for application of intermittent cold *(arrows)* for trigger points (**X**s) in the middle part of the right deltoid muscle, patient seated. **A,** anterior arm position with patient leaning back in a relaxed position against the back support. The operator applies vapocoolant or ice in the pattern indicated and picks up slack in the muscle by passively moving the flexed arm into horizontal adduction as the patient slowly breathes out. Sequential application of coolant is resumed after the patient has slowly taken another deep breath. This cycle continues rhythmically until maximum available range is achieved. **B,** posterior arm position. Sequence and rhythm are similar to **A,** above, but the arm is extended behind the back and adducted as far as muscle tightness or the body will allow. Postisometric relaxation and reciprocal inhibition may often be included to advantage during these lengthening procedures.

14. CORRECTIVE ACTIONS

Any TrPs that refer pain to the deltoid region (and are therefore likely to activate satellite TrPs in the deltoid muscle) should be inactivated. The key muscles most likely to refer in this way are noted in Section 11.

Mechanical stress factors need to be corrected. The patient learns to lift heavy objects with the arm rotated so that the thumb is turned in the direction that unloads the affected part of the deltoid muscle (Section 8).

Similarly, any systemic perpetuating factors (see Chapter 4) should be identified and corrected, especially if the patient responds poorly to TrP therapy.

Activation of latent TrPs by intramuscular injection into the posterior deltoid may be avoided as outlined in Section 7.

The patient should take precautions on stairs, and prevent potential deltoid overload that can result from being forced to quickly grab a hand railing. Traversing stairs slowly while holding onto railings, in addition to visually watching foot placement, may prevent a near fall and recurrence of muscle overload.

Shooting enthusiasts should place a pad in front of the shoulder to minimize the direct trauma of gun recoil.

For continuing relief, daily passive stretching of the affected part of the muscle may be necessary. To self-stretch the anterior part of the deltoid, the patient is taught to do the middle- and lower-hand positions of the In-doorway Stretch Exercise (see Fig. 42.10), and the Against- doorjamb Exercise (see Fig. 30.7) slowly and without forcing. To self-stretch the posterior deltoid, the patient places the arm in the position of Figure 28.4B, grasps the elbow of the affected arm with the other hand and pulls it across the chest, while sitting under a warm shower with the water directed over the muscle.

SUPPLEMENTAL CASE REPORTS

The management of patients with deltoid TrPs is reported by Kellgren[31] and Lange.[34]

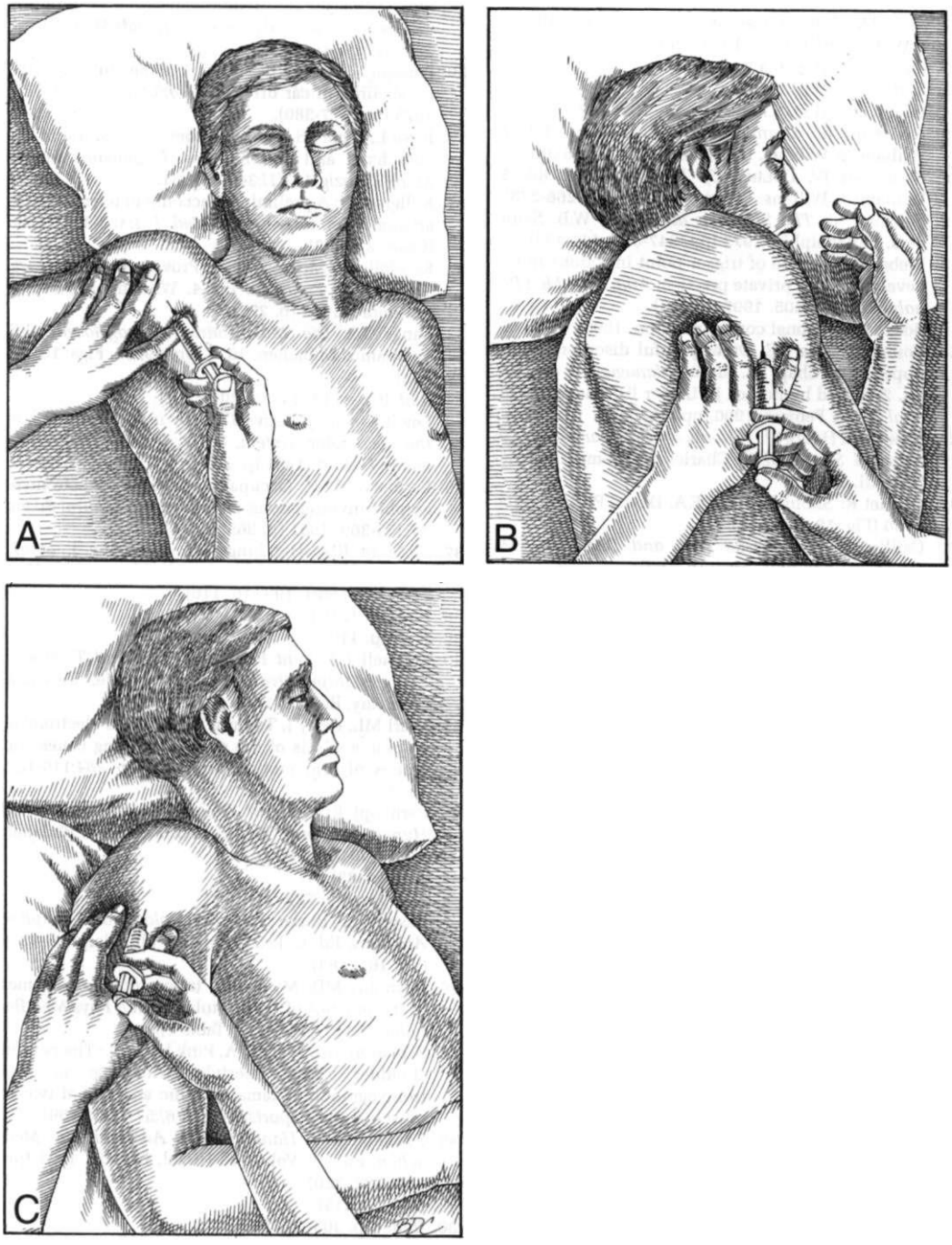


Figure 28.6. Injection of trigger points in the right deltoid muscle. **A,** anterior deltoid, with the patient supine. **B,** posterior deltoid, with the patient lying on the side opposite the involved muscle. **C,** middle deltoid, with the patient supported partially supine. See text for details.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:3 (Fig. 1.2).
2. *Ibid.* p. 385 (Fig. 6.39).
3. *Ibid.* p. 381 (Fig. 6.32).
4. *Ibid.* p. 383 (Fig. 6.35).
5. Anderson JE: *Grant's Atlas of Anatomy.* Ed. 7. Williams & Wilkins, Baltimore, 1978 (Fig. 6-38).
6. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 268-273).
7. Bateman JE: *The Shoulder and Neck.* W.B. Saunders, Philadelphia, 1972 (pp. 347-350, 424-433).
8. Bieber B: The role of trigger point injections in the development of private practice. *Arch Phys Med Rehabil 8(1):197-205,* 1997 (p. 203).
9. Boeve M: Personal communication, 1990.
10. Bonica JJ, Sola AE: Other painful disorders of the upper limb, Chap. 52. In *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, 1990, pp. 947-958.
11. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sports Skill.* Charles C Thomas, Springfield, 111. 1967.
12. Cailliet R: *Shoulder Pain.* FA. Davis, Philadelphia, 1966 (Fig. 19, pp. 82-85).
13. Cailliet R: *Soft Tissue Pain and Disability,* F.A. Davis, Philadelphia, 1977 (p. 152).
14. Christensen E: Topography of terminal motor innervation in striated muscles from stillborn infants. *Am J Phys Med* 38:65-78, 1959 (see pp. 73-74).
15. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 522).
16. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 61).
17. *Ibid.* (Fig. 523).
18. Define E, Hall RM: Active shoulder motion in complete deltoid paralysis. *J Bone Joint Surg* 42-A1:745-748, 1959.
19. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 45-55).
20. Gutstein M: Common rheumatism and physiotherapy. *Br J Phys Med* 3:46-50, 1940 (p. 47).
21. Hagberg M: Electromyographic signs of shoulder muscular fatigue in two elevated arm positions. *Am J Phys Med* 60(3):lll-121, 1981.
22. Hagberg M, Jonsson B: The amplitude distribution of the myoelectric signal in an ergonomic study of the deltoid muscle. *Ergonomics* 28:311-319, 1975.
23. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J* Muscuiosice *Pain* 2(1):29-59, 1994.
24. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Behabil* 73:256-263, 1994.
25. Inman VT, Saunders JB, Abbott LC: Observations on the function of the shoulder joint. *J Bone Joint Surg* 26:1-30, 1944.
26. Ito N: Electromyographic study of shoulder joint. *J Jpn Orthop Assoc* 54:1529-1540, 1980.
27. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W.B. Saunders, Philadelphia, 1991 (p. 84).
28. Jonsson B, Hagberg M: The effect of different working heights on the deltoid muscle: a preliminary methodological study. *Scand J Rehab Med, Suppl.* 3:26-32, 1974.
29. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving. *Ergonomics* 28:375-388, 1975 (pp. 377-380).
30. Jozsa L, Demel S, Reffy A: Fibre composition of human hand and arm muscles. *Gegenbaurs morph Jahrb, Leipzig* 227(2);34-38, 1981.
31. Kellgren JH: A preliminary account of referred pains arising from muscle. B*r Med J* 2:325-327, 1938 (Cases 2 and 3).
32. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 273).
33. Lange M: *Die Muskelharten (Myogelosen):* J.F Lehmanns, Miinchen, 1931 (pp. 49, 66, Figs. 10, 27, 40b).
34. *Ibid.* (Cases 14, 15, 18, 20-22).
35. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (p. 24).
36. Lundervold A: Occupation myalgia. Electromyographic investigations. *Acta Psychiatr Neurol Scand* 26:359-369, 1951 (p. 365, Fig. 5).
37. McMinn RM, Hutchings RT, Pegington J, ef *al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 116).
38. *Ibid.* (p. 121C).
39. *Ibid.* (p. 119).
40. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
41. Pearl ML, Perry J, Torburn L, *et al.:* An electromyographic analysis of the shoulder during cones and planes of arm motion. *Clin Orthop* 284:116-127, 1992.
42. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (p. 11).
43. *Ibid.* (p. 33).
44. *Ibid.* (pp. 54, 72).
45. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 161, 163).
46. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981 (Table 1).
47. Scovazzo ML, Browne A, Pink M, *et al.:* The painful shoulder during freestyle swimming: an electromyographic cinematographic analysis of twelve muscles. *Am J Sports Med 19(6j:577-582,* 1991.
48. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (pp. 280, 282, 320).
49. *Ibid.* (p. 315).
50. *Ibid. (*pp. 303, 322).
51. Steinbrocker O, Isenberg SA, Silver M, *et al:* Observations on pain produced by injection of hypertonic saline into muscles and other supportive tissues. *J Clin Invest* 32:1045-1051, 1953 (p. 1046).
52. Travell J: Ethyl chloride spray for painful muscle spasm. *Arch Phys Med Rehabil* 33:291- 298, 1952 (p. 293).

53. Travell J: Factors affecting pain of injection. *JAMA* 258:368-371, 1955.
54. Travell J, Berry C, Bigelow N: Effects of referred somatic pain on structures in the reference zone. Fed *Proc* 3:49, 1944.
55. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med 11*:425-434, 1952 (p. 428).
56. Winter Z: Referred pain in fibrositis. *Med Rec* 157:34-37, 1944 (p. 4).
57. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment*. Ed. 2. Little, Brown & Company, Boston, 1988 (p. 211, Fig. 12-2).

CHAPTER 29
Coracobrachialis Muscle

HIGHLIGHTS: Trigger points (TrPs) in the coracobrachialis muscle require more skill for their identification and management than those in most other muscles. Involvement of the coracobrachialis muscle usually is not apparent until TrP activity has been resolved in associated muscles, such as the anterior deltoid, biceps brachii (short head) and the triceps brachii (long head). **REFERRED PAIN** from TrPs in this muscle appears over the anterior aspect of the proximal humerus and in an interrupted pattern of pain that extends down the back of the arm and dorsum of the forearm to the back of the hand, but skips the elbow and wrist. **ANATOMY:** Attachments of the coracobrachialis are proximally to the coracoid process and distally to the midportion of the humerus. **FUNCTION** is to assist flexion and adduction of the arm at the glenohumeral joint. **SYMPTOMS** include disabling pain with little restriction in the range of motion. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the coracobrachialis usually occur in conjunction with the involvement of associated muscles. **PATIENT EXAMINATION** reveals a painful Back-rub Test, and pain when the patient attempts to raise the arm into full flexion at the glenohumeral joint and then move the arm behind the ear. **TRIGGER POINT EXAMINATION** is by direct palpation of the coracobrachialis muscle deep to the pectoralis major muscle and medial and deep to the short head of the biceps brachii. The tenderness of TrPs is more distal, and should be distinguished from, trigger areas caused by enthesopathy. **ENTRAPMENT** by this muscle has been observed repeatedly, and is often attributed to compression of the musculocutaneous nerve by a heavily exercised, hypertrophied coracobrachialis muscle. A latent TrP origin of the nerve compression was not tested. **TRIGGER POINT RELEASE** is performed with a stretch and spray technique similar to that for TrPs in the anterior deltoid. When applying TrP pressure release, the taut band of a TrP in the muscle must be distinguished from the adjacent major upper limb nerves. **TRIGGER POINT INJECTION** in this muscle involves an anterior approach through the deltoid muscle with tactile guidance of the needle from the palpating hand after identification of the adjacent neurovascular structure. **CORRECTIVE ACTIONS** include relief of excessive stress from lifting, and home use of the In-doorway Stretch Exercise.

1. REFERRED PAIN
(Fig. 29.1)

Pain is referred over the anterior deltoid area and down the posterior aspect of the arm (Fig. 29.1), concentrating over the triceps brachii, the dorsum of the forearm, and over the dorsum of the hand but often skipping the intervening elbow and wrist joints. The line of pain may extend to the tip of the middle finger. As in other muscles, when the trigger points (TrPs) are more active the extent of referred pain is greater, the pain is more intense, pain is more likely to persist at rest, the TrPs are more tender, the taut bands are more tense, and local twitch responses (LTRs) are more vigorous.

2. ANATOMY
(Fig. 29.2)

Proximally, the coracobrachialis arises from the apex of the coracoid process in common with the tendon of the short head of the biceps brachii (Fig. 29.2), and from the intermuscular septum between the two muscles. ***Distally,*** the coracobrachialis fastens to the medial surface of the humerus just proximal to the midpoint along the shaft of the bone, between the attachments of the triceps and brachialis muscles;[6] distally, the biceps crosses the elbow joint.

The brachial neurovascular bundle passes deep to (behind) the tendinous attachment of the pectoralis minor at the coracoid process and continues down the arm next to the coracobrachialis muscle.

Variations include total absence of the muscle and extension of its humeral attachment to the medial epicondyle.[3]

Supplemental References

Other authors have illustrated the coracobrachialis as seen from the front,[7,8,16,19,27] from the medial aspect,[1] including the muscle's relation to the brachial neurovascular structures,[2,20,28] and in cross section.[11,23]

3. INNERVATION

The coracobrachialis muscle is innervated by a branch of the musculocutaneous nerve that contains fibers from the sixth and seventh cervical nerves and that separates from the nerve trunk before it penetrates the muscle.[6] As the musculocutaneous nerve penetrates the midportion of the coracobrachialis muscle, the nerve may divide the coracobrachialis into clearly defined superficial and deep portions.[3]

4. FUNCTION

The coracobrachialis helps to flex and adduct the arm at the glenohumeral joint.[3,6,13,14,26] When contracted by faradic stimulation while the arm was held in the abducted position, the muscle forcefully drew the humerus toward the glenoid cavity.[10]

This muscle is elongated by both medial rotation and lateral rotation, and has been reported to assist in returning the arm to the neutral position from lateral rotation[3,26] and from medial rotation.[26] The coracobrachialis also has been reported to assist extreme abduction.[3,28] Another author proposed that the coracobrachialis may supply a part of the required adduction stabilization force when the humerus is forced into abduction.[25]

5. FUNCTIONAL UNIT

The coracobrachialis acts synergistically with the anterior deltoid, the short head of the biceps brachii, and the pectoralis major in flexion and adduction of the arm. Antagonists for flexion are the posterior deltoid, latissimus dorsi, teres major, and long head of the triceps.

6. SYMPTOMS

For coracobrachialis TrPs, the primary complaint is upper limb pain, particularly in the front of the shoulder and in the arm posteriorly. The patient experiences pain when reaching behind the body, across the low back, as in the Back-rub Test (Fig. 29.3), which stretches the muscle because of the strong medial rotation with exten-

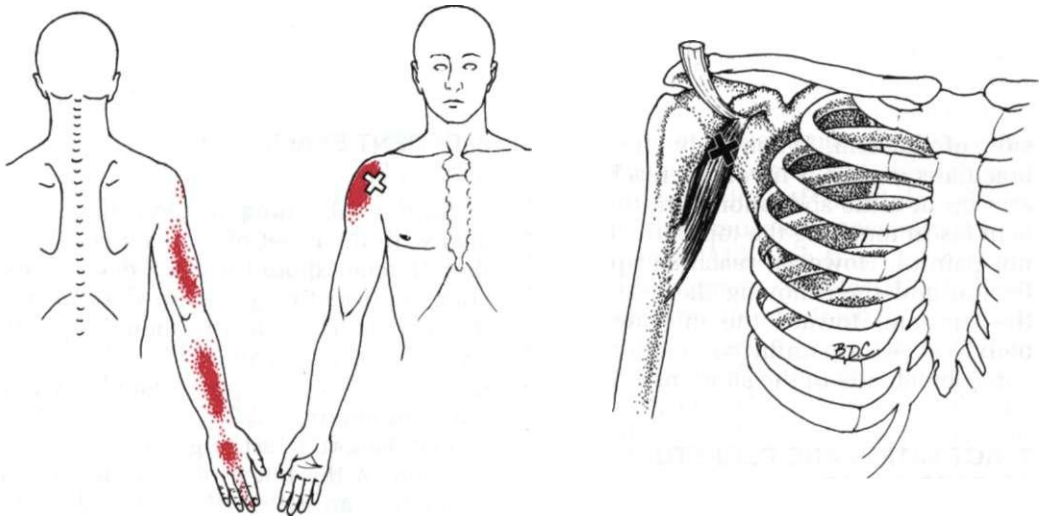


Figure 29.1. Pain pattern *(red)* referred from a trigger point (**X**) in the right coracobrachialis muscle. Trigger points are likely to be found as far distally as the middle of the muscle belly. In patients with milder involvement, the pain may extend only to the elbow.

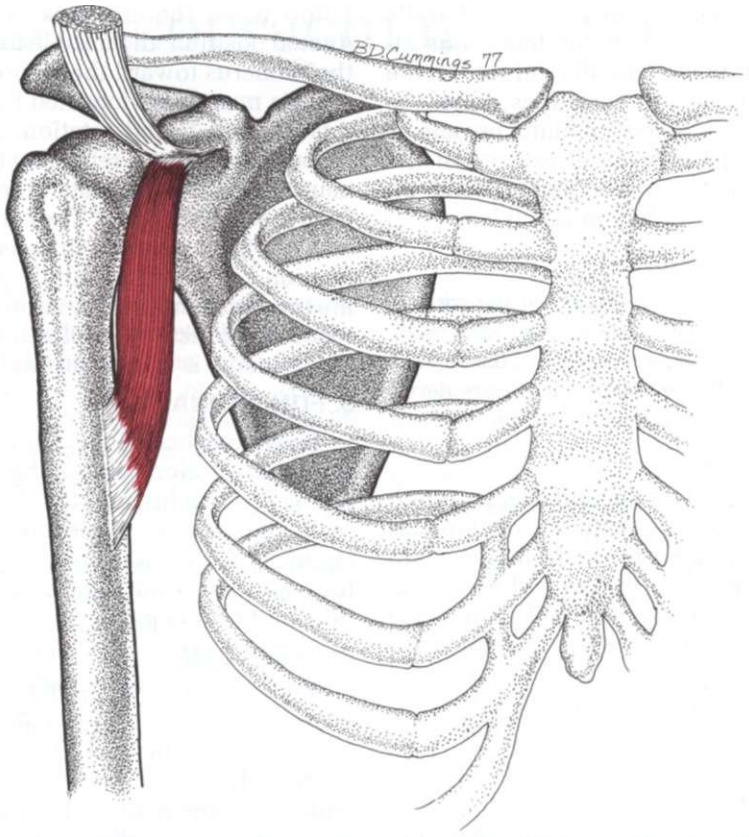


Figure 29.2. Usual attachments of the coracobrachialis muscle *(red):* proximally to the tip of the coracoid process, and distally to a line along the humerus extending almost to midshaft. The short head of the biceps brachii (which has been cut and turned up) arises in common with the coracobrachialis at the coracoid process.

sion of the arm. When only the coracobrachialis muscle is involved, reaching the arm up in some abduction with the elbow bent (as in touching the top of the head) is not painful. However, reaching up in full flexion and then moving the arm behind the ear (and toward the midline of the body) causes a painful contraction of the coracobrachialis in the shortened position.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Active TrPs in this muscle develop secondarily to active TrPs in related muscles of its functional unit, as listed above.

8. PATIENT EXAMINATION (Fig. 29.3)

After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram representing the pain described by the patient. The drawing should be in the style of the pain patterns in this volume, using a copy of an appropriate body form found in Figures 3.2-3.4.

The Back-rub Test (Fig. 29.3) reveals restriction in the range of shoulder motion when there are TrPs in the involved coracobrachialis muscle. This test puts the muscle in a painful position due to the extreme medial rotation and extension of the arm.

With coracobrachialis TrPs, the arm can be flexed as far as the ear, but not behind it. Pain is caused by contracting the muscle in the shortened position.

Flexion of the humerus may be slightly weak. To test the strength of the coracobrachialis, the patient first elevates the arm to about 45° of flexion with lateral rotation. The patient's elbow should be flexed and the forearm fully supinated to minimize biceps assistance. Then the operator applies pressure at the distal humerus, pressing downward and slightly outward (in the direction of extension and slight abduction).[14] Inability to adequately resist such pressure indicates weakness of the coracobrachialis. Maximal resistance effort by the patient is likely to elicit pain if the coracobrachialis muscle harbors active TrPs.

Stretching the involved coracobrachialis by passively extending the arm at the shoulder joint causes pain (particularly when an abduction component is added), as does loading the muscle by resisting active flexion of the arm at the shoulder.[17]

When one tests upper limb range of motion and finds what appears to be a soft tissue restriction, it can be helpful *during the testing* to ask the patient whether he or she feels tightness (or pain) in any particular area and, if so, to touch or point to the area of tightness. Sometimes, palpation of the indicated area of tightness reveals a taut band harboring a TrP. A TrP found in this manner has been referred to as a *relevant* TrP;[4] it can produce dysfunction and it may be latent (producing no pain complaint) rather than active. A relevant TrP in the coracobrachialis may be revealed by passively moving the humerus into simultaneous extension and abduction, particularly if lateral rotation is introduced. The precise location of the perceived tightness can help to distinguish coracobrachialis tension from biceps brachii tension.

When the patient has pain on movement of the glenohumeral joint, the joint should be tested for normal joint play as described by Mennell.[21] When there is restricted arm movement, it is wise also to examine for normal joint play in the acromioclavicular joint and in the sternoclavicular joint.[21]

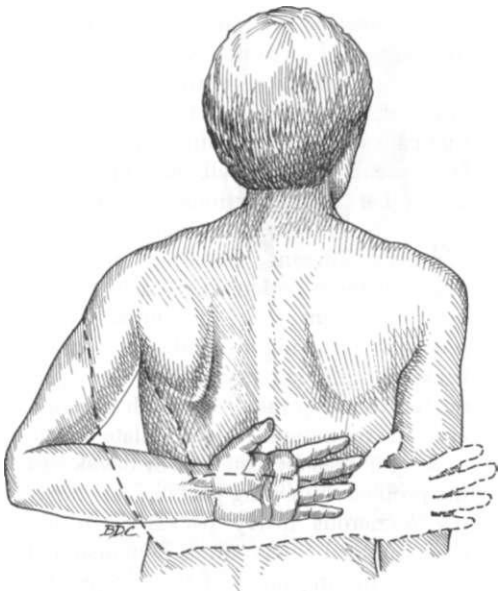


Figure 29.3. Back-rub Test for trigger points in the left coracobrachialis muscle. Before treatment of the coracobrachialis, the patient's knuckles usually can reach only to the midline of the back because of aching pain in the muscle with full medial rotation of the arm in extension. Following successful inactivation of these trigger points, the wrist can reach across the full width of the back (*dotted outline).*

9. TRIGGER POINT EXAMINATION
(Fig. 29.4)

A TrP may have been discovered by passively moving the arm through a lengthened (stretch) range for the coracobrachialis muscle, as described in Section 8 of this chapter. However, involvement of the coracobrachialis is usually discovered when the patient returns following successful inactivation of multiple TrPs in other shoulder muscles, especially the anterior deltoid. Although there is no recurrence of tenderness or detectable LTRs in the muscles previously treated, the patient complains of severe pain, and deep tenderness remains in the region of the anterior deltoid muscle. Careful examination reveals tenderness that lies deeper than the deltoid.

Two areas of tenderness may be encountered in this muscle. Central myofascial TrP tenderness is located approximately midmuscle, and attachment TrP tenderness

is located in the region of the proximal musculotendinous junction (although it also can be distal); the attachment TrP tenderness most likely represents enthesopathy secondary to sustained tension caused by taut bands of the TrP.

Coracobrachialis central TrPs are found when palpating the muscle against the humerus by sliding the finger into the axilla deep to the deltoid and pectoralis major (Fig. 29.4). The tip of the digit encounters the adjacent bellies of the short head of the biceps brachii and, more posteriorly, the coracobrachialis at a level where about half of the biceps fibers have become attached to their common tendon. The axillary neurovascular bundle passes along the coracobrachialis[2] and must be displaced posteriorly to permit the digit to explore the fibers of the coracobrachialis muscle for taut bands by strumming the muscle against the humerus. The neurovascular bundle lies posterior to the attachment of the muscle on the humerus. The central TrPs may be found approximately midmuscle, more distal than the location indicated in Figure 29.1. The location shown in the figure is closer to a trigger area produced by enthesopathy. The attachment trigger area also may feel indurated and respond to digital pressure with referred pain.

Local twitch responses elicited by snapping palpation of the taut band associated with the TrP (avoid the neurovascular bundle) are generally palpable rather than visible in this deep muscle, and they confirm the presence of a TrP.

10. ENTRAPMENT

One would expect that TrPs in the coracobrachialis muscle could cause entrapment of the musculocutaneous nerve as it passes through that muscle en route to the biceps brachii and brachialis.[6,9,28] Clinical symptoms of this entrapment have been well documented in case reports.

Pecina and Bojanic[22] reported on an oarsman who practiced 500 press-ups daily and who presented with reduced size and strength of the biceps brachii muscle, absent biceps tendon reflex, reduced biceps muscle tone, and diminished sensation of the lateral surface of the forearm. Electrodiagnostic testing showed prolonged distal latencies and decreased amplitude of evoked responses in the biceps and brachialis muscles, indicating musculocutaneous nerve compression. Three months after stopping the daily press-ups, muscle mass and strength, and sensation in the forearm had returned. Electrodiagnostic studies showed improvement. Since the coracobrachialis muscle function was unimpaired, entrapment of the musculocutaneous nerve must have been distal to the motor branch to the coracobrachialis. The entrapped part of the nerve was the part that traverses the coracobrachialis muscle.

Additional case reports[5, 18] describe similar painless loss of musculocutaneous nerve function distal to the coracobrachialis muscle following heavy exercise (weight lifting, and building a rock wall) with functional recovery within a few months after cessation of the strenuous activity. Exercise-induced hypertrophy of the coracobrachialis muscle that caused pressure compromise of the nerve as it penetrated the muscle was assumed to be responsible in these cases. No mention was made of examining the coracobrachialis muscle for TrPs. Latent TrPs that cause no clinical pain complaint can be associated with well developed taut bands that produce serious dysfunction.

Reports of four additional cases[12, 15] identify different initiating stressors. Three of these patients gave a history of vigorous extension of the elbow (by repeatedly throwing a football, vigorous backhand playing racquetball, or vigorously extending the elbow with the forearm in pronation to gesticulate while making a speech). The fourth developed the problem following packing and carrying numerous heavy packages. In her case, surgical intervention demonstrated marked impingement of the biceps tendon on the musculocutaneous nerve with full elbow extension. Neurolysis and decompression of the nerve provided relief. The others recovered by avoiding further vigorous activity.

11. DIFFERENTIAL DIAGNOSIS

Entrapment of the musculocutaneous nerve by the coracobrachialis muscle can

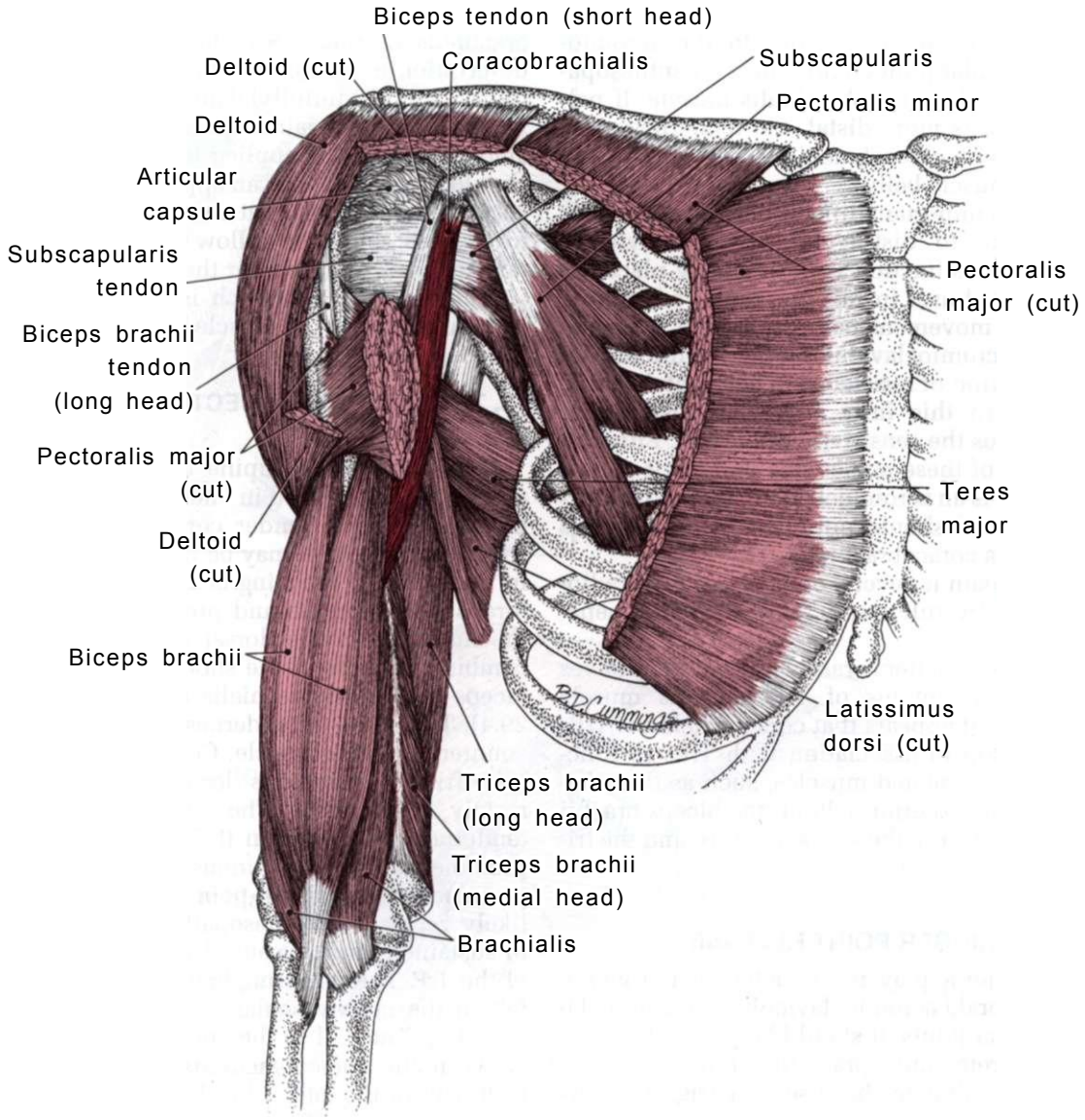


Figure 29.4. Muscular regional anatomy of the right shoulder, seen from the front. The coracobrachialis muscle *(dark red)* crosses superficial to the attachments of the subscapularis, latissimus dorsi, and teres major muscles, but lies deep to the pectoralis major and anterior deltoid muscles. For clarity, the serratus anterior muscle is not shown. The coracobrachialis lies medial to the short head of the biceps, and is palpated for trigger points against the humerus in the anterior axillary fossa, deep to the pectoralis major muscle.

be distinguished from a C_5 or C_6 radiculopathy or from a lateral cord lesion of the brachial plexus by the sparing of the coracobrachialis muscle.

Three cases of isolated tear of the coracobrachialis muscle were reported.[29] All occurred during forceful extension of the arm with the humerus in lateral rotation and abduction.

Diagnoses that can be confusingly similar to coracobrachialis TrPs include C_7 radiculopathy, carpal tunnel syndrome, subacromial bursitis, supraspinatus tendinitis, and, most commonly, acromiocla-

vicular joint dysfunction. Tenderness elicited slightly inferior to the acromioclavicular joint could reflect an enthesopathy of the coracobrachialis muscle. If palpation is more distal, the tenderness may be from coracobrachialis central TrPs in the muscle belly.

An important differential diagnostic procedure to distinguish acromioclavicular joint dysfunction is to passively place the affected arm in full horizontal adduction. This movement compresses the region of the acromioclavicular joint. Additional application of resistance to horizontal abduction in this fully adducted position increases the sensitivity of the test. Either or both of these maneuvers will elicit pain if there is an acromioclavicular joint dysfunction and should not elicit pain if there is only a coracobrachialis TrP.

If pain is severe, one may need to radiologically rule out acromioclavicular separation.

Since patients rarely present themselves with symptoms of TrPs in this muscle alone, it appears that coracobrachialis TrPs develop in association with TrPs in functionally related muscles, such as the anterior or posterior deltoid, the biceps brachii (short head), the supraspinatus, and the triceps brachii (long head).

12. TRIGGER POINT RELEASE

If joint play is restricted in the glenohumeral, acromioclavicular, or sternoclavicular joints, it should be restored.

Stretch and spray are applied in a manner similar to that used for trigger points (TrPs) in the anterior deltoid (see Fig. 28.4A) (the same stretch position is used). For the coracobrachialis muscle, the spray pattern shown for the anterior deltoid is carried closer to the axilla, extended over the back of the arm and forearm, and over the dorsum of the hand to the tip of the middle finger.

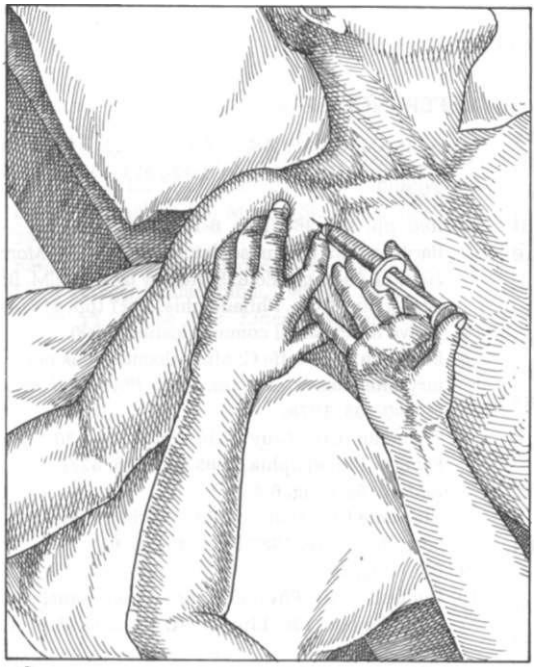
Release of coracobrachialis TrPs using pressure release or other techniques involving local application of pressure requires a knowledge of the close relation of this muscle to adjacent upper limb nerves including the median, ulnar, musculocutaneous, medial antebrachial cutaneous nerves, and the nerve to the coracobrachialis muscle.[22] See the examination description in Section 8. These nerves can be easily (and painfully) damaged by forceful compression against the humerus. The amount of pressure applied for TrP release should be gentle. One can apply light pressure to the TrP to encounter resistance, wait for release, and then follow the tissue to take up slack to encounter the next barrier.

Another form of stretch is to do local manual stretch of the muscle by massaging the muscle lengthwise.

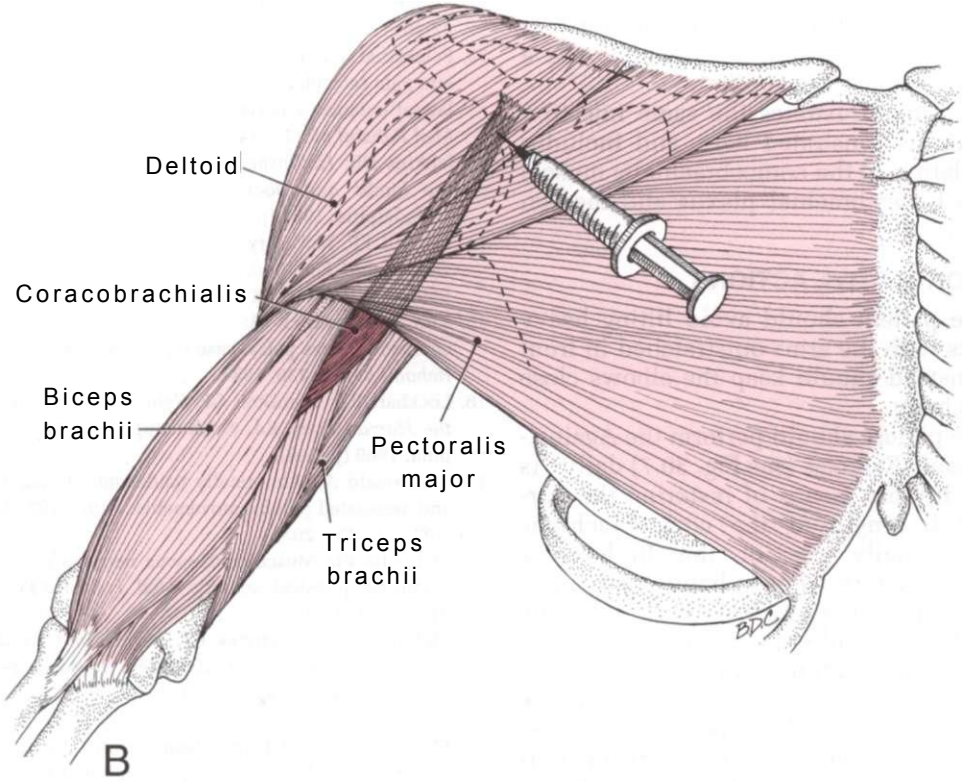
13. TRIGGER POINT INJECTION
(Fig. 29.5)

With the patient supine and with the arm by the side placed in lateral rotation at the shoulder, the tender coracobrachialis trigger points (TrPs) may be palpated deep in the axilla by reaching beneath the pectoralis major muscle and pressing against the humerus on the dorsal aspect of the combined bundle of the short head of the biceps and coracobrachialis muscles (Fig. 29.4). Two areas of tenderness may be encountered in this muscle. Central myofascial TrP tenderness is located approximately midmuscle. The other area of tenderness is located in the region of the proximal musculotendinous junction or proximal attachment point, and most likely represents enthesopathy secondary to sustained tension caused by taut bands of the TrP. The pulsating brachial artery is felt in the neurovascular bundle that lies dorsal and medial to the coracobrachialis, between the coracobrachialis and the attachment of the lateral head of the triceps to the humerus. The needle must avoid this structure which must be clearly identified before proceeding. The needle then is inserted through the pectoralis major or the anterior deltoid, directed toward the tender area that is localized with the operator's other hand.

Figure 29.5 illustrates injection in the trigger area of enthesopathy which, when present, should be injected to expedite relief of pain and recovery of normal function. When this enthesopathy is present, it is essential that midmuscle TrPs in the coracobrachialis and/or short head of the biceps brachii also be inactivated. When



A



B

Figure 29.5. Injection of a tender trigger area of enthesopathy located in the region of the musculotendinous junction. The corresponding trigger point is located more nearly midmuscle and requires essentially the same technique. This attachment trigger area in the coracobrachialis muscle is injected through the anterior deltoid muscle at the level of the greater tubercle of the humerus. The central trigger point in the midmuscle region is more distal and may be reached through the anterior deltoid or the pectoralis major muscle. The neurovascular bundle must be identified before injection and avoided. **A,** injection technique. **B,** schematic diagram showing injection of the coracobrachialis muscle *(dark red)* through the deltoid and pectoralis major muscles.

the needle encounters an active locus of the TrP, it usually elicits a confirmatory LTR. The patient may feel a brilliant flash of referred pain when the needle strikes the TrP; this pain may be similar to that produced by encountering a nerve with the needle. Infiltration of the local anesthetic may cause temporary weakness and anesthesia in the distribution of the musculocutaneous nerve with prompt recovery in 15 or 20 min if 0.5% procaine solution was used for injection. Steroids and long acting anesthetics are *not* recommended in this location because of the proximity of the major neurovascular structures.

The midmuscle TrP is best injected with the fingers of one hand palpating the coracobrachialis posterior to the pectoralis major muscle, taking care not to penetrate the finger with the needle.

Following injection, spray and stretch are applied, three cycles of active full range of motion for that muscle are performed to restore normal function, and a moist warm pack is applied.

Rachlin[24] illustrates the same injection location as illustrated here, and includes the relation of the coracobrachialis muscle to the lower brachial plexus and median nerve.

14. CORRECTIVE ACTIONS

The patient should avoid lifting heavy objects with the arms outstretched in front and instead should keep the elbows close to the body.

The patient should perform the Against-door-jamb Exercise (*see* Fig. 30.7) daily. As an additional means of restoring the normal full length of the coracobrachialis muscle, daily use of the In-doorway Stretch Exercise, lower hand-position, is helpful *(see* Fig. 42.9A). However, one should be careful not to overstretch.

Local application of moist heat to the muscle just before, or after, the passive stretch exercises reduces postexercise soreness. If this soreness is a problem, an alternate-day program may be wise, and may minimize this reaction. At any rate, the patient should be checked to assure that he or she is not stretching too vigorously by using excessive body weight in the stretch.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:37-, 373, 376 (Figs. 6-22, 6-26).
2. *Ibid.* pp. 370, 388 (Figs. 6-17, 6-43).
3. Bardeen CR: The musculature, Sect. 5. In: *Morris's Human Anatomy.* Ed 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (pp. 413, 414).
4. Boeve M: Personal communication, 1990.
5. Braddom RL, Wolfe C: Musculocutaneous nerve injury after heavy exercise. *Arch Phys Med Rebabil* 59:290-293, 1978.
6. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 526, 527).
7. *Ibid.* (p. 520, Fig. 6-45).
8. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 49, 55, 61).
9. *Ibid.* (Fig. 56).
10. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 87).
11. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 32-36).
12. Felsenthal G, Mondell DL, Reischer MA, et *al:* Forearm pain secondary to compression syndrome of the lateral cutaneous nerve of the forearm. *Arch Phys Med Rehabil* 65.139- 141, 1984.
13. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 112).
14. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 267).
15. Kim SM, Goodrich JA: Isolated proximal musculocutaneous nerve palsy: case report. *Arch Phys Med Rehabil* 65:735-736, 1984.
16. Lockhart RD, Hamilton GF, Fyfe FW: *Anatomy of the Human Body.* Ed. 2. J.B. Lippincott, Philadelphia, 1969 (p. 206).
17. MacDonald AJ: Abnormally tender muscle regions and associated painful movements. *Pain* 8:197-205, 1980 (pp. 202, 203).
18. Mastaglia FL: Musculocutaneous neuropathy after strenuous physical activity. *Med J Aust 145 (3-4).*153-154, 1986.
19. McMinn RM, Hutchings RT, Pegington J, *et al: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 126).
20. *Ibid.* (p. 127).
21. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
22. Pecina M, Bojanic I: Musculocutaneous nerve entrapment in the upper arm. *Int Orthop 17(4):232-234,* 1993.
23. Pernkopf E: *Atlas of Topographical and Applied*

Human Anatomy, Vol. 2. W.B. Saunders, Philadelphia, 1964 (Figs. 44, 60, 61).

24. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 330-333).
25. Rasch PJ: *Kinesiology and Applied Anatomy.* Ed. 7. Lea & Febiger, Philadelphia, 1989 (p. 123).
26. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 165, 166).
27. Spalteholz W: *Handatlas der Antomie des Men-schen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (pp. 320, 321).
28. *Ibid.* (p. 753).
29. Wardner JM, Geiringer SR, Leonard JA: Coracobrachialis muscle injury [Abstract]. *Arch Phys Med Rehabil 69:783,* 1988.

CHAPTER 30
Biceps Brachii Muscle

HIGHLIGHTS: **REFERRED PAIN** from trigger points (TrPs) in the biceps brachii is projected mainly upward, over the muscle to the front of the shoulder with spillover pain patterns in the suprascapular region and the antecubital space. **ANATOMY:** Proximally, the attachments are to the superior margin of the glenoid cavity (long head) and to the coracoid process (short head) of the scapula. Distally, the muscle attaches to the tuberosity of the radius. The biceps brachii functions across three joints: the shoulder (glenohumeral), elbow (humeroulnar and humeroradial), and the proximal radioulnar (within the elbow joint capsule). **FUNCTION** of this two-headed, multijoint muscle is complex. The biceps brachii muscle flexes the forearm at the elbow, assists flexion of the arm at the shoulder, and may assist abduction of the laterally rotated arm. It powerfully assists supination of the forearm when the forearm is not fully extended at the elbow. **SYMPTOMS** are restricted motion, superficial aching pain of the anterior shoulder, and sometimes soreness to pressure over the bicipital tendon and at its glenoid attachment. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** occur as the result of acute overexertion or of repeated strain of the muscle, as a satellite of a key TrP, or due to a prolonged fixed position as during surgery. Tenosynovitis of the bicipital tendon may develop secondary to TrPs in the belly of the long head of the muscle. **PATIENT EXAMINATION** for range-of-motion testing can be misleading if the muscle is not lengthened simultaneously across all three of the joints that it crosses. The Biceps-extension Test meets this need. **TRIGGER POINT EXAMINATION** is most effectively performed by pincer palpation with optimal adjustment of muscle tension. **DIFFERENTIAL DIAGNOSIS:** Conditions that mimic biceps TrP findings include bicipital tendinitis, subdeltoid bursitis, C_5 radiculopathy, bicipital bursitis, and glenohumeral arthritis. Related TrPs can develop in the brachialis, supinator, coracobrachialis, and triceps brachii muscles. **TRIGGER POINT RELEASE** can be accomplished with several different manual methods. Using spray and stretch requires that the biceps brachii be passively stretched by abducting the arm to 90°, by extending it posteriorly with the arm laterally rotated at the shoulder joint, and by extending the forearm at the elbow while pronating the forearm. At the same time, the vapocoolant spray is applied cephalad over the muscle and its zone of referred pain. **TRIGGER POINT INJECTION** inactivates the biceps brachii TrPs, but true bicipital tenosynovitis may persist. Injection of the tendon area may then relieve the symptoms. **CORRECTIVE ACTIONS** include lifting objects with the forearms in pronation to unload the biceps brachii muscle. The Against-door-jamb Exercise with respiratory augmentation can effectively stretch the muscle to inactivate and avoid recurrence of biceps brachii TrPs.

1. REFERRED PAIN
(Fig. 30.1)

Trigger points (TrPs) in the biceps brachii are usually found in the midportion of the muscle. They refer pain upward over the muscle and over the anterior deltoid region of the shoulder;[15] occasionally the pain skips to the suprascapular region (Fig. 30.1). These central TrPs also may initiate another additional pattern of milder pain downward in the antecubital space.

Experimental injection of 6% sodium chloride solution into the biceps tendon at the antecubital space in 10 healthy subjects caused pain that was referred locally and also proximally over the biceps muscle (including the acromion in one case). Other phenomena that were referred distally to some part of the volar forearm and hand included deep tenderness, erythema, paresthesia, pallor, and a feeling of weakness.[37]

2. ANATOMY

(Fig. 30.2)

The biceps brachii muscle spans the shoulder, elbow, and proximal radioulnar joints (Fig. 30.2).

Proximally the **long head** of the biceps brachii attaches to the superior margin of the glenoid cavity of the scapula (Fig. 30.2). Its tendon lies in the intertubercular groove, passes through the glenohumeral joint space over the head of the humerus, and attaches to the supraglenoid tubercle at the upper margin of the glenoid cavity. The tendon of the long head can be palpated against the head of the humerus only with the arm in lateral rotation. Otherwise, it is covered by the acromion. The intracapsular portion of the tendon is well visualized by glenohumeral joint arthroscopy. The **short head** attaches ***proximally*** to the coracoid process of the scapula, remaining free of the glenohumeral joint capsule.

Distally the common tendon of both heads of the muscle attaches to the tuberosity of the radius. The attachment faces the ulna when the forearm is supinated,[7] but in pronation the tendon wraps more than halfway around the radius.[33]

The median and radial nerves lie, respectively, along the medial and lateral borders of the distal portion of the biceps and brachialis muscles.[9,30]

The motor endplates in the biceps brachii of a stillborn infant were found to form a distinct band through the middle of the two heads of the muscle.[6] The endplates in a mature muscle formed a somewhat ragged V-shaped band through the middle of the two heads.[2] Postmortem examination of 6 biceps brachii muscles for innervation and corresponding distribution of motor endplates indicated that each head was divided into three distinct longitudinal compartments.[35] The endplate zone of the long head is located slightly more proximal than that of the short head because of the difference in tendon arrangement. The functional significance of multiple compartments has not yet been established.

The number of type 1 (slow twitch) fibers compared to the number of type 2 (fast twitch) fibers in normal biceps

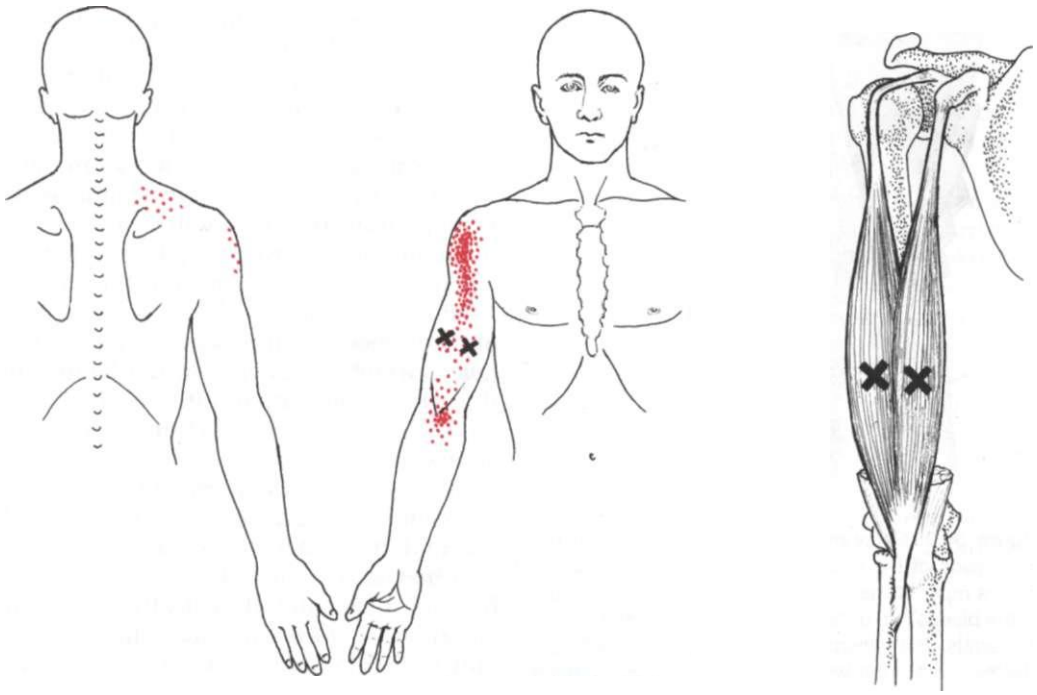


Figure 30.1. Referred pain pattern (essential zone is *solid red,* spillover zone *stippled red)* of central trigger points (**Xs**) in the midportion of the right biceps brachii muscle.

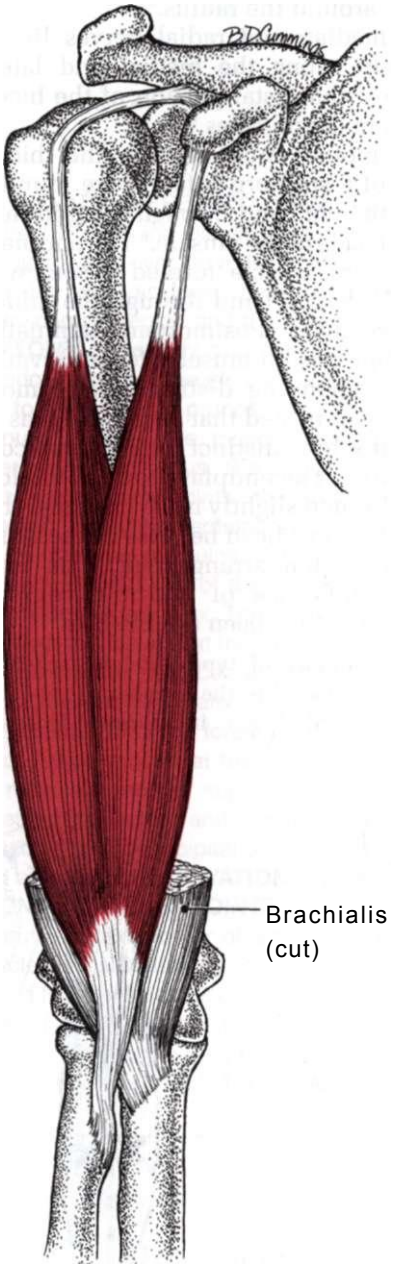


Figure 30.2. Separate proximal attachments of the two heads of the biceps brachii muscle *(red)*, which covers most of the brachialis muscle. The two heads of the biceps join distally to attach to the tuberosity of the radius. The forearm is fully supinated in this figure. The biceps tendon wraps more than halfway around the radius in pronation. The brachialis muscle has been cut for clarity.

brachii muscle is very nearly equal: 52% type 1 fibers in one study,[22] and 55% type 2 fibers in another.[12]

This muscle rarely has anatomical anomalies. A third head is attached at the origin of the coracobrachialis muscle on the coracoid process in less than **1%** of cases (2/476).[25]

Supplemental References

Additional illustrations of the biceps brachii show the relation of the two heads to each other at the shoulder,[8,36] details of its tendinous relations at the shoulder and of its tendinous attachment to the radius,[1,8] and the relation of the biceps brachii to the brachialis.[8]

3. INNERVATION

The biceps brachii muscle is innervated by the musculocutaneous nerve, *via* the lateral cord and by spinal roots C_5 and C_6.[7]

4. FUNCTION

The fact that this muscle has two heads which span three joints helps to explain the complexity of its functions. In summary, the biceps brachii (1) weakly assists flexion of the arm at the shoulder (contribution about 7%),[10] (2) assists abduction at the shoulder when the arm is laterally rotated, (3) flexes the arm at the elbow most vigorously when the forearm is supinated (palm upward), (4) strongly supinates the forearm from the pronated position when the elbow is at least partly flexed, but not when the elbow is extended, (5) the long head of this muscle helps to seat the head of the humerus in the glenoid fossa when a heavy weight is carried in the hand with the arm dependent, and (6) the short head assists horizontal adduction of the arm across the chest.

Anatomically, the biceps brachii acts at the shoulder joint, elbow joint (humeroulnar and humeroradial), and proximal radioulnar articulation (lying within the elbow joint capsule). It flexes the arm at the shoulder,[7,10] flexes the forearm at the elbow,[3,7,10,20,33] and assists forceful supination of the forearm more vigorously when the elbow is flexed to 90° than when it is

straight.[10,40] The long head of the biceps draws the head of the humerus upward into the glenoid fossa.[7, 20, 33] The biceps is in a position to assist flexion at the shoulder when the arm is medially rotated, and to assist abduction of the arm when it is laterally rotated.[20]

When the distal attachment (forearm) is fixed, the biceps brachii flexes the elbow by moving the humerus toward the forearm, as in pull-up or chinning exercises.[24]

The biceps often performs lengthening (eccentric) contractions, for example, when one is required to lower a load from torso level down to the floor.

Function Studies

In studies using electrical stimulation of the entire biceps, this muscle strongly supinated and flexed the forearm.[11] Supination was markedly weaker if the elbow was fully extended, or if only the long head was stimulated. The effect of the loss of biceps function was demonstrated in patients who had selective atrophy of this muscle. Forceful flexion of the forearm at the elbow was achieved by the brachialis and brachioradialis muscles. However, this effort in lifting a heavy weight caused a painful partial dislocation of the humeral head from the glenoid fossa when the additional support of the biceps was absent.[11] Under this condition of lifting a heavy weight, the muscle is needed to keep the head of the humerus seated in the glenoid cavity.

The two heads of the biceps brachii, the brachialis, and the brachioradialis muscles distribute a sustained forearm-flexion load among themselves in an irregular and unpredictable manner.[3] With the elbow bent, motor unit activity in the biceps brachii appears during resistance to supination, but usually disappears when the forearm is then fully extended at the elbow.[3] Electrical activity is vigorous in the muscle during flexion at the elbow when the forearm is supinated, but is markedly inhibited when the forearm is pronated.[3,39] The biceps is the auxiliary which reinforces fast supination or forceful supination against resistance.[40] Motor units are active in the long head during abduction at the shoulder,[33] but only when the arm is held in lateral rotation with the forearm supinated.[3] During flexion at the shoulder, the long head is electrically more active than the short head.[3]

Activity Studies

In a fatigue-tolerance study of arm abduction and flexion to 90° in seven healthy subjects,[17] evidence of significant fatigue appeared more frequently in flexion than in abduction when the arm was held at 90° of elevation.

Sports that require throwing with the arm strongly activate this muscle. An unusually vigorous motor-unit response of the biceps brachii appears near the end of the tennis serve, and also during the basketball spike (a one-leg jump made to block the ball), and during lay-up (a jumping one-handed shot in basketball made off the backboard from close under the basket). Minimal motor unit activity develops during the tennis forehand drive, batting a baseball, and the golf drive.[4]

The biceps is moderately activated by longhand writing and by typing. Typing produces a marked increase in amplitude of biceps electrical activity as the speed of typing increases.[27]

During simulated driving of a car on a country road, electrical activity occurred in the right biceps chiefly when making left turns, and in the left biceps when making right turns. Occasional short bursts of electrical activity were observed in the biceps brachii during simulated driving on a main road.[21,5]

5. FUNCTIONAL UNIT

The biceps functions synergistically with the brachialis and brachioradialis muscles to flex the forearm at the elbow, with the supinator to supinate the pronated forearm, with the anterior deltoid to flex the arm, and with the middle deltoid and supraspinatus to abduct the arm at the shoulder joint. The coracobrachialis

and the clavicular head of the pectoralis major assist the short head in adduction at the shoulder.

The triceps brachii is its chief antagonist.

6. SYMPTOMS

When active TrPs are present in the biceps brachii, the chief complaint is superficial anterior shoulder pain, but NOT deep pain in the shoulder joint, nor pain in the mid-deltoid region. Pain occurs during elevation of the arm above the shoulder level during flexion and abduction.[16] Other symptoms of TrPs are tenderness over the bicipital tendon, diffuse aching over the anterior surface of the arm, but rarely in the antecubital space, weakness, as well as pain, on raising the hand above the head, snapping or grating sounds from the taut long-head tendon on abduction of the arm, and frequently an associated ache and soreness in the upper trapezius region.

If the patient experiences a sudden painful "catch" in the shoulder when abducting the arm in slight extension to 15° or 20°, careful examination may reveal tenderness (enthesopathy) in the region of attachment of the tendon of the long head of the biceps to the glenoid labrum. In these patients, when the *tender* area of enthesopathy presses against the acromion during elevation of the arm, the patient experiences pain that some call an impingement syndrome. Inactivation of the (long head of the) biceps TrPs that are responsible for the enthesopathy relieves the sustained tension responsible for the irritation and permits its spontaneous resolution. Free, full range of motion is thus restored.

In contrast to patients with TrP involvement of the infraspinatus muscle, the patient with biceps TrPs can lie comfortably on the affected side and can reach behind the waistline without pain.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

The biceps shoulder-pain is often activated and perpetuated by overstress during activities like a strong backhand tennis stroke executed with the elbow straight and the forearm supinated to put top-spin on the ball.

Lifting heavy objects with the palm of the hand upward (forearm supinated) may overload the biceps brachii. Other activating stresses include sudden lifting with the arm extended (lifting the hood of a car, or lifting boxes at arm's length); an episodic elbow-flexion load (using an electric hedge clipper); unaccustomed vigorous or repeated supination (turning a stiff doorknob, using a screwdriver); overexertion (shoveling snow); and sudden over-stretching of the muscle (catching a fall with the arm by reaching behind to a railing with the elbow extended).

Frequently repeated activities that can activate and perpetuate biceps TrPs are playing the violin and hard serving in competitive tennis.

The biceps brachii may develop satellite TrPs induced by *key* TrPs in the infraspinatus muscle.[18] Inactivation of the key infraspinatus TrPs is essential for prolonged biceps relief and may be all that is required to inactivate the biceps TrP.

> In one study, biceps brachii TrPs were activated by positioning the supine patient in a way that held the biceps in the stretched position during a prolonged period for ureterolithotomy. The TrPs were inactivated by deep massage of the TrPs and passive stretch,[31] and the patient was relieved of enigmatic pain.

8. PATIENT EXAMINATION (Fig. 30.3)

After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram representing the pain distribution described by the patient. The drawing should be in the style of the pain patterns in this volume using a copy of an appropriate body form found in Figures 3.2-3.4.

Restriction of shoulder or elbow motion due to TrPs in the biceps is not obvious because the muscle crosses three joints, and the muscle must be lengthened across all of them at the same time to test for abnor-

mal tension of its fibers. Limitation of stretch length of the long head of this muscle is tested using the Biceps-extension Test (Fig. 30.3). With the patient seated in a low-backed chair and leaning back to stabilize the scapula against the backrest, the patient's arm is abducted to about 45%. The the elbow is then extended fully and the forearm pronated to stretch the muscle across the elbow region (Fig. 30.3A). Finally, without letting the arm medially rotate at the shoulder, the arm is moved posteriorly into extension. Normally, the arm will extend to the position drawn in black (Fig. 30.3B). If the muscle has been shortened by TrPs, as the stretch increases across the shoulder joint the elbow flexes to relieve the abnormal tension, assuming the position outlined in red (Fig. 30.3B). This compensatory flexion of the elbow indicates a shortened biceps muscle. We find, as Macdonald[28] has reported, that stretching the involved biceps by passively extending the forearm causes pain, as does loading the muscle by actively resisting flexion of the forearm at the elbow.

Weakness of the biceps brachii and brachialis muscles can be identified by testing the strength of elbow flexion in supination and then in pronation of the forearm while the elbow is extended. This change in position has no effect on brachialis strength because it attaches to the ulna, but the biceps is weakened by pronating the forearm if the muscle is already in a lengthened position.

The shoulder, elbow, and radioulnar joints should be examined for normal joint play; if restricted, joint play should be restored.[29] For free movement of the entire

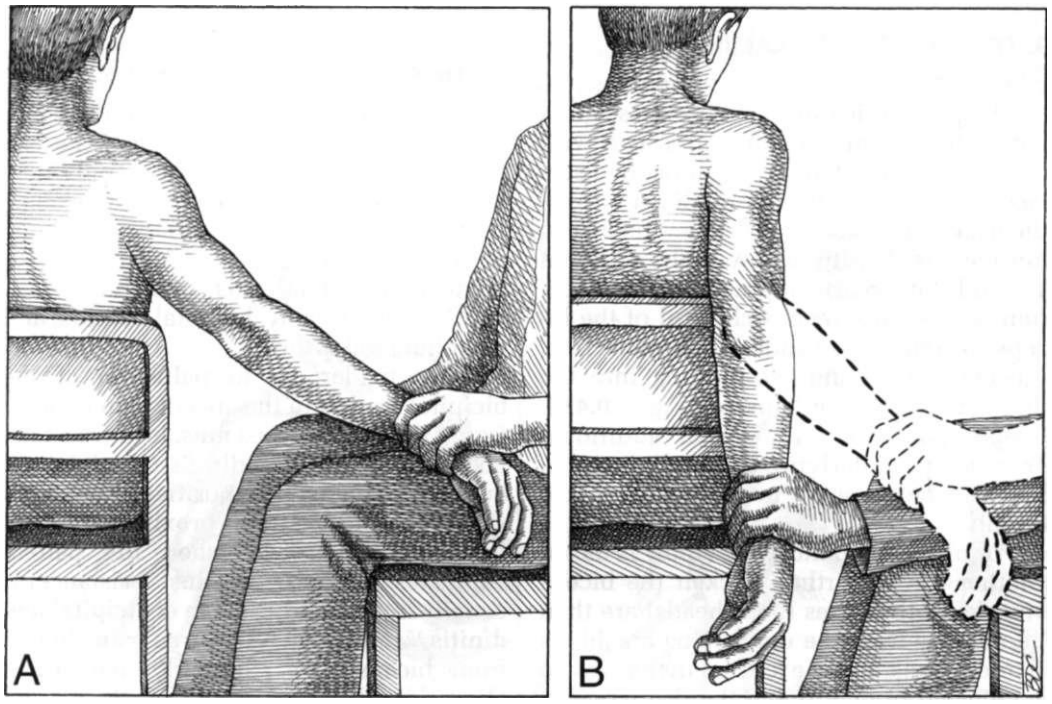


Figure 30.3. Biceps-extension Test for muscle shortening due to myofascial trigger points in the biceps brachii muscle. **A,** initial test position with the forearm pronated, elbow straight, and arm abducted to about 45°. **B,** normal end test position is black. *Red dashed lines* show limited extension at the elbow. As the arm is lowered from abduction and moved into extension at the glenohumeral joint, the elbow flexes to compensate for the shortened biceps.

shoulder complex, normal joint play is needed also in the acromioclavicular and sternoclavicular joints.

To distinguish limitation of elbow extension due to contracture of joint connective tissues from limitation caused by increased tension or limited extensibility of the biceps muscle, the elbow should be extended with the forearm in the pronated position and then it should be passively supinated, releasing biceps muscle tension. If the biceps muscle was limiting the movement, the elbow can extend a bit more. If the joint structures limited the movement, supination has no effect.

Range of motion testing for the biceps (as described above in this section) screens for taut bands with active or latent TrPs. While the passive movements of the arm and forearm are being performed, the examiner asks the patient *where* he or she feels tension and then palpates *there* for a taut band that may be limiting range of motion and thus producing dysfunction.

9. TRIGGER POINT EXAMINATION (Fig. 30.4)

The patient lies supine with the scapula flat on the examining table, or seated with the elbow supported on a well padded surface and with the trunk stabilized against the back of the chair. To slacken the biceps muscle slightly, the elbow is flexed about 15° and the forearm supinated. Flat palpation is used to screen each head of the biceps for the tense bands that harbor TrPs, especially for bands continuing into the distal third of the muscle (Fig. 30.4A). Deeper palpation may reveal additional TrPs in the underlying brachialis muscle; they are more likely to refer pain to the thumb.

For pincer palpation, the elbow is flexed another 15° to further slacken the biceps muscle. Both bellies (both heads) are then lifted away from the underlying brachialis at midmuscle, and the tension in the muscle is adjusted by modifying the degree of elbow flexion to optimize the distinction between a taut band and the surrounding normal muscle tonus. Then, the biceps fibers are rolled between the fingers and thumb to accurately localize any firm bands, nodularity at the TrP, and its spot tenderness. The precise location of the TrP is obtained by pressing along the length of a taut band to pinpoint the spot of greatest tenderness and firmness. Application of pincer palpation (Fig. 30.4B) with a strong snapping movement across a taut band at its point of maximum tenderness is likely to elicit a visible and palpable local twitch response (LTR).

Gerwin *et al.*[14] found that the most reliable criteria for making the diagnosis of myofascial TrPs were the detection of a taut band, the presence of spot tenderness, the presence of referred pain, and the reproduction of the patient's symptomatic pain. For several muscles, agreement on the presence of an LTR was poor, but it was high for the latissimus dorsi. The biceps brachii should be similarly accessible for reliable testing of LTRs by properly trained and experienced clinicians.

10. ENTRAPMENT

No entrapments of the musculocutaneous, median, or radial nerves have been observed due to TrPs in the biceps brachii muscle.

11. DIFFERENTIAL DIAGNOSIS

The referred pain and referred deep tenderness characteristic of biceps TrPs can easily lead to a number of commonly mistaken diagnoses. Conversely, patients suspected of biceps TrPs instead may have one of these other diagnoses. Some of these include bicipital tendinitis, subdeltoid bursitis, C_5 radiculopathy, bicipital bursitis, and glenohumeral arthritis.

Deep tenderness to palpation of the bicipital tendon in the area of pain referred from TrPs in the biceps muscle may be mistaken for bicipital tendinitis or subdeltoid bursitis. Although a positive Yergason's sign (pain referred to the proximal aspect of the bicipital groove when the patient supinates the forearm against resistance) is usually interpreted as a sign of **bicipital tendinitis,**[10] it also can be referred pain elicited from biceps TrPs. Similarly, tenderness elicited by deep palpation over the deltoid muscle but referred from biceps TrPs may be misidentified as **subdeltoid bursitis.**

The spontaneous biceps referred pain pattern fits a **C_5 radiculopathy,** but the patient with pain of myofascial origin has no neurological deficits on physical examina-

tion or electrodiagnostic testing, and does have demonstrable TrPs.

The proximal forearm pain experienced when the forearm is flexed at the elbow with supination, but not felt during flexion with pronation, may be attributed to bursitis of the **bicipital bursa** located at the radial attachment of the biceps. In our experience, this kind of pain is much more likely to be caused by active TrPs in the biceps brachii or supinator muscles, although sometimes the patient also may have the bursitis.

Because TrPs in the biceps brachii may refer pain and tenderness to the region of the glenohumeral joint, these symptoms are easily confused with **rheumatic disease of the joint** unless the biceps muscle is examined for TrPs.[34] The two conditions may coexist.

The presence of **painful biceps tendon instability** can be established by a palpable and painful click as the tendon of the long head of the biceps slides over the lesser tubercle when the arm (while in full abduction and some lateral rotation) is rotated slowly into medial rotation and back into lateral rotation.[10] Biceps tendon instability is *unlikely* to be related to biceps TrPs.

Related Trigger Points

Secondary TrPs commonly develop in the synergistic brachialis and supinator muscles, and also in the antagonistic triceps brachii muscle. Eventually, usually within a matter of weeks, the anterior deltoid, supraspinatus, and upper trapezius muscles succumb to the added stress on these remaining muscles of the biceps brachii's functional unit. Finally, the coracobrachialis may develop secondary TrPs.

12. TRIGGER POINT RELEASE (Fig. 30.5)

This section presents in detail the spray-and-stretch approach. Other release techniques, as described in Chapter 3, Section 12, can be used separately or in combination with spray and stretch and include trigger point pressure release, use of reciprocal inhibition, indirect techniques, and postisometric relaxation (augmented contract-relax). In addition to the general description of postisometric relaxation (PIR) in Chapter 3, detailed instructions and an illustration by Lewit[26] present the PIR technique appropriate for releasing trigger

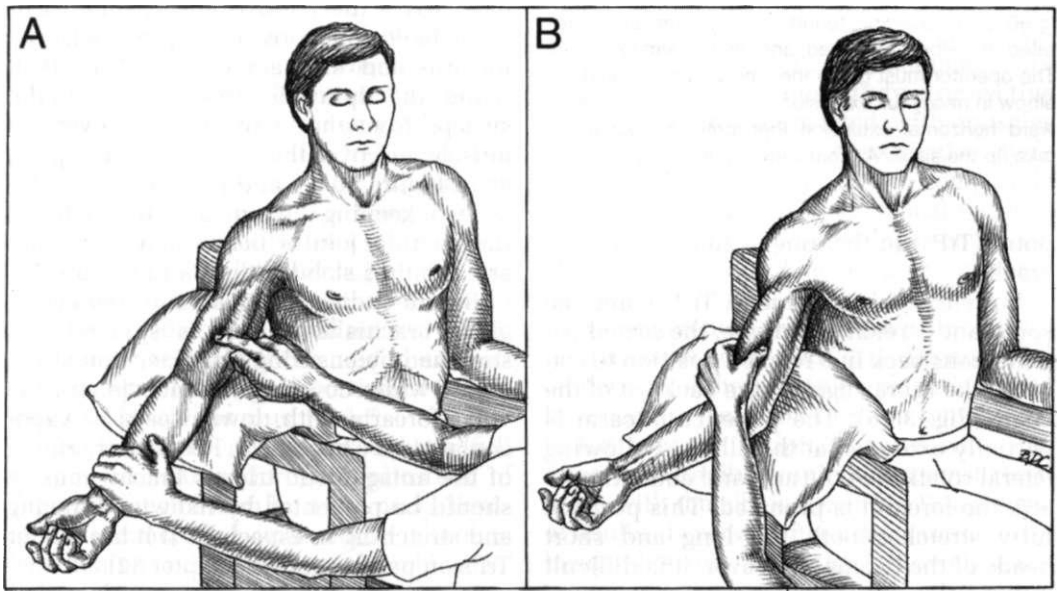


Figure 30.4. Examination of the biceps brachii muscle for trigger points with the patient seated. **A,** flat palpation. The tip of the finger rubs across the fibers. **B,** pincer palpation, which distinguishes between biceps TrPs and underlying brachialis TrPs.

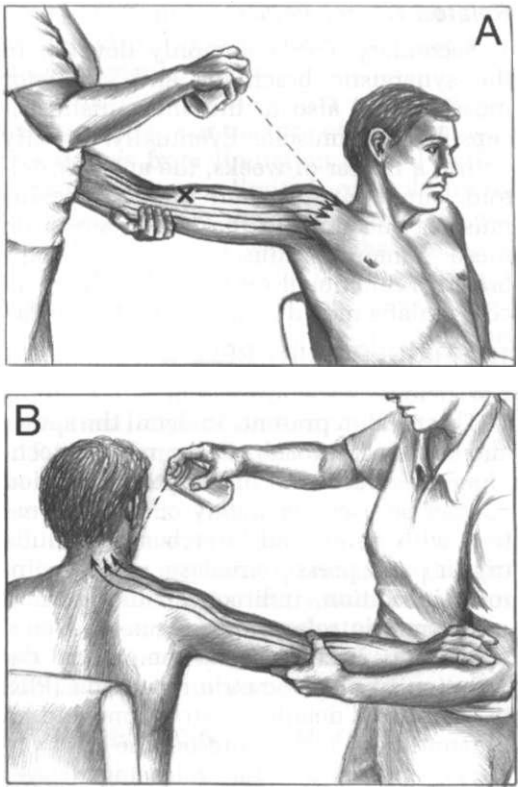


Figure 30.5. The stretch position and spray pattern *(thin arrows)* using cephalad sweeps for a trigger point (x) in the biceps brachii muscle. The arm is abducted to 90° and moved posteriorly with the arm laterally rotated, the elbow extended, and the forearm pronated. The operator must grasp the elbow firmly to hold the elbow in nearly full extension as the arm is moved toward horizontal extension (horizontal abduction) to take up the slack. **A,** front view. **B,** rear view.

point (TrPs) in the long head of the biceps brachii.

To release biceps brachii TrPs using the spray-and-stretch technique, the seated patient leans back in a relaxed position to stabilize the thorax against the backrest of the chair (Fig. 30.5). The patient's forearm is partially extended at the elbow. Following lateral rotation of the arm and abduction to 90°, the forearm is pronated. This position fully stretches both the long and short heads of the biceps. However, it is difficult to hold the position because pronation of the forearm tends to release lateral rotation at the shoulder joint. It is, therefore, necessary to stabilize the patient's elbow with the operator's hand (Fig. 30.5A and B). The jet stream of vapocoolant spray covers the muscle from the elbow cephalad over the front of the shoulder (Fig. 30.5A), and then continues over the upper trapezius to include all of the pain reference zone (Fig. 30.5B). The operator takes up slack that develops in the muscle by maintaining nearly complete elbow extension while moving the arm backward in the direction of horizontal abduction (horizontal extension), as seen in Figure 30.5A. The operator can now interpose a cycle of postisometric relaxation by instructing the patient to press upward and forward gently against the operator's hand and to breathe in slowly. With exhalation, the patient relaxes and the operator takes up any more slack that develops. Cycles of spray and stretch and of postisometric relaxation alternate. The operator avoids having the patient inhale vapocoolant. Additional downsweeps of the spray should start above the TrPs and be applied in a distal direction to cover the front of the elbow and the upper part of the forearm, if that spillover reference region is painful.

To stretch and spray the biceps with the patient supine, the laterally rotated arm hangs over the padded edge of the treatment table; the forearm is pronated. The forearm and arm are extended together, while the spray is applied in parallel sweeps from the elbow upward over the muscle and over the zone of referred pain, as in Figure 30.5A and B. Again, the difficulty of keeping the arm laterally rotated at the shoulder joint while pronating the forearm requires stabilization of the elbow.

Before ending the treatment, the synergistic brachialis muscle is sprayed and stretched by ensuring full extension at the elbow while covering the muscle and the volar forearm with downsweeps of vapocoolant (see Chapter 31). Reactive cramping of the antagonistic triceps brachii muscle should be prevented by likewise spraying and stretching it, especially if it has tender TrPs on palpation (see Chapter 32).

Moist heat is applied promptly to rewarm the cooled skin. The patient then actively moves the shoulder, elbow, and ra-

dioulnar joints through their combined ranges of motion. This fully stretches the biceps and triceps muscles.

Successful inactivation of biceps brachii TrPs often results in the relief of pain and tenderness that may have been attributed to bicipital tendinitis.

13. TRIGGER POINT INJECTION (Fig. 30.6)

If spray and stretch, trigger point pressure release, and/or other release techniques have not fully inactivated these biceps trigger points (TrPs) as evidenced by palpable locations in the midmuscle region that remain tender and refer patient-recognized pain, procaine injection of these remaining active TrPs in the muscle is frequently effective.

For injection, the elbow of the supine patient is flexed to about 45°, and the TrPs precisely located and held firmly in a pincer grasp. The TrPs are injected with 0.5% procaine solution, and the region within the pincer grasp is probed to ensure penetration of all active loci that can produce LTRs.[19] Needle penetrations may be aimed nearly tangential to the humerus, or may be directed perpendicularly toward it, avoiding the medial and lateral borders of the muscle.

This muscle is well suited to the technique of "fast in, fast out" multiple needle insertions as described by Hong.[18]

Also, the TrPs may be located for injection by using flat palpation and straddling them with two fingers of the free hand. The TrPs are held against the underlying brachialis, as in Figure 30.6. Deeper injection may be required to reach associated TrPs in the brachialis muscle (*see* Chapter 31). During injection, the operator should **avoid** the median and radial nerves, which lie, respectively, along the medial and lateral borders of the distal portion of the biceps and brachialis muscles.[9,30]

The injections are followed by a full *passive* stretching during application of vapocoolant and then by a hot pack. Treatment is concluded with *active* alternate lengthening and shortening of the biceps.

Rachlin[32] describes and illustrates injection of biceps brachii TrPs in the appropriate part of the muscle.

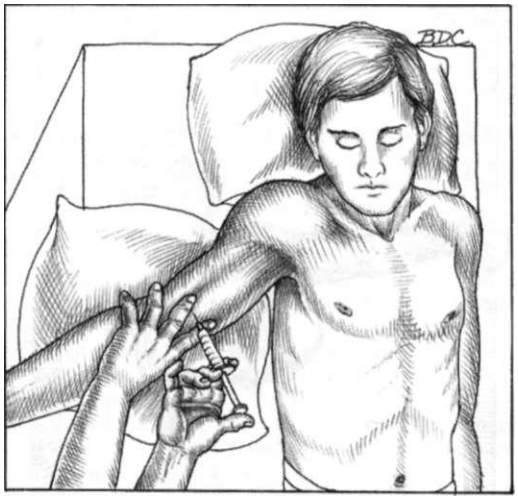


Figure 30.6. Injection of trigger points in the biceps brachii muscle, with the patient supine. The trigger points are most likely to be close to midmuscle.

Additional symptoms, often diagnosed as bicipital tendinitis (tenosynovitis), may be partly due to myofascial pain and tenderness referred primarily from the muscular TrPs, and partly due to tension tenosynovitis (enthesopathy) caused secondarily by these TrPs, which impose sustained abnormal tension on these attachment connective tissues. In one study, connective tissue structures associated with muscles showing symptoms of nonarticular rheumatism (described in terms compatible with myofascial TrPs) histologically exhibited degenerative changes[13] that could account for sensitized nociceptors in these tissues.

A coincidental, primary bicipital tendinitis may be encountered. It is diagnosed by tenderness of the tendon on palpation[38] and by a positive Yergason's test, in which pain is felt over the bicipital groove when the forearm is forcibly supinated against resistance with the elbow flexed.[5,38] When signs of tendinitis persist after inactivation of any biceps brachii TrPs, the synovial space around the tendon may be injected with a short-acting corticosteroid, using the fanwise method of Steinbrocker and Neustadt.[38]

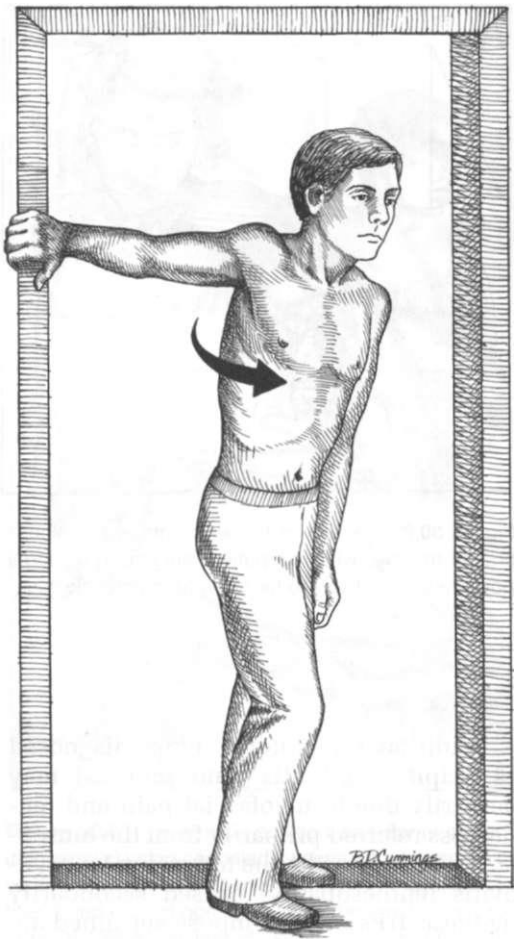


Figure 30.7. Against-doorjamb Exercise for passive stretching of the biceps brachii, anterior deltoid, and coracobrachialis muscles. The patient gradually rotates the torso (*arrow)* to passively stretch these muscles. Slow exhalation during the stretch phase enhances the effectiveness of this exercise. To stretch the biceps fully, the forearm should be extended and pronated with the thumb pointed down, and the arm must be laterally rotated at the shoulder so that the antecubital space faces forward and as far upward as possible.

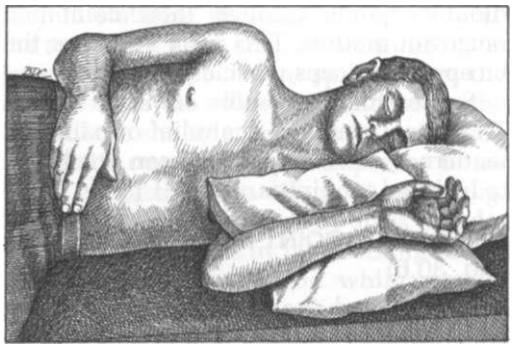


Figure 30.8. The correct sleeping position for a patient with active trigger points in the left biceps brachii muscle. The pillow is positioned to limit flexion at the elbow.

14. CORRECTIVE ACTIONS
(Figs. 30.7 and 30.8)

Following treatment for inactivation of TrPs in the biceps brachii, the patient should passively and gently stretch both heads of the muscle daily by doing the Against-doorjamb Exercise (Fig. 30.7). To do this, the patient laterally rotates the arm at the shoulder joint and pronates the forearm to hook the fingers, thumb down, against the doorjamb. With the hand slightly above shoulder level, the patient rotates the torso away from that arm, applying gentle traction to the straightened elbow, as shown in Figure 30.7. This is done to produce a steady passive stretching of the muscle without jerking. Slow exhalation enhances relaxation and tension release in the muscle during the stretch phase.

The patient with biceps TrP problems should learn to lift and carry objects with the forearms pronated in order to transfer some of the load from the biceps to the brachioradialis and supinator muscles (see Fig. 36.3C).

The patient should avoid sleeping with the elbow tightly flexed by placing a small pillow in the crook of the elbow (Fig. 30.8). This prevents prolonged shortening of the muscle.

SUPPLEMENTAL CASE REPORT

The treatment of a patient with somewhat atypical involvement of the biceps brachii is described by Kelly.[23]

REFERENCES

1. **Agur AM:** *Grant's Atlas of Anatomy.* **Ed. 9. Williams & Wilkins, Baltimore, 1991:408 (Fig. 6.75A).**
2. **Aquilonius SM, Askmark H, Gillberg PG,** *et al:* **Topographical localization of motor endplates in cryosections of whole human muscles.** *Muscle Nerve* **7:287-293, 1984.**
3. **Basmajian JV, DeLuca CJ:** *Muscles Alive.* **Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 268, 269, 277-279).**

4. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sport Skills.* Charles C Thomas, Springfield, 111. 1967.
5. Cailliet R: *Shoulder Pain.* F.A. Davis, Philadelphia, 1966 (p.73).
6. Christensen E: Topography of terminal motor innervation in striated muscles from stillborn infants. *Am J Phys Med* 38:65-78, 1959.
7. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 527, 528).
8. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 29, 31, 49, 53, 55, 61).
9. *Ibid.* (Figs. 67, 68).
10. Curtis AS, Snyder SJ: Evaluation and treatment of biceps tendon pathology. *Orthop Clin North Am* 24(1):33-43, 1993.
11. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 88, 98, 106).
12. Elder GC, Bradbury K, Roberts R: Variability of fiber type distributions within human muscles. *J Appl Physiol* 53(6):1473-1480, 1982.
13. Fassbender HG: Non-articular rheumatism. Chapter 13. In: *Pathology of Rheumatic Diseases,* translated by G. Loewi. Springer-Verlag, New York, 1975 (pp. 307-310).
14. Gerwin RD, Shannon S, Hong CZ, et *al:* Interrater reliability in myofascial trigger point examination. *Pain* 69.65-73, 1997.
15. Gutstein M: Diagnosis and treatment of muscular rheumatism. *Br JPhys Med* 2:302-321,1938 (Cases 1 and 2; Figs. 1, 2; p. 308).
16. Gutstein M: Common rheumatism and physiotherapy. *Br J Phys Med* 3:46-50, 1940 (Case 1, p. 49).
17. Hagberg M: Electromyographic signs of shoulder muscular fatigue in two elevated arm positions. *Am J Phys Med* 60(3):111-121, 1981.
18. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(2):29-59, 1994.
19. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Rehabil* 73:256-263, 1994.
20. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 111).
21. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving, I-III. *Ergonomics* 28:375-388, 1975 (pp. 383-387).
22. Jozsa L, Demel S, Reffy A: Fibre composition of human hand and arm muscles. *Gegenbaurs morph Jahrb, Leipzig* 227:34-38, 1981.
23. Kelly M: Interstitial neuritis and the neural theory of fibrositis. *Ann Rheum Dis* 7:89-96, 1948 (Case 10, p. 94).
24. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 268).
25. Khaledpour VC: Anomalies of the biceps brachii muscle. *Anat Anz* 259:79-85, 1985.
26. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 202-203).
27. Lundervold AJ: Electromyographic investigations of position and manner of working in typewriting. *Acta Physiol Scand* 24:(Suppl. 84), 1951 (pp. 66-67, 80-81, 94).
28. Macdonald AJ: Abnormally tender muscle regions and associated painful movements. *Pain* 8:197-205, 1980 (pp. 202, 203).
29. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
30. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 72, p. 83).
31. Prasanna A: Myofascial pain as postoperative complication [Letter]. *J Pain Symptom Manage 8(7)450-*451, 1993.
32. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 328-330).
33. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Lea & Febiger, Philadelphia, 1967 (pp. 188, 189).
34. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981 (Table 1).
35. Segal RL: Neuromuscular compartments in the human biceps brachii muscle. *NeurosciLett* 240:98-102,1992.
36. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 319).
37. Steinbrocker O, Isenberg SA, Silver M, *et al:* Observations on pain produced by injection of hypertonic saline into muscles and other supportive tissues. *J Clin Invest* 32:1045-1051, 1953 (Fig. 3, p. 1049).
38. Steinbrocker O, Neustadt DH: *Aspiration and Injection Therapy in Arthritis and Musculoskeletal Disorders.* Harper & Row, Hagerstown, 1972 (pp. 44, 46; Fig. 5-6).
39. Sullivan WE, Mortensen OA, Miles M, *et al.:* Electromyographic studies of m. biceps brachii during normal voluntary movement at the elbow. *Anat Rec* 207:243-251, 1950.
40. Travill A, Basmajian JV: Electromyography of the supinators of the forearm. *Anat Rec* 139:557-560, 1961.

CHAPTER 31
Brachialis Muscle

HIGHLIGHTS: **REFERRED PAIN** from trigger points (TrPs) in the brachialis muscle is projected chiefly to the base of the thumb and often to the antecubital region of the elbow. **ANATOMY:** Attachments are to the humerus, proximally, and to the ulna, distally. The **FUNCTION** of this *"workhorse"* of the elbow flexors commonly involves flexion of the forearm toward the humerus. However, with the forearm fixed, when this muscle flexes the elbow joint it rotates the humerus toward the forearm, as in pull-up or chinning exercises. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are caused chiefly by acute and repetitive stress overload. **PATIENT EXAMINATION** reveals aggravation of thumb pain by passive full extension at the elbow. **TRIGGER POINT EXAMINATION** of the brachialis requires that the bulk of the biceps brachii muscle be pushed aside. **ENTRAPMENT** of the sensory branch of the radial nerve may be due to TrP activity of this muscle. **DIFFERENTIAL DIAGNOSIS:** Related TrPs are likely to be found in the brachioradialis, supinator, or adductor pollicis muscles. **TRIGGER POINT RELEASE** by spray and stretch is performed by extending the forearm at the elbow while applying the spray in a down-pattern, assisted by postisometric relaxation and respiratory augmentation. **TRIGGER POINT INJECTION,** to be successful, requires an appreciation of the unexpected thickness of this muscle. **CORRECTIVE ACTION** calls for relieving overload of the muscle and for the patients to learn how to perform self-release of brachialis TrPs.

1. REFERRED PAIN (Fig. 31.1)

Pain is referred from brachialis trigger points (TrPs) chiefly to the dorsum of the carpometacarpal joint at the base of the thumb and to the dorsal web of the thumb (Fig. 31.1), as also noted by Kelly.[16] The most distal trigger area or attachment TrP in the pain pattern drawing of Figure 31.1 is located a few centimeters above the antecubital space and most likely represents enthesopathy secondary to the midfiber TrPs. Spillover pain from these TrPs may cover the antecubital space. The pain that occasionally extends upward over the deltoid muscle is more likely to arise from the most proximal TrPs in the brachialis.

Experimental injection of hypertonic saline into this muscle produced referred pain in the region of the elbow and over the radial aspect of the forearm. The pain was associated with referred tenderness that matched the pain in distribution, duration and severity.[15]

2. ANATOMY (Fig. 31.2)

Proximally, the brachialis muscle attaches to the distal half of the shaft of the humerus anteriorly and to the medial and lateral intermuscular septa. This proximal attachment reaches the distal attachment of the deltoid muscle (Fig. 31.2). **Distally,** the brachialis attaches to the coronoid process on the proximal end of the ulna. However, the overlying biceps brachii attaches distally to the radius (Fig. 31.2).[5]

SUPPLEMENTAL REFERENCES

Other authors have illustrated the brachialis muscle as it is seen from in front,[3,7,27] from the medial aspect,[8] from the medial aspect with associated neurovascular structures,[9,19,23] from the lateral aspect,[1,6,18,22] and as seen in cross section.[2,12,21]

3. INNERVATION

The brachialis muscle is supplied by the musculocutaneous nerve *via* the lateral cord from the C_5 and C_6 roots.[5]

4. FUNCTION

Due to its ulnar, rather than radial attachment, the brachialis performs only one motion, flexion at the elbow joint.[4,11,13,17,26] It is the "workhorse" of the elbow flexors. Like the deltoid, it shows no activity when the dependent arm is heavily loaded with weights.[4] There is fine interplay between the biceps brachii, the brachialis, and the brachioradialis muscles during resisted forearm flexion. The interplay shows striking variability on repeated trials.[4]

When the proximal attachment (humerus) is fixed, the brachialis moves the forearm toward the humerus. With the distal attachment (ulna) fixed, this muscle moves the humerus toward the forearm, as in pull-up or chinning exercises.[17] The brachialis often contracts eccentrically to control (decelerate) the lowering of heavy objects.

Tested during the act of driving a car, the brachialis generally showed a low level of electrical activity that was relatively constant, and only occasionally showed short bursts of more intense activity.[14]

5. FUNCTIONAL UNIT

The brachialis is synergistic with the biceps brachii, the brachioradialis, and with that part of the supinator that functions as an elbow flexor.

The brachialis functions as an antagonist to the triceps brachii.

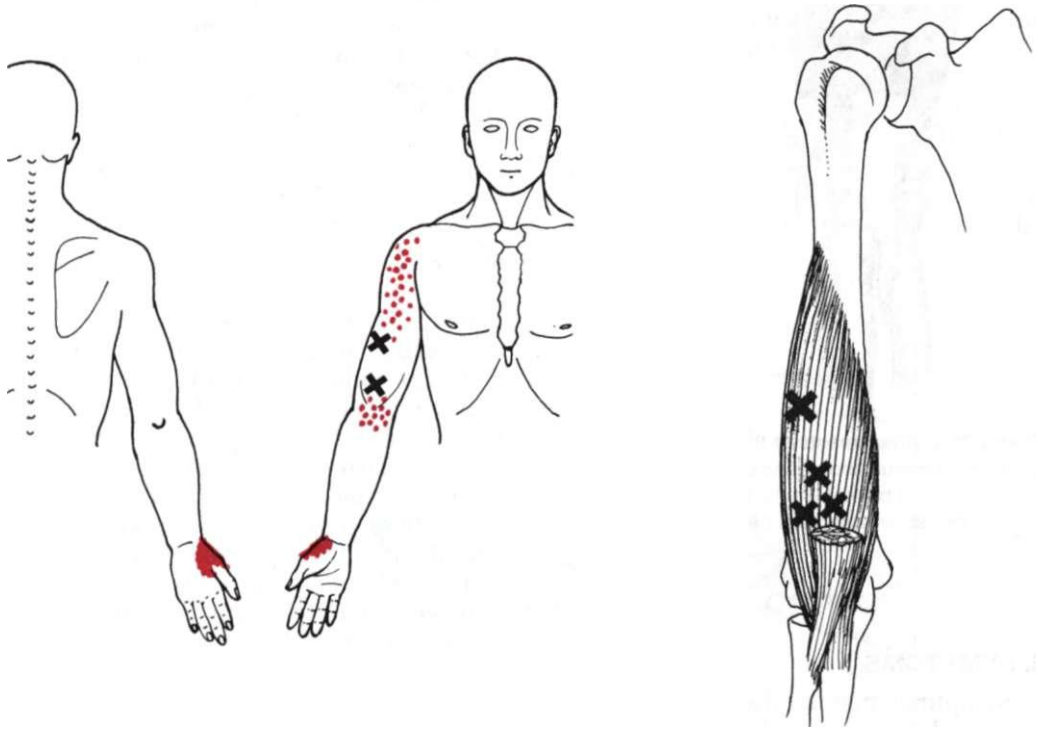


Figure 31.1. The pain pattern (essential portion, *solid red;* spillover portion, *stippled red)* that is referred from trigger points (**Xs**) in the right brachialis muscle. Taut bands associated with midmuscle trigger points may cause entrapment of the radial nerve. The most distal trigger area (attachment trigger point) in the pain pattern drawing probably represents enthesopathy secondary to the midfiber trigger points.

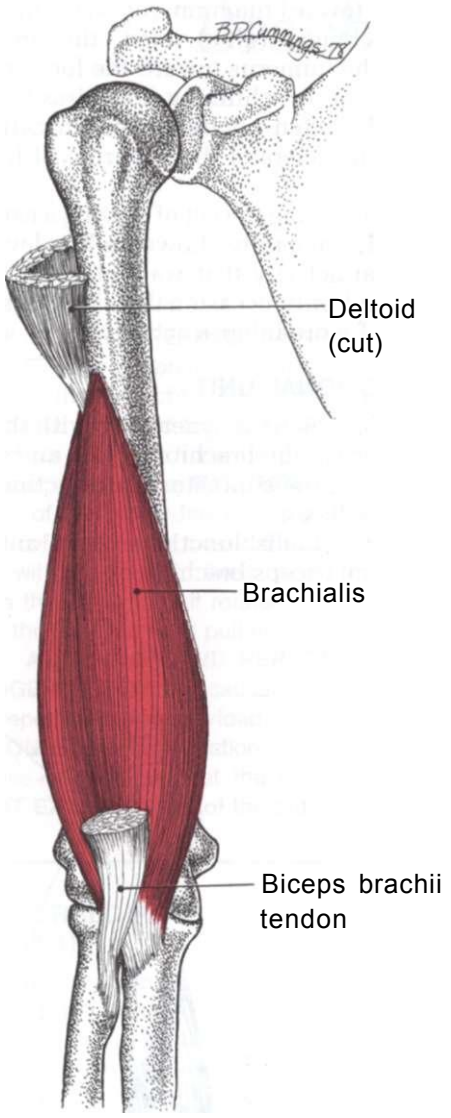


Figure 31.2. Attachments of the right brachialis muscle to the humerus above, and ulna below. The cut end of the overlying biceps brachii tendon appears below. The deltoid, above, also has been cut for clarity.

6. SYMPTOMS

Symptoms may be due to referred pain and tenderness from brachialis TrPs, or secondary to radial nerve entrapment. Referred pain is felt in the base of the thumb at rest and often also with use of the thumb. Diffuse soreness of the thumb is characteristic of its referred tenderness. Pain referred over the anterior deltoid region from brachialis TrPs alone does not lead to impairment of shoulder motion.

Symptoms caused by brachialis entrapment of the superficial sensory (cutaneous) branch of the radial nerve are dysesthesia, tingling, and numbness on the dorsum of the thumb. The aching of referred TrP pain and the symptoms of entrapment are both experienced in the thumb and may be relieved by inactivating the brachialis TrPs.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Brachialis TrPs can be activated and can be perpetuated by continuing stress overload of forearm flexion during heavy lifting. Examples of stress overloads are holding a power tool, carrying groceries, meticulous ironing, and fingering a violin or guitar with the forearm supinated so the biceps brachii is shortened and of not much help. In "tennis elbow," brachialis involvement tends to develop together with that of the biceps brachii after initial activation of TrPs in the supinator (see Chapter 36).

Systemic perpetuating factors are considered in Chapter 4.

8. PATIENT EXAMINATION

After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram representing the pain distribution described by the patient. The drawing should be in the style of the pain patterns in this volume using a copy of an appropriate body form found in Figures 3.2-3.4.

Pain referred from brachialis TrPs is increased by passively extending the elbow fully, although limitation of motion is not a complaint. The range of elbow extension is restricted by only a few degrees, and often is detectable only on comparison with the other arm, or by improvement after treatment. Surprisingly, active motion of the thumb in the pain reference zone usually hurts, but active movement of the elbow does not.

Weakness of the biceps brachii and brachialis muscles can be distinguished by testing the strength of elbow flexion in supination and then in pronation of the

forearm while the elbow is extended. This change in position has no effect on brachialis strength because it attaches to the ulna, but the biceps is weakened by pronating the forearm if the muscle is already in a lengthened position.

Radial nerve compression is indicated when a tingling in the thumb results from pressure exerted on the region where the nerve exits the musculospiral groove and pierces the lateral intermuscular septum (*see* Fig. 32.3). The place to apply pressure is about mid arm, just below the dimple that marks the apex (distal end) of the triangular bulge produced by the deltoid muscle.

The elbow joint should be examined for normal joint play, which needs to be restored if restricted.[20]

9. TRIGGER POINT EXAMINATION (Fig. 31.3)

The patient's elbow is flexed between 30° and 45° and the forearm is supinated to slacken the biceps brachii so the bulk of the biceps brachii can be pushed aside, medially, to palpate the underlying brachialis TrPs (Fig. 31.3). The biceps has more slack if the forearm is placed in supination and is relaxed. Brachialis TrPs can be located in the distal half of the arm (Fig. 31.1) and are likely to refer pain to the thumb and sometimes to the front of the elbow. One of these TrPs may be located deep to the lateral edge of the undisplaced biceps brachii, but others are found toward the middle of the brachialis muscle, sometimes under the biceps brachii. The more proximal TrPs, which refer pain ***up*** the arm, are covered by the biceps muscle.

10. ENTRAPMENT

The symptoms of nerve entrapment include "numbness," hypoesthesia or hyperesthesia, and dysesthesia (as distinguished from the usual deep ache of referred pain). These symptoms, like the referred pain, appear over the dorsum of the thumb and its adjacent web space. This entrapment of the sensory branch of the radial nerve can be caused by a TrP, usually in the lateral border of the brachialis muscle, that produces a taut band of muscle fibers extending to the level where the radial nerve exits the musculospiral groove and pierces the lateral intermuscular septum (*see* Fig. 32.3).

These symptoms of entrapment are relieved by injection of the brachialis TrP, which feels like an almond in the lateral border of the muscle, just proximal to the nerve. The resultant resolution of the taut band and the relief of nerve-entrapment signs and symptoms strongly suggest that muscle shortening associated with the TrPs produced the nerve compression and should be confirmed by sensory nerve conduction velocities before and after relief by treatment.

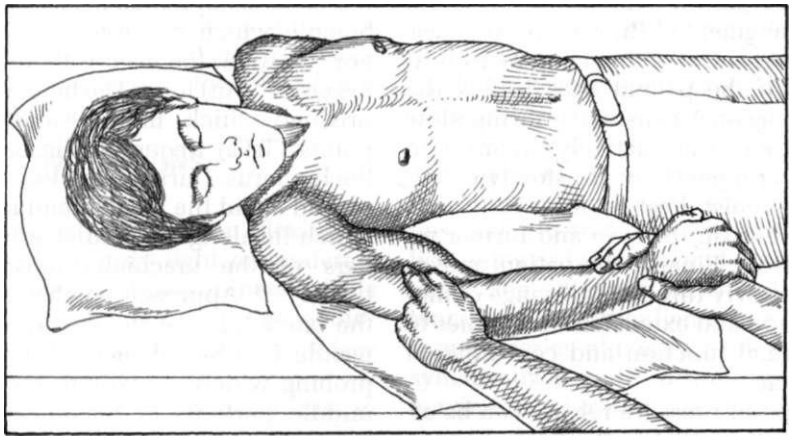


Figure 31.3. Examination of the brachialis muscle for trigger points by pushing the biceps brachii aside in a medial direction in order to reach under it. The biceps has additional slack if the forearm is supinated rather than pronated as shown here.

11. DIFFERENTIAL DIAGNOSIS

Conditions that can produce symptoms confusingly similar to active TrPs in the brachialis muscle include C_5 and/or C_6 radiculopathy, bicipital tendinitis, and supraspinatus tendinitis. An additional consideration would be carpal tunnel syndrome when pain is perceived as isolated over the thenar eminence.

The brachialis is likely to be involved when the biceps brachii, brachioradialis, or supinator muscles harbor active TrPs.

Pain at the base of the thumb also may be referred from TrPs in the supinator, brachioradialis, and adductor pollicis muscles.

12. TRIGGER POINT RELEASE (Fig. 31.4)

Detailed basic instructions for how to perform the various trigger point (TrP) release techniques are found in Chapter 3, Section 12 of this manual.

To apply stretch and spray of the brachialis, the operator rests the distal end of the patient's humerus on a firm support (operator's knee, or armrest of the chair covered by a pillow), as in Figure 31.4. The affected elbow gradually extends as the muscle releases while the vapocoolant spray is applied over the brachialis downward, in the direction of its chief zone of referred pain and on to the end of the thumb. The spray also is applied upward to cover the brachialis again and the anterior deltoid region if pain also is felt there. Release is augmented (first by postisometric relaxation and then by using reciprocal inhibition) if the patient tries to flex the arm gently against resistance during slow inhalation, and then actively attempts to extend the arm gently while slowly exhaling. Warm moist heat is applied to the muscle to rewarm the skin and further relax the muscle. Finally, the patient moves the elbow slowly through full range of motion in flexion and extension three times to restore normal function and coordination of the muscle.

Trigger point pressure release can be effective when applied with the muscle in either a position of slight lengthening or in a position of ease (slightly shortened).

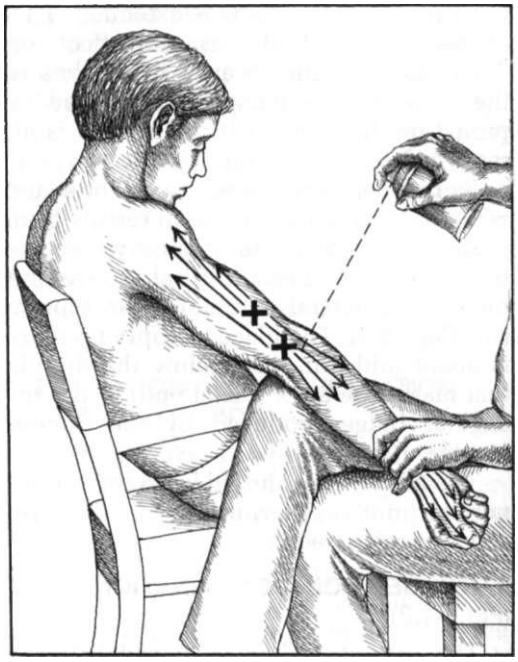


Figure 31.4. Stretch position and spray pattern *(arrows)* for trigger points (Xs) in the brachialis muscle. The elbow is extended by applying counter pressure with the operator's knee at, or just above, the olecranon process while the vapocoolant spray is applied over the muscle and over the dorsum of the thumb and its web.

13. TRIGGER POINT INJECTION (Fig. 31.5)

The arm is flexed approximately 45° and the forearm supinated to slacken the biceps brachii, which is pressed aside, medially. For injection, the needle should be at least 3.8 cm (1 1/2 in) long. The brachialis is a surprisingly thick muscle and its trigger points (TrPs) frequently lie deep, next to the humerus. During injection, the operator should avoid the median and radial nerves which lie along the medial and lateral borders of the brachialis muscle, respectively.[10, 24] Approaching the muscle from the lateral side of the arm (Fig. 31.5), the needle is directed medially and upward, probing widely to explore the lateral and middle portions of the muscle for local twitch responses that identify the location of TrPs to be injected with 0.5% procaine or lidocaine. The needle may *lightly* con-

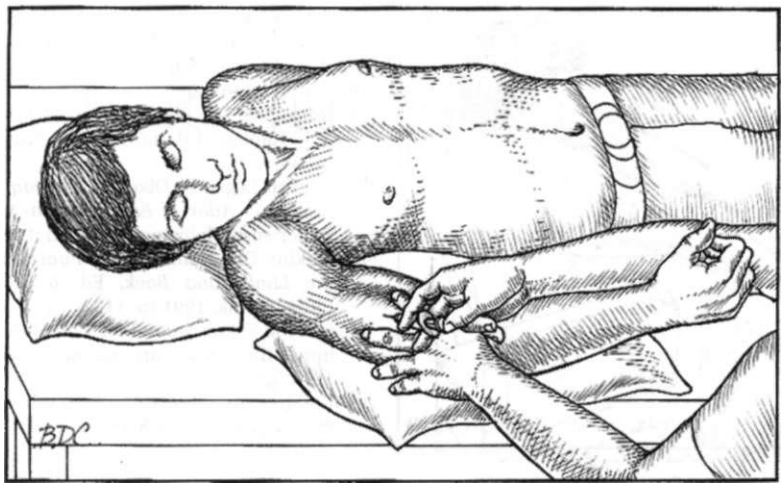


Figure 31.5. Injection of trigger points in the brachialis muscle, with the biceps brachii pushed aside in a medial direction.

tact the humerus, which ensures reaching the full depth of the muscle. If bony contact bends the needle tip so that it "catches" when pulled through the tissue, the needle should be replaced immediately.

After injection, to ensure release of any overlooked TrPs, spray and stretch are applied to the muscle followed by moist heat. Then, the patient moves the muscle through its full range of motion three times to help restore normal coordination and muscle function.

Locations for injection of TrPs in the brachialis muscle are illustrated by Rachlin.[25] The most proximal site illustrated in his Figure 10-50 is most likely an attachment TrP due to enthesopathy rather than a central myofascial TrP.

14. CORRECTIVE ACTIONS (Fig. 31.6)

Stress overload of forearm flexion is avoided by lifting only light or moderate loads, with the forearms supinated. This brings the biceps brachii into play, avoiding additional load on the brachialis (*see* Chapter 30).

The patient learns to place a pillow in the angle of the elbow at night (*see* Fig. 30.8). The pillow prevents sleeping with the arm tightly folded, which is a position that immobilizes the brachialis in a shortened position. Likewise, the elbow should not be held sharply flexed during a long telephone call. One can switch the handset back and forth between hands occasionally, or use a headset that frees the hands.

A purse strap should not hang on the forearm with the elbow bent; the purse may be held in the fingers with the elbow straight, or hung over the opposite shoulder, or best hung on a belt.

When one is playing a musical instrument, like a violin, the elbow should be allowed to hang down straight at every opportunity. The patient should be taught how to keep the brachialis TrPs inactive by applying trigger point pressure release or the self-release procedure described below.

The patient learns to release brachialis TrPs for himself or herself by supporting the humerus just above the elbow, in the position illustrated in Figure 31.6, but at first using only the force of gravity to assist postisometric relaxation without any assistance from the other hand. The patient performs a series of contract-relax maneuvers synchronized with respiration to obtain maximum relaxation, as described in Section 12. After several cycles of postisometric relaxation, additional release and lengthening may be achieved by gently as-

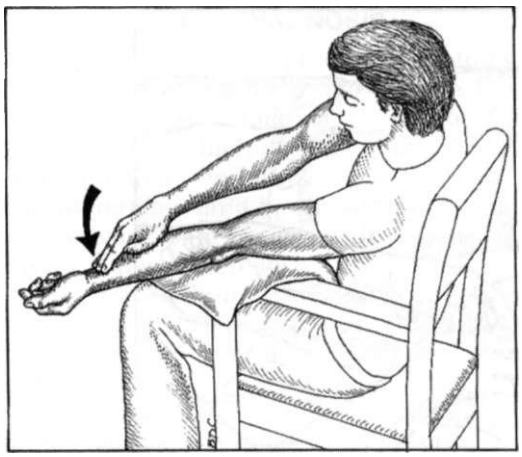


Figure 31.6. Patient performing self-release of brachialis trigger points. See text for details.

sisting gravity with the other hand, as shown in Figure 31.6. This process should NOT be painful with, at most, just a sense of stretch tension. The patient should do several such stretches once or twice daily after soaking the arm and forearm in warm water, or after application of moist heat.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:385 (Fig. 6.39).
2. *Ibid.* p. 389 (Fig. 6.44).
3. *Ibid.* pp. 399, 400 (Figs. 6.57, 6.59).
4. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 240, 263, 264).
5. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 528).
6. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 61).
7. *Ibid.* (Figs. 55, 65, 69).
8. *Ibid.* (Fig. 49).
9. *Ibid.* (Figs. 56, 70).
10. *Ibid.* (Figs. 67, 68).
11. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 98).
12. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 80-83).
13. Jenkins DB: *Hollinsbead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 112).
14. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving. *Ergonomics* 28:375-388, 1975 (pp. 383-386).
15. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3.175-190, 1938 (pp. 187, 188).
16. Kelly M: The nature of fibrositis. I. The myalgic lesion and its secondary effects: a reflex theory. *Ann Rheumatol Dis* 5.1-7, 1945 (Case 1).
17. Kendall FP, McCreary EK, Provance PG: *Muscles, Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 268).
18. McMinn RM, Hutchings RT, Pegington J, *et al: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (pp. 121C, 133F).
19. *Ibid.* (p. 127B).
20. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
21. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (pp. 61, 80).
22. *Ibid.* (p. 58).
23. *Ibid.* (p. 56).
24. *Ibid.* (p. 83).
25. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360, (see pp. 333- 335).
26. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (p. 185).
27. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (pp. 320, 321, 327).

CHAPTER 32
Triceps Brachii Muscle (and the Anconeus)

HIGHLIGHTS: The three heads of the triceps brachii may develop trigger points (TrPs) in five locations, each with its own referred pain pattern. Trigger points occur frequently in this muscle and are commonly overlooked. They increase muscle tension and cause dysfunction, as well as pain. **REFERRED PAIN** from the muscle's TrPs is projected mostly up and down the posterior aspect of the arm and to the lateral epicondyle, more often than to the medial, with spillover pain into the fourth and fifth fingers. It may be projected also to the upper part of the suprascapular region. Trigger points in the long head are a common, but often overlooked, source of pain. **ANATOMY:** the medial and lateral heads attach to the humerus and to the olecranon process of the ulna, thus crossing one joint, unlike the long head which spans two joints. Proximally, the long head of the triceps brachii attaches to the scapula; distally, it forms a two-layer common tendon with all three heads. This tendon attaches at the olecranon process. **FUNCTION** of all parts of the triceps brachii is related to extension of the forearm at the elbow. In addition, the long head adducts, and helps to extend the arm at the shoulder joint. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** are usually due to overload stress of this muscle. **PATIENT EXAMINATION** to identify restricted range of motion requires simultaneous flexion of both the elbow and the shoulder joints, an awkward unnatural position. **TRIGGER POINT EXAMINATION** of the long head requires deep pincer palpation of the muscle adjacent to the humerus. Flat palpation may be used for the other heads. Central TrPs are distinguished from attachment trigger points. **ENTRAPMENT** of the radial nerve may be caused by taut bands in the lateral head. An anomalous anconeus epitrochlearis muscle can entrap the ulnar nerve. **TRIGGER POINT RELEASE** of the triceps brachii by the stretch-and-spray technique requires simultaneous flexion of both the joints that the long head transverses with application of the vapocoolant mainly from the proximal to the distal direction. **TRIGGER POINT INJECTION** of this muscle may be needed to completely inactivate its TrPs and to relieve enthesopathy at attachment TrP_4. **CORRECTIVE ACTIONS** call for modification of activities and mechanical factors that stress this muscle, including the modification of chairs with inadequate elbow support.

1. REFERRED PAIN (Figs. 32.1 and 32.2)

The referred pain patterns of five trigger point (TrP) areas in the three heads of the triceps brachii are shown in Figure 32.1. They occur frequently. The TrPs are numbered in order of decreasing prevalence, based on our experience. In this muscle, it is important to distinguish central TrPs (CTrPs) from attachment TrPs (ATrPs).

TrP_1-Long Head of Triceps (Fig. 32.1A)

Pain and tenderness referred from the long head extends from the central TrP_1 region (Fig. 32.1 A, left side) upward over the posterior arm to the back of the shoulder, occasionally to the base of the neck in the upper trapezius region, and sometimes down the dorsum of the forearm, skipping the elbow. This trigger point region is located in the central portion of the muscle belly.

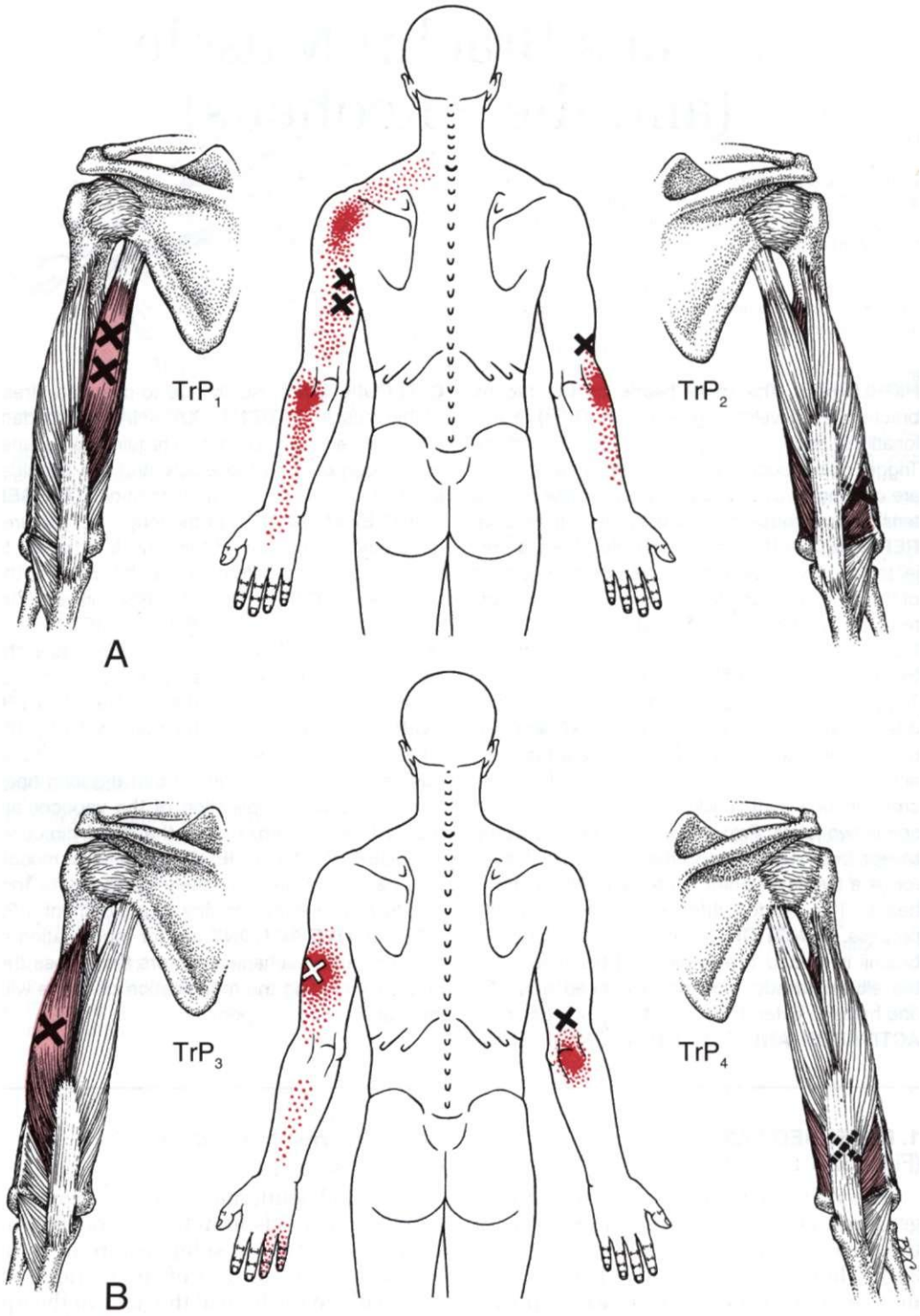


Figure 32.1 Referred pain patterns (*dark red)* from trigger points *(black* or *white* **Xs**) in the triceps brachii muscle *(medium red).* **A,** central trigger point region 1 (TrP_1), in left long head; central trigger point region 2 (TrP_2), in the lateral portion of the right medial (deep) head. **B,** central trigger point region 3 (TrP_3) in the lateral border of the left lateral head; attachment trigger point region 4 (TrP_4), deep under the tendon in the musculotendinous attachment region.

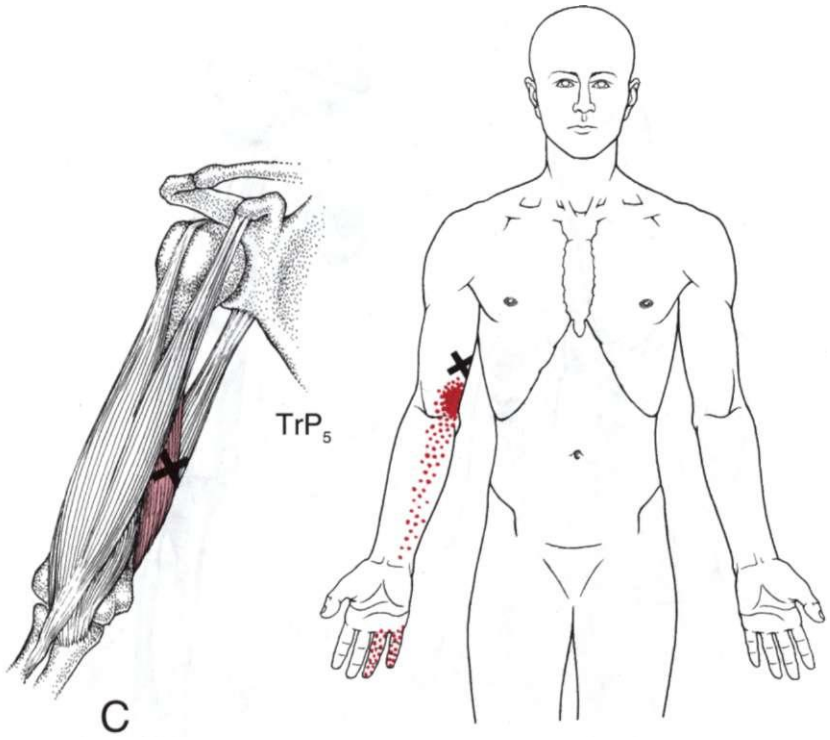


Figure 32.1—*continued.* C, central trigger point region 5 (TrP_5) deep in the medial border of the right medial (deep) head.

TrP_2-Medial Head (Fig. 32.1A)

The next most common triceps TrP, central TrP_2, lies midfiber in the *lateral portion* of the medial head (Fig. 32.1A, right side), in the distal part of the arm. Referred pain and tenderness are projected to the *lateral* epicondyle, and are a common component of the "tennis elbow." Pain also may extend to the radial aspect of the forearm.

TrP_3-Lateral Head (Fig. 32.1 B)

From TrP_3 (Fig. 32.1B, left side), pain and tenderness are referred over the arm posteriorly, sometimes to the dorsum of the forearm, and occasionally to the fourth and fifth digits. Its taut bands may entrap the radial nerve.

TrP_4-Distal Attachment Region (Fig. 32.1 B)

The local tenderness at TrP_4 (Fig. 32.1B, right side) is most likely an attachment TrP secondary to $CTrP_1$, $CTrP_3$, or $CTrP_5$, (which is shown in part ***C*** of Figure 32.1). This TrP_4 may refer pain and tenderness distally to the olecranon process.

TrP_5-Medial Head (Deep Head) (Fig. 32.1 C)

Most easily located by an anterior approach, this central TrP_5 (which is found in the *medial portion* of the medial head) refers pain and tenderness to the *medial* epicondyle. Pain may extend to the volar surface of the fourth and fifth digits and sometimes also to the adjacent palm and middle finger. Winter[43] also included pain along the inner side of the forearm from this TrP site.

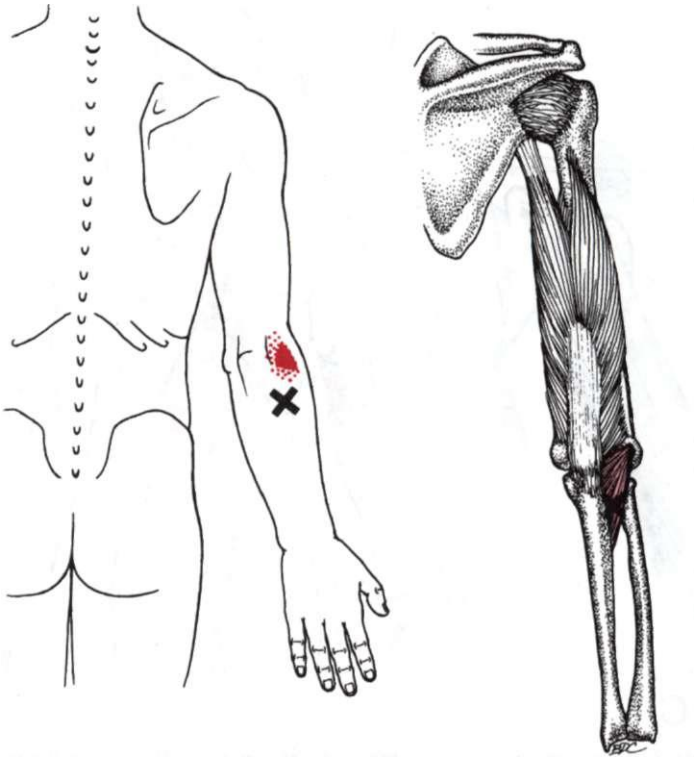


Figure 32.2. Location of a trigger point **(X)** in the anconeus muscle *(light red)* and its referred pain pattern *(dark red)*.

Anconeus (Fig. 32.2)

An active TrP in the anconeus muscle refers pain and tenderness locally to the *lateral* epicondyle (Fig. 32.2).

2. ANATOMY (Fig. 32.3)

The three heads of the triceps brachii muscle attach ***distally*** to the olecranon process of the ulna *via* a common tendon (Fig. 32.3), which begins about the middle of the muscle and consists of a superficial lamina and a deep lamina that join near their insertion. ***Proximally*** the **long head** arises from the infraglenoid lip of the scapula; this head crosses two joints. The **medial head** (sometimes referred to as the deep head) arises from the posterior surface of the humerus ***medial*** and ***distal*** to the radial nerve, and from the intermuscular septum. This head lies deep against the bone, and just above the elbow its attachment covers the posterior humerus both medially and laterally. ***Proximally*** the **lateral head** arises from the posterior surface of the humerus ***lateral*** and ***proximal*** to the radial nerve, and from the lateral intermuscular septum. It bridges the radial nerve and covers much of the medial head (Fig. 32.3C). The medial and lateral heads cross only the elbow joint.[10]

The distribution of fiber types in the triceps muscle was determined by taking at least 13 samples in each of 4 triceps brachii muscles post mortem.[17] Both the lateral head and long head of the triceps had 60% fast twitch (Type II) fibers and 40% slow twitch (Type I) fibers. However, the *medial* head was composed of a higher (60) percent of *slow* twitch fibers compared to only 40% fast twitch fibers. Samples taken near the surface of the muscle and from deep in

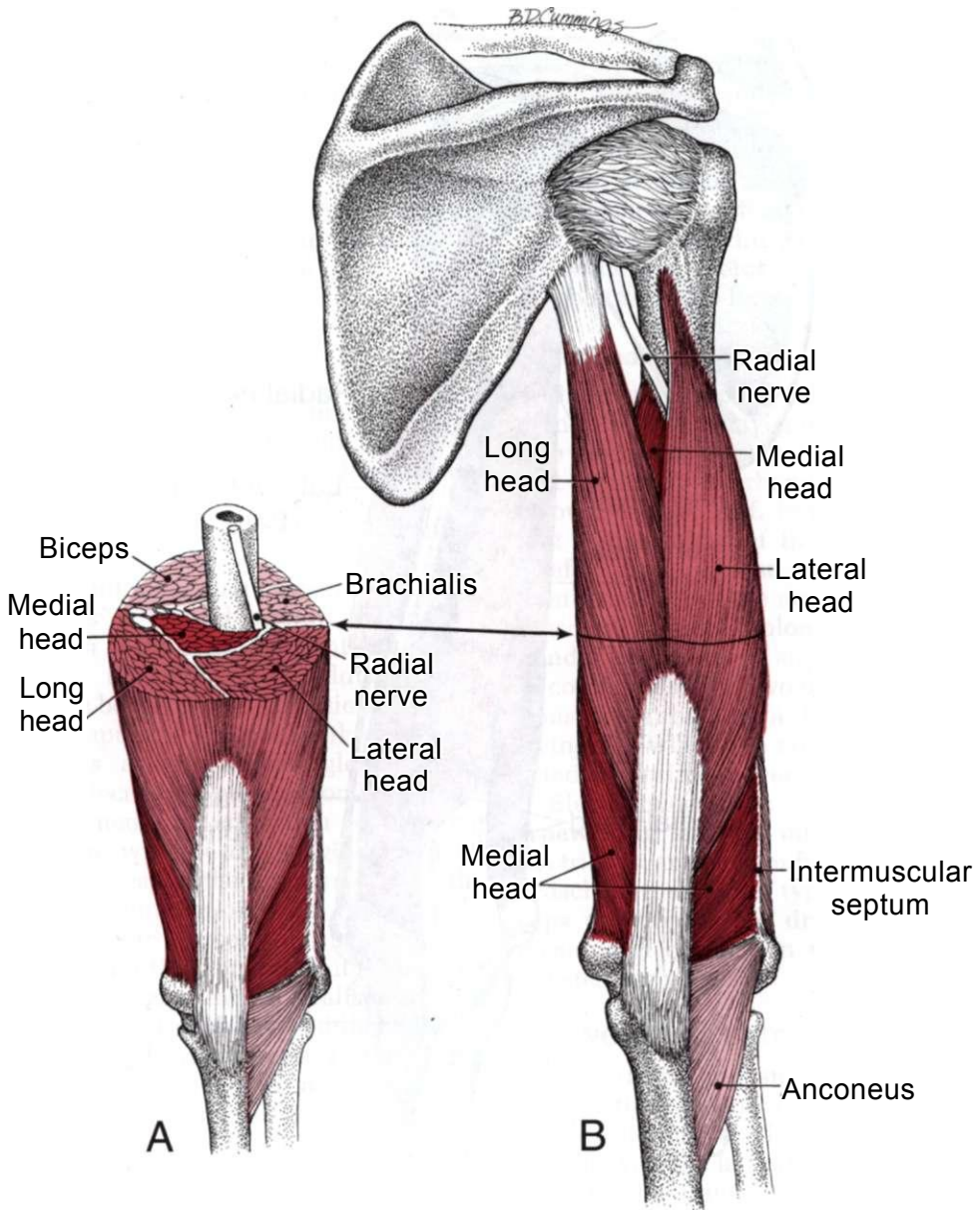


Figure 32.3 **A-B.** Attachments of the right triceps brachii muscle *(two darker reds)* seen from behind and in cross section. The medial (deep) head is *dark red* and the lateral and long heads are *medium red.* The biceps brachii, brachialis, and anconeus muscles are *light red.* **A,** cross section just proximal to the level where the radial nerve penetrates the lateral intermuscular septum, **B,** posterior view of the intact triceps brachii. The horizontal double arrow and *black lines* across the muscle indicate the level of cross section in **A.**

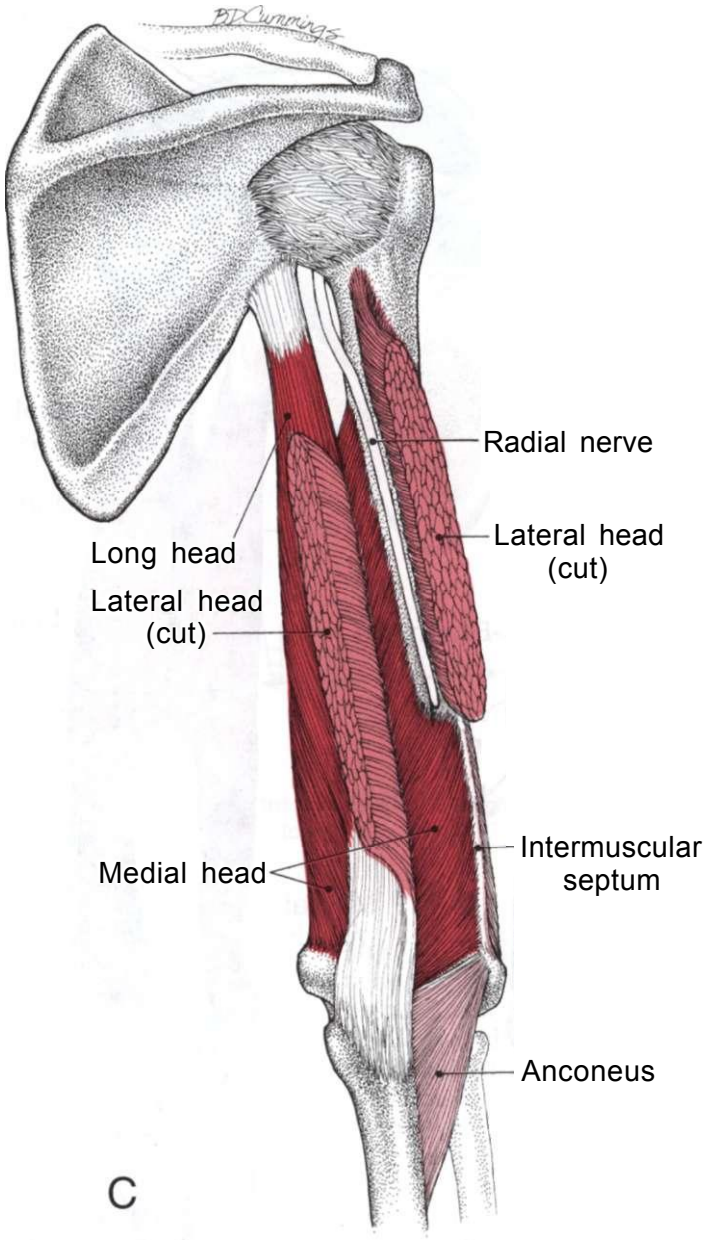


Figure 32.3—*continued* **C,** posterior view with the lateral head cut and reflected, showing the course of the radial nerve, which separates the humeral attachments of the medial and lateral head of the triceps.

the triceps muscles showed no significant difference in this composition.

The **anconeus** muscle appears as an extension of the triceps between the lateral epicondyle and the olecranon process (Fig. 32.3). It attaches ***above*** to the lateral epicondyle and ***below*** to the side of the olecranon process and to the dorsal surface of the ulna.[10]

SUPPLEMENTAL REFERENCES

Other authors have illustrated the triceps brachii as viewed from the medial

aspect,[1, 14] from the lateral aspect,[4,11,34] from behind,[2,12,29,35,41] from behind showing the lateral head reflected to reveal its relation to the radial nerve,[3, 13] and in cross section.[5,18, 33] The anconeus was sometimes included.[6,12]

3. INNERVATION

All heads of the triceps muscle and the anconeus muscle are innervated by branches of the radial nerve *via* the posterior cord of the brachial plexus from spinal roots C_7 and C_8.[10]

4. FUNCTION

All parts of the triceps brachii extend the forearm at the elbow joint.[7,16,21,23,38] However, the medial (deep) head is the workhorse among elbow extensors. It exhibits the earliest and greatest electromyographic (EMG) activity.[7,42] The long head has an additional shoulder-joint function: it adducts[7,21,23, 38] and is said to extend[21,23, 38] the arm at the shoulder joint. On stimulation of the long head, adduction appeared to be the dominant action.[16]

The scapular attachment of the long head influences actions at the glenohumeral joint. Electrical stimulation studies[16] demonstrated that activation of the long head alone, with the arm hanging down, elevated the head of the humerus toward the acromion. Stimulation with the arm abducted to 90° forced the head of the humerus into the glenoid cavity. The long head of the triceps, the pectoralis major and latissimus dorsi all strongly adduct the arm, but the long head counteracts the strong tendency of the other two muscles to pull the head of the humerus downward out of the glenoid fossa.[16] Duchenne found that stimulation of the long head adducted the arm at the glenohumeral joint by drawing the humerus to the scapula without rotating the scapula, whereas stimulating the teres major tended to draw the inferior angle of the scapula toward the humerus without moving the arm.[16] This is not surprising since these two muscles have reverse long and short lever arms resulting in different moments of force at the glenohumeral joint.

The anconeus muscle assists the triceps in extension of the forearm at the elbow.[7] The anconeus was thought by Duchenne[16] to contribute specifically to abduction of the ulna during pronation of the forearm. Perhaps it serves a stabilizing function. It was observed electromyographically to be activated by all index finger movements and to contribute to stabilization of the humeroulnar joint.[40] Other EMG evaluations of anconeus activity concluded that the anconeus, supinator and medial head of the triceps brachii work together to stabilize the elbow joint during pronation and supination of the forearm.[7,42]

Functional Activities

Bilateral triceps brachii muscles were monitored electromyographically with surface electrodes during 13 **sports activities** that included overhand and underhand throws, tennis, golf, baseball hits and 1-foot jumps. Most of the records showed briefer, more intense contraction of the dominant than of the nondominant triceps muscle. The more prolonged activity of the nondominant triceps appeared to function in counterbalance. Two outstanding exceptions were batting a baseball and golf swings, in which the nondominant triceps acted as a prime mover.[8]

Electromyographic monitoring during **typewriting** showed minimal activity in the triceps, and then only as the subject approached maximum typing speed.[25] Triceps activity during **driving** showed no meaningful correlation with deviation of the steering wheel.[22]

5. FUNCTIONAL UNIT

The triceps and anconeus muscles are synergistic extensors of the forearm at the elbow. The long head of the triceps is synergistic with the latissimus dorsi, teres major and teres minor muscles, all of which can act as adductors and extensors of the arm at the shoulder joint, and is synergistic with the pectoralis major in adduction.

As antagonists to the triceps, the biceps and brachialis muscles are prone to develop TrPs (often latent) during chronic TrP involvement of the triceps.

6. SYMPTOMS

The patient is likely to complain of vague, hard-to-localize pain posteriorly in

the shoulder and upper arm. Most patients are unaware of any restriction of arm or forearm motion because of the tendency to keep the elbow slightly flexed, out of the painful range, and to compensate for the slightly reduced reach by additional scapular or body movement. Because of tenderness referred to the medial epicondyle, the elbow may be held away from the side to avoid body contact.

Pain occurs during activity which requires forceful extension at the elbow: in the dominant arm when playing tennis, and in the nondominant arm (elbow held straight) when playing golf. Myofascial elbow pain interferes with either game. As the activity of TrP_2 increases, it is often an important source of pain and loss of function in patients diagnosed as having "tennis elbow" (see Chapter 36).

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Activation of TrPs in the triceps brachii may occur due to overload from overuse of forearm crutches, the stress of a cane that is too long (used because of injury to the back or leg), short upper arms, strain of the muscle in sports (backhand "mis-hit" in tennis), overenthusiastic conditioning exercises (golf practice or push-ups), excessive city driving in a car with manual transmission requiring extensive and repetitive manual gear shifting, or from repetitively pressing tightly bound books on a photocopy machine. Surprisingly, the TrPs in the long head are likely to be activated by sitting for long periods with the elbow held forward in front of the plane of the chest or abdomen and lacking elbow support (e.g., driving a car on a long trip, holding down a sheet of paper with the left hand while writing with the right, or doing needlepoint or other handwork without elbow support). Continuation of this stress will also perpetuate the TrPs.

Triceps brachii TrPs were reported to be activated by jackknife positioning of a patient during nephrolithotomy in a way that held the triceps in the stretched position for a prolonged period of time.[36] The TrPs were inactivated by deep massage of the TrPs and passive stretch, and the patient was relieved of the previously enigmatic pain.

8. PATIENT EXAMINATION (Fig. 32.4)

By increasing muscle tension, TrPs can produce dysfunction. Both active and passive functional arm and forearm movements should be tested.

When the long head of the triceps is involved, the patient is unable to voluntarily adduct that arm against the ear with the elbow held straight (Fig. 32.4); nor can he or she simultaneously fully flex the forearm at the elbow and fully elevate the arm at the shoulder joint, as in the stretch position shown on the subject's right side in Figure 32.6A. This test movement also may be restricted by posterior deltoid TrPs. When full passive stretch across both joints is attempted in this way, the patient may point to a specific area that feels tense or painful; that area is a good place to palpate for TrPs. The patient is unable to fully straighten the elbow against a load when either the medial or the lateral head is involved. Stretching the involved triceps by passively flexing the forearm causes pain, as does loading the muscle by resisting active extension of the forearm at the elbow.[26]

An epicondyle that is painful because of TrPs also is sensitive to tapping because of referred tenderness. Pain in the lateral epicondyle due to activity of triceps TrP_2 often persists in patients with "tennis elbow" after their supinator, biceps brachii and brachioradialis TrPs have been inactivated. Then, residual percussion tenderness of the posterior aspect of the epicondyle indicates that this triceps TrP is probably active.

When the examination suggests the presence of triceps TrPs, it is important to check the related glenohumeral and elbow joints for normal joint play.[30]

9. TRIGGER POINT EXAMINATION (Fig. 32.5).

Gerwin, *et al.*[19] found that the most reliable examination criteria for making the diagnosis of TrPs were the identification of a taut band by palpation, the presence of spot tenderness in the band, the presence of referred pain, and reproduction of the patient's symptomatic pain. Identification of a local twitch response (LTR) by palpa-

tion was unreliable in some muscles. The triceps was not one of the muscles tested in this study, but it is likely to be comparable to the sternocleidomastoid muscle in difficulty. Examinations of the sternocleidomastoid muscle showed a high level of interrater reliability. An LTR is a valuable objective confirmatory finding when present but was the least reliable (most skill-demanding) examination tested.

Figure 32.4. Positive Triceps Brachii Test. If the *long head* contains active trigger points, the patient cannot bring the involved right arm tight against the ear. The shortened triceps muscle will not allow full elevation of the arm at the shoulder joint.

To palpate central TrPs in this muscle, the muscle is lengthened to the beginning of resistance, usually when the elbow is bent 15° or 20°. The arm should be comfortably supported. On the other hand, to examine for tenderness of ATrPs the elbow can be flexed to the point of discomfort. Increasing tension on the region of muscle attachment tends to increase the sensitivity of ATrPs.

Triceps TrP$_1$

This central TrP$_1$ area lies deep in the long head of the triceps at approximately mid-muscle (Fig. 32.1A, left side), a few centimeters distal to where the long head crosses the teres major (*see* Fig. 23.3). The ability of the examiner to locate this TrP is a good test of skill using pincer palpation, which is usually necessary to find it. The fingernails of the examining digits MUST be cut very short. If, after palpation, fingernail marks remain on the skin, the nails were too long. The fingers should encircle the triceps muscle (Fig. 32.5) reaching in until they encounter the humerus (right hand in Fig. 32.5). The long head can be separated slightly from the humerus and its fibers can be rolled between the digits. Clusters of TrPs are often present and are identified by their multiple taut bands, by reproduction of the patient's pain complaint, and often by LTRs.

The tension of the taut bands associated with central TrP$_1$ is likely to contribute to the tenderness of attachment TrP$_4$.

Triceps TrP$_2$

This common contributor to "tennis elbow" pain lies in the distal lateral portion of the medial head of the triceps, 4-6 cm (1 1/2 - 2 1/2 in) above the lateral epicondyle, to which it refers pain in association with other TrPs contributing to a "tennis elbow" myofascial syndrome. This central TrP is found by flat palpation. An LTR can sometimes be seen in a taut band above and behind the lateral epicondyle (Fig. 32.1A, right side).

Triceps TrP$_3$

This central TrP feels like a nodule located by flat palpation at midbelly in the lateral border of the lateral head, just above the point where the radial nerve exits from the musculospiral groove (Figs. 32.1B and

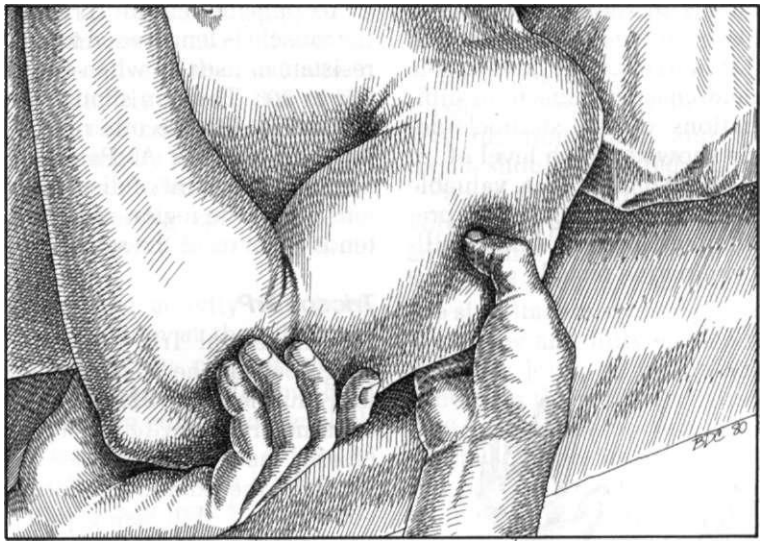


Figure 32.5. Pincer palpation of a central trigger point 1 in the long head of the left triceps brachii muscle. The fingers encircle the long head in a pincer grasp, separating it from the humerus and the adjacent neurovascular bundle. Individual taut bands and their trigger points are located by rolling the muscle fibers between the finger tips. A taut band is tested for a local twitch response by snapping it between the finger tips.

32.3C). The taut band of TrP_3 may entrap the sensory fibers of the radial nerve. In this case, firm palpation along the lateral intermuscular septum, in the region where the radial nerve penetrates the septum, is likely to set off a tingling sensation in the hand. The nodule in the muscle at the TrP lies just above this point of nerve hypersensitivity. The taut bands of $CTrP_3$ may contribute to the enthesopathy of attachment TrP_4.

Triceps TrP$_4$

This attachment TrP_4 (Fig. 32.1B, right side) is found deep in the distal medial head in the region of attachment of the three heads of the triceps, just above the olecranon, to which it refers pain. The enthesopathy producing the tenderness of this ATrP may be secondary to the tension of taut bands associated with triceps TrP_1, TrP_2, TrP_3, and/or TrP_5.

Triceps TrP$_5$

This central TrP is located deep in the medial border of the midfiber region of the medial head just above the medial epicondyle, where it projects its pain and tenderness (Fig. 32.1C). This TrP is found by flat palpation, with the patient lying supine and the arm laterally rotated at the shoulder joint. It also can contribute to the tenderness of attachment TrP_4.

10. ENTRAPMENT
Lateral Head

The TrP_3 nodule is found in the lateral border of the lateral head of the triceps brachii (Fig. 32.1B), just proximal to the exit of the radial nerve from the musculospiral (radial) groove (Fig. 32.3C). Activation of this TrP is often associated with sensory signs and symptoms of compression of the radial nerve. The patient complains of tingling and numbness (dysesthesias) over the dorsum of the lower forearm, wrist, and hand to the base of the middle finger, which lies in the sensory distribution of the radial nerve. By comparison, the aching pain referred from TrP_3 appears in the two "ulnar" (fourth and fifth) digits.

Symptoms of nerve compression may be relieved within minutes to days after an in-

jection of the TrP that releases the responsible taut band of muscle. The local anesthetic solution may temporarily block the radial nerve. This TrP_3 responds poorly to stretch and spray.

Clinical and EMG evidence of radial nerve neuropraxia indicates that entrapment occurred along its passage beneath the triceps muscle. Careful dissection of cadavers revealed in almost every body an accessory part of the lateral head that originated below the spiral groove. The attachment of this slip of muscle to the humerus forms a fibrotic arch of variable snugness over the radial nerve. This arch is distinct from the opening of the lateral intermuscular septum.[24] A patient with a 3-year history of an atraumatic radial paresis progressing to a paralysis was relieved by surgical release of lateral head fibers that attached near the radial nerve.[27] The TrP_3 fibers may tense this arch, contributing to a nerve entrapment.

Anconeus Epitrochlearis Muscle

This anomalous muscle has been reported to be the cause of ulnar compression neuropathy in four patients.[28] They were relieved of symptoms by excision of the muscle. Two other cases of an anomalous anconeus epitrochlearis muscle also were reported.[9]

11. DIFFERENTIAL DIAGNOSIS

Lateral elbow pain that may mimic referred pain from TrP_2 may be caused by entrapment of the radial nerve by the arcade of Frohse or other soft tissues overlying the radial head.[31]

Related Diagnoses

Whenever the diagnoses of "**tennis elbow," lateral or medial epicondylitis, olecranon bursitis,** and **thoracic outlet syndrome** are being seriously considered, the possibility that at least some of the symptoms are being caused by triceps brachii TrPs must be explored. Tennis elbow (lateral epicondylitis) is discussed in detail in Chapter 36, section 11, and thoracic outlet syndrome in Chapter 20, section 11.

Pain referred from the triceps brachii to the vicinity of the elbow joint may be mistakenly attributed to **arthritis.**[39]

Since pain from this muscle may focus on the back of the arm and extend into the hand, it is sometimes erroneously thought to result from a C_7 **radiculopathy.**[39]

The cubital tunnel syndrome is more likely to cause hypoesthesia of the skin in the ulnar distribution of the hand, and weakness and clumsiness of the hand, rather than pain.[15] The cubital tunnel entrapment syndrome is associated with slowing of ulnar nerve conduction through the cubital tunnel, whereas the pain from myofascial TrPs is not.

Any of these conditions may exist and must be diagnosed and treated appropriately.

Related Trigger Points

The synergistic latissimus dorsi, teres major and teres minor muscles often exhibit associated TrPs.

If the elbow pain persists in the lateral epicondylar area after eliminating TrPs in the triceps brachii, then the anconeus, supinator, brachioradialis, and extensor carpi radialis longus muscles may be harboring TrPs that also refer pain to that region.

Key TrPs in the homolateral latissimus dorsi muscle or serratus posterior superior muscle may cause satellite TrPs in the triceps brachii.[20] For lasting release of a satellite TrP, its key TrP must be inactivated.

12. TRIGGER POINT RELEASE (Fig. 32.6)

The stretch and release techniques described in Chapter 3, Section 12 are primarily applicable to triceps *central* trigger points (TrPs). However, many of those direct manual medicine techniques may not be appropriate for the triceps *attachment* TrP_4. The primary therapeutic approach to attachment TrPs is inactivation of associated central TrPs. When the examiner finds tenderness at the attachment point: (1) he or she should palpate from that point back along the induration or taut band (toward the midfiber region of that portion of muscle) to search for a TrP in the more central part of the muscle. (2) If a central TrP is found (as is generally the case), the muscle should be placed in a position of ease (not stretched) and the central TrP should be

treated by nonstretch techniques, such as trigger point pressure release, deep massage, and/or hold-relax, all of which can be preceded by vapocoolant or icing. Indirect techniques can be effective; stretching that places increased tension on the attachments is not desirable when there is an attachment TrP present. (3) If a midfiber TrP is *not* found, then the attachment itself can be treated by ice massage at the attachment region with the muscle in a position of ease, by indirect myofascial release, by phonophoresis to introduce hydrocortisone ointment (which can be preceded and followed by ice massage), or by injection (refer to next section). Injection of attachment TrPs can expedite pain relief.

For inactivation of triceps central TrPs using a spray-and-stretch technique, first the patient is seated and the forearm flexed at the elbow to the point of resistance. A few sweeps of spray are applied as illustrated (Fig. 32.6). After full flexion at the elbow, the long head can be stretched further if needed by passively flexing the arm at the shoulder joint (Fig. 32.6A). For releasing TrPs in the medial and lateral heads, only elbow flexion is necessary because the lateral heads do not cross the glenohumeral joint. The spray or icing is applied from proximal to distal, starting at the latissimus dorsi in the posterior axillary fold and continuing over the triceps brachii, around the elbow, and down the forearm to include the fourth and fifth fingers (Fig. 32.6A). Full flexion at the shoulder joint may be limited also by latissimus dorsi TrPs. In that case, the latissimus should be released before proceeding with the triceps release.

In the position shown in Figure 32.6A, postisometric relaxation (PIR) combined with the use of reciprocal inhibition can be employed to achieve full lengthening of the long head of the muscle. The patient is instructed: (1) to gently press the elbow forward against the operator's resisting hand (isometric contraction phase); (2) to breathe in and hold the contraction for 6 seconds; (3) to breathe out slowly and relax; (4) and to move the arm posteriorly (toward the operator in Fig. 32.6A). This last movement is an active one by the patient, utilizing reciprocal inhibition to further relax the triceps.

Nielsen[32] describes and illustrates in a case report the use of spray and stretch to release TrPs in the long head of the triceps brachii.

To obtain a similar stretch of the triceps brachii muscle in the supine patient, the forearm should be flexed at the elbow and the arm flexed at the shoulder joint to place the supinated hand beneath the shoulder, as in Figure 32.6B. The vapocoolant spray is again applied in parallel sweeps, starting at the latissimus dorsi adjacent to the scapula, covering the triceps distally over the arm and over the elbow to the wrist. In this supine position, as described for the sitting position, PIR can be combined with reciprocal inhibition for effective release of the muscle.

13. TRIGGER POINT INJECTION (Figs. 32.7-32.10)

The basic principles for injection are covered in Chapter 3, Section 13. Note the distinctions between injecting central trigger points (CTrPs) and attachment trigger points (ATrPs) in that chapter. Refer to Section 9 in this chapter for precise palpation of these TrPs.

Rachlin[37] mentions the injection of TrPs in all three heads and illustrates injection of TrPs in the lateral and long heads of the triceps brachii.

TrPr-Patient Supine or Sidelying (Fig. 32.7)

To inject this central TrP by approaching the medial side of the long head of the triceps, the supine patient laterally rotates the arm so that the antecubital space faces up and abducts the arm sufficiently to place the long head on a slight stretch (Fig. 32.7A). The operator encircles the long head of the muscle in a pincer grasp and lifts it away from the underlying bone, away from the adjacent major *blood vessels and nerve,* and away from the lateral head of the triceps (beneath which the radial nerve courses). For injection, the TrP in the palpable band is fixed and injected *between* the tips of the digits. Effective penetration of these TrPs by the needle produces LTRs that are easily seen and can be

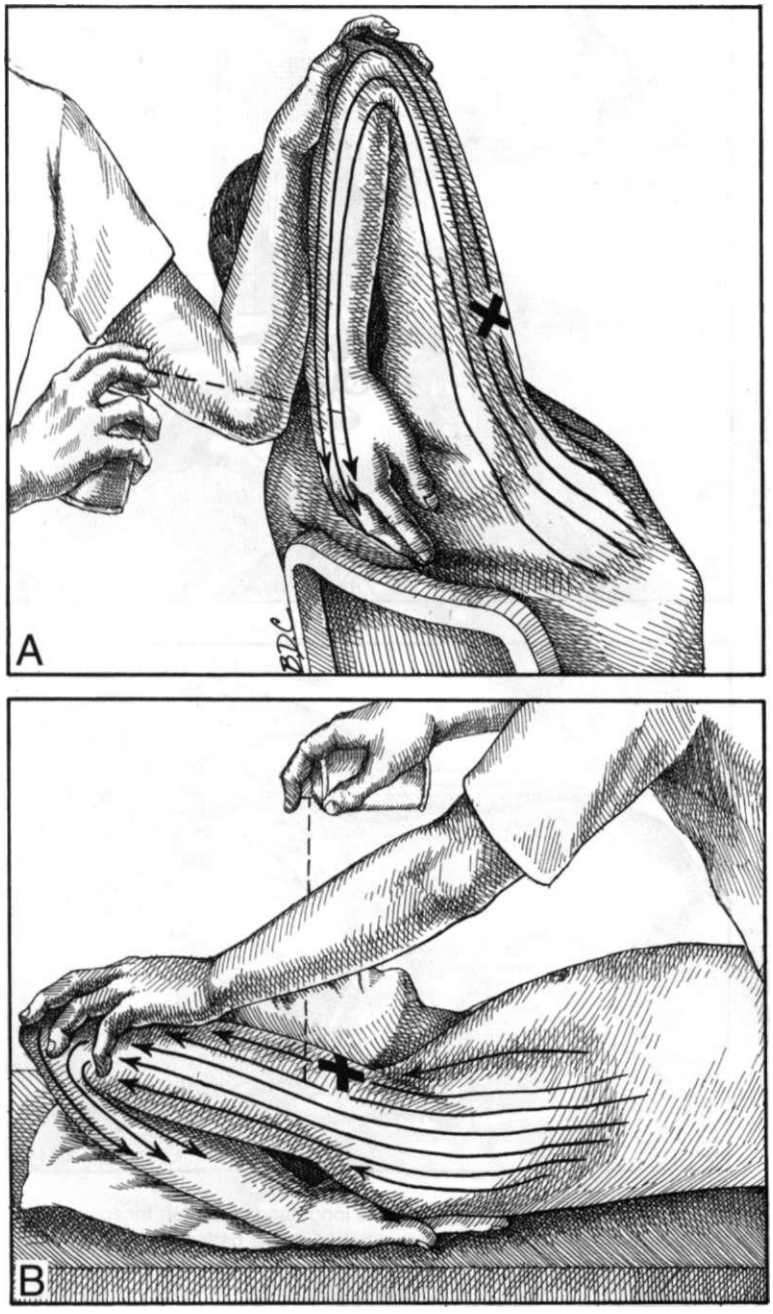


Figure 32.6. Stretch position and spray pattern (*arrows)* for a trigger point **(X)** in the long head of the triceps brachii. This technique is also effective for the other two heads but to stretch them, there is no need to elevate the arm. Only elbow flexion is needed. **A,** patient seated. **B,** patient supine; this position is likely to be more effective because the patient relaxes more completely. See text for description of release by postisometric relaxation combined with reciprocal inhibition.

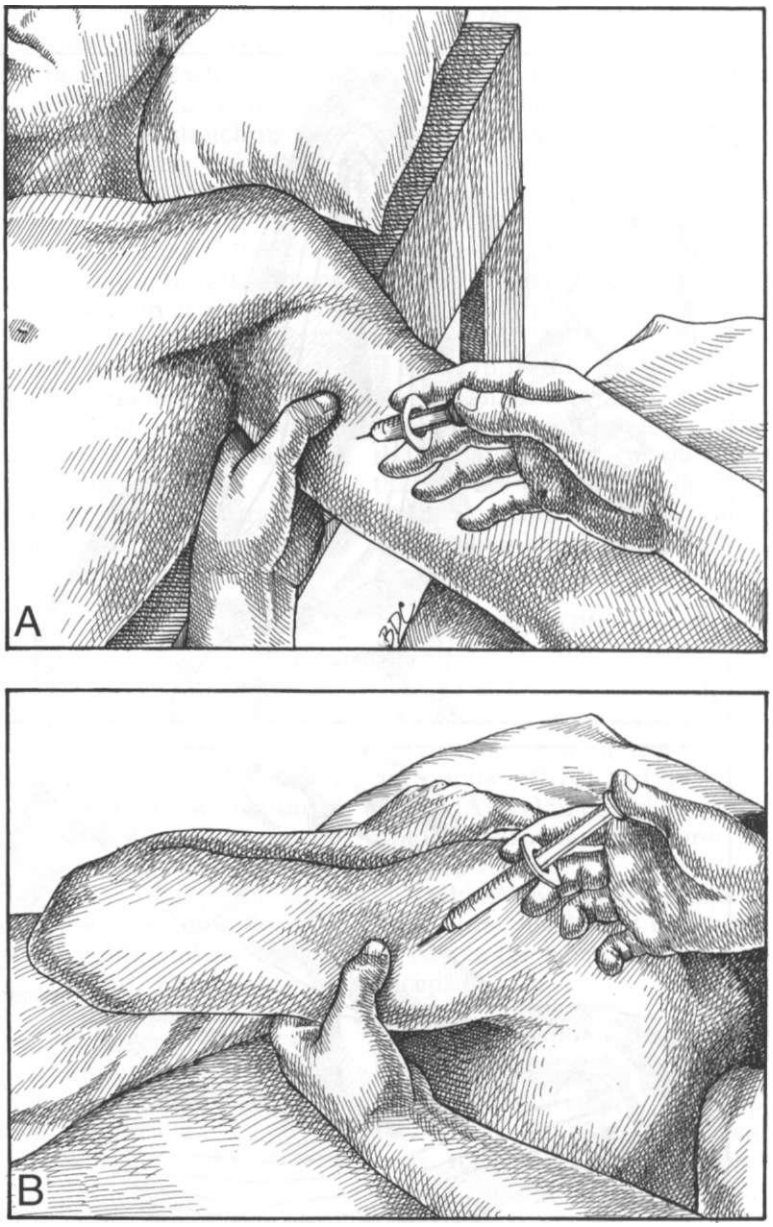


Figure 32.7. Injection of the trigger point 1 region in the long head of the left triceps brachii. A, anterior approach, with the patient supine. **B,** posterior approach, with the patient lying on the uninvolved side.

felt by the encompassing fingers and thumb. The occurrence of these LTRs is very important because they signal effective placement of the needle.

If it is a more convenient position, or if the TrPs are in the lateral part of the long head, this CTrP area can be approached from the lateral aspect of the arm. To do so, the patient lies on the opposite side with the arm to be injected uppermost, facing away from the operator (Fig. 32.7B), permitting the operator to grasp the muscle and inject the TrPs as described above. These TrPs also can be injected through the muscle from the anterior approach.

TrP_2-Patient Sidelying
(Fig. 32.8A)

The patient lies on the uninvolved side with the arm supported on a pillow (Fig. 32.8A). TrP_2 is palpated distally in the lateral border of the medial head, adjacent to the attachments of the extensor carpi radialis longus and the brachioradialis muscles. For injection, the TrP is fixed between the fingers by pressing the muscle on both sides of the TrP against the humerus (Fig. 32.8A).

TrP_3-Patient Sidelying
(Fig. 32.8B)

For injection of central TrP_3, the patient is placed in the same position as described above for TrP_2. The central TrP_3 is located

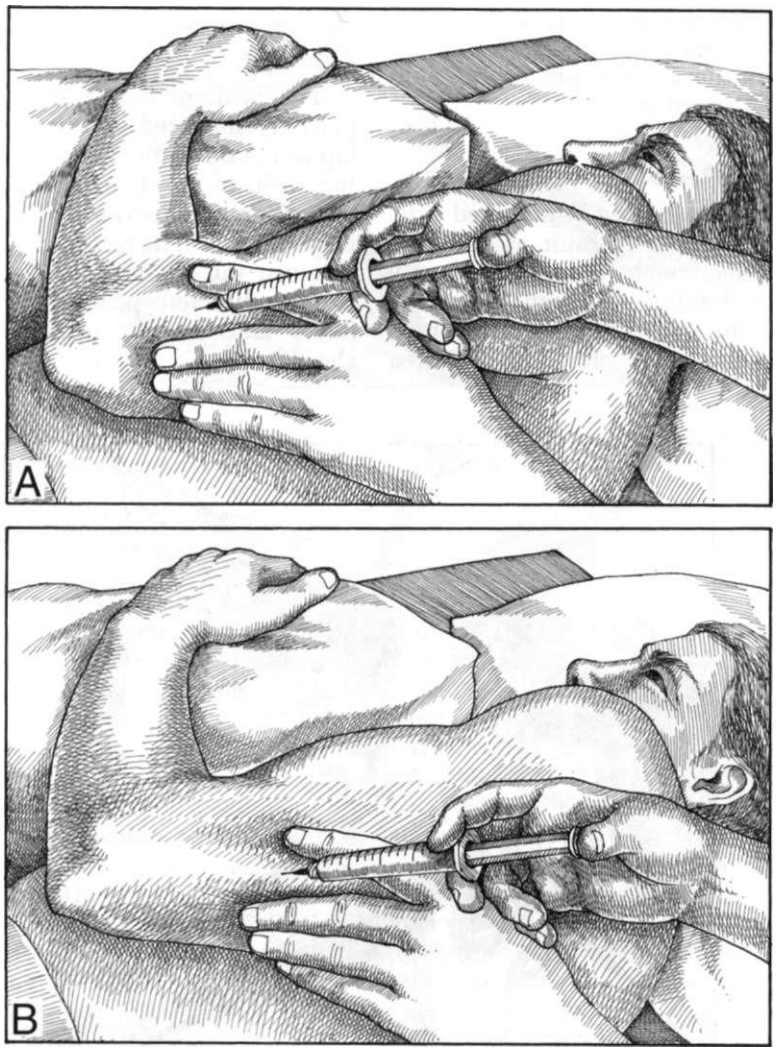


Figure 32.8. Injection of the trigger point 2 and trigger point 3 regions in the left triceps brachii with the patient lying on the right side and the uppermost arm resting on a pillow against the chest wall, elbow bent. **A,** The trigger point 2 region lies in the lateral border of the medial head, distally in the arm; it refers pain and tenderness to the lateral epicondyle. This trigger point is located about three or four fingers breadth proximal to the lateral epicondyle. B, Injection of central trigger point 3 in the more distal fibers in the lateral border of the lateral head; it refers pain locally over the muscle, to the dorsum of the forearm, and to the fourth and fifth digits.

along the lateral border of the lateral head, just above the exit of the radial nerve, which courses beside the brachialis and then beneath the brachioradialis muscle. The needle is inserted tangentially into a thin layer of muscle (Fig. 32.8B) and may be directed either distally or proximally (whichever is more convenient), probing for TrPs in a fan-like pattern.

It is not unusual for some procaine solution to infiltrate the radial nerve and cause a temporary partial nerve block. If the diluted 0.5% procaine solution is used for injection, the nerve recovers its function within 15-20 minutes.

Attachment TrP$_s$-Patient Sidelying

The patient lies on the uninvolved side, facing away from the operator, as in Figure 32.8. This attachment TrP is located only by spot tenderness to deep palpation through the thick aponeurosis of all three heads of the triceps brachii. This TrP area is injected deeply, aiming toward the olecranon process. Penetration of the TrP by the needle is confirmed primarily by the patient's report of a local pain response and of referred pain. Occasionally, the operator feels a local twitch of the muscle when the needle strongly stimulates the sensitized nociceptors in this attachment region. The LTR indicates that the needle is in a TrP region that contributes to the local tenderness and TrP activity.

TrP$_s$-Patient Supine **(Fig. 32.9)**

The patient's laterally rotated and partially abducted arm lies on the padded lap of the operator (Fig. 32.9). Central TrP$_5$ lies deep in the distal medial head of the muscle and is identified by its spot tenderness and LTR. The region of the TrP is fixed between the fingers to inject it, with the needle directed parallel to the muscle fibers and usually upward toward the shoulder.

Figure 32.9. Injection of trigger points (central TrP$_5$ region) in the distal medial head of the left triceps brachii with the patient supine. The arm is laterally rotated, the forearm supinated, and the slightly flexed elbow is supported on a pillow on the operator's lap.

Figure 32.10. Injection of a central trigger point in the midfiber region of the right anconeus muscle of a supine patient. The patient's elbow is flexed slightly and the forearm pronated. The tender trigger point should be localized by the fingers against the ulna, between the olecranon process and the lateral epicondyle,

This TrP is not especially close to the neurovascular bundle, but if one peppers and injects the area too widely while injecting, one can cause a temporary block of the median or ulnar nerve.

Anconeus
(Fig. 32.10)

The arm of the supine patient is supported on a padded surface with the forearm flexed about 45° at the elbow and the hand pronated (Fig. 32.10). For injection, this central TrP is fixed between the fingers of the palpating hand.

14. CORRECTIVE ACTIONS

When typing, writing, reading, etc., the patient should keep the arm vertical, with the elbow behind the plane of the chest and not projected forward. Whenever possible, an armrest of suitable height should support the elbow.

To correct for short upper arms in relation to the torso height, a writing-board with padding glued underneath is used to raise the arms, or the height of the armrests above the seat is increased directly.

If forearm crutches are necessary, their use should be increased gradually to avoid sudden overload of the arm muscles, especially the triceps.

In tennis, the patient may change to a lighter weight racquet or to one not so heavy in the head. Also, it may be helpful to shorten the grip on the racquet handle, which reduces the leverage on this elbow extensor muscle.

Chinning on a bar and push-ups, which easily overload the arm muscles, should be avoided until after recovery and then resumed progressively.

For a home exercise program, the patient should be taught how to stretch the triceps brachii by assuming the patient po-

sition in Figure 32.6A while seated under a warm shower with the water streaming over the muscle.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991, p. 385 (Fig. 6.39).
2. *Ibid.* p. 386 (Fig. 6.40).
3. *Ibid.* p. 387 (Fig. 6.41).
4. *Ibid.* p. 388 (Fig. 6.43).
5. *Ibid.* p. 389 (Fig. 6.44).
6. *Ibid.* p. 403 (Fig. 6.66).
7. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 240, 263, 280, 281).
8. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sports Skill.* Charles C Thomas, Springfield, 111. 1967.
9. Chalmers J: Unusual causes of peripheral nerve compression. *Hand 10*(2):168-175, 1978.
10. Clemente CD: *Gray's Anatomy* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 528, 529, 538).
11. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 56).
12. *Ibid.* (Figs. 57, 59).
13. *Ibid.* (Fig. 60).
14. *Ibid.* (Fig. 61).
15. Craven PR, Green DP: Cubital tunnel syndrome. *J Bone Joint Surg* 62A.-986-989, 1980.
16. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 85, 86).
17. Elder GC, Bradbury K, Roberts R: Variability of fiber type distributions within human muscles. *J Appl Physiol* 53(6):1473-1480, 1982.
18. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 80, 81).
19. Gerwin RD, Shannon S, Hong CZ, *et al:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
20. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(1):29-59, 1994.
21. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 112).
22. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving. *Ergonomics 1*8:375-388, 1975.
23. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 270).
24. Lotem M, Fried A, Levy M, *et al:* Radial palsy following muscular effort. *J Bone Joint Surg* 5323:500-506, 1971.
25. Lundervold AJ: Electromyographic investigations of position and manner of working in typewriting. *Acta Phys Scand 24(Suppl. 84)1-171,* 1951 (pp. 66, 67, 94, 95, 97, 100).
26. Macdonald AJ: Abnormally tender muscle regions and associated painful movements. *Pain* 8.197-205, 1980.
27. Manske PR: Compression of the radial nerve by the triceps muscle. *J Bone Joint Surg* 59A:835-836, 1977.
28. Masear VR, Hill JJ Jr, Cohen SM: Ulnar compression neuropathy secondary to the anconeus epitrochlearis muscle. *J Hand Surg [Am] 13(5).*-720-724, 1988.
29. McMinn RM, Hutchings RT, Pegington J, *et al: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 128).
30. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
31. Minami M, Yamazaki J, Kato S: Lateral elbow pain syndrome and entrapment of the radial nerve. *J Jpn Orthop Assoc* 66:222-227, 1992.
32. Nielsen AJ: Case study: myofascial pain of the posterior shoulder relieved by spray and stretch. *J Orthop Sport Phys Ther* 3:21-26, 1981.
33. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Figs. 44, 61).
34. *Ibid.* (Fig. 57).
35. *Ibid.* (Fig. 59).
36. Prasanna A: Myofascial pain as postoperative complication [Letter], *J Pain Symptom Manage 8(7)450-*451, 1993.
37. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994 (pp. 197-360).
38. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 179, 180).
39. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981 (Tables 1 and 2).
40. Sano S, Ando K, Katori I, *et al.:* Electromyographic studies on the forearm muscle activities during finger movement. *J Jpn Orthop Assoc* 52:331-337,1977.
41. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 322).
42. Travill AA: Electromyographic study of the extensor apparatus of the forearm. *Anat Rec* 244:373-376,1962.
43. Winter SP: Referred pain in fibrositis. *Med Rec* 257:34-37, 1944 (p. 37).

PART 4
FOREARM AND HAND PAIN

CHAPTER 33
Overview of Forearm and Hand Region

INTRODUCTION TO PART 4

This fourth part of the ***Trigger Point Manual*** includes the forearm and hand muscles, and all those that cross the elbow joint, except the anconeus, biceps, brachialis and triceps. This chapter has two sections: Section A presents the Pain and Muscle Guide information. Section B presents Diagnostic Considerations applicable to this part of the body including the **Carpal Tunnel Syndrome, Other Differential Diagnoses,** and **Joint Play.**

SECTION A
PAIN AND MUSCLE GUIDE

This guide lists the muscles that may be responsible for pain in the areas shown in Figure 33.1. The muscles most likely to refer pain to each specific area are listed below under the name of that area. The names of the pain areas identified in the figure are arranged in alphabetical order in the guide. One uses this chart by locating the name of the area that hurts and then by looking under that heading for a listing of all the muscles that might cause the pain. Then, reference should be made to the individual muscle chapters. The number for each chapter is shown in parentheses following the muscle name.

The muscles are listed in such a way that the muscle which is a more frequent cause of pain in an area is listed higher than others. This order is only an approximation; the selection process by which patients reach an examiner greatly influences which muscles are most likely to be involved. **Boldface** type indicates that the muscle refers an essential pain pattern to that pain area. Roman type indicates that the muscle refers a spillover pattern to that pain area. TrP stands for trigger point.

PAIN GUIDE

ANTECUBITAL PAIN

Brachialis (31)
Biceps brachii (30)

BASE-OF-THUMB AND RADIAL HAND PAIN

Supinator (36)
Scaleni (20)
Brachialis (31)
Infraspinatus (22)
Extensor carpi radialis longus (34)
Brachioradialis (34)
Opponens pollicis (39)
Adductor pollicis (39)
Subclavius (42)
First dorsal interosseus (40)
Flexor pollicis longus (38)

DORSAL FINGER PAIN

Extensor digitorum (35)
Interossei (40)
Scaleni (20)
Abductor digiti minimi (40)
Pectoralis major (42)
Pectoralis minor (43)
Latissimus dorsi (24)
Subclavius (42)

DORSAL FOREARM PAIN

Triceps brachii ($TrPs_{1\text{-}3}$) (32)
Teres Major (25)
Extensores carpi radialis longus and brevis (34)
Coracobrachialis (29)
Scalenus minimus (20)

DORSAL WRIST AND HAND PAIN

Extensor carpi radialis brevis (34)
Extensor carpi radialis longus (34)
Extensor digitorum (35)
Extensor indicis (35)
Extensor carpi ulnaris (34)
Subscapularis (26)
Coracobrachialis (29)
Scalenus minimus (20)
Latissimus dorsi (24)
Serratus posterior superior (47)
First dorsal interosseus (40)

LATERAL EPICONDYLAR PAIN

Supinator (36)
Brachioradialis (34)
Extensor carpi radialis longus (34)
Triceps brachii (TrP_3) (32)
Supraspinatus (21)
Fourth and fifth finger extensors (35)
Anconeus (32)

MEDIAL EPICONDYLAR PAIN

Triceps brachii (TrP_5) (32)
Pectoralis major (42)
Pectoralis minor (43)
Serratus anterior (46)
Serratus posterior superior (47)

OLECRANON PAIN

Triceps brachii (TrP_4)(32)
Serratus posterior superior (47)

RADIAL FOREARM PAIN

Infraspinatus (22)
Scaleni (20)
Brachioradialis (34)
Supraspinatus (21)
Subclavius (42)

ULNAR FOREARM PAIN

Latissimus dorsi (24)
Pectoralis major (42)
Pectoralis minor (43)
Serratus posterior superior (47)

VOLAR FINGER PAIN

Flexores digitorum superficialis and profundus (38)
Interossei (40)
Latissimus dorsi (24)
Serratus anterior (46)
Abductor digiti minimi (40)
Subclavius (42)

VOLAR FOREARM PAIN

Palmaris longus (37)
Pronator teres (38)
Serratus anterior (46)
Triceps brachii (TrP_5) (32)

VOLAR WRIST AND PALMAR PAIN

Flexor carpi radialis (38)
Flexor carpi ulnaris (38)
Opponens pollicis (39)
Pectoralis major (42)
Pectoralis minor (43)
Latissimus dorsi (24)
Palmaris longus (37)
Pronator teres (38)
Serratus anterior (46)

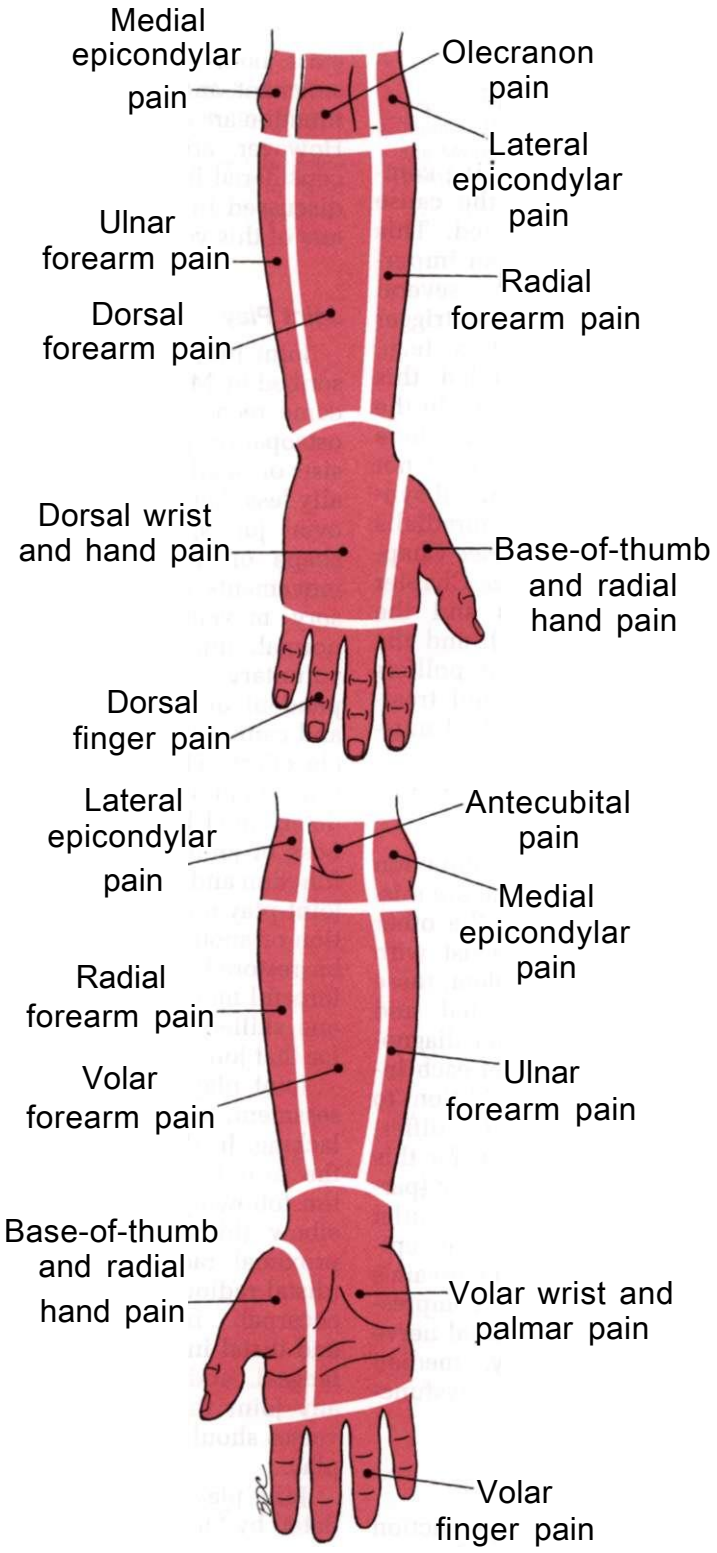


Figure 33.1. The designated areas within the elbow-to-finger region that may encompass pain referred there by myofascial trigger points.

SECTION B
DIAGNOSTIC CONSIDERATIONS

Carpal Tunnel Syndrome

When the median nerve is being compressed in its carpal tunnel, the cause must be identified and corrected. This currently popular diagnosis is an important differential diagnosis for several shoulder girdle and upper limb trigger points (TrPs). Previous chapters (e.g., scaleni, brachialis) have included this syndrome in differential diagnosis. In the forearm and hand region as well, there are TrP sources that mimic some, if not all, of the symptoms of carpal tunnel syndrome. Examples are the brachioradialis and the radial wrist extensors (see Chapter 34), the palmaris longus (see Chapter 37), the flexor carpi radialis and the pronator teres (see Chapter 38), and the opponens pollicis and adductor pollicis (see Chapter 39). Assessment and treatment are covered in the individual muscle chapters.

Other Differential Diagnoses

Trigger points can cause dysfunction and can refer patterns of pain that are mistaken for other conditions. On the other hand, other conditions can coexist with TrPs or can be the primary problem; these conditions must be differentiated and treated appropriately. Differential diagnosis is considered in section 11 of each individual muscle chapter. In addition to carpal tunnel syndrome, among the differential diagnoses to be considered for this region of the body are radiculopathy (particularly C_{5-6}, C_7, C_8, T_1, thoracic outlet syndrome, "tennis elbow" (lateral epicondylitis), osteoarthritis, DeQuervain's stenosing tenosynovitis, nerve compression from a variety of causes (radial nerve entrapment, ulnar neuropathy, median nerve compression), and articular dysfunctions (including subluxation).

Articular Dysfunction

Trigger points and articular dysfunction interact and often coexist. When they coexist, both must be treated. Complete assessment and treatment of articular dysfunction are outside the scope of this book. However, articular dysfunction must be considered in differential diagnosis and is discussed in the individual muscle chapters of this volume.

Joint Play

Joint play and its importance were described by Mennell.[3,4] Since then it has become recognized and appreciated by the osteopathic profession.[1,2] Joint play consists of small involuntary movements (usually less than 3 mm or 1/8 inch) within synovial joints; the range depends on the shape of the joint surfaces. Joint play movements are normally painless accessory movements that are essential for normal, unrestricted, pain-free, range of voluntary movement. Joint play is independent of voluntary muscle contraction and cannot be induced by deliberate muscle effort. Therefore, the presence or absence of joint play movements can only be determined by passive joint examination. Loss of joint play is a form of joint dysfunction and is identified by loss of normal joint-play range of movement in one direction or another. The dysfunction often can be restored to normal with simple, nonforceful mobilization of the joint by someone skilled in the appropriate technique for that joint.

Joint play should be considered in assessment, and it should be restored if lacking. In this forearm and hand region, the examiner needs to consider at least the following articulations in assessment: elbow (humeroulnar, radiohumeral, and proximal radioulnar articulations), wrist (distal radioulnar, radiocarpal, ulnomeniscocarpal, intercarpal), carpometacarpal and distal intermetacarpal, metacarpophalangeal, and interphalangeal. In general, any joint that an individual muscle traverses should be assessed for normal joint play.

Joint play for this region is described in detail by Mennell[4] and by Greenman.[1]

REFERENCES

1. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 99-103, 402-406).
2. Jacobs AW, Falls WM: Anatomy. Chapter 3. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997, pp. 27-43 (see p. 35).
3. Mennell JM: *Back Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1960.
4. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964 (pp. 3-5 and Chapters 4-7).

CHAPTER 34
Hand Extensor and Brachioradialis Muscles

HIGHLIGHTS: The extensor muscles of the hand at the wrist are the extensores carpi radialis longus and brevis, and the extensor carpi ulnaris. The "painful weak grip" muscles are primarily the extensores carpi radialis longus and brevis, and the extensor digitorum. The brachioradialis and supinator also may develop trigger points (TrPs) in association with the radial hand extensors. The active TrPs of these "extensor mass" muscles occur close together in the proximal forearm, distal to but near the lateral epicondyle. **REFERRED PAIN** from TrPs in the extensores carpi radialis longus and brevis appears over the lateral epicondyle, lightly over the dorsum of the forearm, and accents the dorsum of the hand. The extensor carpi ulnaris refers pain to the dorsal surface of the ulnar side of the wrist. The brachioradialis refers pain chiefly to the lateral epicondyle and down over the length of the muscle to the dorsal aspect of the web of the thumb. **ANATOMY:** attachments of the hand extensors are to the region of the lateral epicondyle at the elbow, and distally to various metacarpal bones. The brachioradialis attaches to the shaft of the humerus above the elbow, and distally to the styloid process of the radius. Several variations may occur. **FUNCTION** of the wrist extensors is to synergistically prevent wrist flexion while the finger flexors are being used for grasp. The action of the extensor carpi radialis longus is chiefly radial deviation of the hand. The brevis chiefly extends the hand while the extensor carpi ulnaris primarily deviates the hand toward the ulnar side. The brachioradialis primarily assists flexion of the forearm at the elbow and helps restore the forearm from supination to the neutral position when overcoming resistance. **SYMPTOMS** are usually pain as described above—often diagnosed as a "tennis elbow" syndrome—and an unreliable or weak grip that lets objects fall from the patient's hand. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in these muscles arise from abuse of combined gripping and twisting motions, as in some sports, digging with a trowel in the garden, and using a screw driver. **PATIENT EXAMINATION** that reveals a painful and weak grip when the hand is ulnarly deviated indicates involvement of the extensores carpi radialis longus and brevis. Epicondyle tenderness is commonly present. **TRIGGER POINT EXAMINATION** for a tender nodule in a taut band localizes the active TrPs by pincer palpation of the brachioradialis and flat palpation of the other forearm muscles. **ENTRAPMENT** of either the motor or sensory branch of the radial nerve may be caused by tension of the extensor carpi radialis brevis. **TRIGGER POINT RELEASE** by spray and stretch requires that the extensores carpi radialis and ulnaris muscles are fully lengthened by movement of both the elbow and wrist joints. A proximal-to-distal spray pattern is used. **TRIGGER POINT INJECTION** of these muscles presents no special difficulty when the TrP is accurately located and fixed between the fingers. **CORRECTIVE ACTIONS** include eliminating strain of the involved muscles, establishing a home program of stretch exercises, and the gradual resumption of normal activities after inactivating the TrPs.

1. REFERRED PAIN
(Figs. 34.1 and 34.2)

Radial Hand Extensors
(Fig. 34.1)

Trigger points (TrPs) in the **extensor carpi radialis longus** refer pain and tenderness to the lateral epicondyle (Fig. **34.1**C) and to the dorsum of the hand in the region of the anatomical snuff box, which is often described by the patient as "the thumb."[72, 73] **Extensor carpi radialis brevis** TrPs project pain to the back of the hand and wrist (Fig. **34.IB),** as originally determined in **45** patients.[72] This is one of the most common myofascial sources of pain in the back of the wrist.

Gutstein-Good,[33] who later wrote as Good,[30] reported a case of "idiopathic myalgia," or "muscular rheumatism," in which pain was projected deep in the upper arm with dysesthesia (numbness, pins-and-needles, and painful vibratory sensations) along the forearm to the thumb and index finger. The pain was reproduced by pressure on tender spots in the extensor carpi radialis muscles. Kelly[42, 43] reported three cases of "fibrositis" with pain in the elbow, radiating down the dorsum of the forearm, or to the radial side of the wrist. The pain originated in a tender spot within the extensor muscle mass several centimeters distal to the lateral epicondyle. This is where the authors find TrPs in the extensor carpi radialis longus. Bates and Grunwaldt[11] reported a similar myofascial pain pattern for the extensor carpi radialis muscles in children.

Extensor Carpi Ulnaris
(Fig. 34.1 A)

The extensor carpi ulnaris muscle harbors TrPs less often than the extensores carpi radialis. The referred pain pattern of the extensor carpi ulnaris includes primarily the ulnar side of the back of the wrist (Fig. **34.1A).** Gutstein[32, 33] identified this TrP and pain pattern in a doctor.

Similar pain patterns for these wrist extensor muscles were illustrated by Bonica and Sola.[12]

Brachioradialis
(Fig. 34.2)

The brachioradialis projects its essential pain pattern to the wrist and base of the thumb in the web space between the thumb and index finger (Fig. **34.2).** The brachioradialis, like the underlying supinator, refers pain also to the lateral epicondyle as also illustrated by Bonica and Sola.[12] For the supinator and brachioradialis, this also is an essential pain pattern. Pain referred to the lateral epicondyle from TrPs in either muscle causes the epicondyle to become tender to light tapping on its distal face. Referred pain from the brachioradialis rarely extends to the olecranon process.

The brachioradialis is a thin muscle that immediately overlies the extensor carpi radialis longus. Using flat palpation, it is usually difficult to distinguish which of these muscles is causing the referred pain. Kelly[42] ascribed to the brachioradialis muscle a pattern of pain and tenderness close to the elbow, and of diffuse referred pain and tenderness across the dorsum of the hand. However, in our experience, pain across the back of the hand arises chiefly from associated TrPs in the extensor carpi radialis brevis or longus.

Infusion of 0.2 ml of 5% hypertonic saline into latent TrPs in 60 brachioradialis muscles[31] produced referred pain to the dorsum of the wrist in 35% of infusions and local pain in every test. The area of local pain was approximately twice as large as the area of referred pain. This experimental result confirms the clinically observed referred pain pattern of this muscle and supports the observation that latent TrPs often will produce their characteristic referred pain pattern when compressed.[36] It would be interesting to see if similar injection of *active* TrPs would result in a higher percent of referred pain patterns.

Injection of 1 cc of 20% saline into 15 brachioradialis muscles resulted in no change in *sensory* threshold to electrical stimulation in the cutaneous, subcutaneous, or muscular tissues in the painful region at the site of injection.[74] The authors observed a significantly lowered *pain* threshold in a limited ellipsoidal area in the skin and subcutaneous tissues at the site of injection, and throughout the entire area of painful muscle in the vicinity of the injection. The local tenderness produced by the painful lesion in the brachioradialis muscle produced more ex-

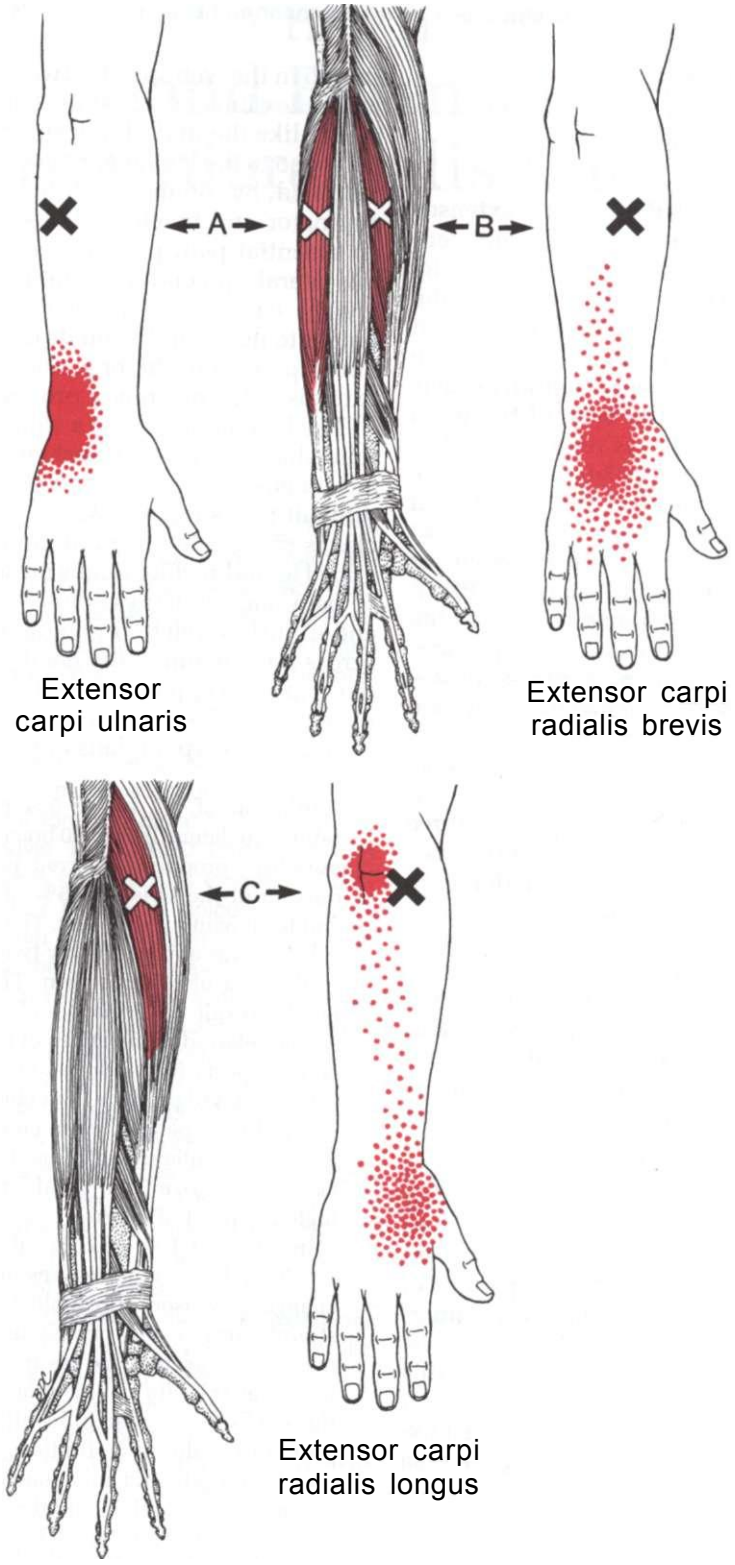


Figure 34.1. Referred pain patterns *(dark red)* and location of central trigger points (**Xs**) in the three primary hand extensor muscles *(medium red)* in the right forearm. **A,** extensor carpi ulnaris. **B,** extensor carpi radialis brevis. **C**, extensor carpi radialis longus.

tensive tenderness in muscle than in more superficial tissues.

2. ANATOMY
(Figs. 34.3 and 34.4)

Radial Hand Extensors
(Fig. 34.3)

The **extensor carpi radialis longus** attaches *proximally* to the distal third of the lateral supracondylar ridge of the humerus, between the lateral epicondyle and the attachment of the brachioradialis muscle (Fig. **34.3A).** The extensor longus attaches ***distally*** to the base of the second metacarpal bone on its dorso-radial aspect. The muscle fibers extend one-third of the length of the forearm, and its tendon the remaining two-thirds.

The *proximal* attachments of the **extensor carpi radialis brevis** (Fig. **34.3B)** lie deep to the belly of its companion, the extensor carpi radialis longus. The attachments of the brevis include the common extensor attachment to the lateral epicondyle, the radial ligament of the elbow, and intermuscular septa between it and adjacent muscles.[14] The belly of the extensor carpi radialis brevis expands to full thickness near the junction of the upper and middle thirds of the forearm, as the more lateral longus muscle dwindles to a tendon.[15,53,57] **Distally** the extensor carpi radialis brevis attaches to the base of the third metacarpal bone on its dorso-radial aspect (Fig. **34.3B).**[14]

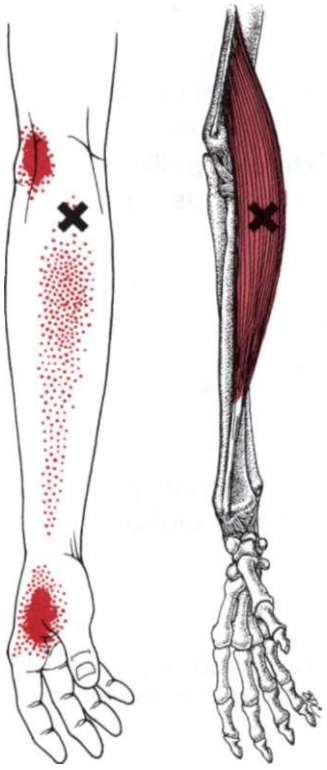


Figure 34.2. Referred pain pattern (*dark red)* and location of central trigger point (**X**) in the right brachioradialis muscle *(medium red).*

Not always clearly described is the fact that proximally the strong aponeurosis of the extensor brevis forms a bridge of fascia, which stretches between the lateral epicondyle and the deep fascia of the dorsal forearm. It may become thickened[29, 45] where the deep (motor) branch of the radial nerve passes beneath it to enter the supinator muscle (Fig. **34.3C).** Usually, the superficial radial nerve has branched off before the deep radial nerve dips beneath the extensor carpi radialis brevis (Fig. **34.3B).** In some cases, however, the nerve divides more distally (Fig. **34.3C),** so that the superficial branch must penetrate the belly of the extensor carpi radialis brevis muscle to return to its course beneath the brachioradialis muscle.[45]

Extensor Carpi Ulnaris
(Fig. 34.3A)

The extensor carpi ulnaris muscle attaches ***proximally*** to the common extensor tendon of the lateral epicondyle and ***distally*** to the ulnar side of the base of the fifth metacarpal bone (Fig. **34.3A**).

Brachioradialis
(Fig. 34.4)

The brachioradialis attaches ***proximally*** to both the lateral supracondylar ridge of the humerus and to the lateral intermuscular septum, distal to where the radial nerve penetrates the septum at mid-arm level (Fig. **34.4).** ***Distally*** the brachioradialis tendon expands laterally as it approaches the styloid process of the radius and connects with the neighboring ligaments.[7] It is then anchored by a tendinous attachment to the styloid process.[7,14,18,50] A variable slip may attach distally to several carpal bones, and to the third metacarpal.[7,14]

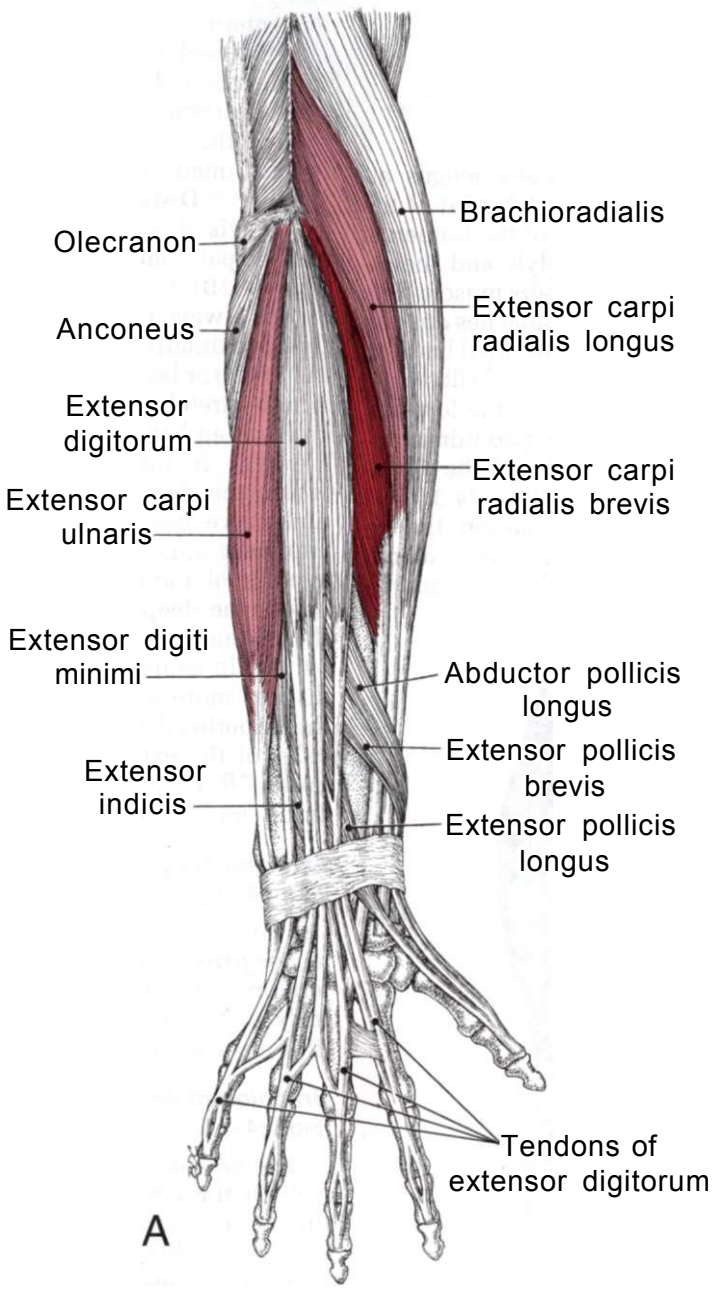


Figure 34.3. The relations of the hand extensor muscles and part of the radial nerve in the right forearm. **A,** dorsal view showing the attachments of the extensor carpi radialis longus and brevis, and extensor carpi ulnaris muscles.

Supplemental References

The radial hand extensors are well illustrated by other authors from the dorsal view,[4,54,57,68] the lateral view,[1,15,53,67] and in cross section.[25,58] The distal attachments at the wrist are shown in detail.[5,16,54,59]

The extensor carpi ulnaris is illustrated in the dorsal view,[4,54,57,68] the lateral view,[53] and in cross section.[24,58] Its distal attachment is also shown in detail.[16,59]

The brachioradialis muscle is depicted in the dorsal view,[4,54,57] the lateral view,[1,15,53,67] the volar view,[3,19,66] and in cross section.[23,58] Details of its distal attachment are shown.[17,54] Other figures show the course of the superficial branch of the radial nerve lying beneath this muscle.[2,20,55]

Architecture of Muscles

Studies of architectural features have included muscle length, sarcomere length and fiber length.[48,49] In these studies, muscle length was measured from the most

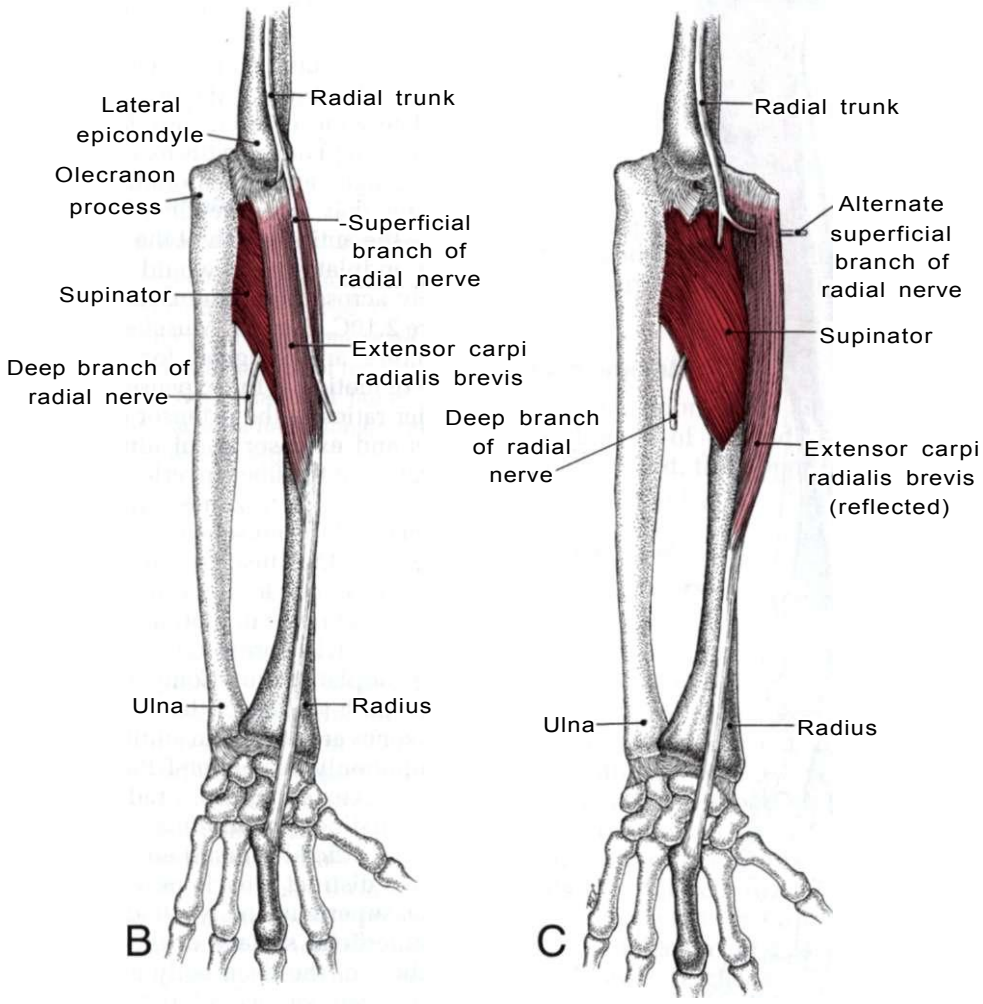


Figure 34.3.—*continued.* **B,** lateral view showing the deep branch of the radial nerve before it passes beneath the fibrous arch formed by the proximal attachments of the extensor carpi radialis brevis *(light red),* and showing the normal course of the superficial (sensory) branch. **C,** variant course of the superficial branch of the radial nerve *through* the (reflected) extensor carpi radialis brevis muscle (Adapted from Kopell HP, Thompson WA: *Peripheral Entrapment Neuropathies.* Ed. 2. Williams & Wilkins, Baltimore, 1963.)

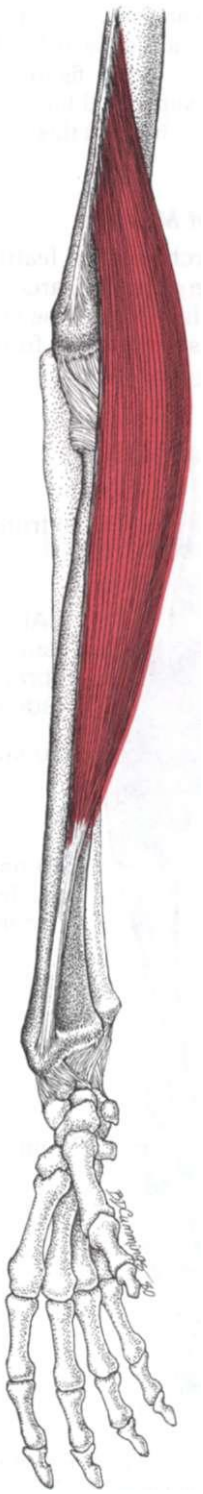


Figure 34.4. The attachments of the right brachioradialis muscle, from the radial view.

proximal muscle fiber attachments to the most distal muscle fiber attachments. Sarcomere length was determined by measuring the diffraction angle of the laser diffraction pattern. Fiber length was determined by measuring a small bundle of fibers isolated from the proximal, middle, and distal regions of the muscle. Fiber length and muscle length were normalized to a standard sarcomere length of **2.2** um. The fiber length of the brachioradialis was outstandingly long **(121** mm) compared to lengths close to **50** mm for the other forearm muscles.

The ratios of fiber length to muscle (belly) length[48,49] are presented in Table **34.1** and are useful for clinical practice because they give an indication of the orientation of the endplate zone in each muscle. The ratios approaching **1,** seen in the extensor carpi radialis longus and brachioradialis muscles, indicate that any one fiber must extend nearly the entire length of the muscle belly. Their endplate zones would appear transversely across the midmuscle region as in Figure **2.10C, E,** and **F.** Muscles with this architecture are designed for velocity and range of motion at the expense of force. The smaller ratios of the extensor carpi radialis brevis and extensor carpi ulnaris muscles indicate that the fibers overlap strongly and that their endplate zones will tend to run the length of the muscle down its middle as in Figure **2.8A.** Muscles with this architecture are designed for force at the expense of velocity and range of motion. The expected location of TrPs corresponds to the location of the endplate zones. Competent histological research studies of the location of endplate zones are needed in adult muscles that are commonly treated for TrPs.

The extensor carpi radialis longus shows distinct *partitioning.*[63] This muscle has a superficial (dorsal) and a deep belly and two distinct muscle nerves. The fibers of the superficial belly attach distally to the superficial surface of the tendon, and the fibers of the deep belly attach distally to the deep surface of the tendon. The proximal nerve supplies primarily the deep belly and the distal nerve primarily the superficial belly. The muscle fibers of the superficial belly are considerably longer than those of the deep belly. The

Table 34.1 ***Fiber Architecture for Five Forearm Muscles***

	Fiber Length/ Muscle Length Ratio'	*Expected Endplate zone Orientation*
Extensor Carpi Radialis Longus	0.82	Nearly transverse
Brachioradialis	0.69	Roughly transverse
Extensor Digitorum Communis	0.42-0.50	Diagonal
Extensor Carpi Radialis Brevis	0.38	Longitudinal
Extensor Carpi Ulnaris	0.28	Longitudinal

"**Fiber length/muscle length ratios approaching 1 indicate that every fiber runs practically the full length of the muscle belly (parallel arrangement) and that the muscle is designed for speed and large range of motion.**
Data taken from Lieber RL, Fazeli BM. Botte MJ: Architecture of selected wrist flexor and extensor muscles. *J Hand Surg 15A:* 244-250, 1990; Lieber RL, Jacobson MD, Fazeli BM, *et al:* Architecture of selected muscles of the arm and forearm: anatomy and implications for tendon transfer. *J Hand Surg 17A (5):* 787-798, 1992.

functional effect of this partitioning needs to be determined.

Anatomical Variations

Extensor Carpi Radialis Longus Variations

One of four types of variations of this muscle was identified in 30% of 375 upper limbs.[46] The chief variations were in the number and arrangement of tendinous attachments to the metacarpal bones. A few extensor carpi radialis longus and brevis muscles were fused.

3. INNERVATION

Hand Extensors

The radial nerve supplies the extensor carpi radialis longus and the brachioradialis muscles as it passes beneath them, proximal to the elbow joint. The nerve also usually divides into superficial and deep branches proximal to this joint. The deep branch of the radial nerve then supplies the extensor carpi radialis brevis and the supinator muscles before turning dorsally and entering the supinator muscle through the opening that sometimes forms an arcade of Frohse. This entrance is an arch of connective tissue in the space between the superficial and deep layers of the supinator (*see* Fig. **36.2B).**[14] The deep branch also gives off the recurrent (epicondylar) nerve, which exits by again passing beneath the archway formed between the two proximal attachments of the extensor carpi radialis brevis muscle (Fig. **34.3B).**[14]

These muscles are supplied by the radial nerve, which receives fibers through all three posterior divisions and the posterior cord.[14] Fibers from spinal nerves C_6 and C_7 supply the extensor carpi radialis longus and brevis, and fibers from C_6, C_7, and C_8 supply the extensor carpi ulnaris muscle.

Brachioradialis

The brachioradialis muscle is supplied by a branch of the radial nerve from the posterior cord, the upper trunk and spinal nerves C_5 and C_6.

4. FUNCTION

Hand Extensors

For effective grasp, these wrist extensors function synergistically to prevent the wrist flexion that the finger flexors would otherwise produce.

There is general agreement[6,9,14,22,39,61] that both the extensores carpi radialis longus and brevis participate in extension and abduction (radial deviation) of the hand, while the extensor carpi ulnaris extends and adducts the hand (ulnar deviation) at the wrist. In addition, the extensor carpi radialis longus assists in flexion at the elbow.[44] Duchenne[22] emphasizes that the extensor carpi radialis longus, which attaches to the second metacarpal bone, mainly abducts the hand. The extensor carpi radialis brevis, which attaches to the third metacarpal, chiefly extends the hand, and the extensor carpi ulnaris, which at-

taches to the ulnar side of the fifth metacarpal, mainly adducts the hand. The extensor carpi radialis longus and the extensor carpi ulnaris muscles, acting together, can extend the hand at the wrist, but do so only when needed to exert strong force.[22,39]

Activation of the hand extensors is essential to a power grip.[61]

> Electromyographic (EMG) monitoring of subjects while they repeatedly pressed a typewriter key at a maximal rate, or wrote with a pencil, showed moderate activity of the finger and hand extensors. At slow rates of typing, the amplitude of this electrical activity dropped to less than one-tenth of that at very rapid rates.[51]
>
> Bilateral EMG monitoring of the radial wrist and finger extensors as a group, and of the brachioradialis muscle separately, was performed with surface electrodes during 13 sports activities. They included overhand throws, underhand throws, tennis, golf, hitting a baseball and 1-foot jumps from the floor. The *extensor group* consistently showed slight to moderate activity, bilaterally similar. The *brachioradialis* frequently showed an activity pattern similar to, but slightly stronger than, that of the hand and finger extensors, especially on the nondominant side. The two exceptions to this relationship were batting a baseball and driving a golf ball. Then, the extensors on the nondominant side showed more electrical activity than did the brachioradialis.[13]

Brachioradialis

Reports on the function of this muscle began with misunderstanding and confusion, some of which still persists. Initially, this muscle was named the "supinator longus," on the assumption that its primary action was supination of the forearm. Duchenne demonstrated clearly by stimulation studies that it functioned chiefly as a flexor at the elbow,[22] which led to its present name, brachioradialis. He also demonstrated that its stimulation brought the forearm to a neutral position from either supination or pronation.

Authors agree that it flexes the forearm at the elbow.[7,14,18,44,50,65] Electromyographically,[8] brachioradialis activity usually is reserved for speedy movement and the lifting of weight by flexing the elbow, especially if the forearm is in the neutral position. However, none of the elbow flexors is used to counteract gravity when a weight is held in the dependent hand with the elbow straight.[8]

The brachioradialis is also the classic example of a "shunt muscle:" it is attached in such a manner that its contraction prevents separation of the elbow joint by centrifugal force during rapid elbow movement. In contrast, the biceps brachii and brachialis, "spurt muscles," accelerate movement at the elbow without counteracting distraction of the elbow joint.

In agreement with Duchenne,[22] textbooks generally state that the brachioradialis returns the forearm to mid-position from pronation or supination.[7,44,65] However, Clemente[14] makes no mention of this function, and Lockhart[50] states that the brachioradialis is never a pronator or supinator of the forearm (which is true only when starting in the neutral position). In a 1957 study that employed bipolar needle electrodes inserted into the middle of the muscle, Basmajian and Latif[10] reported that the brachioradialis could assist either pronation or supination, but only when these motions were resisted.

In an EMG study of two subjects,[64] a monopolar needle electrode recorded electrical activity of the brachioradialis only during resisted pronation, and not during resisted supination. This agrees with Duchenne's early observation[22] that the muscle acted more as a pronator than as a supinator, and with Hollinshead's conclusion[39] that it probably provides limited assistance in pronation, but little, if any assistance in supination. The muscle's use for supination may vary among individuals because of variations in its distal attachment.

> During typewriting (by subjects who demonstrated no resting EMG activity) there was no difference in brachioradialis electrical activity whether the elbow was bent at an acute angle, at a right angle, or at an obtuse angle.[51] Elevation of the typewriter does not create a problem for this muscle, but it does for the shoulder muscles.

During simulated car driving,[40] the brachioradialis and brachialis muscles worked nearly synchronously when most subjects turned the steering wheel to the side contralateral to the muscles. A few subjects apparently did not use these muscles when driving.

The wrist-deviating local twitch response (LTR) observed during examination of the brachioradialis for TrPs, and the kinds of activities that cause TrPs in this muscle, indicate that in some individuals the deepest layer may function to radially deviate the wrist. This movement likely depends upon its variable attachment, occasionally, to the scaphoid, navicular, or third metacarpal bones.[6] This attachment also could make the corresponding muscle fibers more vulnerable than the rest of the muscle to overload. No reference to this action was found in the literature. It is difficult to unambiguously distinguish by palpation the deep brachioradialis fibers from those of the underlying extensor carpi radialis longus, the primary source of this wrist-deviating movement.

5. FUNCTIONAL UNIT

Hand Extensors

For extension of the hand at the wrist, the extensor carpi radialis longus is synergistic with the extensor carpi radialis brevis, the extensor carpi ulnaris, and the finger extensors.

For radial deviation of the hand, the extensores carpi radialis muscles are synergistic with the flexor carpi radialis. For ulnar deviation, the extensor and flexor carpi ulnaris muscles are similarly synergistic.

During flexion of the hand at the wrist, electromyographically, the extensor carpi ulnaris was observed to function as the primary antagonist.[10]

During the grasping of an object, the wrist extensors act synergistically to prevent the flexion of the wrist that the extrinsic finger flexors would otherwise produce.

Brachioradialis

Kinesiologically, synergists with the brachioradialis muscle are the biceps brachii and brachialis muscles. However, in terms of TrP phenomena, the brachioradialis is more closely associated with the extensores carpi radialis longus and brevis, the extensor digitorum, and supinator muscles. These muscles become a functional unit during simple grasp or during combined forearm rotation and grasp with the wrist cocked (held in extension).

6. SYMPTOMS

It is difficult to sharply delineate which symptoms are caused by the radial hand extensors and which are caused by the brachioradialis when more than one muscle is involved. Trigger points in these muscles can produce dysfunction in the form of limited movement and/or weakness, as well as pain.

Pain, as described in Section 1, is a major complaint. The pain is likely to appear first in the lateral epicondyle, and then spread to the wrist and hand. The epicondylar pain, often diagnosed as "tennis elbow" or lateral epicondylitis, is frequently a composite pain that may originate in the supinator, the extensor carpi radialis longus,[21] and/or the extensor digitorum muscles. With involvement of the latter two muscles, patients complain of pain when they attempt a firm grip with the hand in ulnar deviation, such as shaking hands to greet someone. Pain is more likely to be felt if forceful supination or pronation are added to the grasp, as when turning a doorknob, or using a screwdriver.[33]

Weakness of the grip during these movements may be pronounced, so that objects tend to slip out of the hand, particularly when the movement deviates the wrist ulnarward, further weakening the grip. Weakness of the grip results in, for example, letting the head of the tennis racquet drop, loss of control when pouring milk or juice from a carton, or loss of control when drinking coffee just as the cup reaches the lip and is tipped to drink. The muscles act as if the grasp is reflexly inhibited by TrP activity in the simultaneously contracting extensors. An autoinhibition of the extensors also may occur, like that seen when TrPs in the vastus medialis muscle cause buckling of the knee instead of pain. Ivanichev has shown that TrPs in the extensors can produce loss of coordination

and increased fatiguability during repetitive hand flexion and extension.[37] Weakness of the grip is aggravated when the patient with TrPs grasps a large object. However, TrPs in these extensor muscles cause no problem in using scissors, whereas TrPs in the finger flexors do cause a problem.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Myofascial TrPs are activated in the extensor carpi radialis longus and brevis and in the brachioradialis muscles by repetitive forceful handgrip. The larger the object being grasped, and the greater the ulnar deviation of the hand, the more likely the muscles are to develop TrP.

The following examples illustrate how patients have activated TrPs in these muscles: executing a one-hand tennis backhand with the head of the tennis racquet dropped (*see* Fig. 36.6), weeding with a trowel, extensive handshaking, scraping ice off the windshield with a scraper, meticulous ironing of clothes, frisbee-throwing over a prolonged period, and repeatedly lifting a *heavy* large paperweight to test for muscle soreness.[27] This latter activity provides an example of the tendency to test a muscle (already sore with TrPs) by repeatedly activating it. This tendency to test the muscle comes partly from the hope that it is better and won't hurt anymore, and partly from the mistaken idea that exercising it in a painful way will help it to recover. This attitude stems from the "No Pain, No Gain" philosophy, which clearly does NOT apply to TrPs. Voluntarily contracting muscles repeatedly to the point of pain tends to hinder recovery, not help it.

These activities cause referred elbow pain that is frequently called "tennis elbow." The muscles around the elbow that cause this lateral epicondylar pain are likely to develop TrPs in approximately the following sequence: (1) supinator, (2) brachioradialis, (3) extensor carpi radialis longus, (4) extensor digitorum, (5) triceps brachii, (6) the anconeus, and (7) the biceps and brachialis together.

Key TrPs in the scalene muscles can induce satellite TrPs in extensor carpi radialis or extensor carpi ulnaris muscles. In addition, key TrPs in the supraspinatus can cause satellite TrPs in the extensor carpi radialis, and key TrPs in the serratus posterior superior can induce satellite TrPs in the extensor carpi ulnaris.[34]

The supinator muscle usually becomes involved with the brachioradialis, and *vice versa.* One patient, while paddling a canoe, developed lateral epicondylar pain due to TrPs in the brachioradialis of the nondominant forearm. There was less severe involvement of the extensor carpi radialis muscles, but *no* involvement of the supinator. This was an unusual combination.

Lange[47] observed that "writer's cramp" due to Myogelosis was more likely to involve the brachioradialis and forearm extensor muscles than the antagonistic flexors.

The extensor carpi ulnaris, which is seldom required to support a load against gravity, rarely develops TrPs. Its involvement is usually secondary to gross trauma, such as fracture of the ulna, or as part of a "frozen shoulder" syndrome when most of the shoulder muscles and many of the elbow muscles develop TrPs. A "frozen shoulder" can follow dislocation of the shoulder joint, prolonged immobilization of the arm in a cast, or surgery on structures around the shoulder or the elbow joint (*see* Chapter 26, Section 11).

8. PATIENT EXAMINATION

Range of motion of the joints crossed by these muscles should be tested in all planes. During range of motion testing, the shortening due to TrP tension is revealed by tension or limitation of movement when the muscle is lengthened through a combination of the stretch position of all of the joints that it crosses. With the elbow placed in full extension and with the forearm pronated, wrist flexion and ulnar deviation of the hand may reveal restriction in stretch length due to TrP tension of the *extensor carpi radialis longus or brevis.* Flexion with radial deviation at the wrist can reveal restriction in stretch range of motion of the (less frequently involved) *extensor carpi ulnaris,* particularly when forearm supination is added to the test movement. The patient with TrPs often will point to a particular

area and volunteer the information that it hurts or feels tight there. That area is a good location to examine for TrPs (specific examination is described in the next section).

Trigger point involvement of the extensor group of muscles in the forearm, which includes the radial and ulnar hand extensors, the finger extensors and the brachioradialis, can be tested with the *Handgrip Test* as follows: the patient first positions his hand in extension with radial deviation at the wrist in the normal hand-shake position, and then squeezes the examiner's hand. When the hand extensors have TrPs, and the patient attempts to grasp with the wrist extended, placing the extensors in a shortened position, the effort is painful. Attempts to grasp with the wrist flexed also are painful, and are even more weakened than when the wrist is extended.

Identification of the involved muscle is confirmed by eliciting referred pain when the muscle is passively stretched and when it is actively loaded in the shortened position. Macdonald[52] reported that passively stretching an involved extensor carpi ulnaris muscle by flexing and abducting the hand at the wrist caused pain, as did loading the muscle by actively resisting the patient's effort to extend and adduct the hand at the wrist. In addition, testing for the strongest LTR helps to identify which muscle harbors the most active TrPs. The patient should be positioned so that the TrP can be stimulated by snapping palpation and that a twitch response can then be seen or felt.

The TrP origin of the pain is confirmed by the *Compression Test.* The test is performed by strongly and widely compressing the extensor mass of muscles below the elbow in a pincer grasp while conducting the Handgrip Test. This pressure often eliminates the pain response; release of pressure restores the pain during the handgrip. A similar effect may sometimes be obtained by firmly pinching the skin over the muscle mass.

Tapping the lateral epicondyle with the fingertip is likely to demonstrate referred tenderness over the *distal half* of the epicondyle if active TrPs are in the extensor carpi radialis longus, and/or the brachioradialis, and/or the supinator muscles. All of these muscles are attached directly or indirectly through fascia to the lateral epicondyle. Triceps TrPs are located in the arm proximal to the lateral epicondyle, and when they refer pain and tenderness to it, the tenderness appears mainly in the *proximal half of* the lateral epicondyle. Tenderness over the epicondyle can be due to the enthesopathy of attachment TrPs.

Following inactivation of the TrPs in each of these muscles by treatment, these tests no longer evoke referred pain, deep tenderness or LTRs.

Kendall, *et* al.[44] illustrate and describe strength testing of the extensor carpi radialis muscles by resisting the patient's attempt to hold the wrist extended in the radial direction while allowing the fingers to flex. Both muscles are tested with the elbow extended. The brevis alone can be tested with the elbow flexed, a position which makes the longus less effective because it is shortened. The extensor carpi ulnaris is tested by the examiner resisting the patient's attempt to hold the wrist extended in the ulnar direction. In this situation, elbow flexion is not an issue.

If any of the elbow or wrist articulations that are crossed by the muscle under consideration lack normal joint play, inactivation of the TrPs alone will usually not satisfactorily relieve the patient's symptoms. This common type of joint dysfunction can be readily identified and can be corrected as described by Mennell.[56]

9. TRIGGER POINT EXAMINATION
(Figs. 34.5 and 34.6)

Gerwin et al.[28] established that the most reliable criteria for making the diagnosis of myofascial TrPs are the detection of a taut band, the presence of spot tenderness, the presence of referred pain, and reproduction of the patient's symptomatic pain. Although agreement on the presence of an LTR was not good for some muscles, it was high for the middle finger extensor; the muscles covered in this chapter are similarly superficial and should be of similar difficulty. In addition, if the tender region of the TrP feels

like a nodule in the taut band, that is a diagnostic sign of a TrP.

Hand Extensors (Fig. 34.5)

The central TrPs in the extensor carpi *radialis longus* are found in the forearm at nearly the same distance from the elbow as are the TrPs in the brachioradialis muscle, but in the extensor longus they are closer to the ulna. The relaxed, supported forearm is examined by deep pincer palpation, with the hand hanging down over the edge of the support surface and the elbow flexed about 30° (Fig. 34.5A). An LTR from the extensor carpi radialis longus muscle produces strong radial abduction of the hand and some extension at the wrist. Active TrPs are found more often in the extensor longus than in the brevis.

Central TrPs in the extensor carpi *radialis brevis* are located in the muscle mass on the ulnar side of the brachioradialis muscle, distal to those in the longus (Fig. 34.5B). These extensor brevis TrPs lie 5 or 6 cm (a full 2 in) distal to the crease at the elbow. The muscle may be examined by flat palpation against the radius and snapped transversely to elicit its LTR, which produces hand extension with slight radial deviation at the wrist (Fig. 34.5B). Although LTRs are relatively easy to elicit in these muscles and are a valuable confirmation sign, they are not considered an essential finding to make the diagnosis of a TrP.

The extensor carpi *ulnaris* can be located when it stands out clearly from the other forearm muscles as the patient vigorously spreads the fingers. The TrP tenderness is found by flat palpation 7 or 8 cm (about 3 in) distal to the lateral epicondyle and 2 or 3 cm (about 1 in) from the sharp edge of the ulna toward the dorsal surface of the forearm (Fig. 34.1A). An LTR elicited with the hand hanging down, relaxed, causes ulnar deviation of the hand (Fig. 34.5C).

Brachioradialis Muscle (Fig. 34.6)

For palpation of TrPs in this muscle, the patient sits comfortably with the forearm resting on a padded armrest, and with the elbow slightly bent. The brachioradialis muscle is held in a pincer grasp between the thumb and fingers (Fig. 34.6). For injection purposes, it is useful to distinguish TrPs that lie in the deepest brachioradialis fibers (which usually have no effect on wrist motion) from those in the underlying extensor carpi radialis longus fibers, which always radially deviate and extend the wrist; the superficial (sensory) branch of the radial nerve passes between these two muscles. When the patient attempts to flex the forearm against resistance, particularly when the elbow is held at 90° of flexion, the brachioradialis stands out. Using pincer palpation, an examiner frequently can encircle the muscle with the digits and separate it from the underlying extensores carpi radialis longus and brevis. TrPs are usually found only in the deep part of the brachioradialis muscle. Compression of these active TrPs often evokes their characteristic referred pain pattern, primarily to the dorsal web between the thumb and index finger (Fig. 34.2).

10. ENTRAPMENT

The extensor carpi radialis brevis muscle may entrap parts of the radial nerve in either of two ways (Fig. 34.3B and C):

1 If the bridge of fascia between the proximal attachments of the muscle has developed a thickened margin, this hard edge may impinge on the deep radial nerve (forcibly when the forearm is fully pronated),[69] as the nerve passes beneath it to penetrate the supinator muscle.[27,29,38,45]
2 If, as sometimes happens, the sensory fibers branch from the motor fibers *distal* to this bridge of fascia, the sensory branch must penetrate the substance of the extensor carpi radialis brevis to resume its normal course.

The first type of entrapment is less likely to be due to TrP tautness of the extensor carpi radialis brevis than is the second. The first type also is more likely to cause symptoms during forceful pronation by exerting direct pressure on the deep radial nerve. Normally, this first entrapment produces only motor weakness of the muscles innervated by that nerve. These mus-

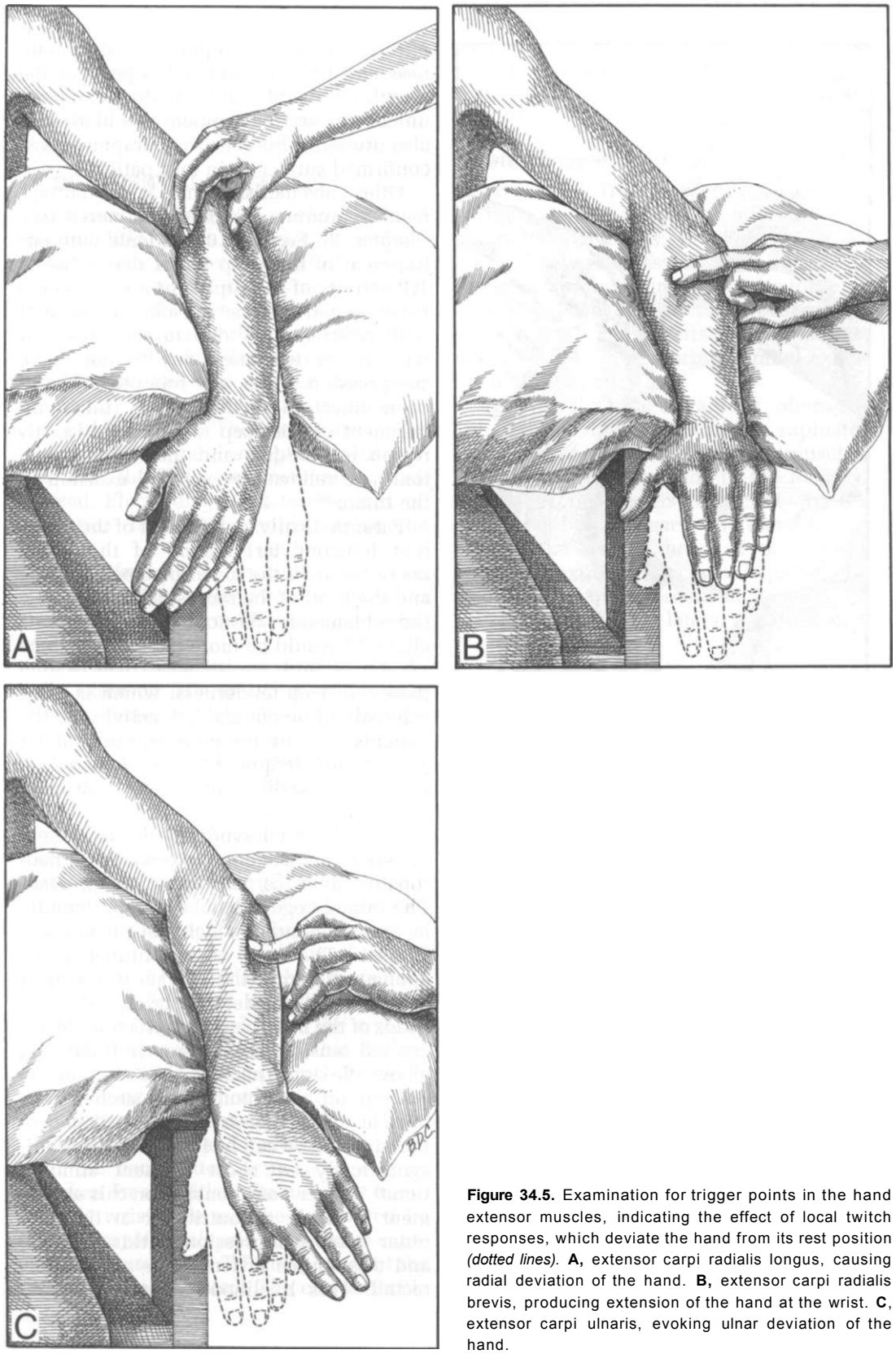


Figure 34.5. Examination for trigger points in the hand extensor muscles, indicating the effect of local twitch responses, which deviate the hand from its rest position *(dotted lines).* **A,** extensor carpi radialis longus, causing radial deviation of the hand. **B,** extensor carpi radialis brevis, producing extension of the hand at the wrist. **C**, extensor carpi ulnaris, evoking ulnar deviation of the hand.

Figure 34.6. Examination of the trigger point area in the brachioradialis muscle. The muscle is held in a deep pincer grasp over the radius, approximately 2 cm (nearly 1 in) distal to the antecubital crease.

cles include the following extensors: the indicis, pollicis longus, pollicis brevis, carpi ulnaris, digitorum, and digiti minimi; it also includes the abductor pollicis longus.

The second mechanism entraps only the superficial (sensory) branch of the radial nerve when it penetrates the belly of the extensor carpi radialis brevis muscle (Fig. **34.3C).**[45] In the presence of this anatomical variation, compression of the nerve by taut bands associated with active TrPs in the extensor carpi radialis brevis can cause purely sensory neuropraxia with numbness and tingling over the dorsum of the thumb and hand, but no motor symptoms unless the first entrapment mechanism is also present. This sensory entrapment was confirmed surgically in four patients.[45]

Other mechanisms may cause entrapment symptoms of the radial nerve (*see* Chapter **36,** Section 10). Patients with entrapment of the deep radial nerve due to TrP activity of the supinator muscle as the nerve penetrates the muscle can present with referred pain due to the TrPs and with motor weakness due to the nerve compression. Both are relieved by procaine injection of the TrPs.[43] Tumor entrapment of the deep radial nerve in this region is pain-free and the motor symptoms are relieved by surgical excision of the tumor.[29]

Parenthetically, entrapment of the recurrent (epicondylar) branch of the radial nerve between the extensor brevis muscle and the head of the radius, which is sometimes blamed for the aching pain of "tennis elbow,"[45] would be more likely to produce numbness and paresthesias than aching pain and deep tenderness, which is characteristic of myofascial TrP activity. In the patients seen by the authors, epicondylar pain is most frequently referred from TrPs in the surrounding muscles, and is rarely of neuritic origin.

Cubital tunnel syndrome is considered the second most common entrapment neuropathy after carpal tunnel syndrome.[26] The tunnel begins distal to the postcondylar groove around which the ulnar nerve courses. The roof of the tunnel is an aponeurotic arch (the arcuate ligament or humeroulnar arcade) that bridges the two heads of the flexor carpi ulnaris muscle. Increased tension of that muscle (including elbow flexion) narrows the tunnel by pulling on the aponeurotic arch of the muscle.[71] Compromise of that segment of the ulnar nerve can be identified electrodiagnostically by short segment stimulation.[41] Trigger point tension on this attachment is likely a contributor at times to ulnar nerve compression at this location, and this source of tension is readily corrected. This likelihood deserves a com-

bined electrodiagnostic TrP identification and TrP treatment research study to determine how commonly TrP tension contributes to cubital tunnel syndrome.

11. DIFFERENTIAL DIAGNOSIS

Differential diagnoses include **lateral epicondylitis** (tennis elbow), C_5-C_6 **radiculopathy** with TrP involvement of the brachioradialis muscle, and C_7 or C_8 radiculopathy with TrP involvement of the wrist extensors. Frequently there is clinical misdiagnosis and confusion between **carpal tunnel syndrome** and referred pain from myofascial TrPs in the hand extensor muscles and brachialis. The patient may have both conditions, and symptoms will persist until the TrP component also is addressed. Electrodiagnostic testing *and* examination for TrPs should resolve the issue.

The differential diagnosis of lateral epicondylitis (tennis elbow) with regard to TrPs is covered thoroughly in Chapter 36, Section 11.

Articular dysfunctions associated with wrist extensor TrPs are volar subluxation of carpal bones and, occasionally, distal radioulnar joint dysfunctions.

The pain and tenderness that is referred from myofascial TrPs to the dorsum of the hand and wrist, especially in the region of the base of the thumb, may easily be mistaken for **tenosynovitis (de Quervain's disease),** which presents similar symptoms.[70] In both conditions, the pain is aggravated by either loading or stretching the involved tendons and muscles. Palpation of the extensores carpi radialis and brachioradialis muscles for TrPs that reproduce the patient's pain largely establishes the myofascial TrP diagnosis. However, this finding does not exclude the additional diagnosis of **coexisting** tenosynovitis until myofascial treatment has been successful.

The wrist pain and tenderness arising from the hand extensor muscles can be mistaken for **arthritis.**[62] On the other hand, arthritic wrist pain may be aggravated by referred myofascial pain from these muscles; the cause of the TrP component of the pain can be eliminated. The osteoarthritic wear-and-tear changes may be only coincidental, and not the cause of the patient's pain.

Related Trigger Points

Myofascial TrPs frequently occur both in the extensores carpi radialis and brachioradialis muscles; involvement of either is likely to be associated with TrPs in the extensor digitorum and supinator muscles. Myofascial TrPs are rarely observed in the extensor carpi ulnaris without at least one TrP in the neighboring parallel extensor digitorum muscle.

TrPs in the brachioradialis often develop secondary to TrPs in the supinator and extensor carpi radialis longus muscles. Involvement then spreads to the long extensors of the fingers, especially to the middle and ring fingers. The distal lateral end of the medial head of the triceps brachii, proximal to the lateral epicondyle, also may develop associated TrPs. These TrPs refer pain to the lateral epicondyle.

12. TRIGGER POINT RELEASE
(Figs. 34.7 and 34.8)

In addition to the spray and stretch technique described in detail here, the other manual release techniques described in Chapter 3, Section 12 are also effective. Trigger point pressure release is simple and effective for these muscles. It can be performed with the muscles in a position of ease (not stretched), and can be combined with other techniques such as postisometric relaxation and the use of reciprocal inhibition. These other manual techniques often can be combined with spray and stretch by starting with a prespray step, or by stroking with ice in the manner described for spray.

Spray and Stretch
(Fig. 34.7)

Hand Extensors. Both the extensores carpi radialis longus and brevis muscles are stretched with the patient either seated or supine, with the forearm extended at the elbow, and with the hand pronated and

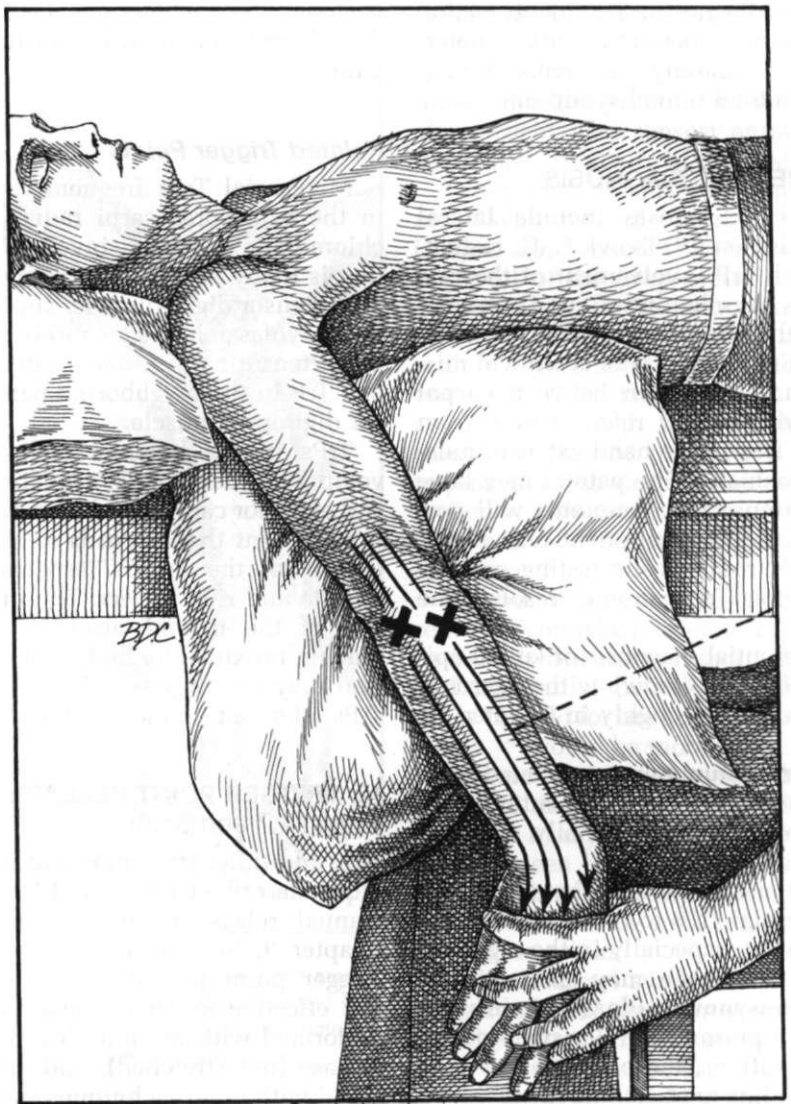


Figure 34.7. Stretch position and spray pattern (arrows) for trigger points (**Xs**) in the hand extensor muscles. The more radial **"X"** identifies the region of an extensor carpi radialis brevis central trigger point. The ulnar **"X"** locates an extensor carpi ulnaris trigger point near its proximal musculotendinous junction.

slack eliminated by flexing the hand at the wrist (Fig. 34.7). During this muscle release, the vapocoolant spray is applied in parallel sweeps over the muscle from the humerus to the hand, covering the epicondyle and distal referred pain areas in the wrist. Muscles with associated trigger points (TrPs), the brachioradialis, finger extensors and supinator, also must be released and can be included along with this spray-and-stretch procedure.

The extensor carpi ulnaris is released by flexing the wrist and moving the hand toward radial deviation without particular concern for elbow extension. Sweeps of the spray are applied in a distal direction, covering the muscle from the lateral epicondyle to the ulnar styloid process, in-

eluding the reference zone at the wrist. As the spray is applied, the clinician takes up slack in the muscle as it develops.

These muscles can also be lengthened by postisometric relaxation combined with reciprocal inhibition. The examiner resists the patient's *gentle* contraction of the wrist extensors (or it can be resisted by gravity) as the patient takes in a deep breath; then as the patient slowly breathes out and relaxes completely, the hand is allowed to drop into flexion. The patient then actively flexes the wrist further and deviates it in the direction that will take up additional slack in the particular muscle under stretch.

Moist heat is applied to the muscles being treated and then the patient moves the hand *slowly* to produce 3 cycles of *full* range of motion.

Brachioradialis (Fig. 34.8). The patient is seated comfortably in a relaxed position with the forearm extended at the elbow and the elbow resting on a padded support. The operator holds the patient's fingers (not hand) so the referred pain zone is exposed to vapocoolant. Full extension of the forearm at the elbow is the primary movement to release the brachioradialis. However, pronation of the forearm compared to the neutral position provides additional stretch by moving the proximal and distal attachments farther apart. The spray is applied as in Figure 34.8. After covering the TrP area, the proximal-to-distal spray pattern detours to cover the lateral epicondyle, then sweeps over the forearm to cover the dorsum of the hand and dorsal web between the thumb and the index finger. Trigger point pressure can be applied during the stretch.

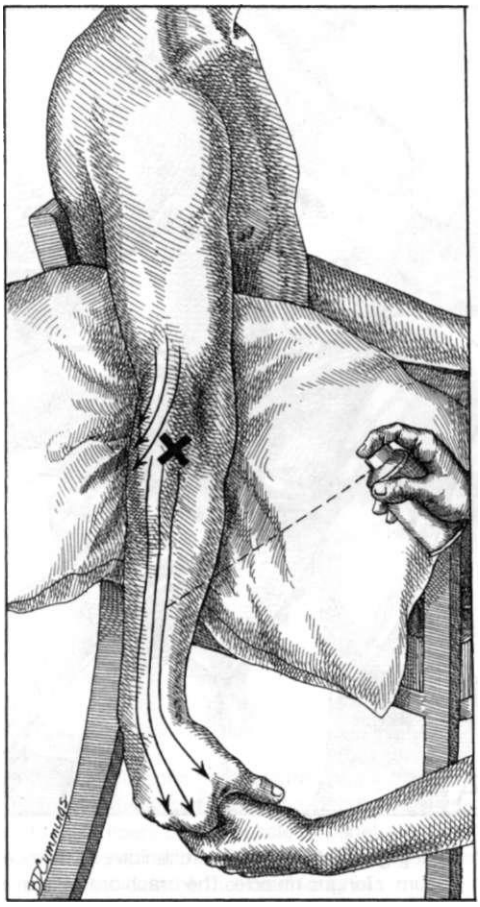


Figure 34.8. Stretch position and spray pattern (arrows) for a central trigger point (**X**) in the brachioradialis muscle. The forearm is placed in pronation, and the elbow is extended against a padded surface to avoid medial rotation at the shoulder. Sweeps of the vapocoolant cover the muscle in the forearm and its referred pain patterns: first, the lateral epicondyle, and second, the dorsum of the hand and the web space between the thumb and index finger. Refer to text for additional details.

13. TRIGGER POINT INJECTION
(Figs. 34.9 and 34.10)

Hand Extensors
(Fig. 34.9)

For injection of the hand extensors, the patient lies supine with the arm resting on a pillow, or other support. Since all three hand extensor muscles are relatively superficial, palpation can precisely localize their trigger points (TrPs) for injection. The operator fixes the extensor carpi radialis **longus** TrP between the index and middle fingers and injects it as shown in Figure 34.9A. The endplate zone is expected to cross the muscle approximately midbelly (Table 34.1). The extensor carpi radialis **brevis** TrP may be 3 or 4 cm (about 1 $^{1}/_{2}$-in) more distal than the longus TrP, and its endplate zone can be expected to run nearly longitudinally most of the length of the muscle belly (Table 34.1).

For injection of the extensor carpi **ulnaris,** the patient's arm is placed with the

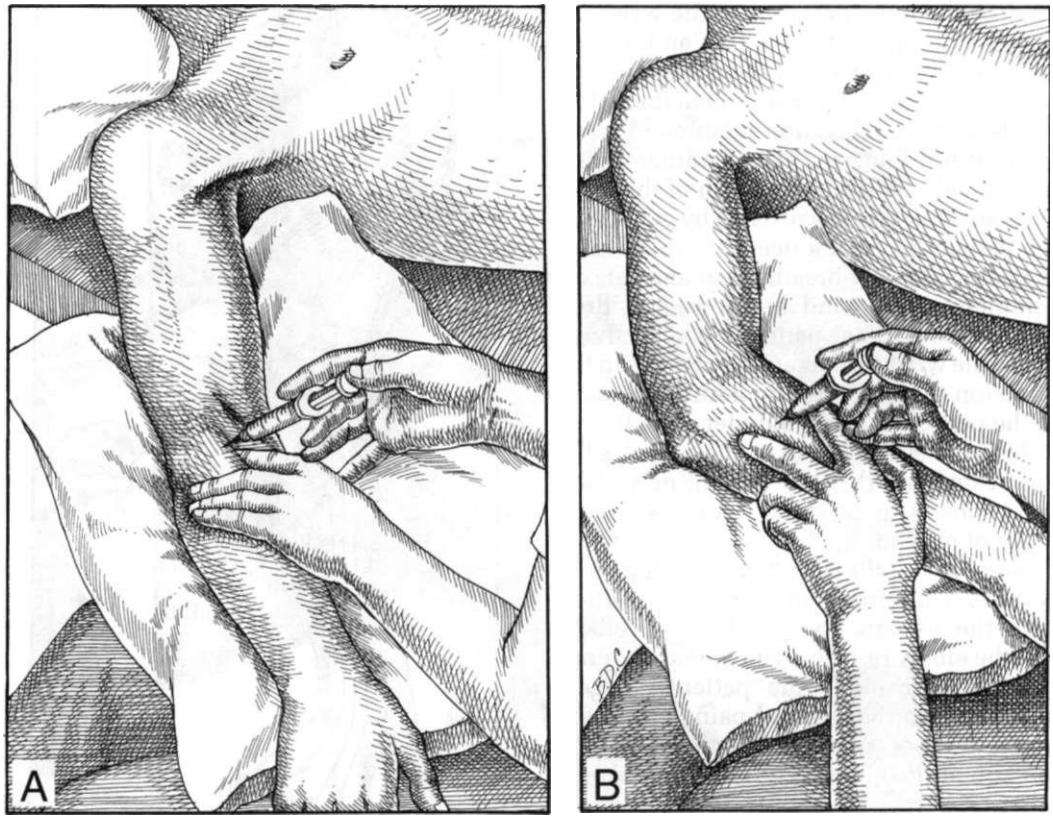


Figure 34.9. Injection technique for two central trigger points. **A,** in the extensor carpi radialis longus muscle. The brachioradialis muscle is displaced to the radial side by the index finger. **B,** in the extensor carpi ulnaris muscle.

lateral epicondyle uppermost (Fig. 34.9B). The TrP is located and one finger placed between the hard edge of the ulna and the nodule in the muscle, and the other finger is placed on the other side of the nodule stabilizing its position for injection.

In all three muscles these TrPs, when impaled by the needle, generally respond with obvious LTRs and characteristic referred pain patterns. After injection, stretch and spray are applied as described above, followed by a hot pack, and then full active range of movement is performed slowly three times.

Cyriax[21] described a similar technique for injecting an extensor carpi radialis muscle with procaine. Rachlin[60] illustrates a location for the injection of a central TrP in the extensor carpi radialis brevis. He also illustrated a location for injecting an attachment TrP in the extensor carpi radialis longus and one in the extensor carpi ulnaris muscle.

Clinical experience[35] has shown that injecting a local anesthetic (lidocaine without steroid) into the attachment TrP region at the proximal tendinous attachment of the extensor carpi radialis longus relieved not only the lateral epicondylitis (tennis elbow) symptoms but also inactivated the central TrP in that muscle that was responsible for the enthesopathy. Apparently there was an important neural feedback mechanism from the attachment TrP that perpetuated the central TrP. A well-designed prospective research study is needed to validate this observation and explore the nature of the feedback loop that appears to be involved. Symptoms of epicondylitis are a common

complaint, the etiology of which is poorly established at present.

Brachioradialis

(Fig. 34.10)

The forearm of the supine patient is supported slightly flexed at the elbow with the forearm pronated. The muscle may be injected by holding the TrP in a pincer grasp between the finger and thumb, as in Figure 34.6, or by using flat palpation, as in Figure 34.10. The endplate zone (where TrPs occur) would be expected to run nearly transversely across the midbelly portion of the muscle (Table 34.1).

When referred pain is evoked in the base of the thumb by a deep injection in the proximal forearm, the TrP may lie either in the brachioradialis or in the underlying supinator. The fact that the sensory branch of the radial nerve may be temporarily blocked by the local anesthetic during this procedure should be explained in advance to the patient.

Rachlin.[60] illustrates injection of a central TrP in the brachioradialis muscle.

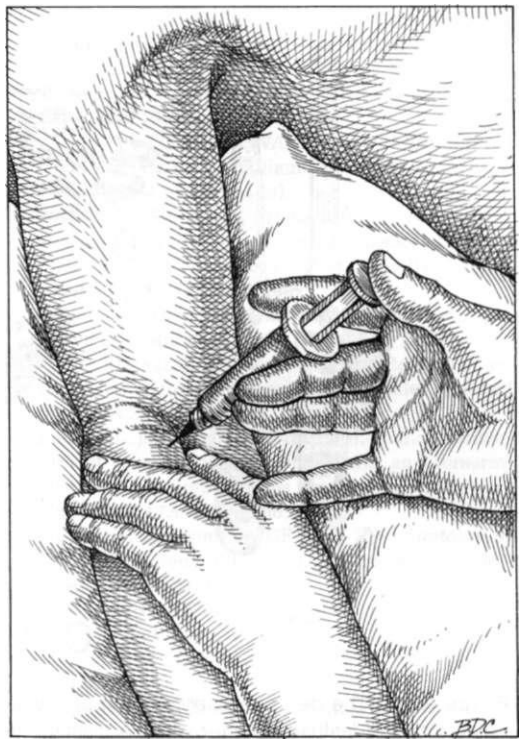


Figure 34.10. Injection of a central trigger point in the right brachioradialis muscle. The needle must reach the deepest fibers of the muscle to penetrate the trigger point. Note that the operator's index finger is now on the radial side of the brachioradialis muscle as compared to the ulnar side in Figure 34.9**A**.

14. CORRECTIVE ACTIONS

(Fig. 34.11)

Hand Extensors

The patient with active TrPs in the radial hand extensors should avoid forceful activity with the hand flexed or in ulnar deviation at the wrist. It may be helpful to adapt certain activities as follows: liquid should be poured from a container by rotating the arm at the shoulder joint, instead of by deviating the hand at the wrist. When playing tennis, the head of the tennis racquet should be angled up. When greeting others for a prolonged period in a receiving line, the hand should be offered with the palm facing upward, and the right and left hand alternated in shaking hands. If work requires stressful twisting motions, a wrist support that prevents hand flexion (Fig. 34.11) can protect these extensor muscles from overload during the course of treatment and recovery.

The patient can easily perform self-stretch of these muscles in the sitting position, with the involved forearm supported on the chair arm and with the hand dropped over the edge of the support. Instructions can be given to the patient for performing postisometric relaxation and for applying trigger point pressure release.

Brachioradialis

The patient should learn to avoid activities which aggravate brachioradialis TrPs, such as digging with a trowel, prolonged shaking of hands, and playing tennis with a racquet that is too heavy. If the activity must be pursued, then the patient should be encouraged to maintain the wrist cocked in extension with radial deviation. This is especially important when playing tennis (*see* Fig. 36.6).

A strap support for the wrist, as shown in Figure 34.11, reminds the patient to rotate the hand from the shoulder and trunk, not at the wrist.

The patient may be taught to self-stretch the brachioradialis muscle by placing the

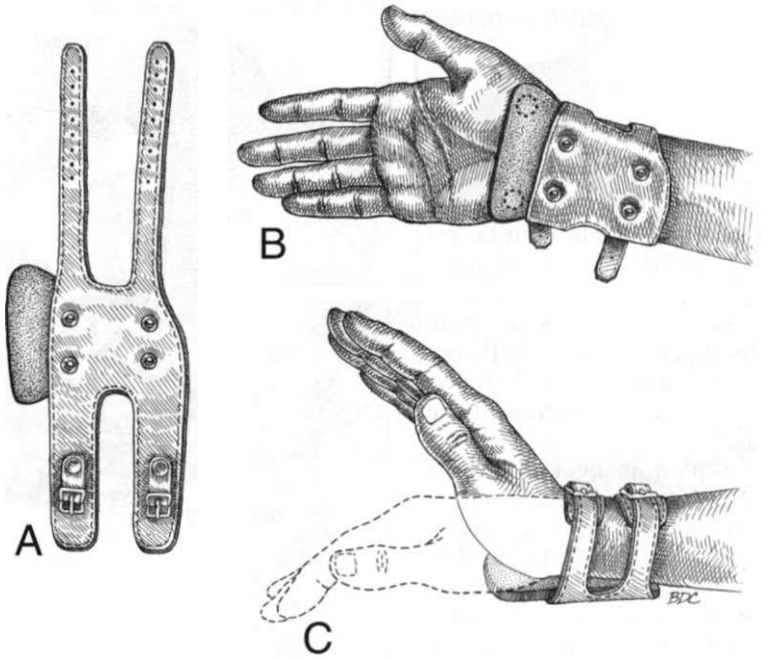


Figure 34.11. The design and application of a wrist brace made of leather or heat-moldable plastic. The brace supports the bony thenar and hypothenar prominences to limit hand flexion at the wrist. This brace relieves the hand extensors of strain during forceful movements that combine grasp, pronation and/or supination by limiting flexion and, to some extent, ulnar deviation at the wrist. It does not limit extension. **A,** pattern of the brace. The outer strap portion is made of flexible leather. The inner piece is made of stiff material. **B,** volar view of the brace strapped into position. The *dotted circles* locate the pisiform bone and base of the first metacarpal, which must be covered to restrict hand flexion effectively. **C**, side view, demonstrating the limits of flexion and extension permitted by the wrist brace.

affected elbow on a support, while seated as in Figure 34.8. The arm must be held laterally rotated at the shoulder so that the antecubital space faces up. The other hand applies the external force to pronate the forearm on the involved side and to stretch the muscle passively to reach the same position as Figure 34.8.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:385 (Fig. 6.39).
2. *Ibid.* pp. 400, 414 (Figs. 6.59, 6.83).
3. *Ibid.* p. 412 (Fig. 6.79).
4. *Ibid.* p. 428 (Fig. 6.103).
5. *Ibid.* p. 430 (Fig. 6.107).
6. Bardeen CR: The musculature, Sect. 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (pp. 421-425).
7. *Ibid.* (pp. 421, 423).
8. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 263, 264, 280, 284).
9. *Ibid.* (p. 290).
10. Basmajian JV, Latif A: Integrated actions and functions of the chief flexors of the elbow. *J Bone Joint Surg* 39A:1106-1118, 1957.
11. Bates T, Grunwaldt E: Myofascial pain in childhood. *J Pediatr* 53.198-209, 1958.
12. Bonica JJ, Sola AE: Other painful disorders of the upper limb. Chapter 52. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990 (pp. 947-958).
13. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sports Skill.* Charles C Thomas, Springfield, Ill. 1967.
14. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 535-538, 1205-1206, 1219-1221).
15. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 61, 74, 75).
16. *Ibid.* (Fig. 77).
17. *Ibid.* (Fig. 114).
18. *Ibid.* (Fig. 80).

19. *Ibid.* (Fig. 66).
20. *Ibid.* (Figs. 67, 68).
21. Cyriax J: *Textbook of Orthopaedic Medicine.* Ed. 5, Vol. 1. Williams & Wilkins, Baltimore, 1969 (pp. 315, 316).
22. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan, J.B. Lippincott, Philadelphia, 1949 (pp. 99, 100, 114-116).
23. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 81-85).
24. *Ibid.* (Sects. 84-85).
25. *Ibid.* (Sects. 81-85).
26. Folberg CR, Weiss AP, Akelman E: Cubital tunnel syndrome. Part I: presentation and diagnosis. *Orthop Rev 23(2):136-144,* 1994.
27. Fraim CJ: Unusual cause of nerve entrapment. *JAMA* 242:2557-2558, 1979.
28. Gerwin RD, Shannon S, Hong CZ, *et al.:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
29. Goldman S, Honet JC, Sobel R, *et al.:* Posterior interosseous nerve palsy in the absence of trauma. *Arch Neurol* 22:435-441, 1969 (p. 440).
30. Good MG: Acroparaesthesia—idiopathic myalgia of elbow. *Edinburgh Med J* 56:366-368, 1949.
31. Graven-Nielsen T, Arendt-Nielsen L, Svensson P, *et al.:* Experimental muscle pain: a quantitative study of local and referred pain in humans following injection of hypertonic saline. / *Musculoske Pain* 5/1J.49-69, 1997.
32. Gutstein M: Diagnosis and treatment of muscular rheumatism. *Br J Phys Med* 2:302-321, 1938 (Fig. 8, Case 8).
33. Gutstein-Good M: Idiopathic myalgia simulating visceral and other diseases. *Lancet* 2:326- 328, 1940 (Fig. 6, Case 7).
34. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(l):29-59, 1994.
35. Hong CZ, Personal Communication, 1997.
36. Hong CZ, Chen YN, Twehous D, *et al.:* Pressure threshold for referred pain by compression on the trigger point and adjacent areas. *J Musculoske Pain* 4(3):61-79, 1996.
37. Ivanichev GA: *[Painful Muscle Hypertonus].* In Russian. Kazan University Press, Kazan, 1990.
38. Jackson FE, Fleming PM, Cook RC, *et al:* Entrapment of deep branch of radial nerve by fibrous attachment of extensor carpi radialis brevis: case report with operative decompression and cure. *US Navy Med* 58:10-11, 1971.
39. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 139-141).
40. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving, I-III. *Ergonomics 18:375-*388, 1975 (pp. 383-387).
41. Kanakamedala RV, Simons DG, Porter RW, *et al:* Ulnar nerve entrapment at the elbow localized by short segment stimulation. *Arch Phys Med Behabil* 69:959-963, 1988.
42. Kelly M: Pain in the forearm and hand due to muscular lesions. *Med J Aust* 2.185-188, 1944 (Figs. 1 and 3, Cases 1 and 5).
43. Kelly M: Interstitial neuritis and the neural theory of fibrositis. *Annals Rheum Dis* 7:89-96, 1948.
44. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 260, 261, 266).
45. Kopell HP, Thompson WA: *Peripheral Entrapment Neuropathies.* Ed. 2. Williams & Wilkins, Baltimore, 1963 (Fig. 54, pp. 138-139).
46. Kosugi K, Shibata S, Yamashita H: Anatomical study on the variation of extensor muscles of human forearm. 6. M. extensor carpi radialis longus. *Jikeikai Med J* 34:51-60, 1987.
47. Lange M: *Die Muskelharten (Myogelosen).* J.F. Lehmanns, Munchen, 1931 (Fig. 38, p. 116).
48. Lieber RL, Fazeli BM, Botte MJ: Architecture of selected wrist flexor and extensor muscles. *J Hand Surg 1*5A:244-250, 1990.
49. Lieber RL, Jacobson MD, Fazeli BM, et *al.:* Architecture of selected muscles of the arm and forearm: anatomy and implications for tendon transfer. *J Hand Surg 17A(5):787-798,* 1992.
50. Lockhart RD, Hamilton GF, Fyfe FW: *Anatomy of the Human Body.* Ed. 2. J.B. Lippincott, Philadelphia, 1969 (p. 215).
51. Lundervold AJ: Electromyographic investigations of position and manner of working in typewriting. *Acta Physiol Scand* 24(Suppl 84):66, 1951 (pp. 66, 67, 80, 131).
52. Macdonald AJ: Abnormally tender muscle regions and associated painful movements. *Pain* 8:197-205, 1980 (pp. 202, 203).
53. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 133).
54. *Ibid.* (pp. 135, 147).
55. *Ibid.* (p. 134).
56. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
57. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Figs. 78, 79).
58. *Ibid.* (Figs. 81, 82).
59. *Ibid.* (Fig. 90).
60. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (p. 348).
61. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 3. Lea & Febiger, Philadelphia, 1967 (pp. 204, 206, 218).
62. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Behabil* 62.111-114, 1981 (Table 1).
63. Segal RL, Wolf SL, DeCamp MJ, *et al.:* Anatomical partitioning of three multiarticular human muscles. *Acta Anat* 142:261-266, 1991.
64. Simons DG, Travell J: Unpublished data, 1978.
65. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. Hirzel, Leipzig, 1922 (p. 325).
66. *Ibid.* (p. 326).
67. *Ibid.* (p. 330).
68. *Ibid.* (p. 332).

69. Spinner M: *Injuries to the major branches of peripheral nerves of the forearm.* Ed. 2. W.B. Saunders, Philadelphia, 1978 (p. 94).
70. Strandness DE Jr.: Pain in the extremities. Chapter 10. In: *Harrison's Principles of Internal Medicine.* Edited by Wintrobe MM, *et al.,* Ed. 7, McGraw-Hill Book Co., New York, 1974 (p. 44).
71. Sunderland S: *Nerves and Nerve Injuries.* Ed. 2. Churchill Livingstone, Edinburgh, 1978.
72. Travell J: Pain mechanism in connective tissue. In: *Connective Tissues, Transactions of the 2nd Conference, 1951.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1952 (pp. 98, 99, Fig. 33A).
73. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med 11:*425-434, 1952 (p. 428).
74. Vecchiet L, Galletti R, Giamberardino MA, *et al.:* Modifications of cutaneous, subcutaneous, and muscular sensory and pain thresholds after the induction of an experimental algogenic focus in the skeletal muscle. *Clin J Pain* 4:55-59, 1988.

CHAPTER 35
Finger Extensor Muscles: Extensor Digitorum and Extensor Indicis

HIGHLIGHTS: **REFERRED PAIN** from the extensor digitorum is projected down the forearm to the back of the hand, and often to the fingers that are moved by the involved muscle fibers. Pain from the extensor indicis is felt most strongly at the junction of the wrist and the dorsum of the hand. Sometimes tenderness in the lateral epicondyle region of the elbow arises from trigger points (TrPs) in the extensors of the ring and little fingers. The thumb extensors seldom develop TrPs. **ANATOMY:** The tendinous expansions and their complicated connections to intrinsic hand muscles provide for an unusual interplay for finger movements. The interlacing variable fibrous bands of the extensor tendons to the fingers limit the specificity with which the extensor muscles can control individual finger movements. Those individual movements depend on lumbricals, interossei, and individual finger flexor control. **FUNCTION** of these finger extensors includes primarily extension of the fingers and of the hand at the wrist, and they provide a synergistic function to permit specific grasp functions of individual fingers. They make an essential contribution to forceful finger flexion. **SYMPTOMS** may include, separately or in combination, pain, weakness, stiffness and tenderness of the proximal interphalangeal joints. Symptoms appear in the finger that corresponds to the involved portion of the extensor muscle group. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** commonly result from too forceful gripping activities, or repetitive finger movements. **PATIENT EXAMINATION** reveals increased muscle tension, muscle weakness, and also pain when the patient attempts to grip an object strongly. **TRIGGER POINT EXAMINATION** demonstrates deep tenderness with central TrP characteristics distal to the lateral epicondyle, in the midfiber portion of the extensor muscle mass. **DIFFERENTIAL DIAGNOSIS** includes consideration of TrPs as the cause of symptoms identified as tennis elbow, key TrPs in other muscles, and in functionally related muscles that include the supinator, brachioradialis and extensor carpi radialis longus muscles. **TRIGGER POINT RELEASE** is most effective if the extensors of the hand and fingers are treated as a group. Both the wrist and the fingers must be fully flexed, as the spray is applied in a proximal-to-distal pattern. **TRIGGER POINT INJECTION** of TrPs in the extensor group should employ a needle technique that also reaches any TrPs in the underlying supinator muscle. **CORRECTIVE ACTIONS** include avoidance of unnecessary muscular strain and the use of a home-exercise program to achieve and maintain a full range of motion.

1. REFERRED PAIN (Fig. 35.1)

Trigger points (TrPs) in these long extensors of the fingers project pain down the dorsum of the forearm to the back of the hand and often into the fingers dorsally. The pain consistently stops short of the ends of the fingers, leaving the last phalanx and nail bed pain-free. (In comparison, the long *flexors* of the fingers project pain to, "and beyond," the finger tips.) Confirming our observations, Gutstein[20] noted tender spots in the forearm extensors distal to the lateral epicondyle that referred pain from the dorsum of the forearm to include the middle and ring fingers.

Extensor Digitorum (Figs. 35.1A and B)

Involvement of the middle finger extensor is extremely common.[20] The pain, which is felt most intensely in the hand, forms a line that extends onto the dorsum of the forearm, wrist and hand, including the metacarpophalangeal (MCP) and proximal interphalangeal (IP) joints of the middle finger. There also may occasionally be an area of pain on the volar side of the wrist (Fig. 35.1**A**). Patients complain of pain in the hand and finger, and of stiffness and soreness in the painful finger joints.[27,51,54] The original report of this pain pattern was based on 38 patients.[51]

The ring finger extensor refers pain similarly to the ring finger.[54] However, unlike the middle finger extensor, TrPs in the ring and little finger extensors are likely also to project pain and tenderness proximally into the region of the lateral epicondyle (Fig. 35.1**B**). When asked whether the pain is felt more on the top or the underside of the fingers, the patient may not be sure, but is likely to show the location by rubbing the dorsal surface of the fingers.

Other authors described the finger extensors as referring pain to the elbow or lateral epicondyle,[19,26] to the forearm,[19,26,27] and to the hand.[26] "Tennis elbow" pain in the region of the lateral epicondyle was associated with signs of TrPs in the finger extensors.[28,29,55]

Kellgren[25] injected 0.2 ml of 6% sodium chloride solution into the belly of a normal extensor digitorum muscle. Pain developed in the dorsal forearm and more severely over the back of the hand. During the sensation of pain, there was slight tenderness to deep pressure, definite tenderness to tapping, but no hypersensitivity of the skin in the painful area.

Extensor Indicis (Fig. 35.1 C)

Central TrPs are found in the midportion of the muscle belly. They refer pain toward the radial side of the dorsum of the wrist and hand, but not into the fingers (Fig. 35.1C).

2. ANATOMY (Fig. 35.2)

Extensor Digitorum (Fig. 35.2A)

This muscle arises ***proximally*** from the lateral epicondyle of the humerus, from intermuscular septa, and from the antebrachial fascia (Fig. 35.2**A**). The extensor digitorum occupies the space on the dorsal surface of the forearm between the extensor carpi radialis brevis and the extensor carpi ulnaris muscles. The three muscles form a common tendon at the lateral epicondyle. The tendons of the extensor digitorum pass deep to the extensor retinaculum through a separate compartment with the extensor indicis.[7]

The tendons of the extensor digitorum are united over the back of the hand by highly variable oblique bands that tend to limit independent movement (Fig. 35.2**A**). The tendinous slips to the index and little fingers are usually joined by heavier tendons from the separate extensor indicis and the extensor digiti minimi muscles, respectively. Many of the extensor digitorum fibers contribute to extension of the middle finger, directly or indirectly, through the oblique bands.[7]

Distally each tendinous slip of the extensor digitorum muscle is bound by fibrous fasciculi to the collateral ligaments of its metacarpophalangeal joint, as the tendon crosses the joint. The tendon spreads into an aponeurotic expansion (also called the extensor hood) to cover the dorsal surface of the proximal phalanx of each finger. Here, it is joined by tendons of the lumbrical and interosseous muscles.[37] This aponeurosis then divides into an intermediate and two collateral slips; the middle one inserts on the base of the second phalanx and the collateral slips continue on to unite and insert onto the dorsal surface of the distal phalanx of each finger.[7]

Extensor Digiti Minimi (Fig. 35.2A)

The extensor digiti minimi is not considered separately in this chapter because its muscle belly is generally connected to the adjacent extensor digitorum muscle.[7] Distally, the extensor digiti minimi joins with the extensor digitorum tendon and

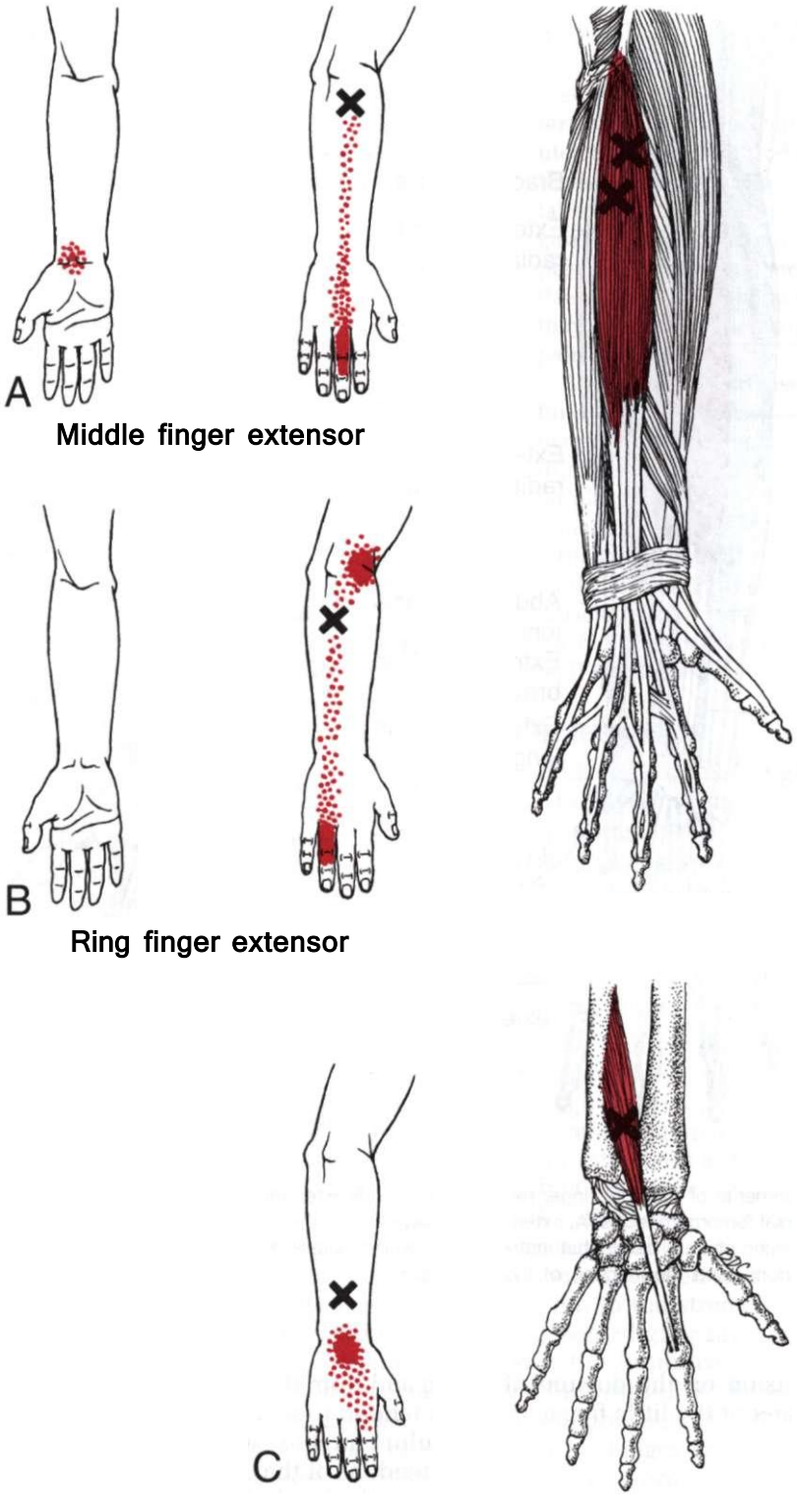


Figure 35.1. Pain patterns *(dark red)* and location of TrPs (**Xs**) in three right digital extensor muscles *(medium red)*. **A,** middle finger extensor. **B,** ring finger extensor. **C,** extensor indicis, dorsal view.

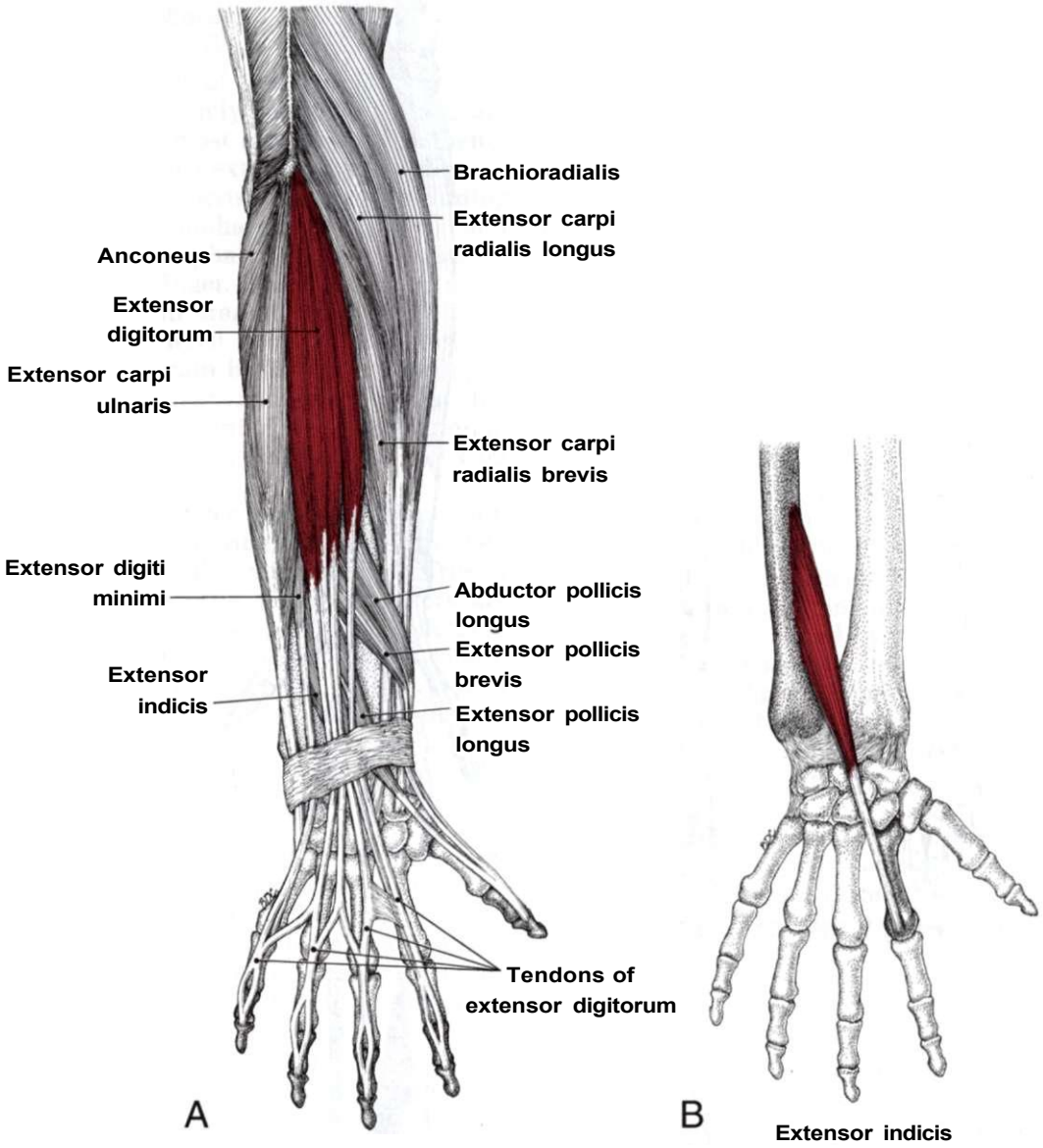


Figure 35.2 A-B. Attachments of the right finger extensor muscles and dorsal forearm muscles. **A,** extensor digitorum *(red),* showing oblique bands that interconnect the distal tendons, and the junction of the extensor indicis tendon with the index finger tendon of the extensor digitorum muscle. **B,** extensor indicis *(red),* which passes beneath the extensor digitorum tendons.

the extensor expansion on the dorsum of the proximal phalanx of the little finger.

Extensor Indicis (Fig. 35.2B)

This muscle arises *proximally* from the dorsal and lateral surface of the body of the ulna and from the interosseous membrane. The tendon passes under the extensor retinaculum in the same compartment with the tendons of the extensor digitorum. *Distally,* at the level of the head of the second metacarpal bone, it joins the ulnar side of the slip of the extensor digitorum muscle

going to the index finger and attaches into the extensor expansion.

Supplemental References

The extensor digitorum muscle is illustrated by other authors from the dorsal view,[1,7,8,49] from the radial aspect,[29,47] and retracted to show its innervation and blood supply.[12] Also shown in detail is the arrangement of its tendons on the dorsum of the hand,[3,7,10,13,30,37,48] and its tendinous attachments to each finger.[4,11] The extensor indicis is seen in the deepest layer of the dorsal forearm muscles.[14,38,50]

The **Type** I **and Type** II **fibers** in both the extensor digitorum communis and the extensor pollicis longus muscles were evenly distributed,[24] as in most skeletal muscles. Type 1 fibers ranged between 46% and 55%, the dominant side consistently showing a lower percentage of type 1, slow twitch fibers than the nondominant side.

Anatomical Variations

The **Extensor Digitorum Brevis Manus** muscle is a relatively rare anatomical variation occurring in 38 (1.1%) of 3,304 hands examined. When present, it is commonly seriously symptomatic (50% of the 38 cases).[17] It is clinically important because it may become painful when overexercised[31] and may be misdiagnosed as a ganglion cyst or tumor, resulting in unnecessary surgery.[31] It originates on the distal margin of the radius or from the dorsal capsule of the wrist joint and inserts on the dorsal aponeurosis of the index finger.[46] This muscle frequently appears as a variation of the extensor indicis proprius because when the extensor digitorum brevis manus is present, the extensor indicis proprius is usually absent.[17] The variant muscle is best demonstrated clinically when the wrist is flexed to 30° and the fingers fully extended.[40] It appears as a prominent mass on the dorsum of the hand near the bases of the first and second metacarpal bones except in the rare cases when it lies deep to the extensor digitorum communis. The diagnosis of the unknown mass can be made by suspecting it and by palpating it for increased tension in response to finger extension, and it can be confirmed by electromyographic examination. It is supplied by the dorsal interosseous nerve. Symptoms are attributed to synovitis caused by constriction of the hypertrophic muscle belly by the distal edge of the extensor retinaculum. Surgical release of the retinaculum generally provides relief.[46] The incidence of TrPs in this muscle is unknown, but if present they could contribute significantly to the pain.

An anomalous **extensor digitorum profundus** muscle caused pain and swelling over the dorsal aspect of the second and third metacarpals of the left hand in a guitar player. Instead of the muscle belly terminating proximal to the extensor retinaculum, it continued under it and extended four centimeters distal to it. The muscle was biopsied under local anesthesia for diagnostic purposes and the patient became asymptomatic.[43]

3. INNERVATION

Both the extensor digitorum and extensor indicis muscles are supplied by the deep radial nerve and the posterior cord, which is formed from all three posterior divisions and all three trunks of the brachial plexus. Both muscles are innervated through spinal nerves C_6, C_7 and C_8.

4. FUNCTION

The extensor digitorum muscle extends all phalanges of the fingers (second through fifth digits),[5,7,30] especially the proximal phalanges,[15] and assists extension of the hand at the wrist.[5,7] It assists in abducting (spreading) the index, ring, and little fingers away from the middle finger.[7,30] All of the extrinsic hand muscles become involved in a power grip, in proportion to the strength of the grip.[5,35] The extensor digitorum acts in conjunction with the lumbricals and interossei to extend the middle and distal phalanges of the second through fifth digits. When the proximal phalanges are held in flexion, the extensor digitorum extends the more distal phalanges, but when the proximal phalanges and the hand are held in extension, then, its contraction has little additional ef-

feet on the last two phalanges.[30,42] These extensors provide an essential synergistic function to permit selective control of individual fingers.

The extensor indicis, in addition to acting on the index finger in the same way that the extensor digitorum acts,[45] may assist in adducting the index finger toward the middle finger,[15,30] because of the angulation of its tendon across the dorsum of the hand.

> Electromyographic monitoring of the hand and finger extensors with surface electrodes was performed during 13 sports, including tennis, golf, baseball, overhand throws, and 1-foot jumps from the floor. All records showed similar motor unit activity bilaterally. The greatest activity appeared in the dominant right forearm during a right-handed golf swing.[6]

5. FUNCTIONAL UNIT

Strong agonist-antagonist interactions are needed between the flexors and extensors of the hand and fingers to produce finger dexterity as well as to produce forceful hand grip. Powerful *flexion* of the distal phalanges requires strong activity also of the *finger extensors.* On the other hand, for the extensor digitorum to extend the interphalangeal joints, the lumbricals and interossei need to function.

The ring and little finger extensors form a functional unit with the supinator for twisting motions, such as opening jar tops and door knobs. Understandably, these three muscles often develop TrPs together.

6. SYMPTOMS

Patients with TrPs in the **finger extensor** muscles complain of pain, as described in Section 1. It may be identified with "tennis elbow," or with arthritis of the fingers.[28,29,55] Early in this century in the days of long skirts when women suffered elbow pain from holding the skirts up, the pain was called epicondylalgia, or brachialgia.[34] The activity has changed, the problem has not. The pain may awaken patients at night.[27] If the firm grip that is required for shaking hands is distressingly painful at the "elbow," TrPs in the extensor muscles of the ring and little fingers are likely to be responsible.

When the **middle finger extensor** alone is involved, the patient may still complain of weakness of the grip, without pain.[53] The finger extensors are essential to a powerful grip, and this weak grip presents another example of the observation that TrPs can inhibit muscular contraction.[53] Muscles learn[52] and can learn dysfunctional behavior but also can be retrained to develop functional behavior.[21]

Symptoms of impaired finger *flexion* may be due to TrPs in the finger *extensor* muscles. Patients may complain of stiffness and tenderness of the proximal interphalangeal joints. Stiffness and painful cramping of the fingers prevented one patient from milking his cows until tender TrPs in his extensor digitorum muscle had been inactivated.[27] A patient seen by Doctor Travell could not type because the ring and little fingers would "not work separately" until the TrPs were injected in the extensor fibers of those fingers.

Patients with TrPs in the *extensor pollicis* complain of difficulty performing skilled hand activities including working in dentistry, writing by hand, and operating a keyboard.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Myofascial TrPs in the finger extensors commonly occur due to such activities as overuse of forceful repetitive finger movements by professional musicians (pianists in particular), carpenters, or mechanics, and frequently stretching a rubber band with the finger extensors. Local infection of the ring finger in a seamstress resulted in a stiff and painful ring finger that was relieved months later by injection of a TrP in the muscle fibers that extended that finger. Activation of finger extensor TrPs by fracture of the forearm has been observed by the authors and was reported by Kelly.[27]

When a finger extensor tendon loses its mooring over the metacarpophalangeal joint, the tendon may be said to "jump its trolley." This is a serious source of muscular strain due to the resultant ulnar devia-

tion of the finger, and the tendon displacement must be surgically repaired for restoration of function.[16]

8. PATIENT EXAMINATION (Fig. 35.3)

Since these finger extensors cross the wrist and all joints of the fingers, the examiner needs to passively flex all those joints to detect restricted range of motion due to TrPs. It is best to fully flex the fingers first, then slowly and gently flex the wrist and finally, move the wrist into ulnar deviation to reveal increased muscle tension caused by taut bands.

Limitation of the *active* range of motion can be tested with the Finger-flexion Test by having the patient flex the interphalangeal joints to bring the tips of the fingers against the palmar pads, while extending the metacarpophalangeal joints (Fig. 35.3). Increased tension of an affected finger extensor muscle due to a TrP results in that finger standing out from the others, away from the palm, such as the middle finger extensor in Figure 35.3. Passive flexion of the finger beyond this point is painful.

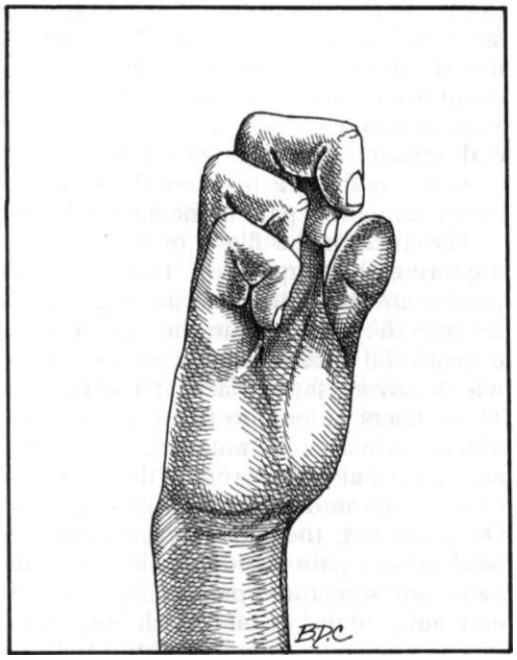


Figure 35.3. Positive Finger-flexion Test, illustrating involvement of only the middle finger extensor muscle. The subject can actively press all of the other finger tips firmly against the palmar pads while the metacarpophalangeal joints are held straight, not flexed.

Weakness due to finger extensor TrPs is detected in the grip during a handshake by testing and comparing both hands simultaneously. This bilateral Handgrip Test is more sensitive when the patient holds the hands in ulnar deviation and flexed at the wrist. This test may reveal weakness without pain when the TrPs are latent.

Tenderness of the proximal interphalangeal joint is commonly associated with the finger stiffness and "soreness" due to finger extensor TrPs, sometimes *without* referred pain in the joint.[53] This may be analogous to the tendinitis associated with TrPs in the fibers of the long head of the biceps brachii (see Chapter 30). Both conditions may be completely relieved by inactivation of the myofascial TrPs in the responsible muscle.

Although the extensor pollicis usually is involved along with other finger extensors, occasionally it alone develops TrPs, which can make it difficult to find the source of symptoms unless this muscle is tested specifically. To test restricted movement due to TrPs in the extensor pollicis, (1) flex the hand passively, (2) pronate the forearm fully, (3) adduct the thumb passively beneath the index finger, (4) passively flex the metacarpophalangeal (MCP) joint, (5) test by passively flexing the interphalangeal (IP) joint. When positive, flexion of the IP joint is limited and causes pain dorsal to the first carpometacarpal joint and radial to the second metacarpal bone. The thumb will tend to move into radial abduction as the IP joint is flexed. When involvement is severe, this tendency for radial abduction may become evident at the end of step 2. This description was supplied by Sachse.[44] Macdonald[36] also observed that loading an involved muscle by actively resisting extension of the thumb caused pain.

The extensor digitorum muscle can be tested for weakness by resisting extension of the metacarpophalangeal joints of the second through fifth digits with the arm resting on a table, as illustrated by Kendall, *et al.*[30] Weakness of this muscle also weakens wrist extension.

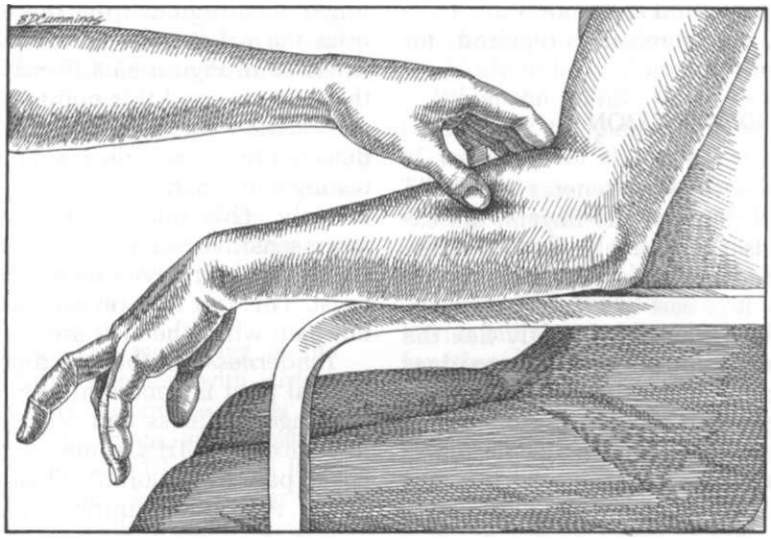


Figure 35.4. Demonstration of a local twitch response produced by snapping palpation of a trigger point nodule in the extensor digitorum fibers to the middle finger. The response of noticeable movement of the middle finger is readily demonstrable in most adults, even without pain symptoms, due to the almost universal presence of *latent* trigger points in this muscle.

Patients suspected of having troublesome TrPs in the finger extensors should be examined for normal joint play in the elbow, wrist and hand. If restricted, normal joint play should be restored.[32,39]

9. TRIGGER POINT EXAMINATION (Fig. 35.4)

Gerwin, *et al.*[17] established that among experienced and trained examiners, reliable criteria for diagnosing myofascial TrPs were the detection of a taut band, the presence of spot tenderness, the presence of referred pain, and reproduction of the patient's symptomatic pain. Although for some muscles tested, local twitch responses (LTRs) were not identified reliably, the **extensor digitorum** in this study scored very high interrater reliability for all examinations including the LTR. It is one of the easier muscles to examine reliably for TrPs.

Nearly all adults have a tender, *latent* TrP in the third finger extensor because it is used in almost every activity of the hand. Snapping palpation of this TrP in the middle-finger extensor produces one of the commonest, most easily elicited, and most easily detected LTRs (Fig. 35.4). This muscle provides a convenient training opportunity for practice in finding a nodule and taut band and for eliciting LTRs. This central TrP is located 3 to 4 cm (about 1 1/2 in) distal to the head of the radius, which lies 2 cm (nearly 1 in) or more distal to the lateral epicondyle (Figs. 35.1**A** and 35.4). Only when the TrP is active does the patient complain of pain in the middle finger.

The TrPs in the fibers of the extensor digitorum that supply the ring and little fingers are difficult to locate (Fig. 35.1**B)** because they are deep in the muscle mass beneath the aponeurosis of origin, part of which covers the surface of the muscle. These fibers lie next to the extensor carpi ulnaris, which is the muscle mass just lateral to the palpable border of the ulna, and close to the underlying supinator muscle. On palpation, these two finger extensors tend to refer pain distally to the wrist and hand, and sometimes proximally to the lateral epicondyle. Local twitch responses, when obtainable, extend the little and ring fingers and confirm the presence of a TrP.

When these central TrPs are present, the attachment of the taut band fibers in the region of the lateral epicondyle frequently is tender to palpation. This tenderness likely identifies the location of an attachment TrP

resulting from the sustained tension caused by the contraction knots in the central TrP.

An active TrP in the **extensor indicis** is found in the middle section of the muscle belly (Fig. 35.1**C**) and, when stimulated by pressure, this central TrP projects pain to the wrist, rarely to the finger. This TrP is seldom found by itself; when the activity of other TrPs has been eliminated and wrist pain persists, an extensor indicis TrP is likely to be the culprit.

Rarely do any of the thumb muscles in the forearm develop TrPs, apparently because the extensor pollicis longus and brevis are minimally involved in grasp activity, and control of the thumb involves only one, rather than two interphalangeal joints.

10. ENTRAPMENT

No entrapments have been observed due to TrP activity in the finger extensor muscles.

11. DIFFERENTIAL DIAGNOSIS

Differential diagnoses for TrPs in the finger extensors include lateral epicondylitis (tennis elbow), C_7 radiculopathy (occasionally C_6 radiculopathy), and DeQuervain's stenosing tenosynovitis. Myofascial TrPs in the finger extensors can be associated with volar subluxations of carpal bones, which must be corrected.

The common diagnosis of tennis elbow or epicondylitis is frequently caused by TrPs in at least one muscle that attaches to the lateral epicondyle; often several of them are involved. Usually (but by no means always), the supinator becomes involved first, followed by the brachioradialis and extensor carpi radialis longus muscles. With the passage of time and spread of the involvement to the middle and ring finger extensors, gripping and hand-twisting motions become painful. At this point, the extensor carpi ulnaris also may develop secondary TrPs. The epicondylitis may begin as an enthesopathy secondary to the central TrPs, but is often not recognized as such, and so the primary TrP cause of the symptoms goes unrecognized and untreated. Tennis elbow is covered as a separate topic in Chapter 36, Section 11.

In addition to these related TrPs, Hong[23] found that key myofascial TrPs in either the scalene muscles or the serratus posterior superior could induce satellite TrPs in the extensor digitorum. These satellite TrPs resolved with inactivation of the key TrPs.

The clinical history of hand difficulties among 100 musicians[22] corresponded to the symptoms characteristic of myofascial TrPs in the wrist and finger extensors, but TrPs were not mentioned in that paper. The symptoms were extensor forearm or dorsal hand pain, loss of facility, and rapid onset of fatigue on repetitive movement. The usual diagnosis was tendinitis of the finger extensors, which could be enthesopathy secondary to central TrPs. The treatment was cessation of playing and no use of the hand for 3 to 6 weeks—a stiff penalty for a musician if it is unnecessary.

12. TRIGGER POINT RELEASE (Fig. 35.5)

For **spray and stretch,** the patient sits in a chair with suitable armrests so that the elbow can be supported in extension and, simultaneously, the hand and fingers can hang fully flexed over the edge with the forearm pronated (Fig. 35.5). Curling only the fingers or bending only the wrist fails to stretch the long extensors of the fingers sufficiently to eliminate their TrPs during vapocooling. Gentle stretching across all finger and wrist joints must be performed simultaneously while parallel sweeps of the spray cover the muscle and its referred pain pattern. The clinician should avoid squeezing the fingers tightly, as doing so can hurt the joints. When trigger points (TrPs) in the ring and little finger extensor muscles refer pain over the lateral epicondyle, an up-sweep (proximal) pattern is added to cover that region also. Moist heat is then applied over the forearm muscles, followed by 3 slow cycles of *full* range of motion in both flexion and extension.

Lewit[32] fully described and illustrated a similar stretch procedure for the extensor digitorum communis that employed postisometric relaxation and is also suitable for home use. The use of postisometric relaxation (described in Chapter 3, Section 12) is highly recommended for release of TrPs in these extensors.

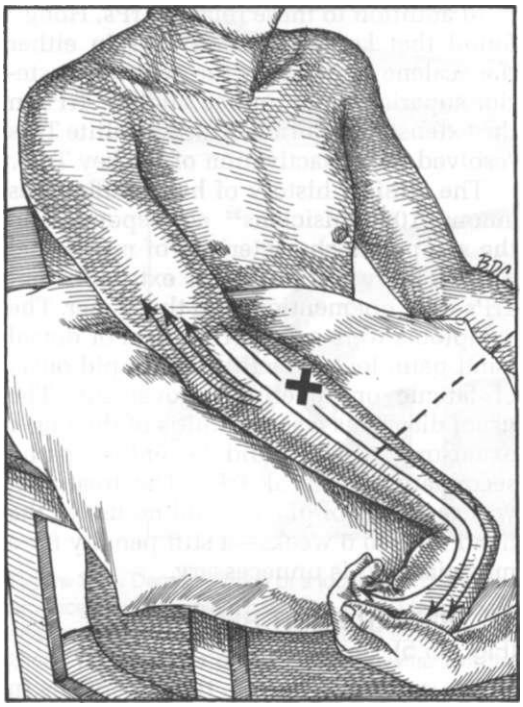


Figure **35.5.** Stretch position and spray pattern *(arrows)* for the entire extensor digitorum muscle. The **"X"** marks the central trigger point region. The downward spray pattern should swing around to include the lateral epicondyle, especially when it also exhibits referred pain and tenderness.

Another effective form of release for TrPs in these muscles is trigger point pressure release (described in Chapter 3, Section 12). The patient can follow the procedure with active flexion which utilizes reciprocal inhibition for additional release.

Following treatment, the patient is encouraged to increase activities gradually, avoiding those that stress the involved muscle enough to make it hurt. Some patients are Spartan and determined to "exercise" and "strengthen" the weak muscle; these patients must be discouraged from purposely repeating painful activities, and thus aggravating their condition.

13. TRIGGER POINT INJECTION (Fig. 35.6)

Other authors have found, as we have, that injection into the site of trigger point (TrP) tenderness in the finger extensors is effective in relieving the patient's myofascial signs and symptoms.[19,26] For injection, the patient lies supine with the arm placed so that the hand and fingers hang down limply, which stretches the finger extensors moderately. After injection, the muscle should be passively stretched to its full range of motion, usually during vapocooling, and moist heat is applied for 5 or 10 minutes. Finally, the patient *slowly* flexes and extends the finger extensors through *full* range of motion for three cycles.

Then, the same activity guidelines apply as after the spray-and-stretch treatment.

Extensor Digitorum

The TrPs in the middle finger extensor are identified by flat palpation and injected with 0.5% procaine solution (Fig. 35.6**A**). Strong LTRs and clear pain patterns, as elicited by examination and needle penetration of the TrPs, are characteristic of this muscle.

The TrPs in the ring and little finger extensors are located between those in the middle finger extensor fibers and the extensor carpi ulnaris muscle. The needle is directed toward the point of deep tenderness (Fig. 35.6**B**). It is not always clear whether the TrP, which is encountered by the needle at considerable depth and which refers pain to the lateral epicondyle, is in the finger extensor or in the underlying supinator muscle. Normal grip strength may return immediately after elimination of these extensor TrPs.[53]

Occasionally, a deep radial (dorsal interosseous) nerve block may inadvertently be produced during injection of these TrPs. The patient should be warned beforehand of possible temporary extensor-muscle weakness, which resolves in 15 or 20 minutes when the dilute 0.5% procaine solution has been injected.

The endplate zone in the extensor digitorum muscle should extend diagonally across the middle of the muscle belly based on the fiber length/muscle belly length ratio of 0.42 to 0.50 for the different digitations.[33] Trigger points can be located anywhere in the endplate zone.

Rachlin[41] illustrates three injection sites in the extensor digitorum communis, the central TrP site in the middle of the muscle, and the two attachment TrPs at the ends of the muscle belly. The sustained tension of the taut bands caused by the

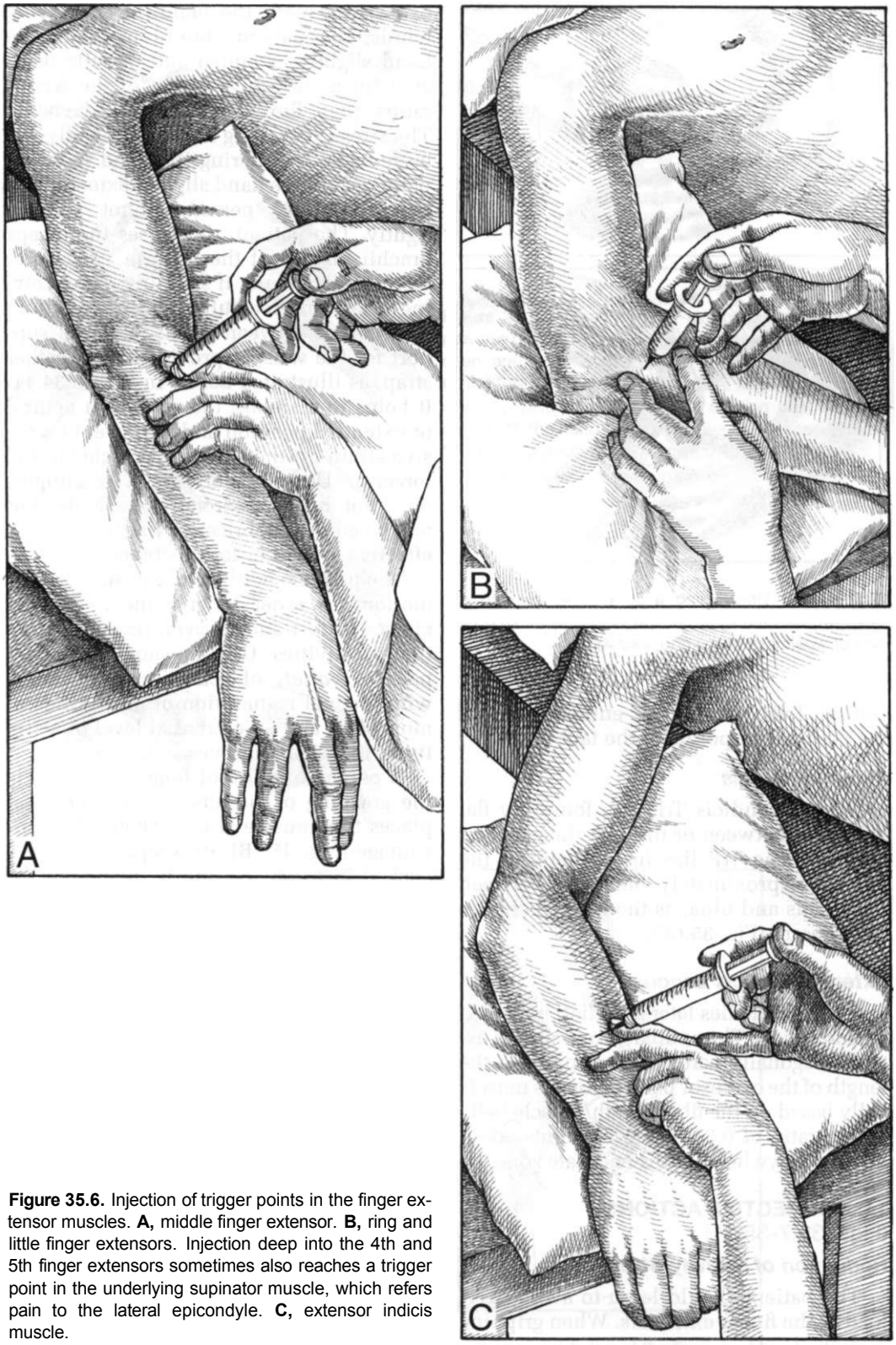


Figure 35.6. Injection of trigger points in the finger extensor muscles. **A,** middle finger extensor. **B,** ring and little finger extensors. Injection deep into the 4th and 5th finger extensors sometimes also reaches a trigger point in the underlying supinator muscle, which refers pain to the lateral epicondyle. **C,** extensor indicis muscle.

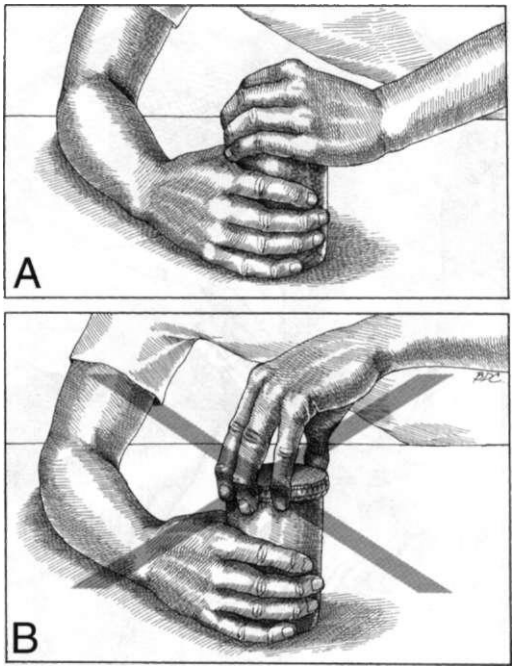


Figure 35.7. Unscrewing a jar top. **A,** position of strength, with the wrist cocked in extension. **B,** position of weakness of the grip, *(red* **X**).

central TrPs can induce enthesopathy at the attachment points of the taut bands.

Extensor Indicis

Extensor indicis TrPs are found by flat palpation between or through the extensor tendons. The TrP lies in the belly of the muscle approximately half way between the radius and ulna, as the muscle crosses the forearm (Fig. 35.6**C**).

Extensor Pollicis Longus

This muscle lies lateral (radial) to the extensor indicis. The endplate zone should extend diagonally across and two-thirds the length of the extensor pollicis longus muscle belly based on the fiber length/muscle belly length ratio of 0.31.[33] Trigger points could develop anywhere in this endplate zone.

14. CORRECTIVE ACTIONS (Figs. 35.7-35.11)

Reduction of Activity Stress

The patient should learn to avoid overload of the finger extensors. When gripping or twisting with the hand, as in playing tennis, the patient should maintain the hand slightly extended and radially deviated (in a cock-up position of the wrist), rather than flexed and ulnarly deviated. The stress of shaking hands repeatedly can be reduced by offering the hand with the palm up and the hand slightly extended, so that the other person cannot squeeze tightly. The patient, thus, uses the biceps brachii instead of the forearm muscles to flex the elbow and, if standing in a receiving line, can gracefully alternate left and right hands between guests. External support for the wrist is provided by a leather strap, as illustrated before in Figure 34.11. It helps to maintain the wrist in a neutral or extended position and to prevent excessive strain on the extensor muscles in the forearm. Unfortunately, such a support may not be commercially available, but may need to be fabricated. Elastic is not as effective as leather for this brace.

The patient should avoid testing painful motions in order to give the muscles a chance to rest and recover, resuming only those activities that do not precipitate pain. A *variety* of activities is desirable, with gradual resumption of more kinds of movement and an increased level of activity as function improves.

A common abuse of finger extensors is the grasping of jar lids in a manner that places this muscle at a mechanical disadvantage (Fig. 35.7**B**). By keeping the wrist cocked in extension and by using the entire arm as a lever (Fig. 35.7**A**), stress on the finger and hand extensors is reduced.

Exercises

The Artisan's Finger-stretch Exercise (Fig. 35.8) and the Finger-flutter Exercise (Fig. 35.9) are especially useful for people who hold their hands in a tense position for long periods of time or perform repetitive finger movements. Examples are those who do fine tool work, piano playing, or longhand writing.

The Artisan's Finger-stretch Exercise begins by placing the forearms pronated in front of the body with the fingers extended and spread apart (Fig. 35.8**A**). As the forearm is slowly supinated, the fingers are flexed, little finger first (Figs. 35.8**B** and **C**),

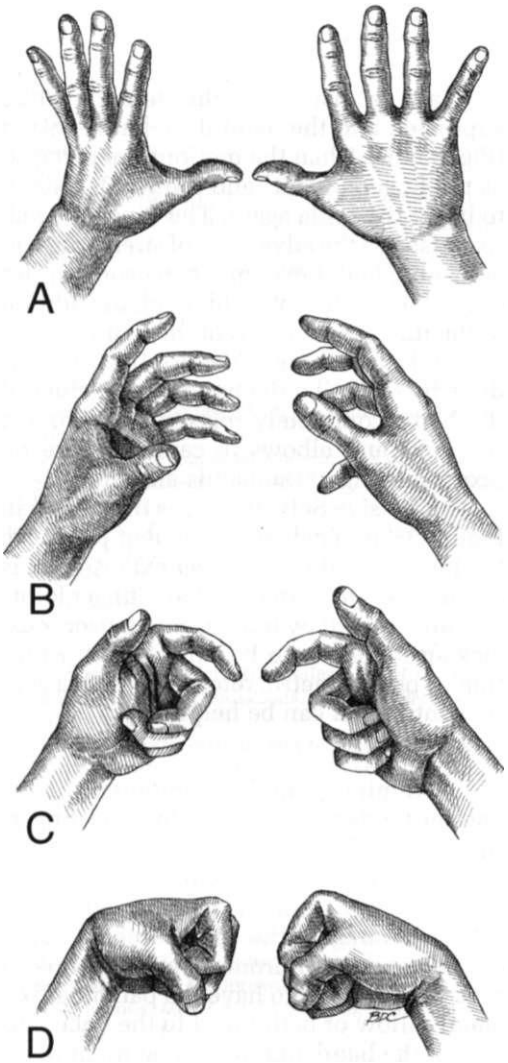


Figure 35.8. Artisan's Finger-stretch Exercise. **A,** the exercise begins with the forearm pronated and the hand open, and the wrist and fingers in full extension. **B** and **C,** the forearm is supinated and the fingers closed in a smooth, continuous movement, starting with the little fingers. **D,** the hand is flexed as the fist is closed forcefully with the thumb overlapping the index finger.

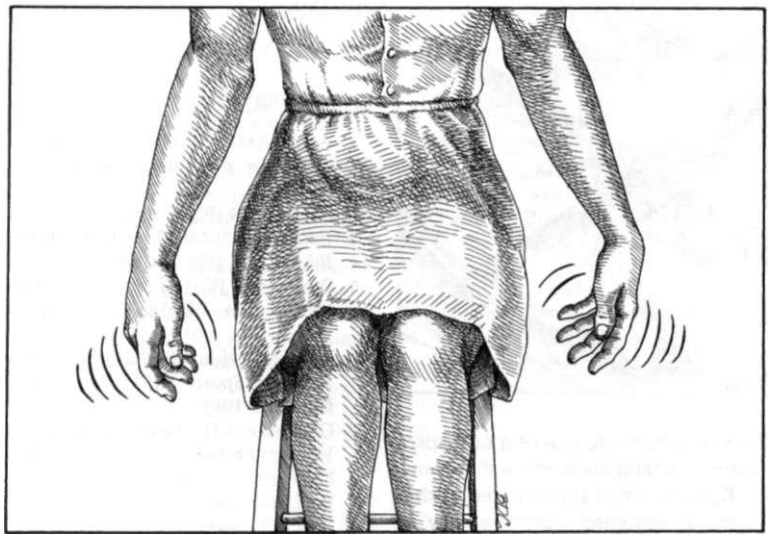


Figure 35.9. The Finger-flutter Exercise demonstrates relaxed passive flutter of the finger and hand extensors by shaking the arm, elbow and forearm.

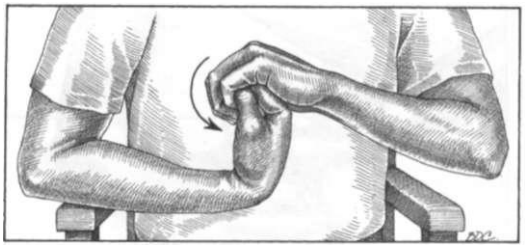


Figure 35.10. Passive Self-stretch Exercise for the right extensor digitorum muscle. The right hand and fingers are fully flexed simultaneously.

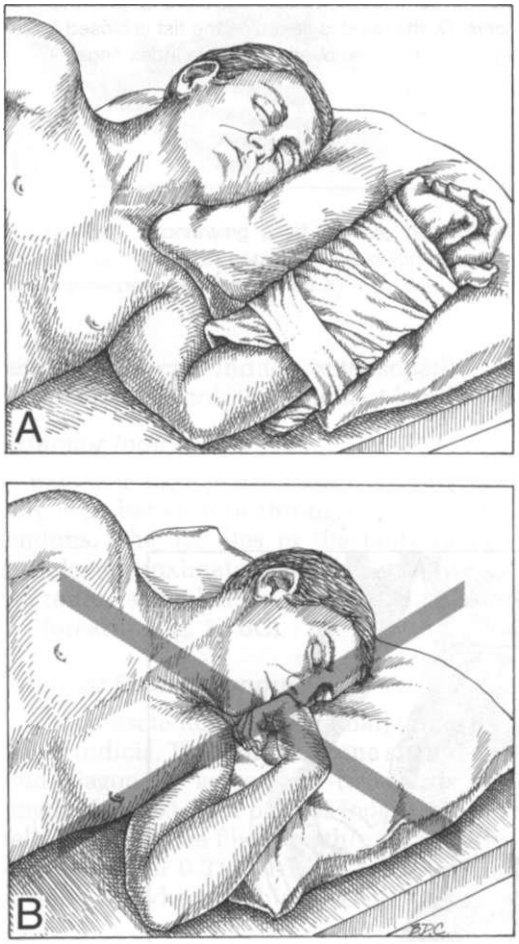


Figure 35.11. Sleep positions. **A,** use of a soft splint to maintain the correct, neutral positions of the elbow, wrist and fingers. **B,** an incorrect position *(red* **X**) that must be prevented, if assumed spontaneously in sleep.

to make a fist with the forearm fully supinated and the hand flexed at the wrist (Fig. 35.8**D**). Then the motions are reversed as the fingers uncurl and the wrists extend to begin the cycle again. The Finger-stretch Exercise has the advantage of stretching and activating both flexor and extensor muscles of the fingers, thumb, and hand, in addition to the intrinsic muscles of the hand.

The Finger-flutter Exercise (Fig. 35.9) is done by dropping the hands to the sides of the body, completely relaxed, and moving the arms and elbows to cause passive relaxed shaking of the hands and fingers.

The Passive Self-stretch, as illustrated in Figure 35.10, enables the patient to relieve the tension of the taut finger extensors. It is essential for both the wrist and finger joints that are crossed by the finger extensor muscles and tendons to be fully flexed. Addition of postisometric relaxation with a gentle contraction can be helpful.

Positioning

Positioning at night is important if the patient tends to hold the hand and fingers in a fully flexed position (Fig. 35.11**B**). This position places the finger extensors in a stretched position for a sustained period and encourages the development of a carpal tunnel syndrome. To avoid this, it may be necessary to have the patient affix a small pillow or bath towel to the volar surface of the hand and forearm at night in order to maintain a neutral mid-position (Fig. 35.11**A**).

REFERENCES

1. **Agur AM:** *Grant's Atlas of Anatomy.* **Ed. 9. Williams & Wilkins, Baltimore, 1991:428 (Fig. 6.103).**
2. *Ibid.* **p. 434 (Fig. 6.114).**
3. *Ibid.* **pp. 429,430 (Figs. 6.105, 6-107).**
4. *Ibid.* **p. 431 (Fig. 6.109).**
5. **Basmajian JV, DeLuca CJ:** *Muscles Alive.* **Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 290, 292-294).**
6. **Broer MR, Houtz SJ:** *Patterns of Muscular Activity in Selected Sports Skill.* **Charles C Thomas, Springfield, Ill., 1967.**
7. **Clemente CD:** *Gray's Anatomy.* **Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 530, 536, 537, 540).**
8. **Clemente CD:** *Anatomy.* **Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 74, 75).**
9. *Ibid.* **(Fig. 61).**
10. *Ibid.* **(Fig. 103).**

11. *Ibid.* (Fig. 116).
12. *Ibid.* (Fig. 78).
13. *Ibid.* (Fig. 104).
14. *Ibid.* (Fig. 77).
15. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 126).
16. Flatt AE: *The Care of the Rheumatoid Hand.* Ed. 3. C.V. Mosby, St. Louis, 1974 (pp. 249-277).
17. Gama C: Extensor digitorum brevis manus: a report on 38 cases and a review of the literature. *J Hand Surg 8(5 Pt. 1)*:578-582, 1983.
18. Gerwin RD, Shannon S, Hong CZ, *et al.:* Interrater reliability in myofascial trigger point examination. *Pain* 69.65-73, 1997.
19. Good MG: The role of skeletal muscles in the pathogenesis of diseases. *Acta Med Scand* 238:285-292, 1950 (p. 287).
20. Gutstein M: Common rheumatism and physiotherapy. *Br J Phys Med* 3:46-50, 1940 (p. 47).
21. Headley BJ: Evaluation and treatment of myofascial pain syndrome utilizing biofeedback. Chapter 5. In: *Clinical EMC for Surface Recordings,* Vol. 2. Edited by Cram JR. Clinical Resources, Nevada City, 1990.
22. Hochberg FH, Leffert RD, Heller MD, *et al.:* Hand difficulties among musicians. *JAMA 249(14):1869-*1872, 1983.
23. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Muscuhske Pain* 2(1);29-59, 1994.
24. Jozsa L, Demel S, Reify A: Fibre composition of human hand and arm muscles. *Gegenbaurs Morph fahrb, Leipzig* 227:34-38, 1981.
25. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3:175-190, 1938 (p. 187).
26. Kelly M: New light on the painful shoulder. *Med J Aust* 2:488-493, 1942 (Case 8, Figs. 3D and 3F).
27. Kelly M: Pain in the forearm and hand due to muscular lesions. *Med J Aust* 2.185-188, 1944 (Cases 2, 7, and 9; Fig. 4).
28. Kelly M: Some rules for the employment of local analgesia in the treatment of somatic pain. *Med J Aust* 2:235-239, 1947 (p. 236).
29. Kelly M: The relief of facial pain by procaine (Novocaine) injections, *J Am Geriatr Soc* 22:586-596, 1963 (Case 3, p. 589).
30. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 254, 255).
31. Kuschner SH, Gellman H, Bindiger A: Extensor digitorum brevis manus: an unusual cause of exercise-induced wrist pain. *Am J Sport Med* 2 7(3):440-441, 1989.
32. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991:147-149, 200-202.
33. Lieber RL, Jacobson MD, Fazeli BM, *et al.:* Architecture of selected muscles of the arm and forearm: anatomy and implications for tendon transfer. *J Hand Surg 17A(5):787-798,* 1992.
34. Llewellyn LJ, Jones AB: *Fibrositis.* Rebman, New York, 1915 (Fig. 35 opposite p. 226; p. 227).
35. Long C, Conrad PW, Hall EA, *et al.:* Intrinsic-extrinsic muscle control of the hand in power grip and precision handling. *J Bone Joint Surg* 52A:853-867, 1970.
36. Macdonald AJ: Abnormally tender muscle regions and associated painful movements. *Pain* 8:197-205, 1980 (pp. 202, 203).
37. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (pp. 135, 146, 147, 150).
38. *Ibid.* (p. 135).
39. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
40. Patel MR, Desai SS, Bassini-Lipson L, *et al.:* Painful extensor digitorum brevis manus muscle. *J Hand Surg 14A(4):674-878,* 1989.
41. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (p. 351).
42. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 200, 203).
43. Reeder CA, Pandeya NK: Extensor indicis proprius syndrome secondary to an anomalous extensor indicis proprius muscle belly. *J Am Osteopath Assoc* 92(3j:251-253, 1991.
44. Sachse J: Personal Communication, 1994.
45. Sano S, Ando K, Katori I, *et al:* Electromyographic studies on the forearm muscle activities during finger movements. *J Jpn Orthop Assoc* 52:331-337, 1977.
46. Shaw JA, Manders EK: Extensor digitorum brevis manus muscle: a clinical reminder. *Orthop Rev* 28(9):867-869, 1988.
47. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 330).
48. *Ibid.* (p. 334).
49. *Ibid.* (p. 331).
50. *Ibid.* (p. 333).
51. Travell J: Pain mechanisms in connective tissue. In: *Connective Tissues, Transactions of the Second Conference, 1951.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1952 (Fig. 33, pp. 98, 99).
52. Travell J: Myofascial trigger points: clinical view. In: *Advances in Pain Research and Therapy.* Edited by Bonica JJ, Albe-Fessard D. Raven Press, New York, 1976 (pp. 919- 926).
53. Travell J, Bigelow NH: Role of somatic trigger areas in the patterns of hysteria. *Psychosom Med* 9:353-363, 1947 (p. 356).
54. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 2 2:425-434, 1952 (p. 428).
55. Winter Z: Referred pain in fibrositis. *Med Rec* 157:34-37, 1944 (pp. 37, 38).

CHAPTER 36
Supinator Muscle

HIGHLIGHTS: "Tennis Elbow" or "epicondylitis," as pain in the lateral epicondyle is often called, is frequently of myofascial origin, usually due to trigger points (TrPs) in the supinator and extensor muscles in the forearm. **REFERRED PAIN** from TrPs in the supinator is projected chiefly to the lateral epicondyle, frequently to the dorsal aspect of the web and base of the thumb, and sometimes to the forearm dorsally. **ANATOMY:** its attachment along the dorsal surface of the ulna at the elbow positions the supinator to wrap around the lateral surface of the radius lateral to its attachment on the volar surface of the radius. The radius acts like a windlass that winds up the supinator and the biceps brachii tendon when the hand is pronated. **FUNCTION** of the supinator is primarily to supinate the forearm, and it secondarily assists flexion at the elbow. **SYMPTOMS** are mainly elbow pain, both at rest and when the arm is used for carrying heavy objects. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the supinator may occur due to stress overload, as when playing tennis, "flipping" a briefcase onto the desk, or turning a stiff doorknob. **PATIENT EXAMINATION** reveals marked referred tenderness to tapping of the lateral epicondyle on the side of tendinous attachment. **TRIGGER POINT EXAMINATION** proceeds by bending the elbow slightly, supinating the forearm, pushing the brachioradialis muscle aside, and palpating the supinator for TrPs against the head and shaft of the radius in the distal antecubital space. **ENTRAPMENT** of the deep radial nerve as it enters the arcade of Frohse may at times be caused by tension on the arcade produced by taut bands of supinator TrPs. **DIFFERENTIAL DIAGNOSIS** is primarily concerned with the tennis elbow syndrome. In addition to TrPs in the supinator muscle, other TrPs contributing to the symptoms often are found in the nearby hand and finger extensors, the brachioradialis, the distal triceps and occasionally the anconeus muscles. The brachialis, biceps and palmaris longus muscles also may become involved, but do not contribute to the lateral epicondylar pain of tennis elbow. For **TRIGGER POINT RELEASE** of the supinator by spray and stretch, the elbow is extended and the forearm pronated, while the vapocoolant spray is applied upward and around the forearm over the muscle, and then down over the dorsal forearm and thumb. Other manual release techniques are also effective. **TRIGGER POINT INJECTION** of central TrPs is begun by directing the needle into the tender spot overlying the head and neck of the radius in the distal antecubital space. Passive lengthening and active range of motion of the supinator follow injection. **CORRECTIVE ACTIONS** include keeping the wrist dorsiflexed and the elbow slightly bent to prevent strain when playing tennis, applying pressure over the TrPs, and carrying packages with the forearm supinated to transfer the load from the supinator to the biceps brachii and brachialis muscles.

1. REFERRED PAIN
(Fig. 36.1)

Trigger points (TrPs) in the supinator muscle refer pain primarily to the lateral epicondyle and the surrounding lateral aspect of the elbow.[56] They also project spillover pain to the dorsal aspect of the web of the thumb and, if sufficiently intense, the pain may include some of the dorsal forearm[57] (Fig. 36.1).

Kelly[29] reported a patient with tenderness in the region of the most common supinator TrP and in the wrist and finger extensors, with numbness in the thumb and tingling in the index and ring fingers. These symptoms, and additional areas of

tenderness in the lower brachialis and in the volar aspect of the wrist, disappeared when the tender spots in the finger extensor group and the supinator were injected with a local anesthetic. Two other patients[28] had the typical supinator pattern of referred pain to the thumb, with relief by injection in the area of the supinator TrPs.

2. ANATOMY
(Fig. 36.2)

The supinator is a flat muscle, the proximal part of which is divided into two layers. The muscle spirals around the lateral (outer) side of the radius to attach ***proximally*** primarily to the dorsal surface of the ulna, and also to the lateral epicondyle of the humerus, to the lateral and ventral ligaments of the radioulnar joint, and to the anterior capsule of the humeroulnar joint (Fig. 36.2B and C).[10] ***Ventrally*** and ***distally*** the fibers form a "Y" shaped attachment to the volar surface of the radius just distal to the tendon of the biceps brachii (Fig. 36.2A). The bare bone between the arms of the "Y" (Fig. 36.2C) separates the proximal portion of the muscle into superficial and deep layers.[4,52] Distally the muscle is undivided. When the forearm pronates, the supinator muscle and the biceps tendon wrap around the radius like a windlass into the space between the radius and the ulna. The deep radial (posterior interosseous) nerve enters between the superficial and deep layers of the muscle beneath a fibrous archway of variable thickness formed by the superficial layer of the supinator muscle. This archway, especially when thickened, is called the arcade of Frohse.[51,52]

"Supinator longus" is an outmoded name for the brachioradialis, and does not refer to the supinator muscle which was called the supinator brevis. The brachioradialis has very limited supinator function.

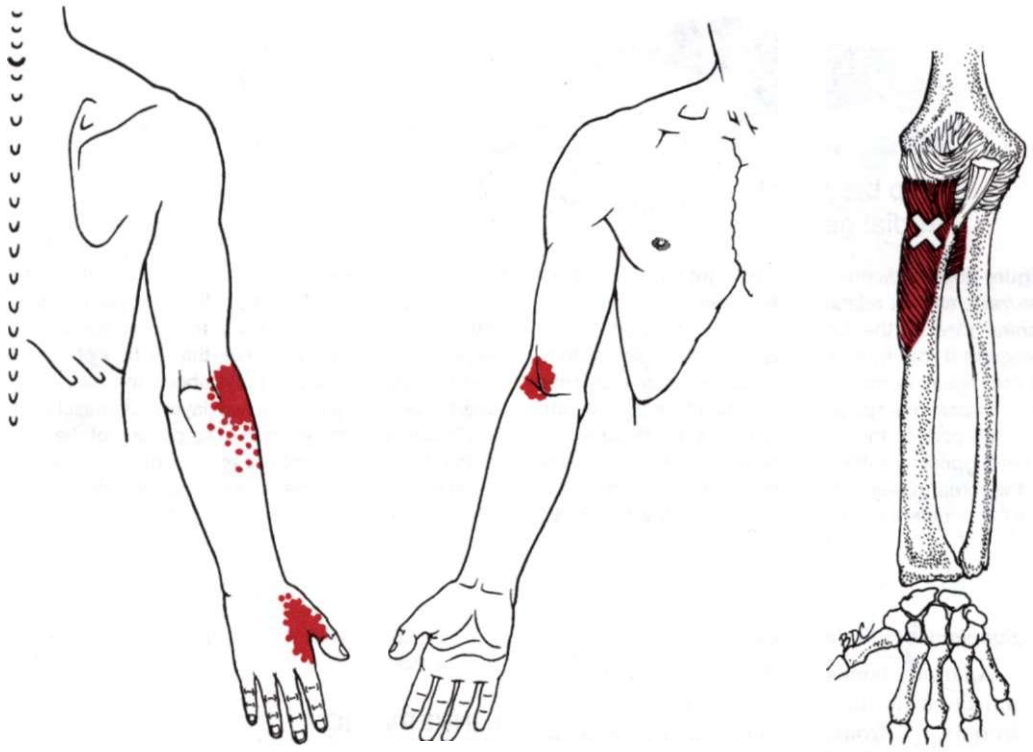


Figure 36.1. Referred pain pattern *(dark red)* of a frequent trigger point (X) in the right supinator muscle.

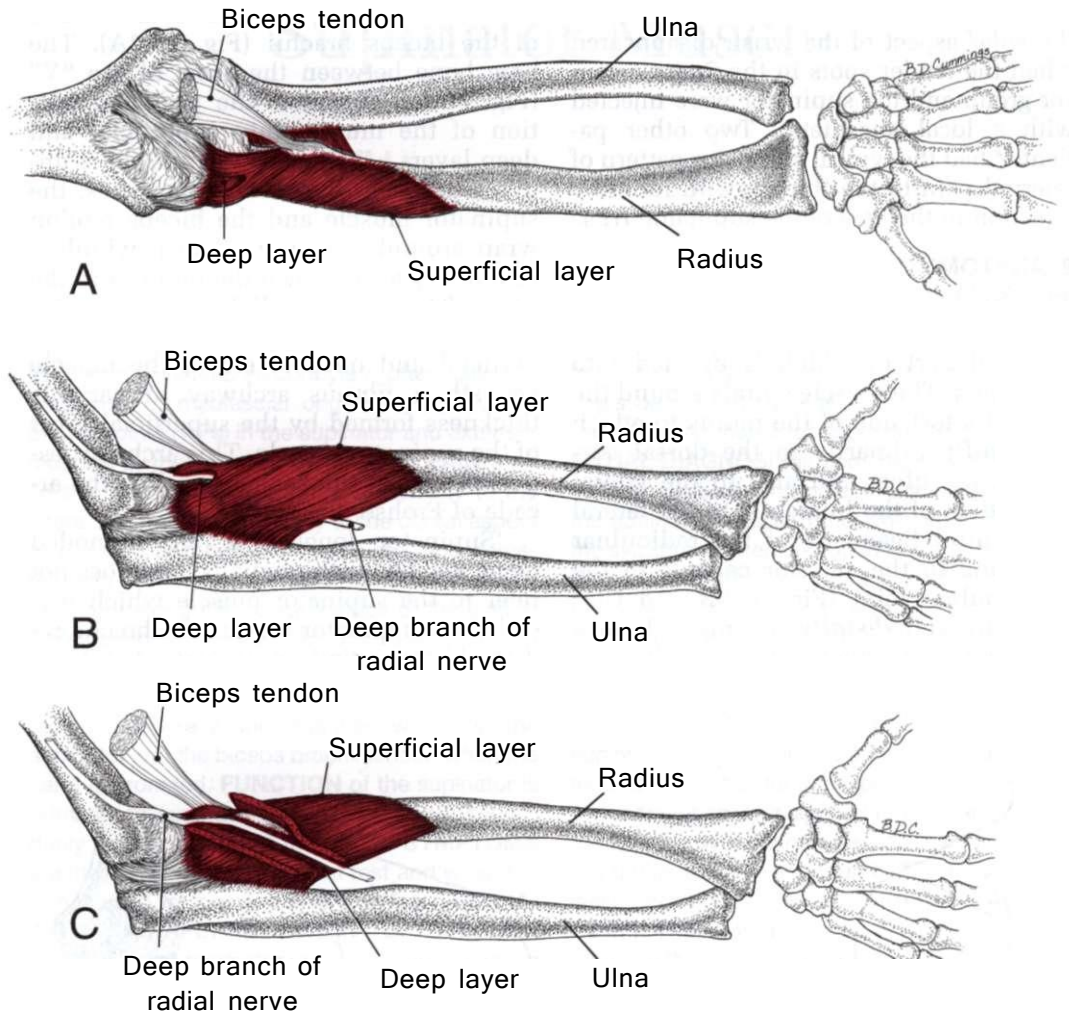


Figure 36.2. Attachments of the right supinator muscle *(red),* and its relation to the deep radial nerve. **A,** *ventral view* of the forearm, hand supinated. In the foreground, the muscle attaches to the volar surface of the radius. In the background, the muscle crosses the interosseous space to its dorsal ulnar attachment. A small part of the deep layer is seen through the arched opening in the superficial layer. **B,** *lateral view* of the forearm, hand in neutral position. The deep radial nerve enters the arched opening in the superficial layer and continues between the two layers of the muscle. C, same view as **B,** with the superficial layer of the muscle reflected to show the deep layer and the nerve. The area of the radius that is free of muscle fiber attachments is seen just above the nerve. This bare bone separates the two layers of muscle and provides space for the nerve. The division of the muscle into two layers does *not* extend into its distal half, where the nerve tunnels through the undivided muscle belly.

Supplemental References

Anatomy books illustrate the supinator muscle from the medial aspect,[33] from in front,[33,35,55] from in front with the radial nerve,[3] from the lateral side,[2,33] from the posterior (dorsal) view,[9, 54] from the posterior view with the deep radial nerve,[13,39] from the medial view,[53] and in cross section.[18,40]

3. INNERVATION

This muscle is supplied primarily by the C_6, partly by the C_5, and sometimes by the C_7 spinal nerves, through the posterior

cord, and finally via the deep (posterior interosseous) branch of the radial nerve.[30] The motor branch to the supinator muscle comes off the posterior interosseous nerve before it enters the muscle."

4. FUNCTION
(Fig. 36.3)

The supinator, as its name implies, is one of the two major supinators of the forearm at the radioulnar joint.[5,17,26,45,58] Supinator activity predominates over biceps activity during unresisted supination of the forearm, and "holds" the forearm in supination.[5, 58] The much stronger biceps assist supination when the forearm is at least slightly flexed at the elbow and when force is needed to overcome resistance to supination.[58] However, the biceps assists very little, if at all, when the elbow is straight. Forceful supination, therefore, requires at least a slight degree of elbow flexion.

Based on electromyography, forceful elbow flexion with the forearm pronated, as in Figure 36.3C, inhibits contraction of the biceps (which is a supinator) and, therefore, tends to load the supinator, brachioradialis and brachialis muscles. Forceful elbow flexion in the supinated position of Figure 36.3A tends to load the biceps and to unload the supinator. The supinator also assists flexion of the forearm at the elbow when the forearm is held intermediate between supination and pronation,[49] as in Figure 36.3B.

The supinator fibers that attach to the anterior capsule of the humeroulnar joint contribute primarily to elbow flexion, rather than to supination; they pass between the deep radial nerve and the biceps tendon. The epicondylar fibers also may contribute to flexion.

5. FUNCTIONAL UNIT

Supination is augmented by the biceps brachii during increased effort. The supinator is synergistic with the forearm flexors, as described in Section 4, above.

The chief antagonist to the supinator is the prime pronator, the pronator quadratus; the secondary antagonist is the pronator teres.[5]

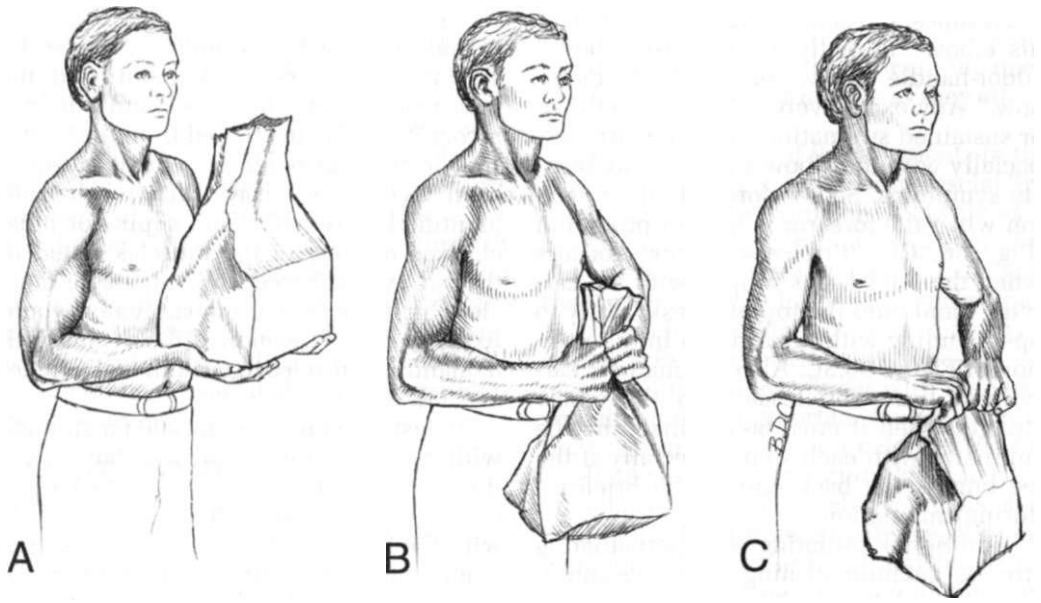


Figure 36.3. Three hand positions for carrying a heavy object with the elbow flexed. **A,** forearms supinated, which loads the biceps brachii and unloads the supinator. **B,** hands in the neutral position, which loads both muscles. **C,** forearms pronated, which tends to unload the biceps and to load the brachialis, brachioradialis, and the few fibers of the supinator that contribute to elbow flexion.

6. SYMPTOMS

Patients with active TrPs in the supinator muscle complain of aching pain in either, or both, the lateral epicondyle and the dorsal surface of the web of the thumb. Pain is caused by activities like carrying a heavy briefcase with the elbow fully extended, playing tennis, and other movements listed under Section 7, below. These patients also are likely to experience continuing elbow pain at rest following such activities. In our experience, nearly every patient with lateral epicondylar pain and tenderness has an active supinator TrP; the supinator is the muscle most frequently contributing to the pain of "tennis elbow."

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Symptoms of "tennis elbow" may occur when the player mis-hits the ball "off-center," twisting the racquet with the elbow completely extended (more likely in backhand, than in forehand strokes). During full elbow extension the biceps cannot assist the supinator to resist the added force. Supinator strain may occur when resisting unexpected pronation or when executing an extremely forceful supination.

At times, the commonly used term "tennis elbow" is really a "briefcase elbow," "door-handle elbow," or "dog-walker's elbow." Any excessively forceful, repetitive, or sustained supination of the forearm, especially with the elbow straight, may initiate symptoms. So can forceful elbow flexion when the forearm is held in pronation (Fig. 36.3C). "Briefcase elbow" occurs when the briefcase is flipped with the carrying hand onto the top of a desk, ready to open, ending with the forearm in the position of Figure 36.3C. Also traumatic is carrying a heavy briefcase with the elbow straight when it must be stabilized by the supinator with each step, especially if the leg bumps the back end of the briefcase during ambulation.

Additional initiating and perpetuating stresses include turning stiff doorknobs, wringing clothes when doing laundry, meticulous ironing, unscrewing a tight jar lid by movement only at the wrist, walking a large dog pulling on a leash, handshaking in long receiving lines at receptions (as by politicians), erasing chalk lines on a blackboard, washing walls by hand, and raking leaves.

8. PATIENT EXAMINATION

The examiner should test for supinator stretch length by simultaneously pronating the forearm and extending it at the elbow. This test can, but does not always, show restriction if the supinator alone harbors TrPs.

Tapping the lateral epicondyle elicits the exquisite tenderness of enthesopathy caused by taut-band tension of the central TrPs. Referred tenderness of the web of the thumb also may be present when the web is squeezed. The combination of epicondylar tenderness and pain at the base of the thumb strongly suggests an active supinator TrP. Thumb motion is usually not restricted and often is not painful. The Hand-to-shoulder-blade Test (see Fig. 22.3) shows slight restriction, and causes pain as described in Section 1, above. A handshake with a firm grip becomes painful when extensor muscles of the wrist and fingers have secondarily developed active TrPs.

Gerwin, *et al.*[21] established that, among experienced and trained examiners, three reliable criteria for diagnosing myofascial TrPs were the detection of a taut band, the presence of spot tenderness, and *patient's recognition* of pain elicited from the tender spot in the taut band. In several muscles, local twitch responses (LTRs) were not identified as reliably. The supinator muscle was not one of the muscles tested in this study, but based on comparable muscles that were tested, the supinator would likely be one of the more difficult and skill-demanding muscles to examine reliably for an LTR.

To test the supinator muscle for strength with minimum interfering assistance from the biceps brachii, have the supine patient extend the elbow along the side of the body with the hand and forearm in a neutral position and resist a supination effort by the patient. Increased muscle tension due to TrPs can be tested by fully supinating the forearm against resistance, looking for painful limitation of full supination.

When a supinator TrP is suspected, the radioulnar (proximal and distal), radiohumeral, and humeroulnar joints should each be tested for normal joint play and if restricted, normal joint play should be restored.[31, 34] The first two joints are most critical for normal supinator function.

9. TRIGGER POINT EXAMINATION

The most frequent location of supinator TrPs is close to the attachment of the superficial layer of the supinator muscle on the ventral aspect of the radius, which, in turn, is just lateral and somewhat distal to the biceps tendon (Fig. 36.1). The brachioradialis is slackened by flexing the elbow slightly (15° to 30°) and this muscle is pushed aside laterally. The forearm is fully supinated, otherwise the TrPs may be hidden by the ulna. In the supinated position, the supinator TrPs lie directly over the radius and immediately beneath the skin between the biceps tendon and the brachioradialis muscle. Both muscular landmarks are readily identified by asking the patient to flex the forearm against resistance. Snapping palpation of very active TrPs may occasionally produce a confirmatory supination twitch response of the hand in spite of the shortened position of the muscle.

A second, deeply situated, supinator attachment TrP also may be found by pressing downward against the ulna on the lateral side of the forearm close to the radius as the muscle approaches its attachment where the lateral joint capsule meets the ulna. This TrP is evidenced by tenderness to *deep* palpation through the mass of the hand extensor muscles, especially through the extensor carpi ulnaris longus, 4 or 5 cm (nearly 2 in) distal to the lateral epicondyle, and 1 or 2 cm (about 3/4 in) distal to the head of the radius. This second TrP is sometimes associated with deep radial nerve entrapment.

10. ENTRAPMENT

Entrapment of the radial nerve as it traverses the supinator muscle may or may not produce symptoms often identified as tennis elbow. The clinician should note that: (1) the painless weakness of muscles supplied by the radial nerve is usually caused by a tumor,[22] (2) a painful tennis elbow without muscular weakness or signs of entrapment (usually diagnosed as tennis elbow or lateral epicondylitis) is often caused by myofascial TrPs without radial nerve compromise, and (3) the mixture of tennis elbow pain and evidence of radial nerve entrapment in the region of the supinator muscle suggests the possibility of both nerve entrapment and supinator TrPs. Tennis elbow is covered in the next section, Differential Diagnosis, and the painful radial nerve entrapment group is considered here.

Surgical reports of radial nerve entrapment make it clear that frequently the problem occurs as the deep radial (posterior interosseus) nerve enters the supinator muscle (Fig. 36.2B and C). An anatomical study showed that the proximal edge of the superficial layer of muscle fibers formed a tendinous thickened border in 30% of 50 "normal" adult arms.[52] Hong, *et al.*[25] found a slightly lower percentage, 2 thickened borders in 10 arms. This fibrous arch is also known as the Arcade of Frohse. The thickened arch was much more common in patients who received an operation for a supinator syndrome than in "normal" arms (10 of 12 patients). The nerve enters the arcade about 1 cm lateral to the biceps tendon. Here, the nerve lies against the anterior capsule of the radiohumeral joint, cushioned slightly by the fibers of the deep layer of the supinator muscle as they attach to the joint capsule.

The descriptions of these patients in the surgical literature suggest a myofascial TrP component of the problem as well as a nerve entrapment component. Thirty two of forty eight surgical patients had been treated previously for lateral epicondylitis,[24] which is commonly caused by myofascial TrPs (see Section 11). Resisted extension of the middle finger with the wrist straight and unsupported caused pain at the origin of the common extensor tendon in all 48 elbows in one study[47] and in 21 of 50 elbows in another study.[24] This sign could have been from an enthesopathy (attachment TrP) of the hand and finger extensor muscles rather than a supinator entrapment. Resisted supination weakness in 26 of 50 elbows[24] also could fit a supinator TrP (the supinator motor nerve branches off

before it enters the muscle[11]). Tenderness over the epicondyle in 43 of 50 elbows[24] could be referred from a supinator TrP.

The most commonly used relatively successful surgical procedure was division of the arcade of Frohse relieving the deep radial nerve of any entrapment pressure[24, 32, 47] plus various additional procedures reported by different authors. Relief obtained in this way plus the observation of nerve indentation by the thickened arcade of Frohse in all 33 elbows in one study[47] and in 34 of 50 elbows in another[24] confirms that some degree of nerve entrapment was occurring at that point. Why this particular anatomical configuration should develop problems well into adulthood after many years without symptoms is not so clear.

This operation should relieve radial nerve entrapment by the muscle, but would not be likely to inactivate any supinator TrPs, which would account for incomplete pain relief by the surgical procedure alone. On the other hand, following surgery, patients have a period of muscle rest that could help spontaneous recovery and they may be more careful of activities involving forceful supination, reducing this perpetuating factor.

One paper analyzed the results of a nonsurgical approach.[25] Posterior interosseous neuropathy was confirmed electrodiagnostically in 15 patients who received unidentified conservative therapy. All recovered within 5 years without surgery. Another author emphasizes that this supinator entrapment is caused purely by soft tissues and does not involve a bony limitation of space.[27]

Supinator TrPs can cause entrapment of the deep radial nerve if those supinator fibers that are attached to an arcade with a thick tendinous edge are shortened by activity of the deep supinator attachment TrP (described in the previous section) and create tension on the arcade of Frohse. That this TrP lies close to the nerve is evidenced by occasional temporary local anesthetic block of the nerve when that TrP is injected.

Clinically we find that inactivation of all local myofascial TrPs relieves the pain, and inactivation of the supinator TrP on the ulnar side of the nerve usually relieves the deep radial nerve entrapment, without surgical intervention. Patients with a well developed arcade may be more vulnerable to entrapment of the radial nerve by supinator TrPs.

Not one paper could be located that reported systematic examination of patients with this entrapment for TrPs and evaluated the results of releasing the TrPs. Competent research studies of this type are sorely needed.

11. DIFFERENTIAL DIAGNOSIS

Differential diagnoses of the symptoms caused by supinator TrPs include tennis elbow or lateral epicondylitis, entrapment of the posterior interosseous nerve, C_5-C_6 radiculopathy, and DeQuervain's stenosing tenosynovitis. Tennis elbow is covered in detail in this section.

Frequently, recurring articular dysfunction at the distal radioulnar joint is associated with TrPs in the supinator muscle.

Arthritis of either articulation at the elbow is a possible, but unlikely, cause of pain localized to the lateral epicondyle. It should be diagnosed by radiologic examination.

Tennis Elbow (Lateral Epicondylitis or Radial Epicondylalgia)

Tennis elbow is a common disease entity that plagues a large proportion of the athletic population[41] and 40% to 50% of recreational tennis players.[46] It occurs primarily in those between 30 and 55 years of age.[20] The literature on this subject omits a convincing explanation for the symptoms, which suggests that a major cause may have been overlooked. The observation that a latent TrP is found in the third finger extensor in most mature adults may be relevant. Recognition of the contribution by myofascial TrPs should help greatly to provide the missing explanation. It is now generally accepted that the symptoms are caused by repetitive microtrauma to the musculotendinous unit causing inflammatory and degenerative tissue damage.[43] This description fits the enthesopathy of attachment TrPs that result from the chronic tension of taut bands of central TrPs.

The symptoms of tennis elbow can come from TrPs in the supinator and/or hand and finger extensor muscles. As reviewed above, if the supinator is involved,

the diagnosis may be entrapment of the radial nerve as it enters that muscle and is commonly approached by surgical release of the arcade of Frohse and division of the superficial portion of the supinator muscle. When the extensor muscles attached to the lateral condyle are involved, their attachment is often severed surgically to relieve the TrP tension that often causes the lateral condyle enthesopathy. The surgery in both situations may be unnecessary if the responsible TrPs were identified and inactivated.

Conservative (Nonoperative) Treatment. Cyriax[15] identified four varieties of "tennis elbow." The *tenoperiosteal* variety was explained as a partial tear at the ligamentous attachment of the hand and finger extensor muscles to the lateral epicondyle, which produced a painful scar[35] and could correspond to attachment TrPs of these muscles. This was treated with local triamcinolone injection and complete rest of the upper extremity for a week. The *muscular* variety required injection of 0.5% procaine solution precisely into the tender point in the "extensor carpi radialis" belly, corresponding to central TrPs of that muscle. The *tendinous* variety was described as a lesion in the "body of the tendon," (presumably the common extensor tendon) at the level of the head of the radius. Surgical exploration of this area with removal of tissue revealed microscopic rupture of the origin of the extensor carpi radialis brevis with abortive regeneration.[37] It was treated with four to eight sessions of massage and could correspond to attachment TrPs of that muscle. The *supracondylar* variety displayed a tender point along the supracondylar ridge above the lateral epicondyle at the origin of the extensor carpi radialis longus muscle. It, too, was relieved by deep massage and would be compatible with attachment TrPs of that muscle.

In two studies, most of the patients with tennis elbow responded well to conservative treatment and did not require surgery (82% of 339 patients[14] and 96% of 871 patients).[19] In the series that identified the conservative measures, the authors limited use of the hand to avoid painful activities, applied a dorsiflexion wrist splint, and gave local injections of steroids directly into the most tender area over the origin of the flexor or extensor muscles.[14]

The use of a nonelastic fiber arm band that is lined with foam rubber to prevent slippage was sufficient to relieve symptoms in 12 of 40 patients. The remaining 28 required injection of steroid and local anesthetic into the tender tissues distal to the lateral epicondyle (TrPs not mentioned). The band was adjusted to be snug and comfortable with the forearm muscles relaxed.[19] This technique is useful until the responsible TrPs have been inactivated. The skin pressure seems to reduce the intensity of TrP activity, similar to the technique described for the sternocleidomastoid muscle.

An analysis of 12 reasonably well-designed studies on corticosteroid injection for lateral epicondylitis,[1] concluded that corticoid injections appear to be relatively safe and seem to be effective in short term (2-6 weeks). The limited time of effectiveness can be explained by treatment only of the attachment TrPs at the site of enthesopathy but neglecting inactivation of the responsible central TrPs. The controlled study of 109 patients by Solveborn, *et al.*[50] is a good example of this short-term response to steroid and analgesic injection of only the region of enthesopathy.

Surgical Treatment. Garden[20] reported good results in 44 of 50 elbows by tenotomy of the extensor carpi radialis brevis or Z-lengthening its tendon. Bosworth[7] reported good results in most of 62 patients by releasing the common extensor origin at the epicondyle in combination with a variety of other procedures. Surgery of varying extensiveness for "tennis elbow" is reported enthusiastically, including excision of the proximal attachment of the extensor carpi radialis brevis,[37] a medio-lateral incision to the bone in the tender area through a stab wound,[35] division of the deep fascia that covers the extensor group of muscles distal to the epicondyle,[42] surgical release of the common origin of the radial hand extensors,[48] and extensive removal of tendinous and joint tissue in the painful area.[8]

The common denominator of these surgical approaches is release of hand extensor, and sometimes finger extensor tendons. No research could be found that critically examined how frequently the surgery for tennis elbow might be unnecessary if the TrPs responsible for the enthesopathy were assessed and inactivated.

Etiology. Tennis elbow is commonly attributed to microtrauma to the musculotendinous unit with degenerative and inflammatory changes[38] at the origin of the extensor carpi radialis brevis produced by repeated large impact forces created when the ball hits the racquet in a backhand stroke.[46] Mechanical analysis does not support this theory. Rather, this muscle is heavily loaded (40%-70% of voluntary contraction) throughout active play.[46] The pathology fits an overload-induced myofascial TrP etiology well. This microtraumatic origin was further substantiated by a recent study[43] that showed a strong positive correlation between magnetic resonance imaging and histopathological changes of neovascularization, disruption of collagen, and mucoid degeneration without inflammation. This histological picture is compatible with enthesopathy secondary to taut bands of central TrPs in forearm muscles.

Related Trigger Points

With the "tennis elbow" symptoms of pain and tenderness in the region of the lateral epicondyle, TrPs are often found also in the triceps brachii, in the lower end of the lateral margin of its medial head (TrP_2), in the long extensors of the fingers, the extensor carpi radialis longus and brevis, and the brachioradialis muscles. When all of these TrPs have been eliminated, an anconeus TrP may still cause lateral epicondylar pain and tenderness to tapping.

Additional muscles that may become involved as part of the supinator's functional unit, but which do not refer pain to the lateral epicondyle, are the brachialis, biceps brachii (TrPs in the distal third of the muscle), and sometimes the palmaris longus.

12. TRIGGER POINT RELEASE (Fig. 36.4)

The stretch and release techniques described below and in Chapter 3, Section 12 are applicable to *central* trigger points (TrPs) in the supinator muscle. The primary therapeutic approach to *attachment* TrPs is to inactivate the *central* TrPs that are causing them by treating the muscle in a position of ease (not stretched) using TrP pressure release, postisometric relaxation, and indirect techniques.

For application of **stretch and spray,** the patient's elbow rests on a padded armrest, or over the operator's knee. This support permits full elbow extension. As the forearm is fully pronated to take up any slack in the supinator muscle, the elbow is supported to prevent medial rotation at the shoulder. After several initial sweeps of spray, gentle pressure is applied to follow release of muscle tension as the vapocoolant spray is again swept upward and outward diagonally over the forearm following the muscle fibers from the TrP area to the region of the lateral epicondyle (Fig. 36.4A). Then, downsweeps are applied over the dorsum of the forearm and web of the thumb (Fig. 36.4B).

As part of the stretch-and-spray procedure, tension should be released in adjacent muscles likely to have developed associated TrPs, and which are likely to protest painfully after release of the supinator. Stretch and spray are applied to the biceps and brachialis, brachioradialis, extensor carpi radialis, and triceps muscles and to any associated TrPs in the finger extensors.

Trigger point pressure release is applied by gently holding TrPs in the taut supinator muscle against the underlying radius. It is most effective when combined with spray and stretch. This procedure is followed by three slow cycles of *full* active supination and pronation of the forearm. Moist heat is applied promptly to treated regions.

Following successful treatment, the elbow should straighten to a degree of extension not obtainable before, and the Hand-to-shoulder-blade Test (*see* Fig. 22.3) should be normal if no other TrPs are restricting it. *All* tenderness to tapping on the lateral epicondyle should be gone. If not, residual TrPs may remain in the supinator, or in nearby muscles, especially the anconeus and triceps.

Supinator central TrPs respond well to **postisometric relaxation.** This technique for the supinator was well described and illustrated by Lewit.[31] It is also valuable as a self-treatment at home.

For more rapid relief of attachment TrPs, **iontophoresis or phonophoresis** of a steroid may be considered, *after* the central TrPs have been inactivated by techniques (such as the ones mentioned in the first paragraph of this section) that put minimal stress on the muscle's attachments.

13. TRIGGER POINT INJECTION (Fig. 36.5)

For injection of central trigger points (TrPs) in the midfiber part of the muscle (Fig. 36.1, *right side)* the patient is placed in the same position as for examination, and a 22- to 27-gauge, 3.8-cm (1 1/2-in) needle is directed proximally into the TrP just lateral to the attachment of the biceps brachii tendon, where maximum tenderness is found in a nodule on palpation (Fig. 36.5); it is often difficult to see or feel an LTR in this muscle except through the needle. When the forearm is supinated, the deep radial nerve passes through the muscle lateral to this TrP area (Fig. 36.2B and C) and, thus, is not usually encountered during TrP injection.

Rachlin[44] illustrates injection of the central TrP region in this muscle.

Injection of attachment TrPs (located at the musculotendinous junction close to the joint capsule) with analgesic can expedite resolution of the local tenderness and the return to full function after the corresponding central TrPs have been inactivated.

It is wise to probe the tender area thoroughly, searching for all of the sensitized locations in the TrP. No focal TrP tenderness should remain after the injection.

Injection is followed promptly by stretch and spray and then by active pronation and supination to restore full normal muscle length. The elbow area is then rewarmed with a hot pack. The injection and stretch can be repeated in a few days, if necessary.

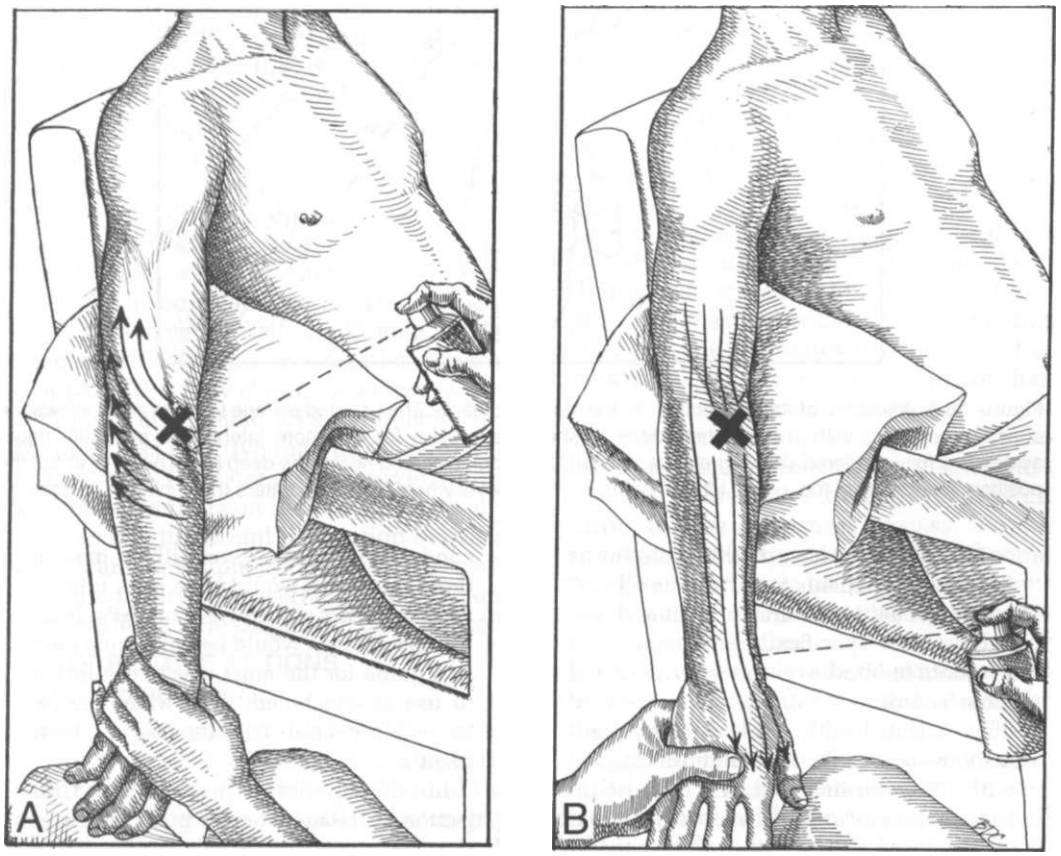


Figure 36.4. Stretch position and spray pattern (*arrows)* for trigger points (**Xs**) in the supinator. **A,** the elbow must be supported to maintain effective elbow extension and pronation of the forearm. The operator blocks medial rotation at the shoulder by resting the medial epicondyle against the elbow support. The upsweep spray pattern covers the muscle and lateral epicondyle. **B,** the down-sweep spray pattern covers the muscle and its referred pain pattern on the forearm and at the base of the thumb, dorsally.

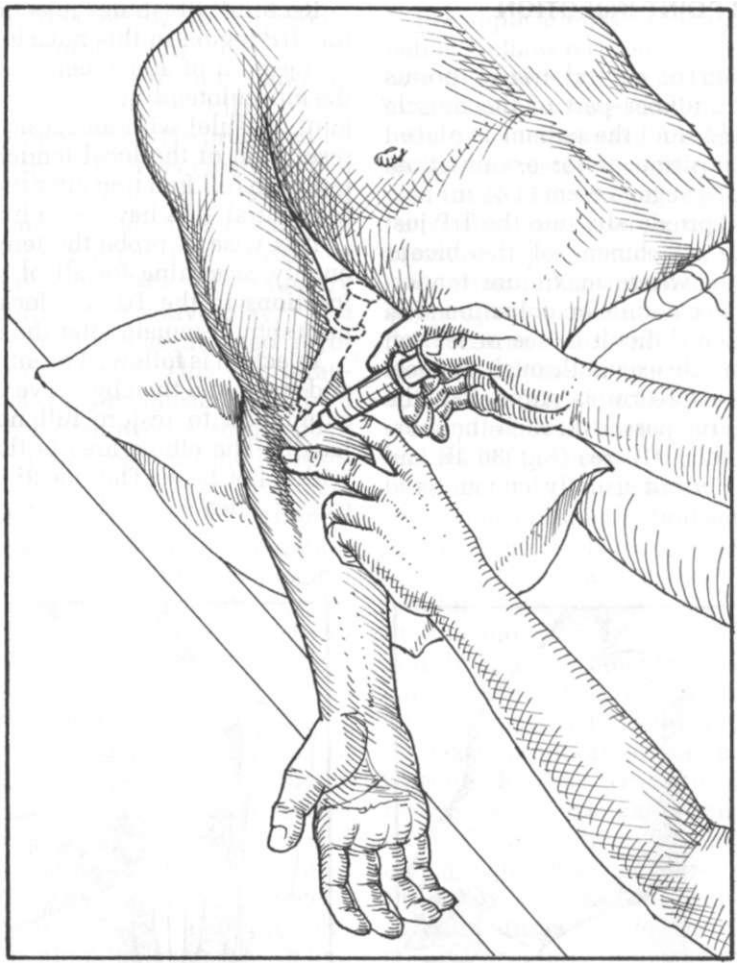


Figure 36.5. Injection of trigger points in the right supinator muscle, with the brachioradialis pushed aside. The fully rendered drawing shows the syringe position for injecting the usual trigger point in this muscle. The ghosted syringe *(dashed lines)* shows the approach for the more lateral and proximal trigger point located nearer the deep radial nerve at about the level where the nerve enters the supinator muscle.

To assess the effect of what solution is injected, 95 patients with "tennis elbow" were injected in the area of pain and tenderness (not specifically supinator TrPs) in a double-blind experiment with one of three solutions.[16] Ninety-two percent of those injected with 1 ml of methylprednisolone acetonide, 20% of those injected with 1% Xylocaine, and 24% of those injected with 0.9% saline were either cured or improved.[16] Thus, the corticosteroid was much more effective in this study. The most likely site of these injections would have been in the region of extensor carpi radialis brevis enthesopathy close to the condyle (often identified as epicondylitis). Therefore, very likely, most of these injections would have been into attachment TrPs and not central TrPs. If so, the conclusions would be applicable only as a guide for the most effective solution to use at attachment TrPs when the responsible central TrP site has not been identified and treated.

In other studies of "tennis elbow," injection of the most tender point with corticosteroid and lidocaine together was effective in more than half of 202 cases;[6] injection of triamcinolone acetate alone afforded relief in 66% of patients.[36] The relatively good immediate results with steroid injection would be expected at at-

tachment TrPs, but the results were short in duration[1] (which would also be expected if the central TrPs were neglected).

Effective elimination of central TrP activity by direct injection with a local anesthetic or saline requires precise targeting of the TrPs so that needle contact elicits a local twitch response or a pattern of pain recognized as familiar to the patient. With needle penetration of central TrPs, we see no advantage and some disadvantage to adding steroid in the injection solution. However, in many cases, the addition of steroid may be appropriate for *attachment* TrP injections.

It is becoming clear that the taut bands caused by TrPs in the forearm extensor muscles place a chronic strain on their tendinous attachments at the lateral epicondyle, producing enthesopathy that eventually could produce the structural changes previously described in Section 11. Inactivation of the responsible TrPs would seem to be a simpler initial approach than surgery, and has been found to be effective clinically. A well-controlled prospective research study of the TrP origin of tennis elbow is urgently needed.

"Tennis elbow" syndromes also have been treated with acupuncture at motor points,[23] The endplate zone of motor points is also where central TrPs are located. To the extent that the acupuncture needles are used to impale TrPs, they should be effective. Dry needling of attachment TrPs is probably considerably less effective than dry needling of central TrPs. The lateral epicondyle would be a convenient region in which to critically evaluate this important distinction between central and attachment TrPs with a well-controlled, blinded research study and careful definition of the diagnostic criteria used.

14. CORRECTIVE ACTIONS (Figs. 36.6 and 36.7)

Tennis players should keep the wrist slightly extended and the elbow slightly bent (Fig. 36.6A). Allowing the head of the racquet to drop (Fig. 36.6B) reduces grip strength. With slight extension and no ulnar deviation of the hand at the wrist, the increase in strength protects the supinator from mis-hit overload and is easily demonstrated on a grip-strength meter. Slight extension places the forearm flexors at some mechanical advantage. Ulnar deviation places the ring and little finger flexors at a mechanical disadvantage. The bent elbow provides biceps assistance in supination and helps to prevent supinator overload. The two-handed backhand stroke protects the supinator by preventing complete elbow extension during the stroke. Tennis players who use a two-handed backhand have much less trouble with tennis elbow.[46]

If the player still has difficulty with the racket slipping in the hand because the grip is weak, the size of the racquet handle should be reduced so that the fingers wrap fully around it. Otherwise, the extensors, especially those of the ring and little fingers, which are essential for a strong grip, function at a disadvantage. A weak grip permits the racquet to turn in the hand when the ball is mis-hit off-center, causing sudden muscle strain. The additional effort required to keep a tight grip on a large handle further strains the finger extensors.

Elbow pain often begins when a person gets a new racquet that is too heavy, that has a larger handle, or is unbalanced and is too heavy at the head end. The position of the grip on the racquet may be shortened to reduce the length of the lever arm against which the forearm muscles must operate.

Tennis players with this elbow problem should not play on consecutive days, but should rest the supinator muscle until the postexercise soreness from overuse has worn off, usually in a day or two.

A snug figure-8 elastic support may be worn that encompasses the muscles just above and below the elbow, but leaves an opening for the olecranon process. Such a support is sold in sporting goods and some drug stores, and may be worn during tennis, gardening, etc. This provides supporting counter pressure over the supinator and other vulnerable elbow muscles, and discourages full elbow extension. A similar solution was described by Froimson.[19]

A patient with "briefcase elbow" may find it better to carry the briefcase tucked under the arm with the elbow bent, and should avoid flipping the briefcase onto the desk; it should be placed there in two steps: (1) lift it on top of the desk and (2) with *two* hands, lay it flat for opening.

For some activities, wrist-rotation stress may be avoided temporarily by using the

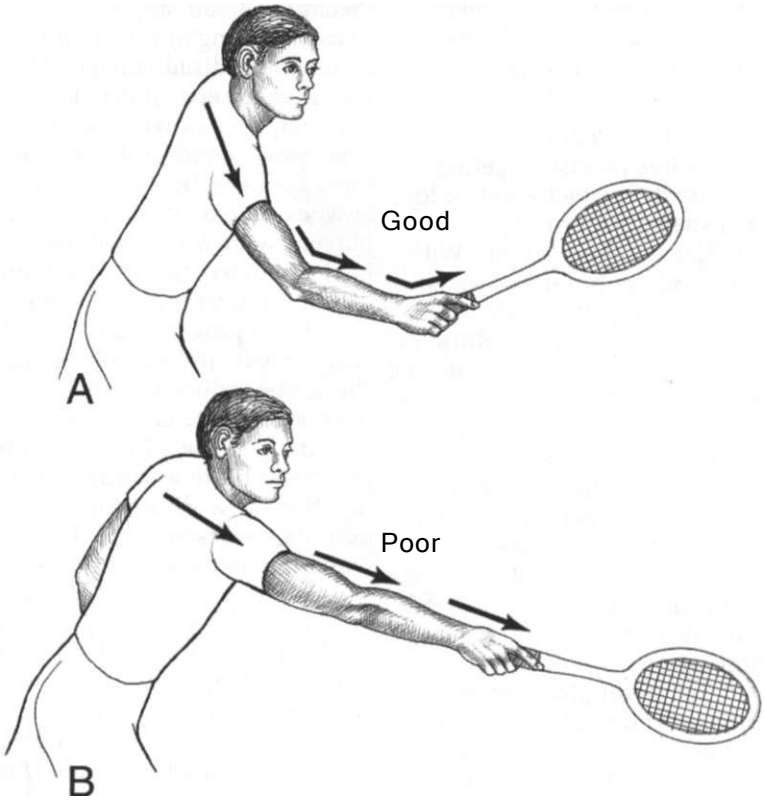


Figure 36.6. Use and misuse of the tennis racquet (backhand stroke). **A,** good position. The elbow is slightly bent and the wrist cocked in radial extension to raise the head of the racquet. **B,** poor position. The elbow is straight and the wrist dropped, which overloads the supinator muscle during supination at the end of the stroke and weakens the grip,

other hand or by using the affected hand differently. Instead of wringing washed clothes, they may be pressed against the bottom of the sink to drain the water from them. Raking leaves and walking a large dog that pulls on a leash should be discontinued. If shaking hands in a receiving line is unavoidable, the right and left hands are alternated from person to person, reaching across with the opposite arm to the next guest in line; the hand is presented with the *palm up* to gracefully avoid a friendly crushing hand grip.

The patient with supinator TrPs should learn to carry packages with the forearms supinated (Fig. 36.3A) rather than pronated (Fig. 36.3C); this substitutes the biceps for the supinator as an assistant to the brachialis to flex the elbow when lifting loads. The biceps is much stronger than the supinator for this purpose.

For a strengthening and conditioning isotonic exercise, the forearm is alternately supinated and pronated, holding a weight (Fig. 36.7). A progressive program increases the weight of the object as strength improves. This exercise is started after the elbow pain and soreness due to TrPs have subsided. It increases the supinator's tolerance to future activity.

15. CASE REPORTS

Dr. Travell saw a patient who presented with a right "tennis elbow." Six months before, the patient had been out on skis, using ski poles, and that same day he had played several hours of paddle tennis, when he noticed elbow pain.

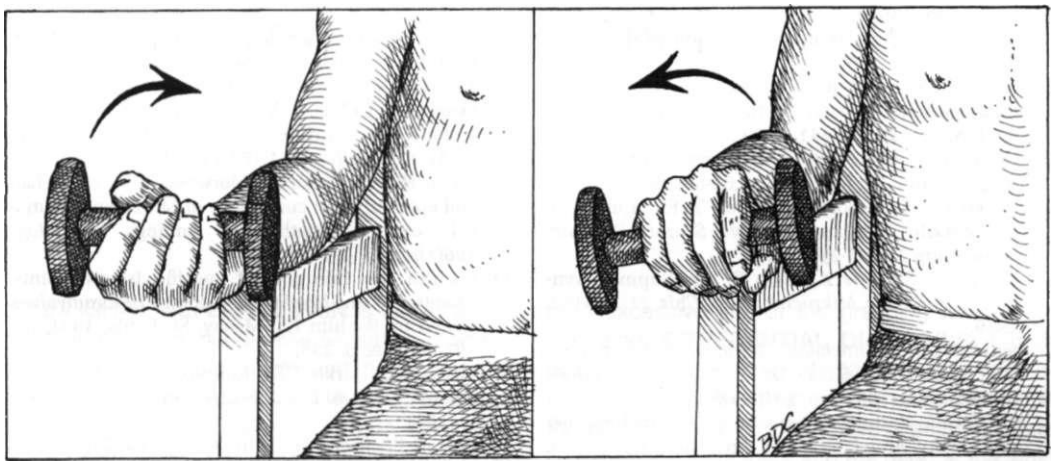


Figure 36.7. Isotonic exercise to strengthen and condition the right supinator muscle. The forearm is alternately supinated *(left)* and pronated *(right)* while holding a weight, which is increased progressively as strength improves.

He had previously received the usual treatment and several injections into the olecranon bursa. The syndrome was a major calamity to the patient, because it had "changed his life style." He could not play tennis, or swing an axe, or run his power saw. He was rapidly learning to become left-handed. On examination, he had the expected myofascial TrPs: the worst was in the supinator, next was the brachioradialis, followed by the 3rd and 4th finger long extensors, and finally, the brachialis. The triceps was not involved. Trigger points in two muscles referred pain directly to the lateral epicondyle: the supinator and the 4th finger extensor, as was expected. Other muscles had the usual TrPs, which Dr. Travell described in advance, to the patient's amazement. Following injection of TrPs with 0.5% procaine, the patient's symptoms and the referred tenderness felt on tapping the epicondyle were completely eliminated.

REFERENCES

1. Assendelft WJ, Hay EM, Adshead R, *et al:* Corticosteroid injections for lateral epicondylitis: a systematic overview. *Br J Gen Pract 46(405j:209-2W,* 1996.
2. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:434 (Fig. 6.114).
3. *Ibid.* p. 415 (Fig. 6.84).
4. Bardeen CR: The musculature, Sect. 5. In: *Morris's Human Anatomy,* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (p. 426).
5. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 284-286, 290, 292-294).
6. Bernhang AM: The many causes of tennis elbow. *NY State J Med* 79:1363-1366, 1979.
7. Bosworth DM: Surgical treatment of tennis elbow. *J Bone Joint Surg 47 A* 1:1533-1536, 1965.
8. Bowden BW: Tennis elbow. *J Am Orthop Assoc* 78:97-98, 101-102, 1978.
9. Clemente CD: *Gray's Anatomy.* Ed. 31. Lea & Febiger, Philadelphia, 1985 (p. 539).
10. *Ibid.* (pp. 538, 539).
11. *Ibid.* (p. 1221).
12. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 77).
13. *Ibid.* (Fig. 78).
14. Coonrad RW, Hooper WR: Tennis elbow: its course, natural history, conservative and surgical management. *J Bone Joint Surg 55A(6):1177-1187,* 1973.
15. Cyriax J: *Textbook of Orthopaedic Medicine.* Ed. 5, Vol. 1. Williams & Wilkins, Baltimore, 1969 (pp. 312-316).
16. Day BH, Govindasamy N, Patnaik R: Corticosteroid injections in the treatment of tennis elbow. *Practitioner* 220:459-462, 1978.
17. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 99, 100).
18. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 83,84).
19. Froimson AI: Treatment of tennis elbow with forearm support band. *J Bone Joint Surg 53A(1):*183-184,1971.
20. Garden RS: Tennis elbow. *J Bone Joint Surg 43B(1):*100-106, 1961.

21. Gerwin RD, Shannon S, Hong CZ, *et al:* Interrater reliability in myofascial trigger point examination. Pain 69:65-73, 1997.
22. Goldman S, Honet JC, Sobel R, *et al:* Posterior interosseous nerve palsy in the absence of trauma. *Arch Neurol* 23:435-441, 1969.
23. Gunn CC, Milbrandt WE: Tennis elbow and acupuncture. *Am J Acupunc* 5:61-66, 1977.
24. Hagert CG, Lundborg G, Hansen T: Entrapment of the posterior interosseous nerve. *Scand J Plast Re-constr Surg* 22:205-212, 1977.
25. Hong VG, Steffens K, Koob E: Das Supinatorsyn-drom. *Handchir Mikrochir Plast Chir* 23:147-152, 1989.
26. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 141, 142).
27. Kaplan PE: Posterior interosseous neuropathies: natural history. *Arch Phys Med Behabil* 65:399-400,1984.
28. Kelly M: Pain in the forearm and hand due to muscular lesions. *Med J Aust* 2:185-188,1944 (Cases 1 and4).
29. Kelly M: The nature of fibrositis. I. The myalgic lesion and its secondary effects: a reflex theory. *Ann Rheum Dis 5:1-7,* 1945 (p. 3, Case 1).
30. Kendall FP, McCreary EK, Provance PG: *Muscles, Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 264, 389).
31. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heine-mann, Oxford, 1991 (pp. 149, 150, 200).
32. Lister GD, Belsole RB, Kleinert HE: The radial tunnel syndrome. */Hand Surg* 4:52-59, 1979.
33. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 136).
34. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964 (p. 68).
35. Murtagh JE: Tennis elbow: description and treatment. *Aust Fam Physician* 7:1307-1310, 1978.
36. Nevelbs AB: The treatment of tennis elbow with tri-amcinolone acetonide. *Curr Med Res Opin* 6:507-509, 1980.
37. Nirschl RP, Pettrone FA: Tennis elbow: the surgical treatment of lateral epicondylitis. *J Bone Joint Surg* 61A:832-839, 1979.
38. Ollivierre CO, Nirschl RP: Tennis elbow. Current concepts of treatment and rehabilitation. *Sports Med* 22(2).133-139, 1996.
39. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2, W.B. Saunders, Philadelphia, 1964 (Fig. 79).
40. *Ibid.* (Fig. 81).
41. Plancher KD, Halbrecht J, Lourie GM: Medial and lateral epicondylitis in the athlete. *Clin Sport Med* 15(2):283-305, 1996.
42. Posch JN, Goldberg VM, Larrey R: Extensor fas-ciotomy for tennis elbow: a long-term follow-up study. *Clin Orthop* 335:179-182, 1978.
43. Potter HG, Hannafin JA, Morwessel RM, *et al:* Lateral epicondylitis: correlation of MR imaging, surgical, and histopathologic findings. *Radiology 196(1):43-46,* 1995.
44. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (see p. 336).
45. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (p. 187).
46. Roetert EP, Brody H, Dillman CJ, *et al.:* The biome-chanics of tennis elbow. An integrated approach. *Clin Sport Med 14(l):47-57,* 1995.
47. Roles NC, Maudsley RH: Radial tunnel syndrome: resistant tennis elbow as a nerve entrapment. *J* Bone *Joint Surg* 54B(3).499-508, 1972.
48. Rosen MJ, Duffy FP, Miller EH, *et al.:* Tennis elbow syndrome: results of the "lateral release" procedure. *Ohio State Med J* 76:103-109, 1980.
49. Simons DG, Travell JG: Unpublished data, 1979.
50. Solveborn SA, Buch F, Mallmin H, *et al:* Cortisone injection with anesthetic additives for radial epi-condylalgia (tennis elbow). *Clin Orthop* 336:99-105, 1995.
51. Spinner M: The Arcade of Frohse and its relationship to posterior interosseous nerve paralysis. *J Bone Joint Surg* 50B(4);809-812, 1968.
52. Spinner M: *Injuries to the Major Branches of Peripheral Nerves of the Forearm.* Ed. 2. W.B. Saunders, Philadelphia, 1978 (pp. 80-94).
53. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 324).
54. *Ibid.* (p. 328).
55. *Ibid.* (pp. 321, 327).
56. Travell J: Basis for the multiple uses of local block of somatic trigger areas (procaine infiltration and ethyl chloride spray). *Miss Valley Med J* 73:12-21,1949 (p. 18, Fig. 4).
57. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 33:425-434,1952 (p. 428, Fig. 6).
58. Travill A, Basmajian JV: Electromyography of the supinators of the forearm. *Anat Rec* 339:557-560, 1961.

CHAPTER 37
Palmaris Longus Muscle

HIGHLIGHTS: **REFERRED PAIN** is felt as a distinctive, prickling, needle-like sensation over the palm. Tenderness of the palm and the progression of contracture are frequently relieved when trigger points (TrPs) in the palmaris longus are inactivated. **ANATOMY:** this highly variable muscle attaches proximally to the medial epicondyle of the humerus and distally to the palmar fascia. **FUNCTION** is chiefly to cup the palm, and it also can assist flexion of the hand at the wrist. **SYMPTOMS** are pain and tenderness in the palm that interfere with the use of tools. Contracture of the palmar fascia also may be present. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** occur from excessive use of the grasping function of the hand. **DIFFERENTIAL DIAGNOSIS:** trigger points in this muscle are identified by their distinctive prickling rather than aching pain. **TRIGGER POINT RELEASE** of the muscle is accomplished by extending the fingers and the hand at the wrist while applying the spray in a distal pattern. This treatment can be effectively supplemented with trigger point pressure release of the palmaris longus TrPs. **TRIGGER POINT INJECTION** is relatively simple using flat palpation to localize the TrPs. **CORRECTIVE ACTIONS** entail avoiding activities that overload the palmar cupping function or that traumatize the palm.

1. REFERRED PAIN
(Fig. 37.1)

Like another muscle, the platysma, which also acts primarily on cutaneous tissue, the trigger points (TrPs) in the palmaris longus refer a superficial, needle-like prickling pain rather than the deep-tissue aching pain of most other muscles. The referred pain pattern centers in the palm (Fig. 37.1). It extends to the base of the thumb and to the distal crease of the palm, but not into the digits. The prickling sensation feels as if it is produced by many fine needles. The spillover pattern may extend to the distal volar forearm.

2. ANATOMY
(Fig. 37.2)

The palmaris longus arises ***above*** chiefly from the medial epicondyle of the humerus, and inserts ***below*** into the triangular palmar aponeurosis and the transverse carpal ligament (Fig. 37.2). At the wrist, its tendon passes superficial to the flexor retinaculum. The tendon stands out clearly when the hand is actively flexed and the palm cupped, because the tendon ends in the hand as the palmar aponeurosis (see Fig. 37.3).[22]

Normally, the palmaris longus is a slender fusiform muscle with its belly located in the proximal half of the forearm between the flexor carpi radialis and the flexor carpi ulnaris muscles. It overlies the flexor digitorum superficialis; however, it is anatomically highly variable. Variations include congenital absence (often bilateral), a distally placed muscle belly, a double-bellied muscle, and a distally placed anomalous muscle that may show a variety of attachments.[10,34] The incidence of total absence ranges from 12.7%-20.4% in studies of occidental and black persons, but only from 2.2%-3.4% in Orientals. Bilateral absence is nearly twice as common as the absence of just one muscle. Either the right or left muscle is equally likely to be missing.[34] Absence is slightly more common in females than males and in whites than blacks. It may be inherited as a sex-linked dominant trait.[34] Anomalies other than absence occur in approximately 9% of individuals.

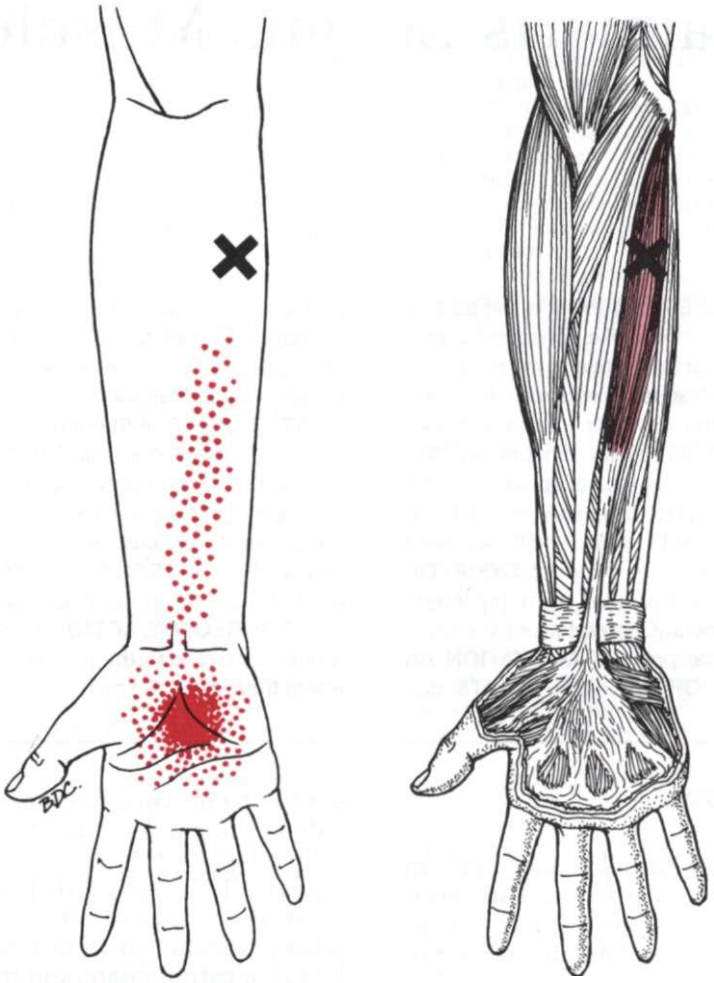


Figure 37.1. Patterns of the referred prickling sensation *(dark red)* arising from a central trigger point (**X**) in a right palmaris longus muscle *(light red)* in its usual configuration. The referred sensation is described as a superficial painful prickle, rather than an aching pain. The belly of this variable muscle, and therefore its trigger points, may lie high or low in the forearm.

The palmar aponeurosis comprises two layers. A superficial layer of longitudinal fibers extends directly from the palmaris longus tendon at the wrist to the fingers. There, the fibers fan out in bundles to cover the flexor tendons of each finger and often of the thumb. Some of the superficial fibers attach to the skin of the flexor crease at the base of the fingers. Others continue into the digits to merge with the digital sheaths. The rest of the distal superficial fibers arch as bands transversely across the underlying tendons and muscles. The deep layer, which consists mainly of transverse fibers, blends with the transverse metacarpal and transverse palmar ligaments. The fibers of the two layers of aponeurosis intertwine.[2]

Two cases of what appeared to be carpal tunnel syndrome were found to have a variation of the palmaris longus in which the tendon passed beneath, rather than above, the volar carpal ligament.[7] Three other cases proved to have anomalous distal bellies of the palmaris longus which compressed the median nerve against the

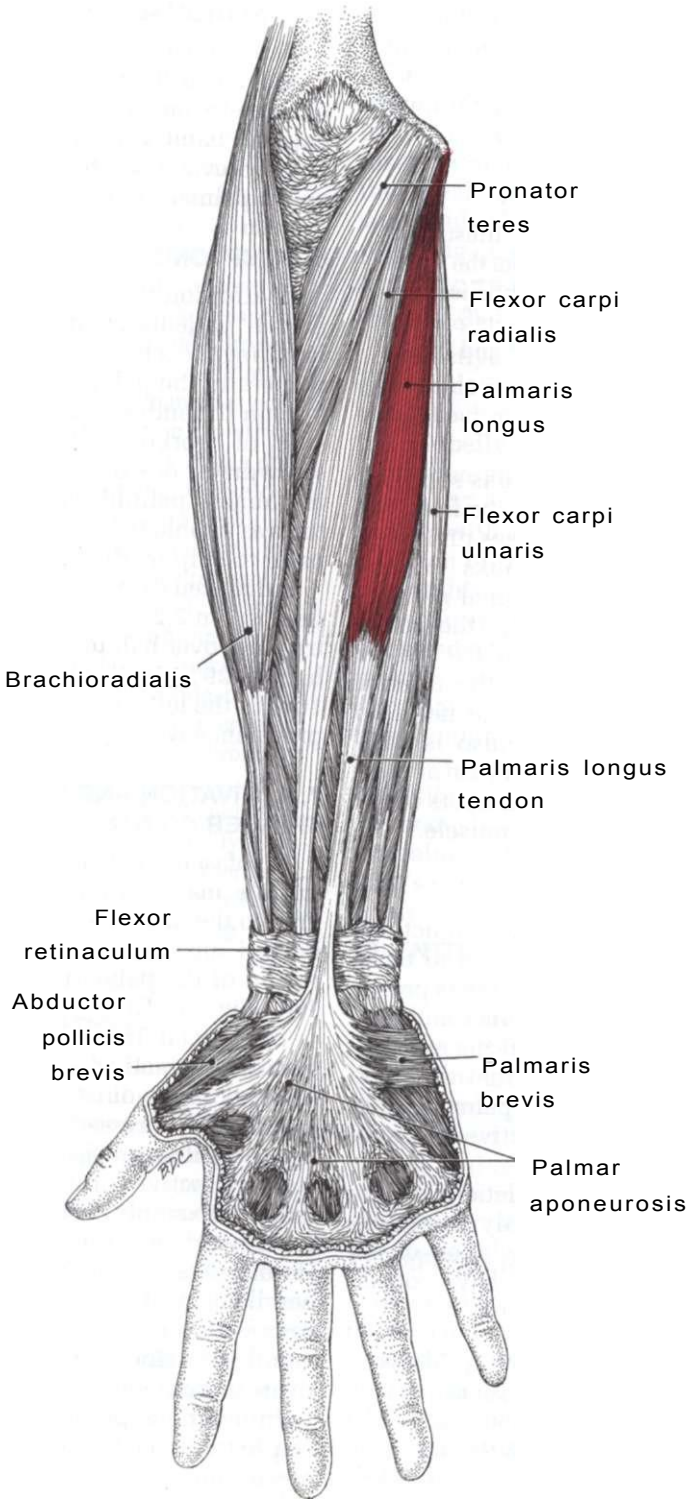


Figure 37.2. Ventral forearm muscles including the usual attachments of the palmaris longus muscle *(red).* It originates at the medial epicondyle, and attaches distally to the palmar aponeurosis. The superficial layer of the palmar aponeurosis has fibrous bands that extend into the fingers and often to the thumb.

underlying tendons.' All were relieved by surgical decompression. Additional examples of nerve entrapment at the wrist by variations of the palmaris longus muscle are presented in Section 10.

SUPPLEMENTAL REFERENCES

A number of authors have illustrated the palmaris longus muscle from the volar view,[10,1,23,27,39,42] and others in cross section.[1,8,29] Some have detailed its palmar fascial attachments,[10,11,24,28,40] and many of its variations.[2,34]

3. INNERVATION

The palmaris longus muscle is supplied by a branch of the median nerve from the lateral cord, through the anterior divisions, the upper and middle trunks of the brachial plexus, and either spinal roots C_6 and C_7,[9,31] roots C_7 and C_8 (the usual arrangement),[14,17] or C_7, C_8 and T_1.[39] Fibers from C_8 and T_1 pass through the inferior trunk and the medial cord. The nerve to the palmaris longus muscle also is variable; it may penetrate the flexor carpi radialis muscle,[14] or the superficial fibers of the flexor digitorum superficialis muscle.[5]

4. FUNCTION

The palmaris longus muscle functions to flex the hand at the wrist and to tense the palmar fascia. It probably assists pronation of the hand against resistance and may assist flexion of the forearm at the elbow.

Two authors[17,31] reported that the muscle tenses the fascia of the palm, which, anatomically, is its distinctive primary function.

Duchenne,[12] upon stimulation of the palmaris longus, observed only hand flexion without pronation or deviation of the hand to either side. Authors have consistently noted this flexor function.[5,6,9,17,31]

Beevor[6] observed that the palmaris longus contracted with the flexor carpi radialis as the hand was pronated against resistance; others agreed with this pronator function.[5,16,17] Because of the muscle's attachment to the medial epicondyle of the humerus, some authors propose a possible weak, flexor action at the elbow.[5,17]

5. FUNCTIONAL UNIT

The palmaris longus has no antagonistic muscle. The thenar and hypothenar muscles are synergistic with it by helping to cup the hand but, of these, only the palmaris brevis also attaches to the subcutaneous palmar fascia.

6. SYMPTOMS

In addition to pain, as described in Section 1, patients complain of difficulty in handling tools because of soreness and tenderness in the palm and frequently call attention to tender nodules there. The pressure of working with the handle of a screwdriver or trowel in the palm becomes intolerably painful. For instance, a sculptor was unable to pound his chisel with his palm to chip marble.

Advanced cases may exhibit palmar contracture. In 2,278 cases of Dupuytren's contracture, over half had contractures in both hands, 29% in the right hand only, and 16% in the left hand only. The maledemale prevalence was reported as 6:1[38] and 8:1.[20]

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Myofascial TrPs in the palmaris longus muscle may develop as satellites of key TrPs in the distal medial head of the triceps brachii muscle,[15] which refer pain to the region of the palmaris longus muscle *[see* TrP_5, Fig. 32.1C).

Myofascial TrPs in the palmaris longus also may be activated by direct trauma, as by a fall on the outstretched hand. Use of a tool forcibly pressed or held firmly in the cupped palm can aggravate, and may initiate, TrP activity in the palmaris longus muscle. Examples are gardening and using a screwdriver or other carpenter's tool. Holding a tennis racquet with the end of the handle against the palm, and leaning on a cane with an angular, rather than a round, handle pressing into the palm also may activate or perpetuate TrPs in this muscle.

In our experience, patients with Dupuytren's contracture commonly have one or more active TrPs in the fibers of the palmaris longus muscle, although there are no experimental data to tell if the TrPs and the contractures are etiologically related.

DUPUYTREN'S CONTRACTURE

Authors agree that heredity is a factor in the development of the contracture, but have become increasingly negative toward repeated trauma as a primary cause.[20,38] Patients, on the other hand, tend to make the latter association because of the referred palmar tenderness. Contracture is more likely to be encountered in those who do ***not*** perform regular manual labor than in those who do.[20] The novice worker is apt to maintain the palm in the ***tightly*** cupped position for longer periods when holding a tool, while the skilled craftsman does not.

The prevalence of Dupuytren's contracture is reported to rise sharply in the 4th decade; it is higher in patients with alcoholism, epilepsy and diabetes mellitus. The condition may be associated with increased sympathetic tone, and frequently also with the reflex sympathetic dystrophy of the shoulder-hand syndrome.[38]

Initially, tender nodular thickenings usually appear on the ulnar side of the palm, short of the distal palmar crease. The nodules develop within the fibrofatty tissue superficial to the palmar aponeurosis.[38] Next, fibrous bands extend centrifugally from these nodules.[20,38] Finally, the palmar aponeurosis develops nontender, contracted, dense fibrous bands which hold the fingers flexed, crippling the hand. These stages usually overlap, and progression may stop at any point.[21]

8. PATIENT EXAMINATION

The patient cups the hand vigorously (as in Fig. 37.3) to make the tendon stand out at the wrist, superficial to the transverse carpal ligament. The prominence of the tendon depends on the degree of wrist flexion or extension. This becomes evident when the strongly cupped hand is moved slowly from extension to flexion. To the examiner, this tendon, which may disappear with hand relaxation, verifies the existence of the palmaris longus muscle, and helps the patient to see and feel the relationship between the fibrotic palmar fascia and the palmaris longus muscle. Palpation of the muscle during contraction helps to identify variations of the usual structure.

Central TrPs of the superficial palmaris longus are found in the middle of the muscle belly, which is usually located in the proximal half of the forearm (Fig. 37.1). Palpation of a palm that is developing Dupuytren's contracture reveals discretely tender nodules with a background of diffuse referred palmar tenderness, which is the usual "soreness" in response to pressure. Only the TrP-referred sensation has a prickling quality.

9. TRIGGER POINT EXAMINATION (Fig. 37.3)

An active TrP in this muscle is located in a palpable nodule of a taut band that can be rolled back and forth between two fingers in the midbelly region of the muscle. There is also often an attachment TrP as shown in Figure 37.3. The tender TrP usually responds with a local twitch response seen as a flexion of the hand at the wrist. Stimulation of this TrP by pressure often elicits the projection of referred prickling pain in the pattern described in Section 1. However, if intense *spontaneous* pain is present due to maximal hyperactivity of this TrP, its further stimulation by digital pressure cannot increase the already maximum referred sensation. In this case, the examiner may erroneously assume that the tender spot in the palmaris longus is not related to the patient's complaint. This same error also can occur in other muscles.

10. ENTRAPMENT

No nerve entrapments have been observed due to TrPs in this muscle. However, anatomical variations are likely to cause median nerve entrapment at the wrist[4,13,26,37] or ulnar nerve entrapment in the region of the ulnar tunnel at the wrist.[32,33,35] Increased tension and nodular enlargement characteristic of TrPs in one of these variant muscles could aggravate the entrapment symptoms.

11. DIFFERENTIAL DIAGNOSIS

The volar wrist and hand pain and tenderness may tempt some clinicians to diagnose the symptoms caused by palmaris longus TrPs as carpal tunnel syndrome, in which case, inactivation of the TrPs will relieve the patient's symptoms. When the palmaris longus anomalously extends under the carpal ligament, TrPs in it can cause a genuine carpal tunnel syndrome. Active TrPs in such a muscle would in-

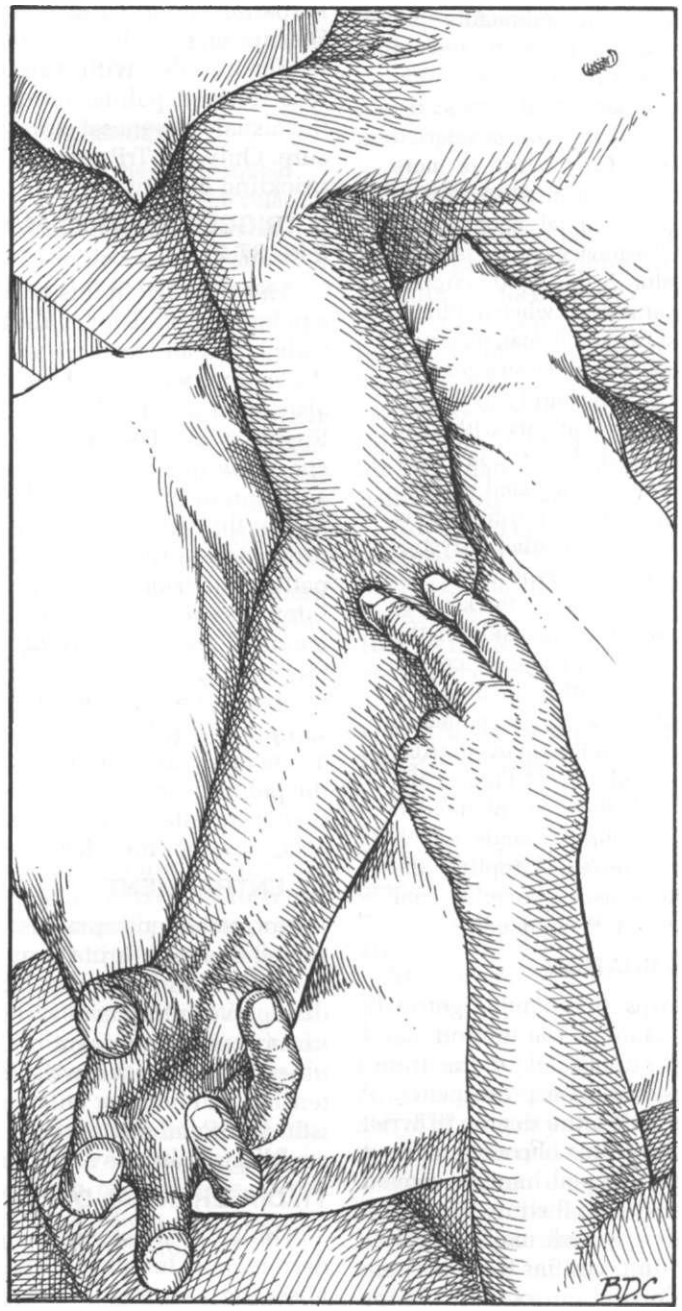


Figure 37.3. The strongly cupped hand illustrates the major function of the palmaris longus muscle. The active contraction with the wrist neutral makes its superficial tendon stand out at the wrist. A region in the muscle that is likely to contain a proximal attachment trigger point lies between the two examining fingers.

crease tendon tension and tend to aggravate the carpal tunnel symptoms.

Because of its distinctive prickling pain, this TrP syndrome is usually easily distinguished from other painful conditions of the volar wrist and hand such as referred pain from TrPs in the flexor carpi radialis, pronator teres, and the brachialis muscles.

Anomalous palmaris longus muscles or attachments can cause distal forearm pain,[36] compression neuropathy,[18] or a "dead feeling."[41]

Active TrPs in the palmaris longus are frequently associated with TrPs in the hand and finger flexors. However, the palmaris TrPs are rarely associated with TrPs in the muscles that refer pain to the elbow, as in "tennis elbow."

Loss of joint play in the wrist articulations should be indentified and corrected.[25]

12. TRIGGER POINT RELEASE
(Fig. 37.4)

In addition to the spray-and-stretch technique described here, other techniques including postisometric relaxation, reciprocal inhibition and contract-relax as described in Chapter 3, Section 12 are also effective for release of *central* trigger points (CTrPs) in the palmaris longus muscle. The primary therapeutic approach to *attachment* TrPs (ATrPs) is to inactivate the CTrPs that are causing them.

To release these TrPs by spray and stretch, the patient is seated with the forearm of the affected side supported on a padded surface. The fingers and hand are extended (Fig. 37.4), while parallel sweeps of the vapocoolant spray are applied in a distal direction over the mus-

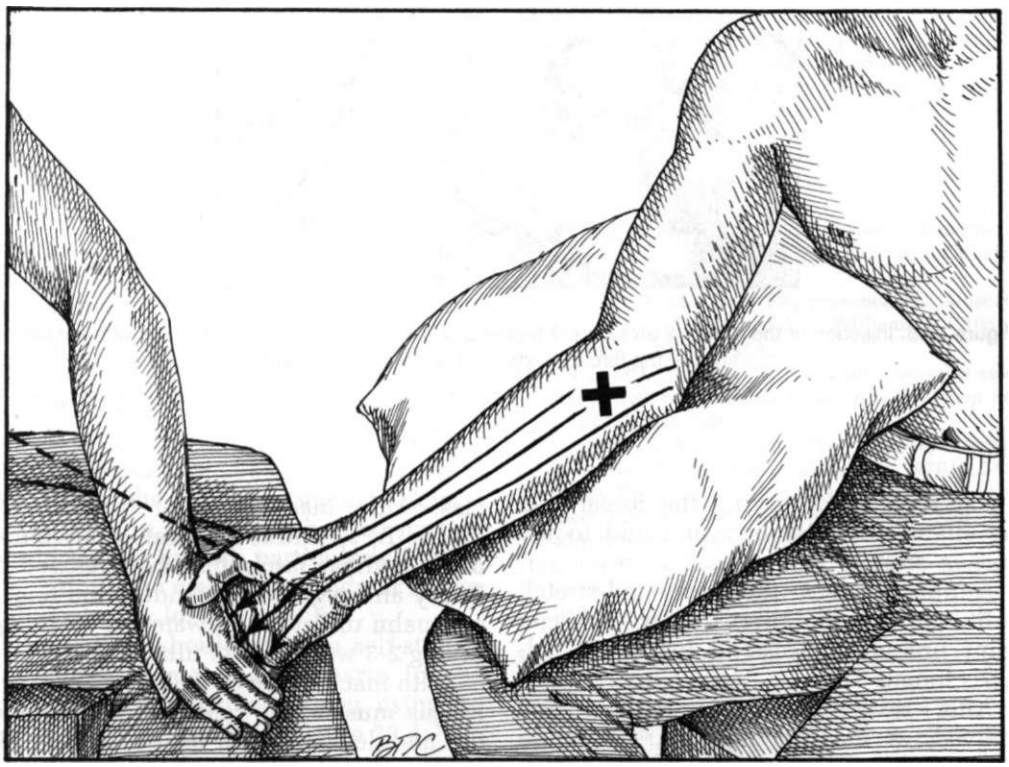


Figure **37.4.** Stretch position and spray pattern *(arrows)* for the palmaris longus muscle. To fully stretch the muscle, the operator simultaneously extends the patient's fingers and the hand at the wrist.

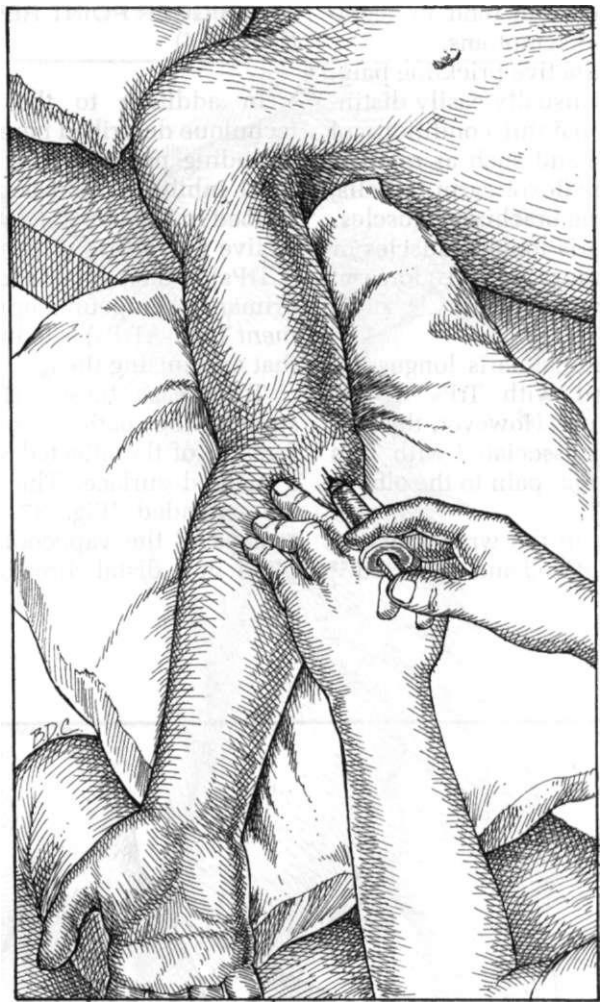


Figure 37.5. Injection of the proximal attachment trigger point in the palmaris longus muscle with the patient supine, the forearm extended and well supported.

cle and palm. Extending the forearm at the elbow normally does not add to the passive stretch.

Applications of the spray-and-stretch technique may be alternated with trigger point pressure release to inactivate palmaris longus TrPs.

After this muscle is stretched and sprayed, or its TrPs injected, the entire group of forearm flexor muscles, particularly the hand and finger flexors, is then stretched and sprayed to eliminate any associated TrP involvement of parallel muscles. After inactivation of the palmaris longus TrPs, mild to moderate contractures of the palmar fascia may be stretched by firmly and regularly extending the fingers and palm under warm water, or while applying 2-3 watts/cm^2 of ultrasound.[38]

With inactivation of TrPs in the palmaris longus muscle, the referred tenderness of the nodules and palm may disappear immediately. The further the fibrotic contracture has progressed, the greater the likelihood that the fibrosis and local tenderness will persist after TrP inactivation.

13. TRIGGER POINT INJECTION
(Fig. 37.5)

The patient lies supine with the affected elbow in extension. After locating any trigger points (TrPs) in the palmaris longus by palpation (Fig. 37.3), each TrP is probed and injected with 0.5% procaine solution (Fig. 37.5). Immediately after the TrP injection, passive stretching of the muscles is carried out, again with the application of vapocooling and moist heat. Inactivation of the TrPs and restoration of full muscle length relieves the prickling palmar pain and releases the sustained tension that the taut muscle fibers placed on the palmar aponeurosis. Full active range of motion expedites the return of normal function.

Rachlin[30] illustrates three TrP sites in the palmaris longus muscle, one CTrP in the middle of the muscle belly, and an ATrP at each end of the muscle.

Troublesome palmar nodules that remain after inactivation of palmaris longus TrPs are likely to resolve more rapidly if each is injected with about 0.3 ml of soluble steroid, such as dexamethasone sodium phosphate diluted with 2% procaine to prepare approximately 2 ml of a 0.5% solution of steroid. Sigler[38] recommended the injection of steroid *only* in the early stages of nodule development.

This treatment may stop the progression of fibrosis, but cannot reverse advanced palmar contracture. Surgical recommendations range from simple cutaneous fasciotomy and nodule excision to limited fasciotomy. Radical fasciotomy is rarely, if ever, indicated.[38]

14. CORRECTIVE ACTIONS

The patient must avoid the activities listed in Section 7 that activate and perpetuate TrPs in the palmaris longus muscle.

Any TrPs in the triceps brachii muscle should be inactivated, particularly if they refer pain to the medial epicondylar region of the palmaris longus muscle.

The patient should learn to self-stretch the palmar fascia and palmaris longus muscle in a warm bath or shower, using the stretch position shown in Figure 37.4.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:441 (Fig. 6.123).
2. *Ibid.* pp. 412 (Figs. 6.79, 6.80).
3. Backhouse KM, Churchill-Davidson D: Anomalous palmaris longus muscle producing carpal tunnel-like compression. *Hand* 7:22-24, 1975.
4. Bang H, Kojima T, Tsuchida Y: A case of carpal tunnel syndrome caused by accessory palmaris longus muscle. *Handchirurgie* 20.141-143, 1988.
5. Bardeen CR: The musculature, Sect. 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (p. 432).
6. Beevor CE: Muscular movements and their representation in the central nervous system. *Lancet* 2:1715-1724, 1903 (pp. 1718, 1719).
7. Brones MF, Wilgis EF: Anatomical variations of the palmaris longus, causing carpal tunnel syndrome. *Plast Reconstr Surg* 62:798-800, 1978.
8. Carter BL, Morehead J, Wolpert SM, *et al.: Cross-Sectional Anatomy.* Appleton-Century-Crofts, New York, 1977 (Sects. 53-39).
9. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 531, 532, 544, 545).
10. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 65-67).
11. *Ibid.* (Figs. 106, 121).
12. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (p. 120).
13. Giunta R, Brunner U, Wilhelm K: Bilateral reverser Musculus palmaris longus-seltene Ursache eines peripheren N.-medianus-Kompressionssyndroms [Bilateral reversed palmaris longus-a rare cause of peripheral median nerve compression syndrome. Case report]. *Unfallchirurg* 96(10):538-540, 1993.
14. Hollinshead WH: *Anatomy for Surgeons.* Ed. 3, Vol. 1, *The Head and Neck.* Harper & Row, Hagerstown, 1982 (pp. 393, 394).
15. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(2):29-59, 1994.
16. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W.B. Saunders, Philadelphia, 1991 (pp. 125-127).
17. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 253).
18. Lahey MD, Aulicino PL: Anomalous muscles associated with compression neuropathies. *Orthop Rev* 15(4).199-208, 1986.
19. Langman J, Woerdeman MW: *Atlas of Medical Anatomy.* W.B. Saunders, Philadelphia, 1978 (p. 241).
20. Larsen RD, Posch JL: Dupuytren's contracture with special reference to pathology. *J Bone Joint Surg* 40A.773-793, 1958 (pp. 773, 774).
21. Lieber RL, Jacobson MD, Fazeli BM, *et al.:* Architecture of selected muscles of the arm and forearm: anatomy and implications for tendon transfer. *J Hand Surg 17A(5):787-796,* 1992.
22. Maragh H, Boswick JA Jr: Dupuytren's disease. *Cont Ortho* 8:69-76, 1984.
23. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 133E).
24. *Ibid.* (pp. 139, 140).
25. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.

26. Meyer FN, Pflaum BC: Median nerve compression at the wrist caused by a reversed palmaris longus muscle. *J Hand Surg 12A(3)*369-371, 1987.
27. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 75).
28. *Ibid.* (Fig. 84).
29. *Ibid.* (Figs. 82, 83).
30. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (see p. 339).
31. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 197, 199).
32. Regan PJ, Feldberg L, Bailey BN: Accessory palmaris longus muscle causing ulnar nerve compression at the wrist. *J Hand Surg 16A(4):736-738,* 1991.
33. Regan PJ, Roberts JO, Bailey BN: Ulnar nerve compression caused by a reversed palmaris longus muscle. *J Hand Surg 13B(4):406-407,* 1988.
34. Reimann AF, Daseler EH, Anson BJ, *et al.:* The palmaris longus muscle and tendon. A study of 1600 extremities. *Anat Rec* 89:495-505, 1944.
35. Robinson D, Aghasi MK, Halperin N: Ulnar tunnel syndrome caused by an accessory palmaris muscle. *Orthop Rev 18(3):*345-347, 1989.
36. Ryu J, Watson HK: SSMB syndrome. Symptomatic supernumerary muscle belly syndrome. *Clin Orthop* 216:195-202, 1987.
37. Schlafly B, Lister B: Median nerve compression secondary to bifid reversed palmaris longus. *J Hand Surg 12A(3):371-373,* 1987.
38. Sigler JW: Dupuytren's contracture. Chapter 81. In: *Arthritis and Allied Conditions.* Ed. 8. Edited by Hollander JE, McCarty DJ Jr. Lea & Febiger, Philadelphia, 1972 (pp. 1503-1510).
39. Spalteholz W: *Handatlas der Anatomie des Men-schen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 235).
40. *Ibid.* (p. 335).
41. Thomas CG: Clinical manifestations of an accessory palmaris muscle. *J Bone Joint Surg* 40A:929, 1958.
42. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 322).

CHAPTER 38
Hand and Finger Flexors in the Forearm

Flexores Carpi Radialis and Ulnaris, Flexores Digitorum Superficialis and Profundus, Flexor Pollicis Longus (and the Pronator Teres)

HIGHLIGHTS: **REFERRED PAIN** from each finger flexor is experienced throughout the length of the digit which it flexes. Pain may be experienced "beyond" the tip of the digit, extending like lightning. Trigger points (TrPs) in the hand flexors refer pain that centers on the volar wrist crease. **A** trigger finger is an annoying, but painless dysfunction that appears to be caused by restriction of the flexor tendon and is relieved by procaine injection into the tender point beneath the tendon just proximal to the corresponding metacarpal head. **ANATOMY:** attachments of the finger flexors are mostly to the medial epicondyle proximally and individually to the middle and terminal phalanges of each finger distally. The flexors of the hand also arise from the medial epicondyle. The flexor carpi ulnaris inserts on the pisiform bone, and the flexor carpi radialis inserts on the bases of the second and third metacarpal bones. **FUNCTION** of the hand flexors is to flex and deviate the hand at the wrist. The finger flexors help to flex the hand at the wrist. The flexor digitorum superficialis selectively flexes primarily the middle phalanges and the profundus flexes primarily the distal phalanges. The **FUNCTIONAL UNIT** of the finger flexors includes the finger and hand extensors, contraction of which is required for effective grasp. **SYMPTOMS** include pain on forceful use of scissors and when cupping and supinating the hand to receive coins placed into it. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** results from repetitive or prolonged strong gripping, or from repeated strenuous twisting and pulling movements with the fingers. **PATIENT EXAMINATION** reveals tightness of individual muscles and pain projected to the end of the finger tips when the hand flexors are stretched and when the finger flexors are then passively stretched individually. **ENTRAPMENT** of the ulnar nerve may be caused by or aggravated by TrPs in the flexor carpi ulnaris, in the flexor digitorum superficialis, or in the flexor digitorum profundus muscle. Entrapment of the median nerve may be caused by or aggravated by TrPs in the pronator teres or flexor digitorum superficialis muscles. **DIFFERENTIAL DIAGNOSIS** includes medial epicondylitis, ulnar neuropathy, carpal tunnel syndrome, osteoarthritis of the wrist, and cervical radiculopathies. **TRIGGER POINT RELEASE** by stretch and spray is accomplished by fully extending the hand and fingers while applying the vapocoolant in a distal pattern. Other techniques also are effective. **TRIGGER POINT INJECTION** is often not required for TrPs in the hand and finger flexor muscles, but may be required to relieve a trigger finger or trigger thumb. **CORRECTIVE ACTIONS** call for avoiding prolonged, tight gripping, and for establishing good forearm muscle relaxation and self-stretch habits by regular use of the Artisan's Finger-stretch, the Finger-extension, the Finger-spreading, and/or the Finger-flutter Exercise.

1. REFERRED PAIN (Fig. 38.1)

The pain patterns illustrated in this chapter are based on a local twitch response (LTR) to identify the muscle being injected and the patient's report of the distribution of pain induced by needle penetration of the trigger point (TrP).

Winter[80] described TrPs in the flexors of the hand and fingers near their common attachment to the medial epicondyle as a frequent source of referred pain. Good[34] depicted the pain as projecting to the volar aspect of the wrist, or to the corresponding digit. Good[36] also attributed idiopathic myalgia (descriptions compatible with myofascial TrPs) of the elbow to referred pain from localized myalgic areas, some of which were in the flexors of the wrist and fingers. He relieved the symptoms by procaine injection of the myalgic spots.

Hand Flexors (Fig. 38.1 A)

An active TrP in the flexor carpi radialis refers pain and tenderness that center in the radial aspect of the volar crease of the wrist, with some spillover into the adjacent forearm and palm (Fig. 38.1A, *left).*

An active TrP in the flexor carpi ulnaris refers pain and tenderness to the ulnar side of the volar aspect of the wrist with similar spillover (Fig. 38.1A, *right).*

Finger Flexors (Fig. 38.1 B)

No distinction is made between the referred pain patterns of the flexores digitorum superficialis and profundus. A TrP in these fibers refers pain to the same digit that the fibers activate. For example, a TrP in the fibers of the middle finger flexor projects pain through the length of the middle finger (Fig. 38.1B, *left).* Similarly, TrPs in fibers that flex the ring and little fingers project pain throughout those digits (Fig. 38.1B, *right).* Pain is frequently described as an explosive pain that "shoots right out the end of the finger, like lightning." This pattern differs from the pain referred from the finger extensors, which stops short of the end of the digit. When patients with active TrPs in the flexors are asked whether the pain is more on the top or underside of the finger, they are likely to rub the volar aspect and reply, "I don't know." The movement reveals their answer.

Kellgren[42] reported that injection of 6% salt solution into the flexor digitorum profundus muscle produced metacarpophalangeal (MCP) joint pain that was indistinguishable from the pain caused by injecting 0.3 ml of the same solution directly into the same joint space of the opposite hand. The similar nature of the joint pain due to these two sources causes confusion between the pain of articular disease and that referred from myofascial TrPs in the finger muscles.

Pain referred to the hypothenar eminence and the 5th knuckle (MCP joint), induced by injection of 0.2 ml of 6% salt solution into the flexor digitorum profundus muscle, persisted despite *total anesthesia* of the painful structures by local anesthetic block of the ulnar nerve at the wrist.[42] This observation is compatible with the central mediation of referred pain (by the convergence-projection mechanism[64]) from TrPs as described in Chapter 2, Section C. The referred pain in this experiment was not dependent on impulses arising in the pain reference zone and a significant part of the afferent nerve discharges caused by the irritant saline solution in the muscle, and of those perceived as pain from the reference area, must have followed a common pathway in the central nervous system. One might describe this as a phantom pain. (The pain referred to the hypothenar eminence may have been due to inadvertent saline infiltration of the flexor carpi ulnaris muscle as well.)

Long Thumb Flexor (Fig. 38.1 C)

When the flexor pollicis longus (Fig. 38.1C) harbors an active TrP, it projects pain throughout the volar aspect of the thumb to its tip (and "beyond").

Pronator Teres (Fig. 38.1 D)

The pronator teres TrPs refer pain deep in the volar radial region of the wrist and also of the forearm (Fig. 38.1D).

Other Authors

In their illustrations of the referred pain patterns for all of these flexor muscles located in the forearm, Bonica and Sola[12] strongly emphasized local pain in the region of the TrP and minimize the pain referred to the wrist and beyond. Rachlin,[62] on the other hand, emphasized the more distal pain pattern of the flexor digitorum superficialis but did not include the other hand and finger flexors.

Trigger Finger

The painless phenomenon of a trigger finger, a "trick" or "locking" finger, consists of the finger sticking in the flexed position until it is extended by an external force. This condition responds to injection of a tender spot deep in the fascial sheath, which is apparently responsible for the constriction of the flexor tendon near the MCP joint. The constriction may ensnare a knot-like enlargement of the tendon itself. Such a fascial band that might anchor the tendon is described just short of the end of the distal palmar synovial sheath for digits two, three and four.[21] This condition may also respond to firm pressure applied to the spot of restriction. The enlargement may be due to a local inflammatory reaction.

2. ANATOMY (Fig. 38.2)

Hand Flexors (Fig. 38.2A)

The **flexor carpi radialis** muscle is subcutaneous and nearly centered on the volar side of the forearm between the pronator teres, which crosses the forearm above it on the radial side and the palmaris longus, which tends to overlap it on the ulnar side (Fig. **38.2A).** This radial hand flexor attaches ***above*** to the medial epicondyle *via* the common tendon and to intermuscular septa. The muscle belly extends only to the mid-forearm. Its tendon attaches ***below*** mainly onto the base of the second metacarpal bone, with a slip extending to the base of the third metacarpal bone.

The **flexor carpi ulnaris** muscle lies superficially along the volar side of the sharp edge of the ulna. ***Proximally*** it attaches by two heads: the humeral head attaches to the medial epicondyle of the humerus *via* the common tendon and the ulnar head fastens to the medial margin of the olecranon and to the proximal two-thirds of the dorsal border of the ulna through an aponeurosis shared in common with the extensor carpi ulnaris and flexor digitorum profundus, and to intermuscular septa. ***Distally*** its tendon attaches to the pisiform bone.[21]

Finger Flexors (Figs. 38.2B and C)

Proximally, the **flexor digitorum superficialis** (sublimis) comprises three heads: humeral, ulnar and radial (Fig. **38.2B).** The humeral head attaches to the medial epicondyle of the humerus *via* the common tendon and to intermuscular septa. The ulnar head attaches to the medial side of the coronoid process of the ulna, proximal to the attachment of the pronator teres, beneath the humeral head and the radial head attaches to the oblique line of the radius, between the attachments of the biceps brachii and pronator teres muscles. The median nerve passes beneath the fibrous archway between the attachments of the ulnar and radial heads.[29] This muscle covers most of the volar forearm, beneath the palmaris longus muscle and flexores carpi muscles (Fig. **38.2B).**[21]

The tendons at the wrist, and to some extent the fibers of the flexor digitorum superficialis, lie in a deep and a superficial plane. The superficial plane carries tendons to the middle and ring fingers, and the deep plane to the index and little fingers.

Distally, at the first phalanx, each tendon of the **flexor digitorum superficialis** divides to pass around the deep tendon of the flexor profundus, as each superficialis tendon attaches to the sides of a middle phalanx.

The fibers of the **flexor digitorum profundus** (Fig. **38.2C)** extend through the proximal half on the ulnar side of the forearm. The muscle attaches ***above*** to the proximal three-fourths of the volar, medial and dorsal surfaces of the ulna to an aponeurosis shared by the flexor and extensor carpi ulnaris, to the medial side of the coronoid process of the ulna, and to the ulnar half of the interosseous mem-

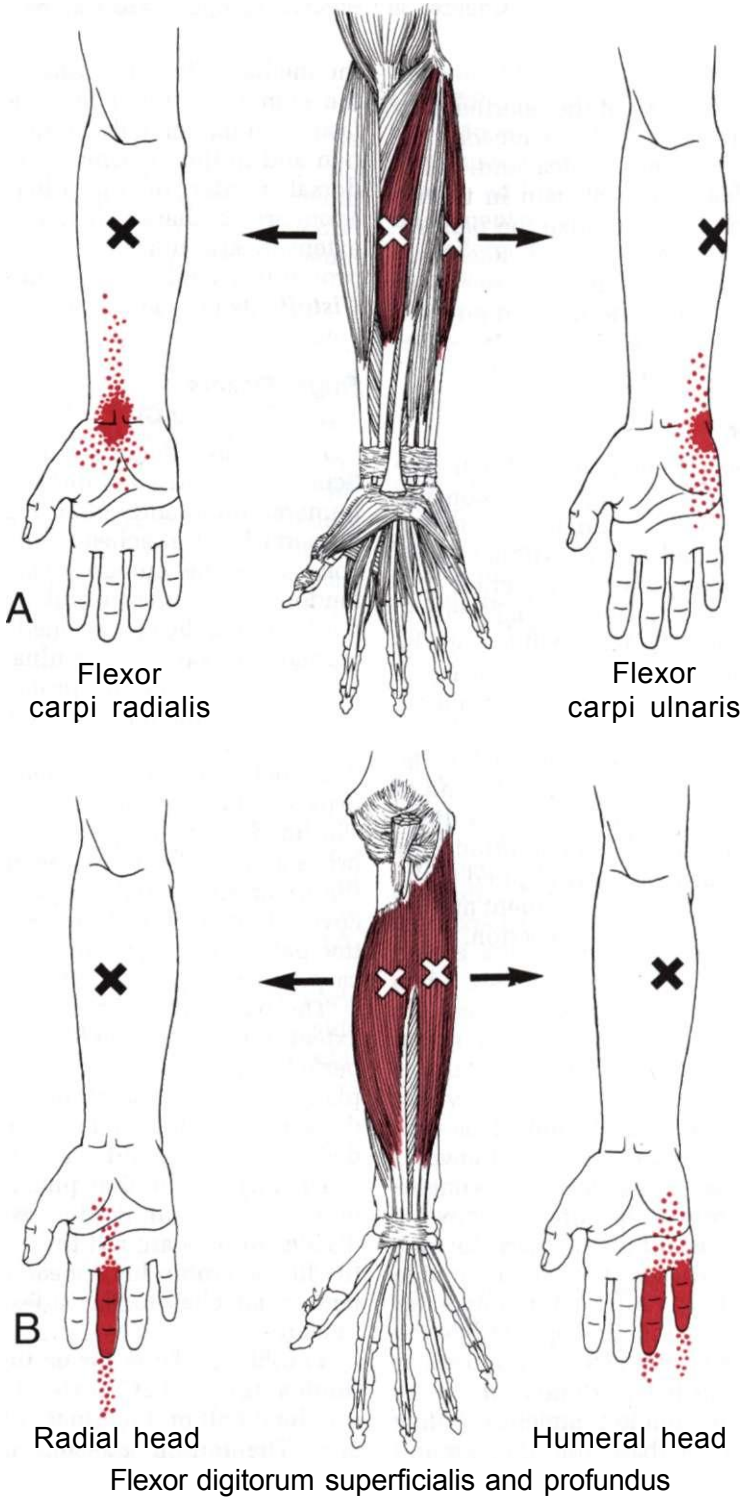


Figure 38.1. Composite referred pain patterns (*dark red)* and location of central trigger points (**Xs**) in the right hand and finger flexors *(medium red)* for all muscles except the flexor pollicis longus. In the flexor pollicis longus, the ***X*** designates an attachment TrP **A,** flexor carpi radialis and flexor carpi ulnaris. **B,** flexor digitorum superficialis and profundus: *left*—superficialis middle finger pattern; *right*—superficialis 4th and 5th digit patterns and profundus pattern. The index finger pattern, not shown, is comparable.

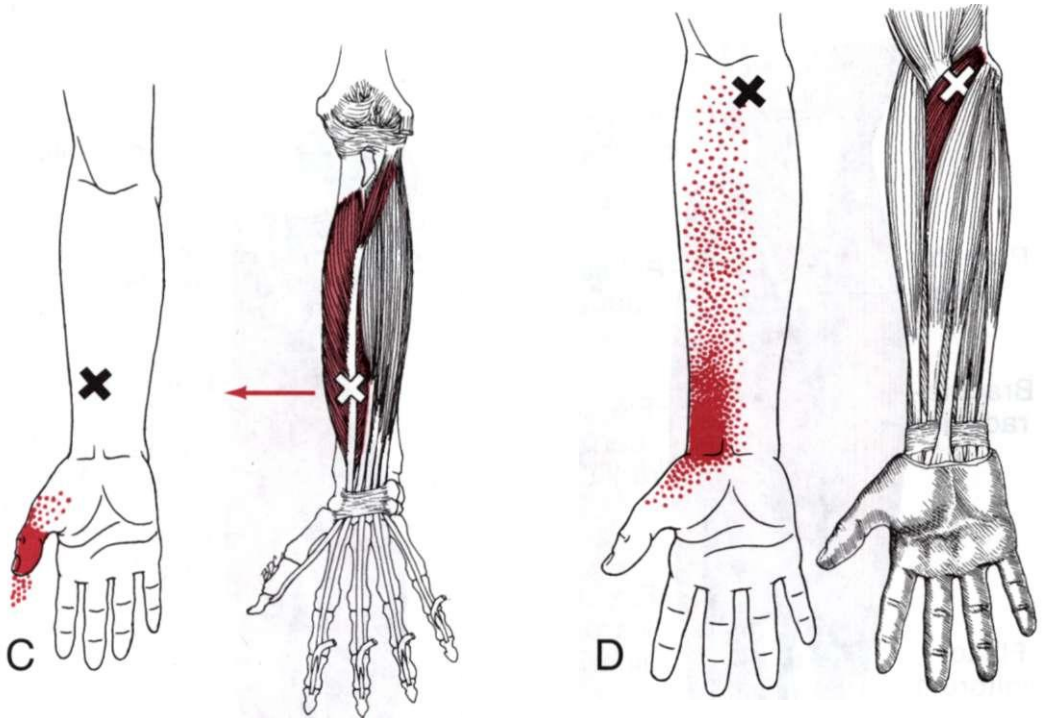


Figure 38.1—*continued.* **C,** flexor pollicis longus. **D,** pronator teres.

brane. Each tendon fastens ***below*** onto the base of the terminal phalanx of the respective finger.[21]

The **flexor pollicis longus** (Fig. 38.2C) extends throughout the forearm under more superficial muscles, chiefly on the radial side. It attaches ***proximally*** to the radius, the adjacent interosseous membrane, and by a slip to the humerus and ***distally*** to the base of the distal phalanx of the thumb.[21] The belly of the flexor digitorum superficialis covers both the deep finger flexor and the long thumb flexor muscles.

The **pronator teres** attaches ***above*** and ***medially*** by two heads. The humeral head fastens proximal to the medial epicondyle and to adjacent fascia. The ulnar head fastens to the medial side of the coronoid process of the ulna, and the median nerve enters the forearm between these two heads. The muscle attaches ***below*** and ***laterally*** to the lateral surface of the radius at its midpoint in the forearm.

Locations and Structure

The specific location of the muscle bellies of the four digitations of the flexor digitorum superficialis are described and illustrated.[11] The muscle bellies for the second and fifth digits are relatively distal and those of the third and fourth digits largely proximal to them.

The architectural arrangement of fibers in the flexor carpi radialis (see below for compartmentalization) and in the flexor carpi ulnaris[48] reveals similar fiber lengths (51 and 41 mm). However, the ulnar muscle is much more pennate (12°) than the radial one (3.1°). This is reflected in the ratios of fiber length to muscle length of .19 and .31 respectively. Extensor forearm muscles show a much larger range of length ratios.[49] The flexor carpi ulnaris favors force rather than speed and should have an endplate zone that would be nearly longitudinal from almost one end of the muscle to the other (see

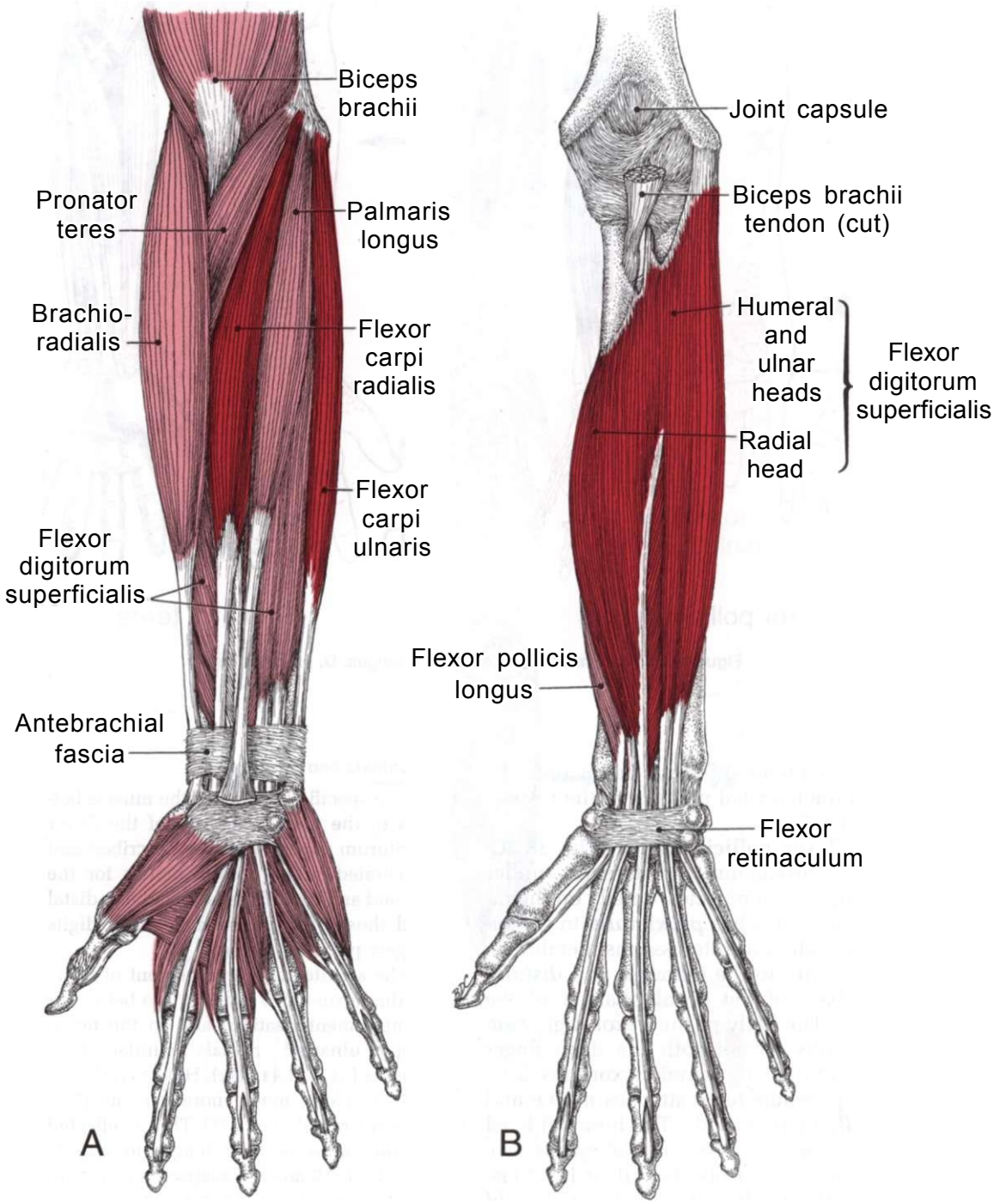


Figure 38.2. Volar view of the right upper extremity showing the attachments of the hand and finger flexors in the forearm. **A,** flexor carpi radialis and flexor carpi ulnaris are *dark red,* other muscles including the pronator teres are *medium red.* **B,** flexor digitorum superficial (*dark red).* The ulnar head lies unseen beneath the humeral head.

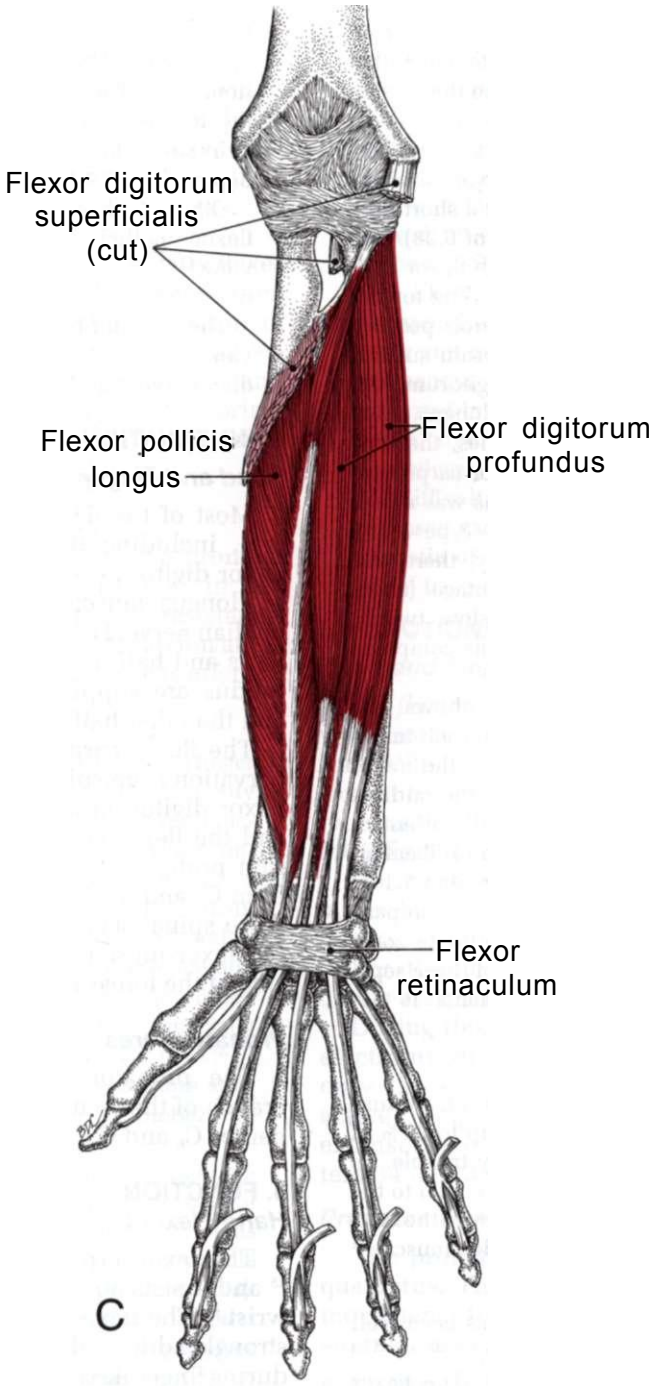


Figure 38.2—*continued.* **C,** flexor digitorum profundus and flexor pollicis longus *(dark red)* and cut end of flexor digitorum superficialis *(light red).*

Fig.2.8A). On the other hand, the flexor carpi radialis is structured for speed rather than force and would be expected to have a diagonal endplate zone that would likely be broken by the three separate compartments of the muscle.[66]

The pronator teres has a "strength" architecture similar to the flexor carpi ulnaris (pennate angle 10° and a short fiber length/muscle length ration of *0.28)*. The finger flexors and long thumb flexor have intermediate architecture tending toward more of the force configuration progressively from the flexor digitorum superficialis through the flexor digitorum profundus to the flexor pollicis longus.

As in most skeletal muscles, the fiber type distribution in the flexor carpi radialis and flexor pollicis longus was nearly evenly distributed between Type I and Type II fibers.[40] Interestingly, there was consistently a lower percentage (about 6% difference) of type I (slow twitch) fibers on the dominant side as compared to the nondominant side.[12]

The flexor carpi radialis shows evidence of three compartments, each innervated by a separate branch of the motor nerve. Fibers inserting along the midline of the tendon are longitudinally oriented. A medial and a lateral group of fibers attach along the sides of the tendon.[66] It is characteristic for each of such compartments to have a separate endplate zone. The functional significance of these separately innervated compartments is not known at this time.

Variations

An accessory flexor digitorum profundus indicis is not unusual (up to 20% of bodies) but rarely causes any trouble. Its muscle belly ordinarily is proximal to the wrist in close approximation to the normal flexor digitorum profundus muscle.[79]

Supplemental References

The flexor carpi radialis has been well illustrated in the volar view,[1,12,27,58,67,72] and in cross section.[4,16,24,59] The flexor carpi ulnaris has been shown in the volar view,[1,2,21,26,27,52,58,67,72] in the lateral view,[28] and in cross section.[4,17,24,59]

The flexor digitorum sublimis has been clearly illustrated in the volar view[2,5,22,26,27,29,54,58,61,68,71,72,75] and in cross section.[4,18,24,59] The flexor digitorum profundus has been drawn in the volar view,[3,5,22,25,26,53,54,6,'69,75] and in cross section.[4182559] The fibrous loop that restrains the flexor tendons at the point of constriction in the trigger finger also has been depicted.[30,31,55,60,73]

Other authors have illustrated the flexor pollicis longus in the volar view,[3,22,25-27,52,53,58,68,67,72,74] and in cross section.[4,19,24]

The pronator teres is portrayed in volar view[1,23,27,52,58,67,69,72] in relation to the median nerve,[29] and in cross section.[4,59]

3. INNERVATION

Hand and Finger Flexors

Most of the flexor muscles in the forearm, including the flexor carpi radialis, flexor digitorum sublimis and flexor pollicis longus muscles, are supplied by the median nerve. However, the flexor carpi ulnaris and half of the flexor digitorum profundus are supplied by the ulnar nerve, and the other half, by the median nerve.

The flexor, carpi radialis derives its innervation from spinal nerves C_6 and C_7, the flexor digitorum sublimis from C_7 and C_8, and the flexor carpi ulnaris, flexor digitorum profundus and flexor pollicis longus from C_8 and T_2.[21] Thus the most caudal of these spinal segments innervate the deepest flexor muscles and those on the ulnar side of the forearm.

Pronator Teres

The pronator teres is supplied by a branch of the median nerve through spinal nerves C_6 and C_7.

4. FUNCTION

Hand Flexors

The flexor carpi radialis flexes the hand[21,63] and assists abduction of the hand at the wrist.[63] The flexor carpi ulnaris flexes and strongly adducts the hand,[21,63] and is active during finger-flexion movements.[65] An EMG study[51] substantiated these functions.

Finger Flexors

The flexor digitorum superficialis primarily flexes the middle phalanx of each

finger, but also flexes the proximal phalanx, as well as the hand at the wrist.[63]

The flexor digitorum profundus primarily flexes the terminal phalanx of each finger, and also all the other phalanges and the hand.[21,63] It is used not so much for wrist flexion, as for gross closure of the fist at all joints simultaneously.[8]

Long Thumb Flexor

The flexor pollicis longus initially flexes the terminal phalanx of the thumb, then the proximal phalanx with adduction of the metacarpal bone,[21] and eventually assists in flexion and abduction of the hand at the wrist.[63] Normal flexion of this prime mover requires coordinated activity of four other thumb muscles.[7]

Pronator Teres

The pronator teres assists the pronator quadratus, the primary pronator, in fast movements and to overcome resistance. The pronator teres also assists flexion at the elbow, but only when resistance is offered.[9]

Activities

Motor unit activity of the hand and finger flexor muscles was monitored bilaterally with surface electrodes during 13 sports activities that included overhand throws, underhand throws, tennis, golf, hitting a baseball, and 1-foot jumps in basketball. Examination of the records showed moderate to strong activity, which was bilaterally similar in pattern, but of higher amplitude on the dominant right side, especially when the hand was gripping a handle.[13]

Exercise normally produces more stiffness (reduction in range of motion) in older subjects.[20]

Lundervold[50] studied the electrical activity in the muscles of 135 subjects, 63 of whom had "occupational myalgia" (signs and symptoms including pain and muscle tenderness that strongly suggested TrPs). He found that the symptomatic subjects were much more likely than pain-free subjects to show continuous, larger amplitude motor unit activity when striking a typewriter key repetitively with one finger. When muscular tenderness and pain involved the flexors in the forearm on one side only, typing with the *asymptomatic* arm greatly increased the sustained motor unit activity of these flexor muscles on the symptomatic side, which were "at rest." Subjects with symptoms also were more likely than normal subjects to respond to needle electrode insertion in the muscle with marked motor unit activity (muscle tension) that slowly subsided over a minute or more; normal subjects usually showed little or no such muscle-tension response. Symptomatic subjects were more likely than were pain-free subjects to respond with increased and sustained motor unit activity to psychic stress (gruff commands), insufficient light, a cold draft, and to loud noise. The motor units in the involved forearm flexor muscles were clearly more excitable, and exhibited difficulty relaxing under stress. This increased excitability seems to be a characteristic of motor units that have active loci of trigger points at their endplates.

5. FUNCTIONAL UNIT

Hand and Finger Flexors

All *flexion* movements of the fingers involve some activity of the *extensor* digitorum. When the fingers are held in extension at the interphalangeal joints, only the interossei and the lumbricales produce MCP flexion.[8]

During hand flexion at the wrist, the palmaris longus assists the finger and hand flexors.

For thumb flexion, the flexor pollicis brevis assists the flexor pollicis longus.

During flexion of the hand at the wrist, electromyographic records showed that only the extensor carpi radialis was an active antagonist.[8] The hand and finger extensors function as described in Chapters 34 and 35.

Pronator Teres

The pronator teres assists the pronator quadratus. The brachioradialis may assist movement toward pronation from full supination *(see* Chapter 34).

6. SYMPTOMS

Hand and Finger Flexors

Patients with TrPs in the flexor muscles of the forearm report difficulty in using scissors for cutting heavy cloth or for gar-

dening, or in using tin shears. In contrast, patients with active TrPs in the extensor forearm muscles and "tennis elbow" report no problem with the use of scissors.

Active TrPs in the finger flexors interfere with the placement of curlers in the hair and with the placement of a hair clasp at the back of the head.

Patients with active TrPs in the pronator teres are likely to be unable to supinate the cupped hand, as when coins are placed into it. The combined motion of full supination, slight extension and cupping of the hand becomes prohibitively painful. These patients usually compensate by rotating the arm at the shoulder, thus overloading the shoulder muscles.

Trigger Finger

This phenomenon, also called "locking finger," is a painless but very annoying locking of the digit in the flexed position, despite a maximum active effort to extend the finger; the digit must be extended passively by an external force.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

A posture or activity that activates a TrP, if not corrected or if it is continued, can also perpetuate the TrP. In addition, many preexisting structural and systemic factors (see Chapter 4) can perpetuate a TrP when it has been activated by an acute or chronic overload.

Satellite TrPs may develop in the flexor carpi radialis from key TrPs in the pectoralis minor muscle.[38] Satellite TrPs may develop in the flexor carpi ulnaris from key TrPs in the pectoralis minor, latissimus dorsi, or serratus posterior superior muscles.[38]

Trigger points in these hand and finger flexors are *not* aggravated by the fine pincer movements that tend to activate TrPs in the intrinsic hand muscles, but rather by abuse of gross gripping movements. The skier who grips ski poles hard for long periods, and the carpenter who tightly grips small-handled tools are likely to activate these TrPs.

The finger flexor muscles may develop active TrPs as a result of driving a car with the fingers tightly gripping the steering wheel, especially when the hand grasps the top of the wheel so that the hand is flexed at the wrist. Symptoms are especially likely to occur after long, intense driving.

The passive-stretch position for treatment of the finger *extensors,* that places the fingers and hand in full flexion, can cause sudden shortening activation of latent TrPs in the hand and finger flexors.

Activation of the flexor pollicis longus TrP causes symptoms that have been termed "weeder's thumb." This TrP is activated by forceful rocking, twisting and then pulling motions, all of which can strain this and other thumb muscles.

The pronator teres TrP can be activated as the result of a fracture at the wrist or elbow.

The locking of a trigger finger appears to be due to a nodule in the tendon being caught by the constriction of the annular band that anchors the tendon sheath.[14] The precise mechanism that causes the nodule in the tendon is not clear. It may be caused by a TrP in the lumbrical muscle. One patient reactivated a trigger finger (middle digit) by the continuing use of a cane, the angled head of which pressed on the trigger-finger sore spot just proximal to the head of the third metacarpal bone.

8. PATIENT EXAMINATION (Fig. 38.3)

When testing for TrP dysfunction, painful restriction of range of motion is more sensitive and more specific to TrPs than is weakness. All of the hand and finger flexors can be screened for restriction at one time by fully supinating the forearm with the fingers (including the distal phalanges) and hand fully extended. The long thumb flexor can be tested by extending the wrist and thumb.

The Finger-extension Test can screen both hands at once by first placing the finger tips of the right and left hand together (Fig. **38.3A),** and then pushing the palms tightly against each other while bringing the forearms into as straight a line as possible (Fig. **38.3B).** Active TrPs in the flexor muscles then are revealed by a feeling of tightness in the muscle and pain in the pain reference areas specific to the involved muscles (Section **1).**

Involvement of individual finger flexor muscles can be tested by passive extension

of each digit first by extending the wrist and just the middle phalanx, and then both the middle and distal phalanges for painful limitation of extension.

If weakness is an issue, individual muscles can be tested as clearly described and illustrated by Kendall, *et al.*[43]

The wrist articulations and the metacarpophalangeal and interphalangeal joints should be tested for normal joint play and if restricted, they should be released.[47,56]

9. TRIGGER POINT EXAMINATION

The central TrPs in these flexor muscles are located in the midfiber portions of the muscle bellies, as shown in Figure 38.1. Both the flexor carpi radialis and ulnaris muscles are sufficiently superficial for their TrPs to be identifiable by spot tenderness of a nodule in a taut band and by eliciting pain that is familiar to the patient as the patient's pain complaint.[33] To elicit LTRs, the forearm is supinated and the hand must hang

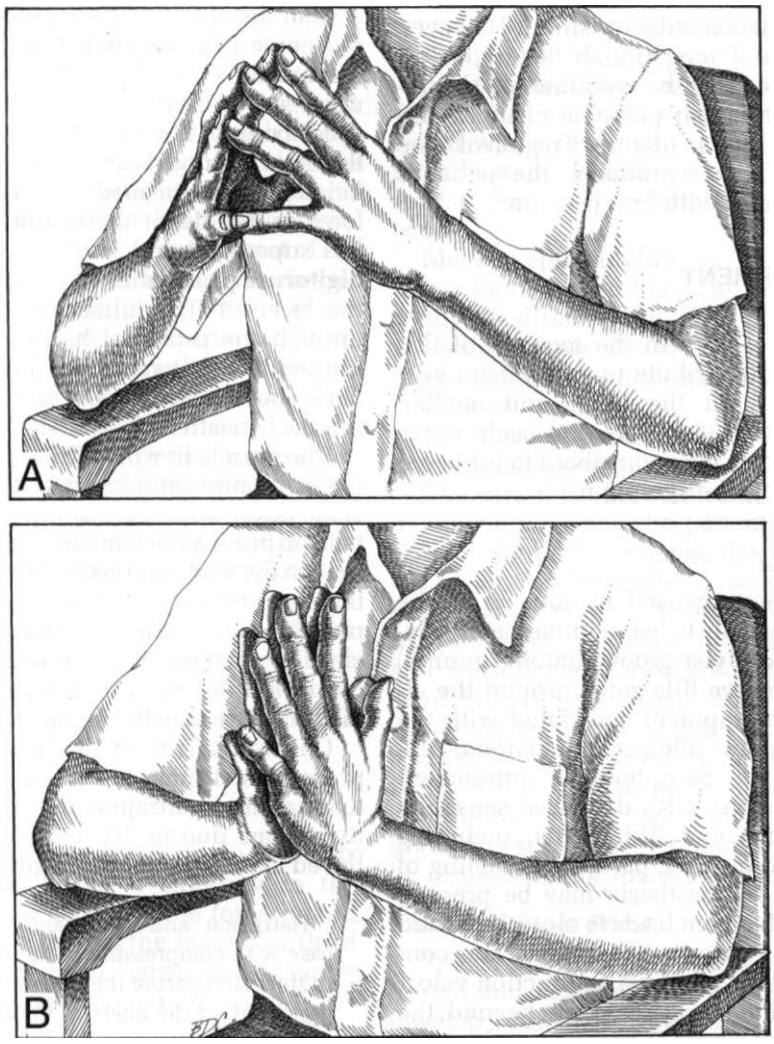


Figure 38.3. The Finger-extension Test showing some tightness of the hand and finger flexors. **A,** starting position. **B,** nearly normal extension. The final position must have the palms together and both forearms in a horizontal line for a completely negative, normal test.

Table 38.1 ***Muscles of the Forearm that may Develop TrPs which Cause Entrapment of the Ulnar or Median Nerve***

ULNAR NERVE
Flexor carpi ulnaris
Flexor digitorum superficialis
Flexor digitorum profundus
MEDIAN NERVE
Pronator teres
Flexor digitorum superficialis

limply in the extended position. However, the finger and long thumb flexors are so deeply placed that the examiner may be unable to distinguish palpable changes and may be able only to identify a region of deep tenderness that reproduces the patient's pain complaint with firm pressure.

10. ENTRAPMENT (Fig. 38.4)

Myofascial TrPs in the muscles of this chapter can contribute to entrapment syndromes in both the ulnar and median nerves. Table 38.1 lists for each nerve which muscles may contribute to (or be responsible for) the symptoms.

Ulnar Nerve

Entrapments caused by muscle in the forearm are likely to begin immediately distal to the condylar groove (cubital tunnel) which the nerve fills going around the elbow. Any entrapment associated with the cubital tunnel is often called a *cubital tunnel syndrome.* Symptoms of entrapment commonly begin with disturbed sensation in the fourth and fifth digits, including dysesthesia, burning pain and a feeling of numbness. Hypoesthesia may be present. Motor involvement leads to clumsiness and weakness of the grip. The diagnosis is confirmed by delayed nerve conduction velocity across, and to a lesser extent beyond, the point of entrapment.[41] The region of entrapment usually can be identified in this way as somewhere beyond the distal end of the condylar groove and within the first third of the forearm. Electromyographic determination of which muscles show neuropathic changes may further localize the lesion.

The ulnar nerve exits the upper arm through the medial intermuscular septum, to pass through a groove behind the medial epicondyle (Fig. **38.4A).** The nerve is held in this groove by a fibrous expansion of the common flexor tendon, which forms the roof of the cubital tunnel. From there, it enters the forearm beneath an aponeurotic arch formed by the humeral and ulnar heads of the flexor carpi ulnaris muscle,[21] commonly called the humeroulnar arcade. In **130** cadaver elbows, the arcade lay from **3** to **20** mm distal to the medial epicondyle and the nerve next coursed through the flexor carpi ulnaris muscle for **18** to **70** mm.[15] The ulnar nerve next occupies the triangular space bounded by three flexor muscles: the **flexor carpi ulnaris** covers the space superficially toward the medial (ulnar) side of the forearm, the **flexor digitorum superficialis** lies superficial and lateral, and the **flexor digitorum profundus** lies beneath, deep to the nerve.[16] The ulnar nerve continues through the proximal half of the forearm sandwiched between the flexor carpi ulnaris above it and the flexor digitorum profundus beneath it (Fig. **38.4B).**

The muscle in which TrPs would be most likely to cause entrapment of the ulnar nerve is the flexor carpi ulnaris. First, by taut band tension pulling the humeroulnar arcade tight against the nerve and second by compressing the nerve between taut bands of TrPs in the muscle where the nerve penetrates the muscle. These muscular entrapments are in addition to the causes of ulnar nerve entrapment that are usually enumerated.[44]

Clinically, TrPs in the flexor digitorum profundus at times also seem to contribute to ulnar nerve entrapment; how is not clear. Symptoms due to TrP entrapment are relieved by inactivating all contributing TrPs.

> Harrelson and Newman[37] reported a case with compression of the ulnar nerve in the distal part of the forearm due to hypertrophy of the fibers of the flexor carpi ulnaris muscle, which attached to the deep side of the distal 7 cm (2 3/4-inches) of the tendon, before the tendon attached to the pisiform bone. Excision of the mus-

cle relieved the patient's symptoms and neurological deficits. TrP tension may have been contributing to symptoms. Inactivating the TrPs would have required less drastic treatment.

A variation of the *flexor digiti quinti* caused ulnar nerve compression.[78]

Median Nerve

Median nerve entrapment below the elbow is most likely to cause paresthesias and hyperesthesia of the third and fourth digits sometimes including additional digits on either side[10] and is commonly called the *pronator teres syndrome.*[10,32] The nerve normally passes between the humeral and ulnar heads of the **pronator teres** beneath the fibrous arch between the two heads but sometimes pierces the humeral head.[32] The nerve then passes beneath the aponeurotic arch of the **flexor digitorum superficialis** that bridges between its radial and humeroulnar heads and clings to the underside of that muscle.[3] Myofascial TrPs might promote entrapment of the median nerve in both muscles by myofascial taut bands that increased the tension of the aponeurotic arch against the nerve and by direct compression of the nerve by taut bands of TrPs where the nerve penetrates the humeral head of the pronator teres.

Although clinical experience indicates TrPs can cause some of these entrapments, well planned case studies of the TrPs that include full electrodiagnostic documentation and adequate pre- and post-treatment outcome measures are sorely needed.

The presence of an anomalous **flexor digitorum superficialis indicis** muscle caused an acute carpal tunnel syndrome that was relieved by freeing the muscle from the median nerve.[7]

Radial Nerve

The presence of an anomalous **flexor carpi radialis brevis** originating in the proximal radial aspect of the forearm and inserting at the base of the second or third metacarpal has been implicated in anterior interosseous nerve compression.[45]

11. DIFFERENTIAL DIAGNOSIS

Differential diagnoses that are commonly identified when myofascial TrPs in flexor muscles of the forearm are responsible for or contribute to the symptoms include medial epicondylitis, ulnar neuropathy, carpal tunnel syndrome, osteoarthritis of the wrist, C_5 radiculopathy (when there are TrPs in the flexor pollicis longus muscle), C_7 radiculopathy with TrPs in the radial head of the flexor digitorum superficialis, and C_8 or T_1 radiculopathy with TrPs in the humeral head of the flexor digitorum superficialis muscle. There is also erroneous identification of thoracic outlet syndrome with active TrPs in the proximal part of the flexor digitorum superficialis, as some practitioners are prone to apply the term thoracic outlet syndrome to any disturbance of the fourth and fifth digits in the presence of a normal or nonfocal neurologic examination.

Articular dysfunctions likely to be associated with TrPs in this group of muscles include a distal radiocarpal dysfunction and/or dorsal subluxation of the carpal bones.

Medial epicondylitis is basically the same kind of problem as lateral epicondylitis discussed in Chapter 36, Section 11 under Tennis Elbow. However, in this case, a different condyle and different muscles are involved.

Ulnar neuropathy is reviewed in the preceding Section 10.

The **carpal tunnel syndrome** is likely to be diagnosed when the patient has active TrPs in the pronator teres, flexor carpi radialis, and/or brachialis muscles. The referred pain from even more distant TrPs in the sternocleidomastoid, infraspinatus and subscapularis muscles have tempted some to make the carpal tunnel diagnosis. A median nerve conduction study and examination of the muscles for TrPs establishes if one or both of the diagnoses are appropriate. Rarely, an anomalous flexor digitorum superficialis indicis muscle may be responsible for the symptoms.[7,39]

Related Trigger Points

Trigger points in the parallel flexores digitorum and flexores carpi muscles tend to develop together. However, TrPs may appear in the flexor carpi radialis alone following an elbow fracture, or comparable trauma.

Active TrPs in the finger flexors may develop as satellites to TrPs in muscles of the

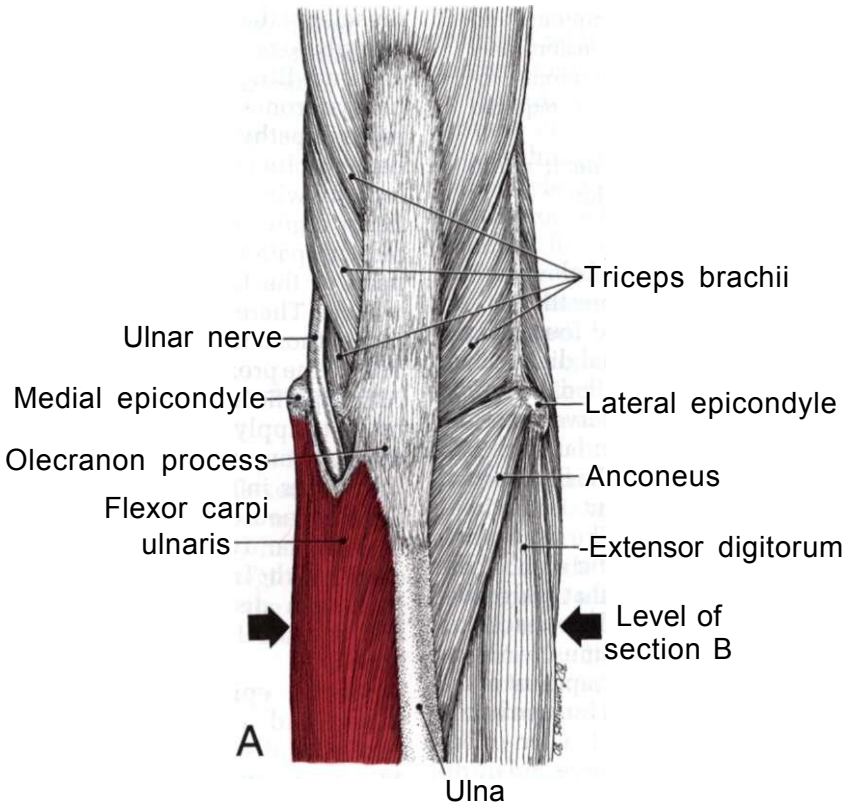


Figure 38.4. Dorsal view of the normal relation between the right ulnar nerve and the flexor carpi ulnaris muscle *(dark red)*. **A,** the tendinous arch between the muscle's humeral and ulnar heads, through which the ulnar nerve passes, is called the cubital tunnel.

shoulder and neck that refer pain into the volar forearm, especially when TrPs in these upper muscles tend also to cause nerve entrapment, such as the scalene or pectoralis minor muscles.

Myofascial TrPs in the flexor pollicis longus tend to develop independently of active TrPs in the other forearm flexor muscles.

12. TRIGGER POINT RELEASE (Fig. 38.5)

In addition to the spray-and-stretch technique described here, other techniques (including trigger point pressure release, reciprocal inhibition, postisometric relaxation, and contract-relax as described in Chapter 3, Section 12) are also effective for release of *central* trigger points (CTrPs) of the flexor muscles in the forearm. The primary therapeutic approach to *attachment* TrPs is to inactivate the CTrPs that are causing them.

Spray and Stretch

For treatment of involved flexores digitorum and flexores carpi radialis and ulnaris muscles, the patient lies comfortably with the elbow resting on a padded surface and the forearm supinated. The hand hangs over the edge of the support, so that the hand and fingers can be passively extended simultaneously, as the hand is pressed into full supination (Fig. 38.5A). Unless all three positions are established together, full stretch of the flexors is not obtained. Immediately before and while the muscles are being stretched, the vapocoolant spray is applied in parallel sweeps

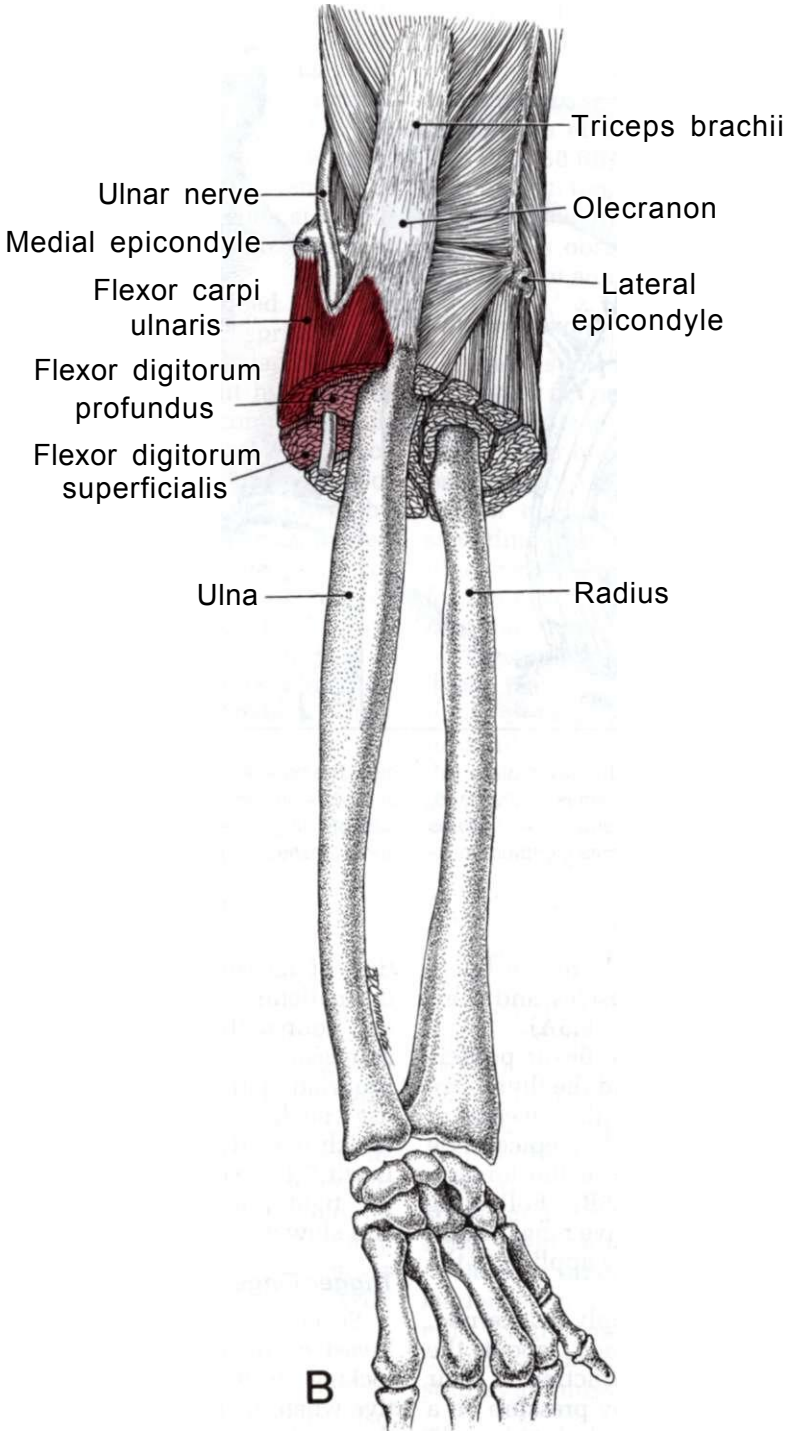


Figure 38.4—*continued.* **B,** cross section showing the relation of the ulnar nerve to the flexor carpi ulnaris *(dark red),* and the flexores digitorum superficialis and profundus muscles *(light red).* The section is several centimeters below the elbow in the region of the trigger points that may cause the nerve entrapment.

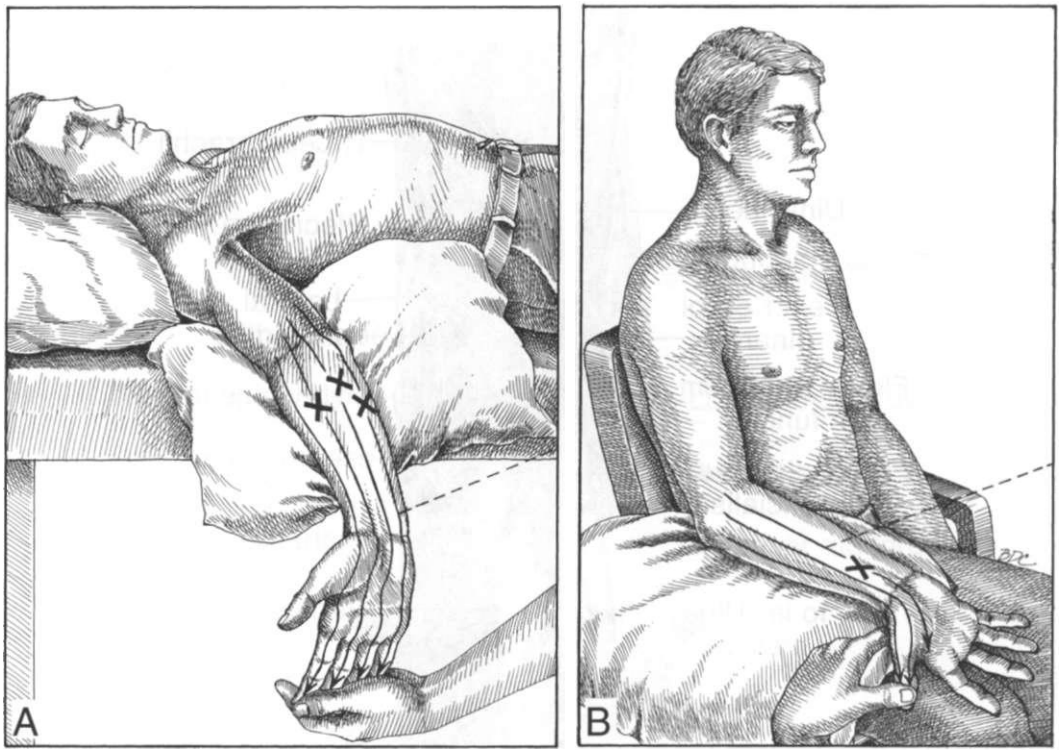


Figure 38.5. Stretch position and spray pattern *(arrows)* for trigger points (**Xs**) in muscles of the hand, thumb and finger flexors in the forearm. **A,** flexores carpi radialis and ulnaris, and flexores digitorum muscles. The patient's hand is supinated, elbow extended, and both the hand and fingers are fully extended. The patient's fingers are included in the downsweeps of spray *(clashed line).* **B,** flexor pollicis longus muscle.

from the medial epicondyle to the finger tips over the involved muscles and their referred pain patterns (Fig. 38.5A).

To stretch and spray the flexor pollicis longus muscle, the hand and the thumb are extended similarly, while the sweeps of spray travel from the medial epicondyle down over the radial side of the forearm and the thumb (Fig. 38.5B). Following three slow cycles of full active range of motion, moist heat is promptly applied to the volar forearm.

These patients are deeply concerned, and sometimes misinformed, about the cause of their pain. Reproduction of their pain during examination by pressure on a TrP demonstrates that the pain is primarily muscular in origin, and is therefore reassuring to them. After treatment, the demonstration of freedom from pain during a repeat of the Hand-grip Test *(see* Chapter 34, Section 8) and the normalization of measured grip strength reassures the patients that their pain has a myofascial source that is amenable to effective management. The patient must learn an appropriate self-stretch technique for home use such as postisometric relaxation, which is well described and illustrated by Lewit.[47] It involves a gentle contraction of the tight muscle followed by relaxation and slow exhalation.

Trigger Finger

Several techniques are available for noninvasive treatment of trigger finger. The locking mechanism seems to be less effective when the tendon is loaded (placed under tension). With the finger in the locked position, first have the patient flex it a bit more, then apply active resistance to place tension on the tendon, and then have the patient gradually let the finger extend while maintaining the tension. Simply pulling the

finger back into normal rest position passively seems, if anything, to aggravate the trigger mechanism, but this loading technique seems to facilitate its return to normal.

Sometimes firm pressure applied to the tender spot where locking occurs will restore normal function, as if the tendon or tendon sheath had become edematous locally and needed some help to return to normal.

Tsuyuguchi, *et al.*[76] described applying to **65** children a modified coil spring splint which maintained the interphalangeal joint in neutral extension or slight hyperextension. The authors reported complete healing in an average period of **9** months. Since there were no controls and the period of disability before treatment is unknown, the chance that they would have recovered spontaneously during this long period of compromised function is unknown. No mention was made of a trial of injection. Eight of the **65** patients required surgery.

A similar treatment employs a flexible splint placed around the proximal interphalangeal joint to restrict flexor action of that finger for a period that is long enough to significantly reduce the frequency of triggering.[70] This approach also restricts function.

13. TRIGGER POINT INJECTION (Fig. 38.6)

Usually, hand and finger flexor muscles respond well to stretch and spray. Their trigger points (TrPs) often do not require injection, except TrPs that aggravate an ulnar nerve entrapment at the elbow, and those responsible for trigger fingers. Injection is usually effective unless perpetuating factors have been overlooked. After TrP injection, residual muscle tension may be released by spray and stretch, and in any case must be followed by three slow cycles of full active range of motion of the wrist and fingers together.

Hand Flexors

Injection of TrPs in these muscles also has been found effective by others.[12,35] To inject a TrP in the **flexor carpi radialis,** the elbow of the supine patient is extended and the hand supinated. When the active TrP has been located by flat palpation, it is injected with **0.5%** procaine solution (Fig. **38.6A),** and then passively stretched before the hot pack is applied.

To inject an active TrP in the **flexor carpi ulnaris** muscle, the supine patient is asked to flex the elbow and laterally rotate the arm (Fig. **38.6B).** Since this TrP is quite superficial, it, too, is located by flat palpation and injected under direct tactile control. An LTR is observed when the needle encounters an active location in the TrP.

Finger Flexors

Tender spots in the superficial flexors are located by flat palpation and the area of focal tenderness is injected. The TrPs in the deep finger flexor muscles are usually located approximately **3** cm (about **1** 1/2-in) distal to the medial epicondyle. These deep TrPs are sometimes responsible for entrapment of the ulnar nerve, and are injected as illustrated for the flexor carpi ulnaris (Fig. **38.6B),** except that they lie deeper and require penetration to at least **2** cm (nearly **1** in); this depth reaches beyond the flexor carpi ulnaris, into the flexor digitorum sublimis or profundus. One obtains the impression that there may be a family of TrPs in several muscles. In ridding this muscular region of TrP activity, it is not uncommon to cause a temporary block of the ulnar nerve; the local anesthesia disappears in **15-20** min when **0.5%** procaine solution has been used.

Every muscle that was injected should be extended to full range of motion at once. First passively, usually during vapocooling, and then actively through three slow cycles of *full* range of motion in *both* directions. Moist heat is applied promptly.

Rachlin[62] illustrated the injection of a central TrP and the location of an attachment TrP in the flexor digitorum superficialis, and a central TrP location in the flexor carpi radialis longus.

Despite much clinical and theoretical evidence supporting this approach of specific TrP treatment for TrPs in these flexor muscles, adequately controlled research studies are missing and needed.

Trigger Finger

A trigger finger may be promptly and permanently relieved by injection, but the return of full function may not occur for several days following treatment. The needle tip is aligned with the midline of the fin-

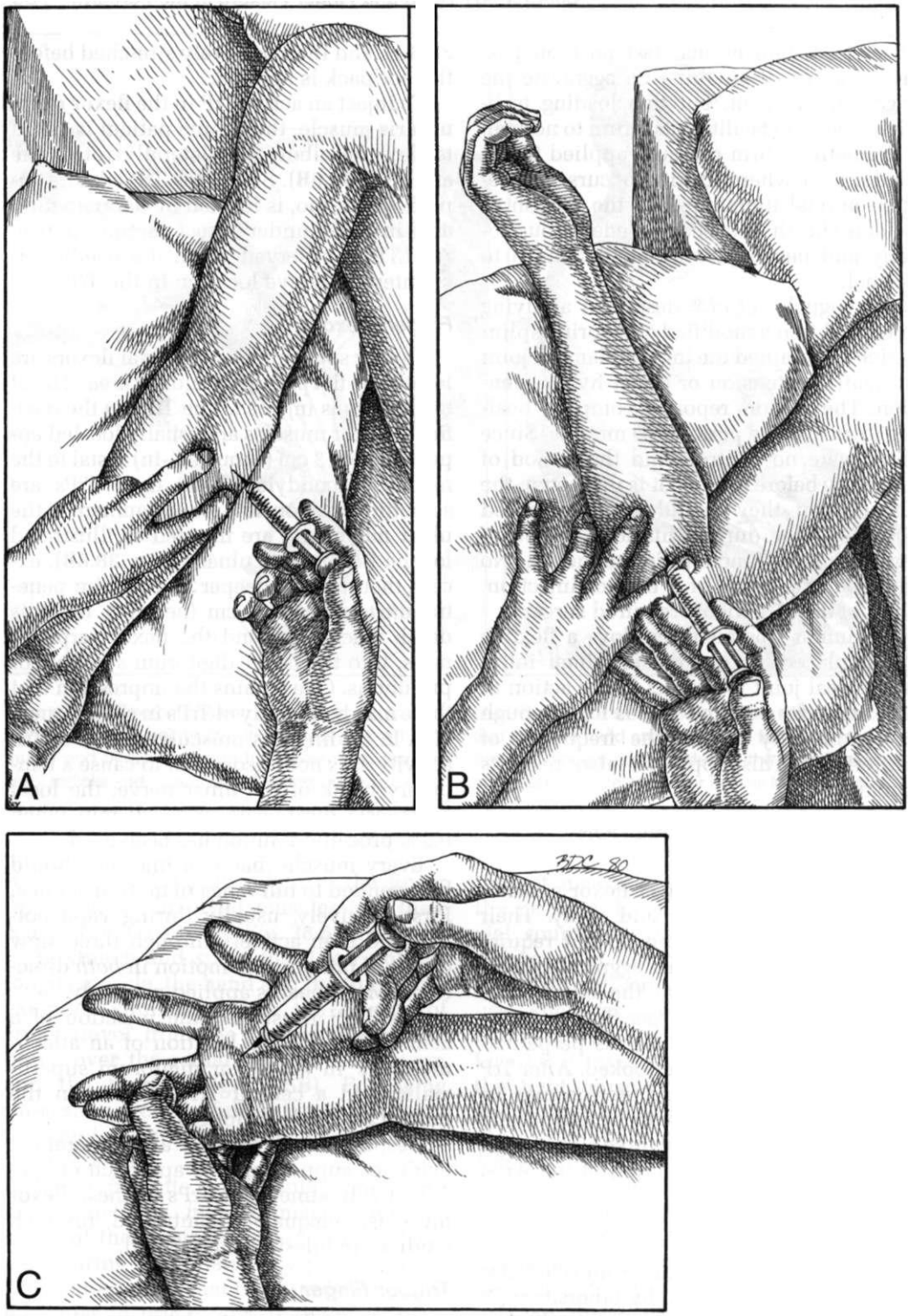


Figure 38.6. Injection technique for trigger points in the hand flexors, and for a trigger finger. **A,** flexor carpi radialis, with the elbow straight. **B,** flexor carpi ulnaris, with the forearm flexed and the arm laterally rotated to provide convenient access to this muscle. **C,** injection to relieve a trigger finger. The injection apparently releases a fibrous ring or swelling of the tendon that ensnares the flexor tendon of the middle finger.

ger and is inserted in the center of the tender point, apparently deep in the restricting fibrous ring around the flexor tendon, in the midline, just proximal to the head of the metacarpal bone (Fig. 38.6C). Injection of 1-1.5 ml of 0.5% procaine solution precisely into the tender spots suffices. No LTR is observed. This TrP must be purely fascial, and not located within muscular tissue.

A trigger thumb, present in one patient for 10 years following trauma to the upper extremity, was immediately and permanently unlocked by a single injection. This result is an example of Dr. Travell's experience. Trigger thumb is considered in detail in Chapter 39, Section 8.

A controlled double-blind prospective study of 41 patients with trigger finger or trigger thumb of at least 3 months' duration compared the result of injecting only 1% lignocaine or 20 mg or methylprednisolone acetate with lignocaine.[46] The success rate was 60% for injection of analgesic with steroid and 16% without steroid suggesting that an inflammatory component contributes to the symptom. An uncontrolled study of 68 cases of trigger finger showed a similar response rate of 60% to as many as three injections of equal parts of betamethasone and 0.5% lidocaine.[57] The authors were concerned about the possibility of tendon rupture with multiple injections, but did not specifically report any ruptures.

14. CORRECTIVE ACTIONS

When prolonged gripping, such as tightly holding a ski pole or a steering wheel, activates TrPs in the flexor muscles, the patient should learn to relax the grip frequently, to pronate the hand rather than holding it supinated, and to stretch the muscles at frequent intervals by positively reinforcing the desired behavior. Relaxation is aided by occasionally doing the Artisan's Finger-stretch Exercise (see Fig. 35.8) or the Finger-flutter Exercise (see Fig. 35.9). Grasping the sides of the steering wheel halfway between the top and bottom places the wrist in a more neutral position.

If the patient rows on a crew or paddles a canoe, he should fully open the fingers on the return stroke while holding the oax or paddle between the thumb and palm in order to relieve tension and to stretch the flexor muscles. For those playing racquet games, the wrist should be held in a neutral or slightly "cock-up" position and should not allow the racquet to droop. A patient with latent TrPs in the flexor muscles should learn to keep the hand, as well as the forearm, supported on the armrest when sitting and not to let the hand dangle over the end, thus avoiding leaving the hand and finger flexors in a shortened position.

When treating the hand and finger *extensors* by stretch and spray, a painful shortening activation of TrPs in the flexor muscles can be avoided by routinely applying stretch and spray to them. This additional treatment fully releases muscle tension from latent TrPs in the flexor muscles that are prone to activation.

In general, TrP activity in the hand and finger flexors can be avoided, or treated, by daily use of four different hand exercises: the Finger-extension Exercise (Fig. 38.7),

Figure 38.7. The Finger-extension Exercise is a self-stretch passive movement for inactivating trigger points and relieving tension in the hand and finger flexor muscles. The right forearm under stretch is well supported and relaxed.

the Artisan's Finger-stretch Exercise *(see* Fig. 35.8), the Finger-flutter Exercise *(see* Fig. 35.9), and the Interosseous-stretch Exercise *(see* Fig. 40.6).

The trigger finger is apparently a fascial entrapment which seems not to be influenced by muscle-stretch exercises. Recurrent heavy pressure on the tender spot against the metacarpal head, as by a cane or the handle of a tool, should be avoided.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:412 (Fig. 6.79).
2. *Ibid.* p. 413 (Fig. 6.81).
3. *Ibid.* pp. 413-415 (Figs. 6.81, r6.83, 6.84).
4. *Ibid.* p. 440 (Fig. 6.122A).
5. *Ibid.* p. 424 (Fig. 6.99).
6. Al-Qattan MM, Duerksen F: A variant of flexor carpi ulnaris causing ulnar nerve compression. *J Anat* 380:189-190, 1992.
7. Ametewee K, Harris A, Samuel M: Acute carpal tunnel syndrome produced by anomalous flexor digitorum superficialis indicis muscle. *J Hand Surg 10B(l):83-84,* 1985.
8. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 290, 294).
9. *Ibid.* (pp. 280, 281).
10. Bayerl W, Fischer K: [The pronator teres syndrome. Clinical aspects, pathogenesis, and therapy of a non-traumatic median nerve compression syndrome in the space of the elbow joint]. *Handchirurgie ll(2):91-98,* 1979.
11. Bickerton LE, Agur AM, Ashby P: Flexor digitorum superficialis: locations of individual muscle bellies for botulinum toxin injections. *Muscle Nerve* 20:1041-1043, 1997.
12. Bonica JJ, Sola AE: Other painful disorders of the upper limb. Chapter 52. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, 1990, pp. 947-958.
13. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sports Skill.* Charles C Thomas, Springfield, Ill., 1967.
14. Cailliet R: *Soft Tissue Pain and Disability.* FA. Davis, Philadelphia, 1977 (p. 188, Fig. 155).
15. Campbell WW, Pridgeon RM, Riaz G, *et al:* Variations in anatomy of the ulnar nerve at the cubital tunnel: pitfalls in the diagnosis of ulnar neuropathy at the elbow. *Muscle Nerve 14(8):733-738,* 1991.
16. Carter BL, Morehead J, Wolpert SM, *et al.: Cross-Sectional Anatomy.* Appleton-Century- Crofts, New York, 1977 (Sects. 53-58).
17. *Ibid.* (Sects. 53-59).
18. *Ibid.* (Sects. 53, 54, 56-63).
19. *Ibid.* (Sects. 56-61).
20. Clarkson PM, Dedrick ME: Exercise-induced muscle damage, repair, and adaptation in old and young subjects. *J Gerontol* 43(4):M91-M96, 1988.
21. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 531-535).
22. *Ibid.* (p. 534, Fig. 6-53).
23. *Ibid.* (p. 531, Fig. 6-51).
24. *Ibid.* (p. 530, Fig. 6-50).
25. *Ibid.* (p. 533, Fig. 6-52).
26. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 69).
27. *Ibid.* (Fig. 66).
28. *Ibid.* (Figs. 74, 75).
29. *Ibid.* (Fig. 68).
30. *Ibid.* (Fig. 108).
31. Ferner H, Staubesand J: *Sobotta Atlas of Human Anatomy.* Ed. 10, Vol.1, *Head, Neck, Upper Extremities.* Urban & Schwarzenberg, Baltimore, 1983 (p. 360).
32. Fuss FK, Wurzl GH: Median nerve entrapment. Pronator teres syndrome. Surgical anatomy and correlation with symptom patterns. Surg *Radiol Anat 12(4):267-271,* 1990.
33. Gerwin RD, Shannon S, Hong CZ, *et al.:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
34. Good MG: What is "fibrositis?" *Rheumatism* 5:117-123, 1949 (pp. 120, 121; Fig. 3).
35. Good MG: Acroparaesthesia—an idiopathic myalgia of elbow. *Edinburgh Med J* 56:366- 368,1949 (Case 1).
36. Good MG: The role of skeletal muscles in the pathogenesis of disease. *Acta Med Scand* 138:285-292, 1950 (p. 287).
37. Harrelson JM, Newman M: Hypertrophy of the flexor carpi ulnaris as a cause of ulnar- nerve compression in the distal part of the forearm. *J Bone joint Surg* 57A.-554-555, 1975.
38. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(1):29-59, 1994.
39. Hutton P, Kernohan J, Birch R: An anomalous flexor digitorum superficialis indicis muscle presenting as carpal tunnel syndrome. *Hand 13(1):85-86,* 1981.
40. Jozsa L, Demel S, Retry A: Fibre composition of human hand and arm muscles. *Gegenbaurs morph Hahrb, Leipzig* 227:34-38, 1981.
41. Kanakamedala RV, Simons DG, Porter RW, *et al:* Ulnar nerve entrapment at the elbow localized by short segment stimulation. *Arch Phys Med Rehabil* 69:959-963, 1988.
42. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3:175-190,1938 (pp. 179,188, 189).
43. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 240, 256-259).
44. Kopell HP, Thompson WA: *Peripheral Entrapment Neuropathies.* William & Wilkins, Baltimore, 1963 (pp. 113, 114, 116).
45. Lahey MD, Aulicino PL: Anomalous muscles associated with compression neuropathies. *Orthop Rev* 15(4):199-208, 1986.
46. Lambert MA, Morton RJ, Sloan JP: Controlled study of the use of local steroid injection in the treatment of trigger finger and thumb. *J Hand Surg 17B(1):69-*70, 1992.
47. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (p. 146-148, 202).
48. Lieber RL, Fazeli BM, Botte MJ: Architecture of selected wrist flexor and extensor muscles. *J Hand Surg 1*5A:244-250, 1990.

49. Lieber RL, Jacobson MD, Fazeli BM, *et al.:* Architecture of selected muscles of the arm and forearm: anatomy and implications for tendon transfer. *J Hand Surg 17A(5):7S7-798,* 1992.
50. Lundervold AJ: Electromyographic investigations of position and manner of working in typewriting. *Acta Physiol Scand* 24:(Suppl. 84), 1951.
51. McFarland GB Jr, Kursen UL, Weathersby HT: Kinesiology of selected muscles acting on the wrist: electromyographic study. *Arch Phys Med Rehabil* 43.165-171, 1962.
52. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 133E).
53. *Ibid.* (p. 133F).
54. *Ibid.* (pp. 140A, 141A, 142B).
55. *Ibid.* (No such figure).
56. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
57. Otto N, Wehbe MA: Steroid injections for tenosynovitis in the hand. *Orthop Rev 15(5):*290-293, 1986.
58. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Figs. 75, 76).
59. *Ibid.* (Figs. 81, 82).
60. *Ibid.* (Figs. 86, 87).
61. *Ibid.* (Figs. 85).
62. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (p. 342).
63. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 185, 197, 199, 200, 206).
64. Ruch TC, Patton HD: *Physiology and Biophysics.* Ed. 19. W.B. Saunders, Philadelphia, 1965 (pp. 375, 378).
65. Sano S, Ando K, Katori I, *et al.:* Electromyographic studies on the forearm muscle activities during finger movements. *J Jpn Orthop Assoc* 53:331-337, 1977.
66. Segal RL, Wolf SL, DeCamp MJ, *et al.:* Anatomical partitioning of three multiarticular human muscles. *Acta Anatomica 1*42:261-266, 1991.
67. Spalteholz W: *Handatlas der Anatomie des Men-schen.* Ed. 11, Vol. 2, S. Hirzel, Leipzig, 1922 (p. 326).
68. *Ibid*. (p. 327).
69. *Ibid*. (pp. 328, 329).
70. Swezey RL: *Arthritis: Rationale and Therapy and Rehabilitation.* W.B. Saunders, Philadelphia, 1978 (Fig. 57, p. 86).
71. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2. Vol. 1. Macmillan, New York, 1919 (pp. 321, 323).
72. *Ibid*. (p. 322).
73. *Ibid*. (p. 333).
74. *Ibid*. (p. 324).
75. *Ibid*. (pp. 331, 335, 336).
76. Tsuyuguchi Y, Tada K, Kawaii H: Splint therapy for trigger finger in children. *Arch Phys Med Rehabil 64:75-76,* 1983.
77. Weathersby HT, Sutton LR, Krusen UL: The kinesiology of muscles of the thumb: an electromyographic study. *Arch Phys Med Rehabil* 44:321-326,1963.
78. Weeks PM, Young VL: Ulnar artery thrombosis and ulnar nerve compression associated with an anomalous hypothenar muscle. *Plast Reconstr Surg* 69(1):130-131, 1982.
79. Winkelman NZ: An accessory flexor digitorum profundus indicis. *J Hand Surg 8(1):70-* 71, 1983.
80. Winter Z: Referred pain in fibrositis. *Med Rec* 257:34-37, 1944 (p. 4).

CHAPTER 39
Adductor and Opponens Pollicis Muscles (Trigger Thumb)

HIGHLIGHTS: "Weeder's thumb" is a painful disability of the thumb that is primarily due to active trigger points (TrPs) in the adductor and opponens pollicis muscles. The pain patterns and treatment approach for the opponens pollicis are similar to those of the abductor pollicis brevis and flexor pollicis brevis muscles. The latter two muscles lie partly over the opponens and are difficult to distinguish from it by palpation. **REFERRED PAIN** from both the adductor and opponens pollicis muscles projects to the radial and palmar aspects of the thumb; the opponens pollicis also may refer pain to the radial side of the palmar aspect of the wrist. **ANATOMY:** the medial attachment of the oblique head of the *adductor pollicis* is to the carpometacarpal region of the index and middle fingers. Medially, the transverse head attaches to the shaft of the third metacarpal bone. Laterally, both heads fasten to the base of the proximal phalanx of the thumb. The *opponens pollicis* extends from the trapezium bone of the wrist and the flexor retinaculum in the heel of the hand to wrap partially around and attach to the first metacarpal bone. The **FUNCTION** of the adductor pollicis is to adduct the thumb toward the index finger, while the opponens pollicis is essential in bringing the thumb pad across the palm to touch the pads of the ring or little fingers (opposition). **SYMPTOMS** due to active TrPs in these muscles are thumb pain during activity and, if severe, at rest, with awkwardness of pincer grip between the thumb and fingers. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in these muscles may be caused by strong, prolonged pincer gripping, as when sewing, weeding, writing longhand, and opening jar tops. **PATIENT EXAMINATION** should include a check for a Heberden's node on the ulnar side of the interphalangeal joint of the thumb, a node that is often associated with TrPs in the adductor pollicis. "Trigger thumb" usually is caused by a TrP located beside, and radial to, the flexor pollicis longus tendon, just proximal to the first metacarpophalangeal (MCP) joint. **DIFFERENTIAL DIAGNOSIS** should distinguish opponens pollicis referred pain from carpal tunnel syndrome. **TRIGGER POINT RELEASE** by spray and stretch requires maximal spread of the thumb away from the index finger while extending the thumb. Vapocoolant spray is swept radially over the thenar eminence and thumb, and proximally over the wrist. Trigger point pressure release of TrPs in the opponens pollicis can be helpful. **TRIGGER POINT INJECTION** of the adductor pollicis employs pincer palpation and digital needle guidance. The opponens pollicis requires flat palpation. "Trigger thumb" is relieved by injection of the tender point just radial to a point of possible ensnarement of the flexor pollicis longus tendon by the thickened flexor sheath at the distal end of the first metacarpal bone. **CORRECTIVE ACTIONS** include home exercises, such as, the Adductor Pollicis-stretch, the Opponens Pollicis-stretch, the Finger-flutter and the Finger-extension Exercises. These movements provide important intermittent relief during activities that require sustained or vigorous contraction of the thumb muscles.

1. REFERRED PAIN (Fig. 39.1)

Adductor Pollicis

An active trigger point (TrP) in the adductor pollicis muscle causes aching pain along the outside of the thumb and hand at the base of the thumb distal to the wrist crease (Fig. 39.1A). The spillover pain area hits the palmar surface of the first metacarpophalangeal (MCP) joint, and may include most of the thumb, thenar eminence, and dorsal web space.[3,41]

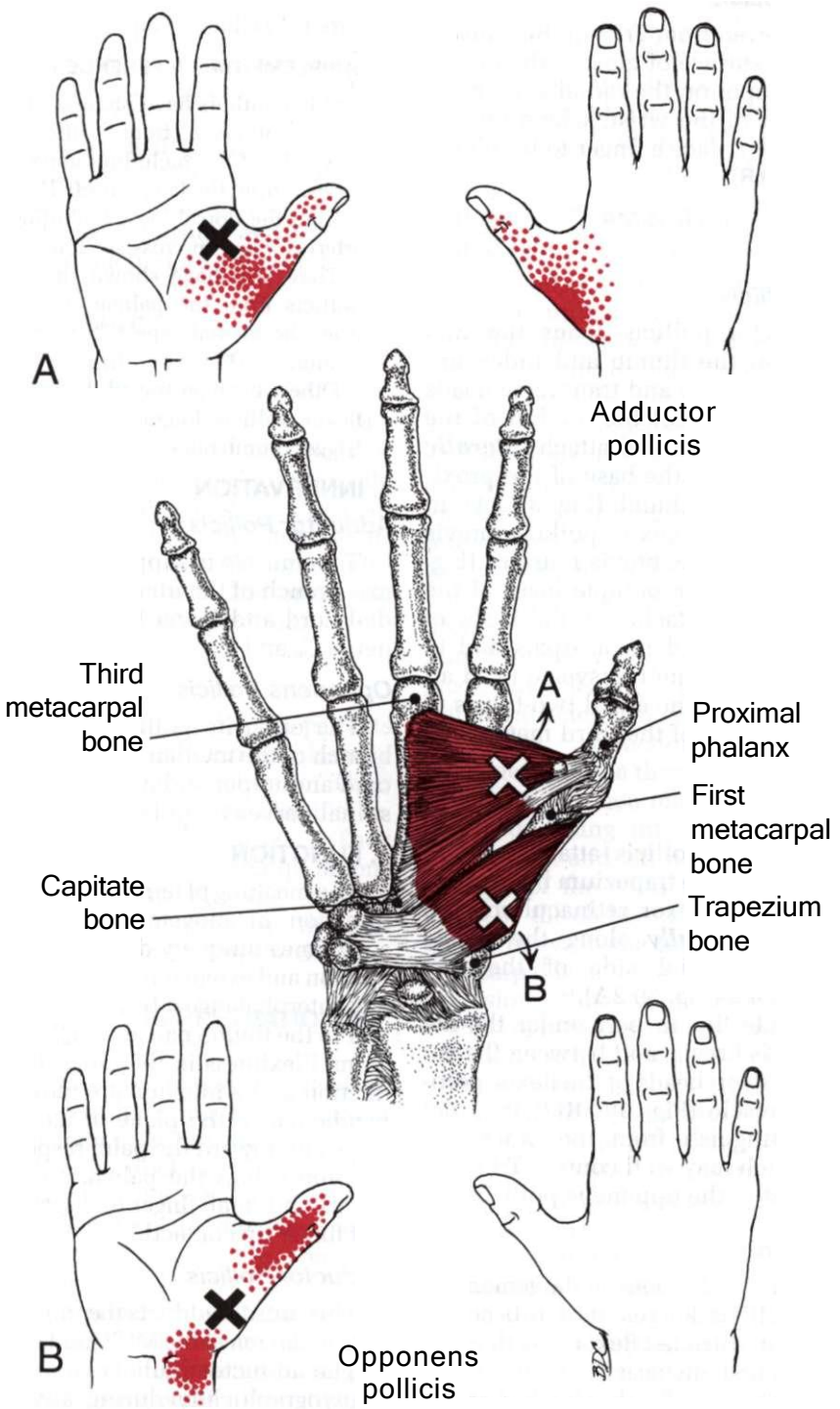


Figure 39.1. Referred pain patterns *(dark red)* and location of trigger points (**Xs**) for two thumb muscles *(medium red),* right hand. **A,** adductor pollicis. **B,** opponens pollicis.

Opponens Pollicis

Pain is referred from TrPs in this muscle to the palmar surface of most of the thumb and also to a spot on the radial side of the palmar aspect of the wrist, where the patient is likely to place a finger to locate the pain (Fig. 39.1B).

2. ANATOMY (Fig. 39.2)

Adductor Pollicis

The adductor pollicis spans the web space between the thumb and index finger. Both the oblique and transverse heads lie beneath (dorsal to) the tendon of the flexor pollicis longus and attach ***laterally*** to the ulnar side of the base of the proximal phalanx of the thumb (Fig. 39.2A), in common with the flexor pollicis brevis and abductor pollicis brevis muscles (Fig. 39.2B). ***Medially*** the oblique head of the adductor pollicis attaches to the bases of the second and third metacarpals and to the capitate bone. The transverse head attaches ***medially*** to the distal two-thirds of the palmar surface of the third metacarpal bone (Fig. 39.2A).[12]

Opponens Pollicis

The opponens pollicis attaches ***medially*** to a ridge on the trapezium bone of the wrist and to the flexor retinaculum, and ***laterally*** and ***distally*** along the whole length of the radial side of the first metacarpal bone (Fig. 39.2A).[12]

This muscle lies in part under the abductor pollicis brevis, and between the superficial and deep heads of the flexor pollicis brevis muscle (Fig. 39.2B).[12] It is not easy to distinguish from the other two muscles, which may well contain TrPs that are attributed to the opponens pollicis.

Trigger Thumb

Apparently, a bulbous enlargement of the flexor pollicis longus tendon becomes ensnared by a restricted flexor sheath at the head of the first metacarpal bone, where the tendon becomes firmly attached to the thumb after it has passed over the adductor pollicis and between the two heads of the flexor pollicis brevis muscle (Fig. 39.2B).[13] This triggering phenomenon is similar to that described for the tendons of the finger flexors (*see* Chapter 38).

SUPPLEMENTAL REFERENCES

Other authors have pictured the adductor pollicis from the palmar view[2,3,12,16,26,35,36] including nerves and arteries,[13] from the lateral (radial) aspect,[4,15,27] from the dorsal view including related arteries,[20] and in cross section.[1,10,17,30]

They also have shown the opponens pollicis from the palmar view,[3,5,12,16,35,37] from the medial aspect,[19,27] and in cross section.[11,17,30]

Others have portrayed the region of the flexor pollicis longus tendon where the trigger thumb phenomenon occurs.[2,14,18,28]

3. INNERVATION

Adductor Pollicis

This muscle is supplied by the deep palmar branch of the ulnar nerve from the medial cord and lower trunk through spinal nerve C_8 and T_1.

Opponens Pollicis

The opposite pollicis is supplied by a branch of the median nerve from the lateral cord and upper and middle trunks through spinal nerves C_6 and C_7.

4. FUNCTION

The meaning of terms used to describe the direction of movement are specific and sometimes uniquely defined for the thumb. Flexion and extension movement at the MCP and interphalangeal (IP) joints is perpendicular to the thumb nail and in the plane of the palm. Flexion is in the ulnar direction. Abduction and adduction are movements perpendicular to the plane of the palm, away from, and toward the palm respectively. Opposition brings the palmar surfaces of the thumb and small finger in direct contact (not just finger tip contact).[6,12,22,23]

Adductor Pollicis

This muscle adducts the thumb. It also assists in flexion at the MCP joint of the thumb.

The adductor pollicis is activated electromyographically during any adduction, opposition, and MCP flexion,[40] and especially during forceful opposition of the thumb, which rotates the thumb to face the other fingers.[6]

Opponens Pollicis

The opponens muscle of the thumb abducts,[9] flexes,[6, 22] and rotates the metacarpal bone of the thumb into a position of opposition.[6,21-23]

Electromyographically, the opponens was consistently active during opposition of the thumb and, surprisingly, was moderately active during extension, and markedly active during abduction, of the thumb.[7]

5. FUNCTIONAL UNIT

The abductor pollicis brevis, flexor pollicis brevis, and the opponens pollicis generally act together synergistically. The adductor and extensors of the thumb are their antagonists. Functionally, this group of abductors and their antagonists act in conjunction with the first dorsal interosseous and extrinsic finger muscles for forceful index-finger pinch, and with the opponens digiti quinti for forceful opposition.

6. SYMPTOMS

In addition to pain (Section 1), patients with active TrPs in these thumb muscles may complain that the thumb is "clumsy." Their handwriting often has become illegible because they "can hardly hold a pen." They have trouble with the fine manipulations necessary for buttoning clothing, sewing, drafting and painting that require the prehensile pincer grip provided by the thumb.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

A common syndrome, "weeder's thumb," is caused by activation of TrPs in these muscles when the patient pulls well-rooted weeds, like dock or plantain. The trouble arises when the patient repeatedly firmly grasps the base of the weed in a strong pincer grip, twists the weed to loosen the root, and then exerts an even stronger pincer grip to pull it. Sustained, unrelieved tension will activate these TrPs when using a fine paintbrush, sewing, or writing longhand—especially if writing requires pressing firmly with a ball-point pen that is held perpendicular to the paper.

When TrPs result from the stresses on muscle that are imposed during the fracture of a bone in the hand, patients may say, "Of course it hurts, I had a fracture there years ago." They do not realize that the hand should be pain-free when the bone has healed. They are unaware that the continuing pain is probably due to residual myofascial TrPs in the hand muscles.

8. PATIENT EXAMINATION (Fig. 39.3)

Since deep tenderness in the web space of the thumb may be referred from the scalene, brachialis, supinator, extensor carpi radialis longus, or brachioradialis muscles, these should be checked first for active TrPs. If these muscles are involved, they should be treated *before* attempting to inactivate TrPs in the thumb muscles; the tenderness in the region of the thumb, if referred, may disappear following inactivation of TrPs in the distant forearm and arm muscles. In the "weeder's thumb" syndrome, TrPs in the first dorsal interosseous muscle usually respond to treatment immediately, leaving the more complex thumb muscles still causing symptoms.

Flexion, adduction, and abduction movements of the thumb are weaker on the affected side when one of these muscles is involved, taking into account differences due to right and left hand dominance. The strength of the adductor pollicis is easily tested by the ability to hold a piece of paper tightly between the thumb and the second metacarpal bone. Abduction, and especially extension, of the thumb are often painful.

Pain and tenderness referred to the first MCP joint from TrPs in the adductor pollicis muscle are easily mistaken for evidence of joint disease if the myofascial origin of the symptoms is not recognized.[32] On the other hand, pain and dysfunction of the MCP and interphalangeal joints may be due to loss of joint play which should be identified and corrected.[29]

Heberden's nodes have been observed on the ulnar (inner) side of the thumb. When a node is present there, an associated TrP is nearly always found in the adductor pollicis muscle. This muscle adducts the thumb, much as the palmar interossei adduct the fingers, and the association with the Heberden's node probably has a similar basis (*see* Chapter 40).

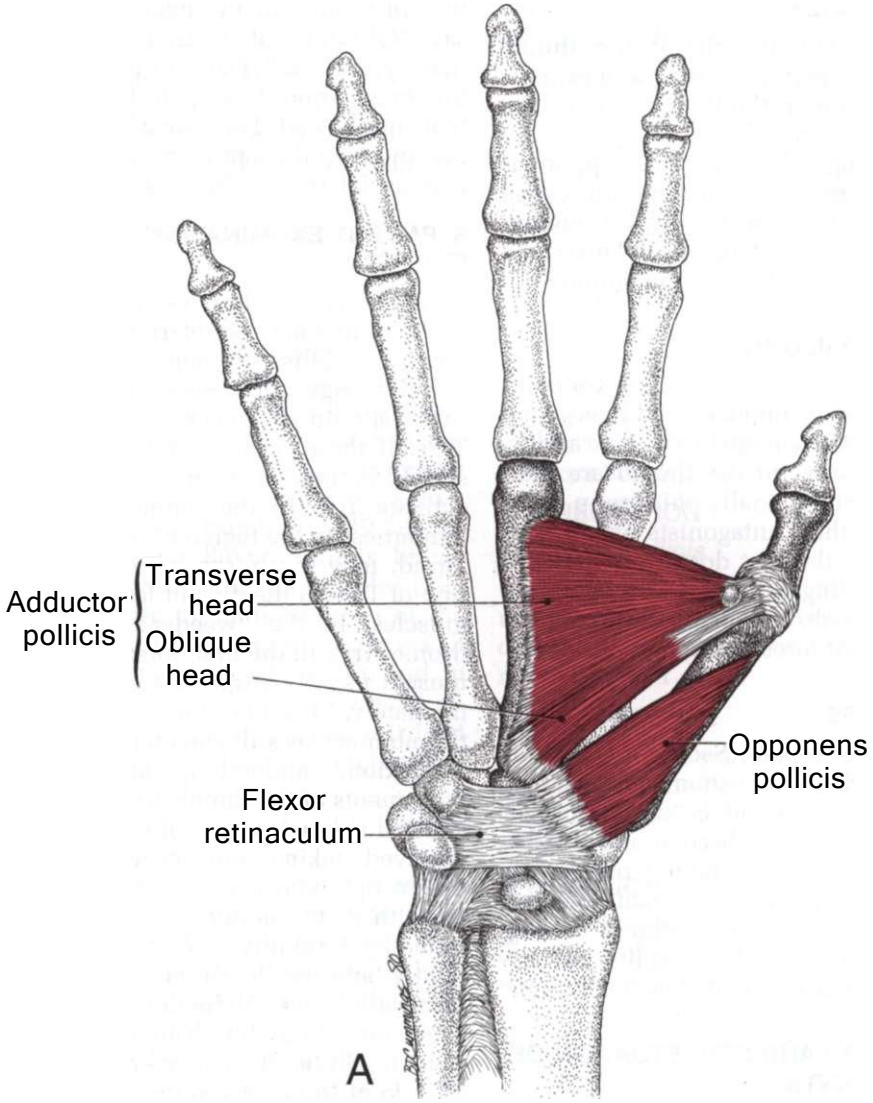


Figure 39.2. Attachments of thumb muscles. **A,** the adductor pollicis and opponens pollicis *(dark red)* after removal of the flexor pollicis brevis and abductor pollicis brevis muscles.

Trigger Thumb (Fig. 39.3)

The phenomenon of "trigger thumb" is identified by the patient's inability to extend the thumb without external assistance after flexing it; the thumb "locks" in flexion. The corresponding phenomenon, trigger finger, is considered in detail in Chapter 38, Sections 6 and 12.

The cause of the problem is associated with a tender spot located lateral to the tendon of the flexor pollicis longus, possibly in the flexor pollicis brevis. To locate this TrP, the patient supinates the forearm, fully extends the MCP joint of the thumb, and then alternately flexes and extends the distal phalanx, while the examiner identifies the tendon (Fig. 39.3). To identify the tendon of the flexor pollicis longus, the examiner places a finger against the bulge of the MCP joint, pressing on the space between the flexor pollicis brevis and the ad-

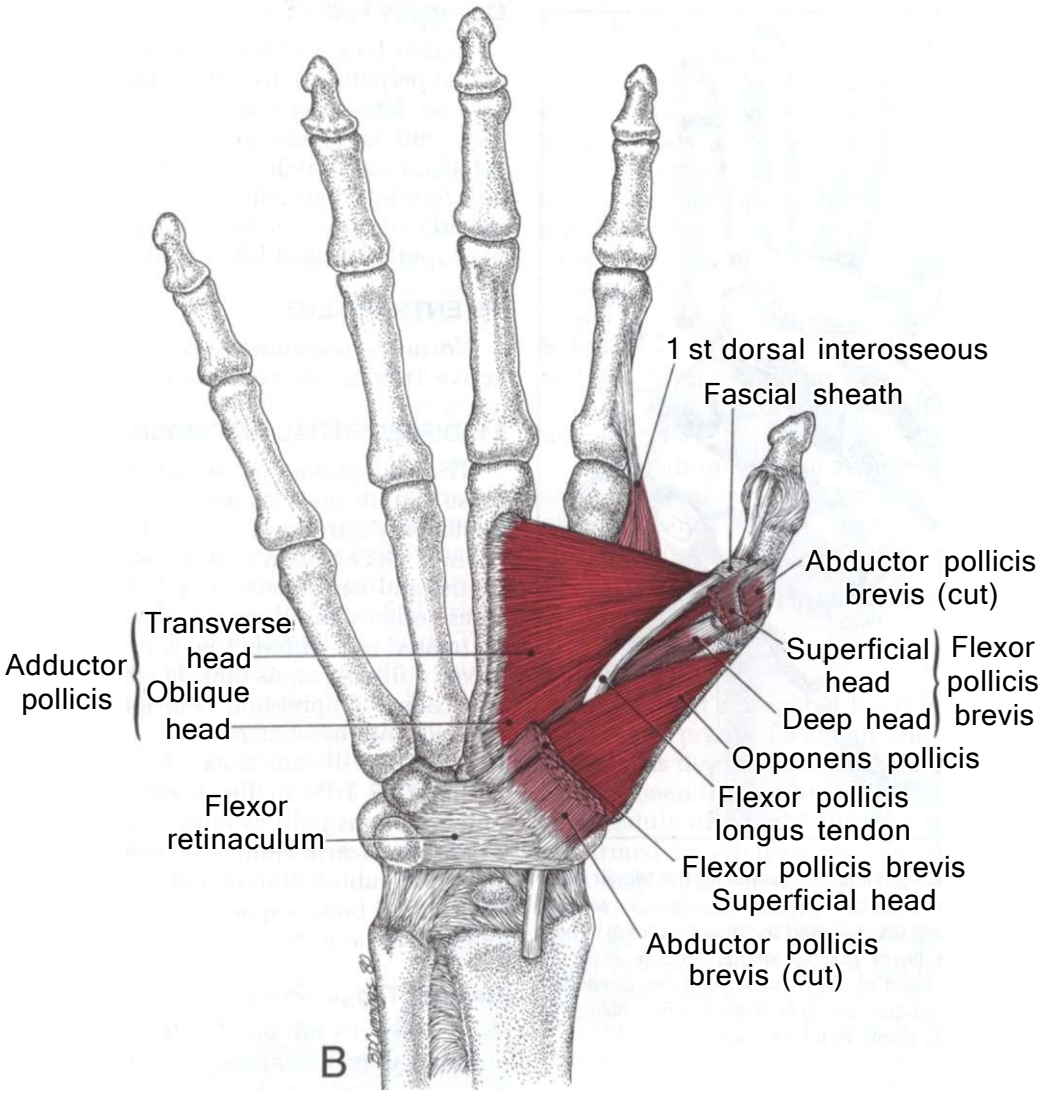


Figure 39.2–*continued.* **B,** course of the tendon of the flexor pollicis longus muscle with restraining fascial sheath at the head of the first metacarpal close to the metacarpophalangeal joint, and the cut attachments of the overlying *(light red)* flexor pollicis brevis and abductor pollicis brevis muscles.

ductor pollicis muscles where the tendon of the flexor pollicis longus enters the fascial sheath of the thumb (Fig. 39.2B). As the patient moves the distal phalanx back and forth, the cord of the subcutaneous tendon is located proximal to where it enters the anchoring arch of fibers at the head of the first metacarpal bone in the region of the "trigger" phenomenon. The TrP tenderness usually is located several millimeters *lateral [radial)* to the tendon, just proximal to the bony bulge of the MCP joint.

Locking of the interphalangeal joint of the thumb may be caused by a sesamoid bone of that joint.[9]

Of 30 patients presenting with trigger thumb, 25 were followed to spontaneous resolution without treatment. Five demanded treatment. The average duration of symptoms to spontaneous recovery was 6.8 months (range 2-15).[34]

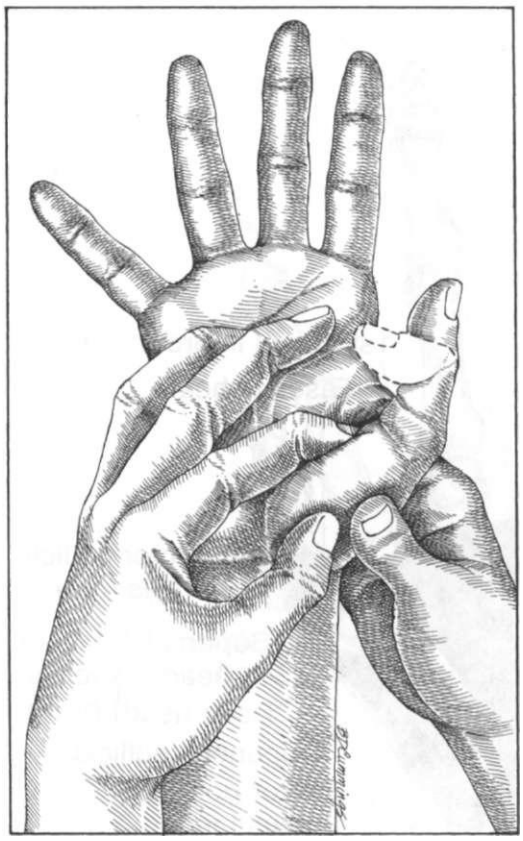


Figure 39.3. Technique for palpating the trigger point of a "trigger thumb." The distal phalanx is wiggled back and forth (as indicated by "ghosting" of it) to help identify the flexor pollicis longus tendon. Pressure against the head of the metacarpal bone, radial (lateral) to the tendon, elicits spot tenderness. Needle in Figure 39.5**C** points to tender spot.

9. TRIGGER POINT EXAMINATION

Adductor Pollicis

With the patient seated comfortably and the hand pronated and relaxed, the web space of the thumb is examined by pincer palpation, through the dorsal approach. The first dorsal interosseous muscle, which lies superficial to the transversely oriented adductor fibers, is pushed aside. The nodule with exquisite spot tenderness in a taut band, referred pain that the patient recognizes, and (for skilled examiners) local twitch responses are elicited from active TrPs of the adductor pollicis muscle.

Opponens Pollicis

Active TrPs in this muscle are identified by flat palpation across the direction of the muscle fibers over the thenar eminence (Fig. 39.2A). When the TrP is deeply located, a local twitch response is more difficult to elicit than when the TrP lies in the superficial abductor or flexor pollicis brevis superficial head fibers (Fig. 39.2B).

10. ENTRAPMENT

No nerve entrapments are attributed to active TrPs in these muscles.

11. DIFFERENTIAL DIAGNOSIS

The symptoms produced by TrPs in these thumb muscles are most commonly mistakenly attributed to carpal tunnel syndrome, DeQuervain's stenosing tenosynovitis, and carpometacarpal osteoarthritis. These other conditions can exist and must be treated in a different way. An accessory flexor pollicis longus muscle when present can cause compression neuropathy of the anterior interosseous nerve.[24]

Articular dysfunctions that can relate strongly to TrPs in the adductor pollicis and opponens pollicis muscles are those at a carpometacarpal joint, the most likely being volar subluxation of a metacarpal bone on a carpal bone, especially at the first carpometacarpal joint.

Related Trigger Points

Active TrPs are nearly always found in the first dorsal interosseous muscle when they are present in the adductor and opponens pollicis. Repeatedly, one gains the impression that the thumb muscles are involved primarily, and the first dorsal interosseous is affected secondarily, due to its synergistic function.

The flexor pollicis brevis and abductor pollicis brevis muscles eventually are also likely to become involved.

12. TRIGGER POINT RELEASE (Fig. 39.4)

To release trigger point (TrP) involvement in the adductor and opponens pollicis muscles using spray and stretch, the forearm is supinated while resting on a supporting surface that permits full thumb

extension (Fig. 39.4) and adduction to lengthen the opponens, and then the thumb is abducted to the onset of resistance to lengthen the adductor. The vapocoolant spray is applied in parallel sweeps across the palm, toward and over the radial surface of the thumb, while the adductor and opponens pollicis muscles are extended to take up the slack. A proximal spray pattern is added across the radial side of the wrist to cover the pain pattern of the opponens pollicis. Three slow full cycles of active range of motion are followed by application of moist heat.

Another manual release is to lengthen the opponens pollicis muscle as illustrated in Figure 39.4, but instead of applying intermittent cold, the clinician applies trigger point pressure release on the TrP and combines this with contract-relax by having the patient contract the muscle during the application of pressure and then fully relax while the clinician takes up the slack. This step of application of pressure with voluntary contraction, of course, can be alternated with application of intermittent cold. When properly coordinated this approach can be remarkably effective. It may be several hours before local tenderness begins to subside and the next day or two until the thumb becomes less symptomatic. It may be necessary to repeat this treatment every day or two for several times before the TrP is completely inactivated and painless normal thumb function returns. A few patients are able to learn how to do this complicated but effective procedure as self-treatment. This procedure teaches the person doing it to himself or herself a lot about how TrPs respond to this kind of manual treatment. It helps the person to get a "feel" for an optimal treatment technique.

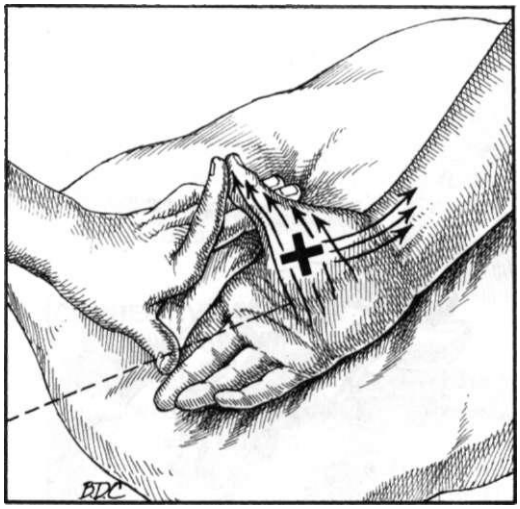


Figure 39.4. Stretch position and spray pattern *(arrows)* for a trigger point in either the adductor or opponens pollicis muscle. The **"X"** locates the adductor pollicis trigger point. The spray sweeps across the palm and thenar eminence to the end of the thumb. The up-pattern of spray across the wrist is added when the opponens pollicis is involved.

Spray and stretch of these muscles are not always as effective as TrP injection.

Trigger thumb is not released by spray and stretch alone. Sometimes application of trigger point pressure on the tender spot at the point of restriction is effective.

13. TRIGGER POINT INJECTION (Fig. 39.5)

Adductor Pollicis

The patient's pronated hand is palpated for trigger points (TrPs) in the adductor pollicis, as described in Section 9. When a TrP has been located by its spot tenderness in a nodule of a taut band and sometimes confirmed by eliciting an LTR, the operator's finger presses against it from the palmar side to fix it and provide guidance (Fig. 39.5A). As the needle is directed toward this guiding finger, it should pass to the radial side of, or perhaps penetrate, the first dorsal interosseous muscle. Following the injection, the muscle is passively stretched while release of the muscle is aided by sweeps of vapocoolant, three slow cycles of full active range of motion and followed by application of moist heat.

Opponens Pollicis

When a TrP in this muscle has been located by flat palpation (Section 9), it may be injected as illustrated in Figure 39.5B. This injection is also illustrated by Rachlin.[31] The muscle is then passively stretched during vapocoolant application (Fig. 39.4), moved through full range, and the skin rewarmed.

Trigger Thumb

The flexor pollicis longus tendon, and the tender area apparently responsible for

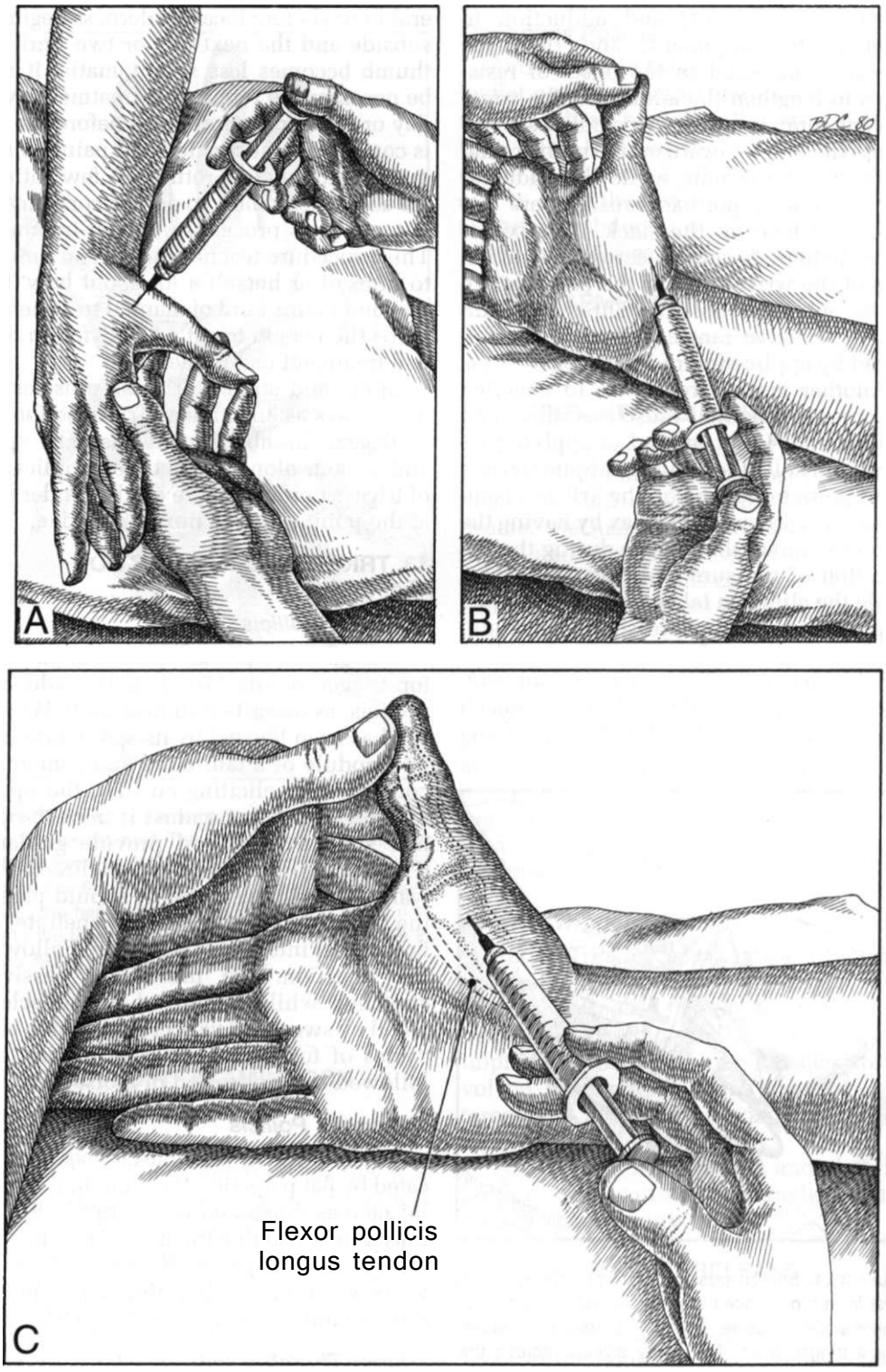


Figure 39.5. Techniques of trigger point injection. **A,** dorsal approach for the adductor pollicis muscle. **B,** palmar approach for the opponens pollicis. **C,** injection for "trigger thumb."

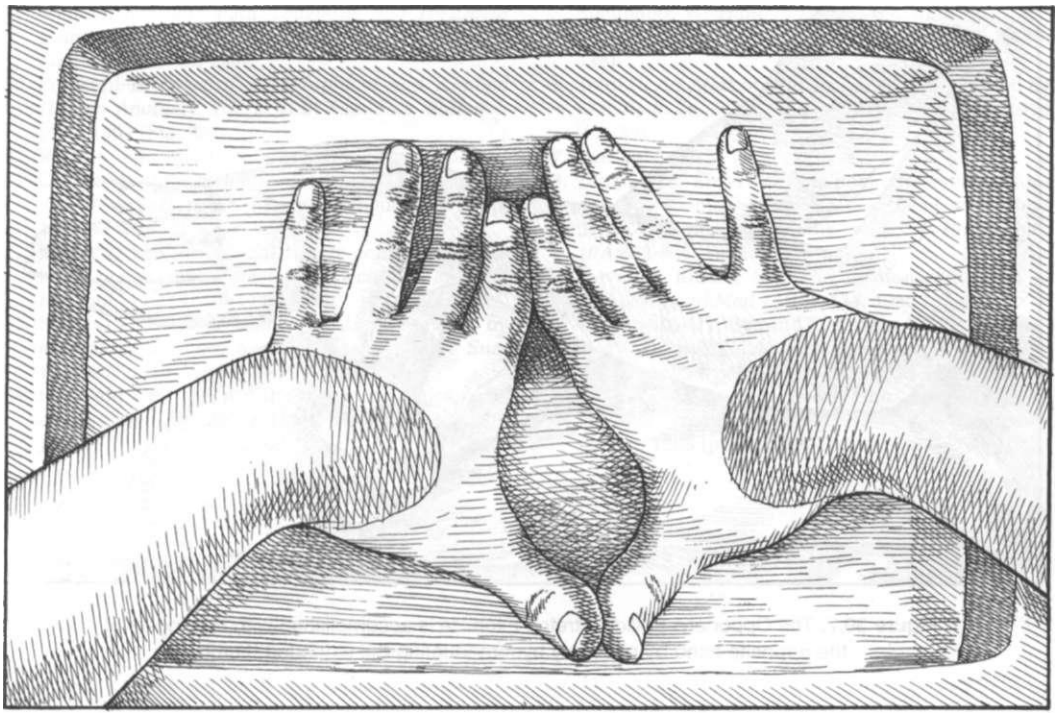


Figure 39.6. The Adductor Pollicis-stretch Exercise is performed by pressing the thumb and index finger apart on each hand, in a basin of warm water.

its ensnarement are first located by palpation. With the thumb fully extended as described in Section 8, the tender spot is injected as illustrated in Figure 39.5C. The needle probes widely down to the head of the first metacarpal bone, lateral and deep to the tendon, which usually need *not* be injected to eliminate the locking mechanism in the thumb.

14. CORRECTIVE ACTIONS (Figs. 39.6 and 39.7)

The patient should avoid persistent, vigorous weeding by limiting the time spent, by alternating hands in this activity, or by loosening the dirt with a spading fork before pulling the weeds out. He or she should learn to use a soft felt-tip pen, which requires much less pressure on the paper than does a ball-point pen, especially when the latter is held in the up-ended position. The hours spent doing needlepoint continuously should be limited.

The patient can also be taught to do the Adductor Pollicis-stretch Exercise (Fig. 39.6) by placing the hands in a basin of warm water, while pressing the thumbs and index fingers of both hands against each other, to achieve full passive abduction and extension of the thumbs.

The Opponens Pollicis-stretch Exercise (Fig. 39.7) is performed by fully extending and then passively adducting the thumb, with the fingers of the opposite hand providing the external force. A different line of muscle fibers (specifically, the opponens) is stretched if less extension is applied during the hyperadduction stretch. This stretch is more effective if done under a warm shower or with the hands in warm water.

Reactivation of "weeder's thumb" can be avoided by having the patient frequently interrupt the gardening activity with the Artisan's Finger-stretch Exercise (see Fig. 35.8). Artisans who use tools that require sustained muscular tension for fine finger control also are taught to pause and break

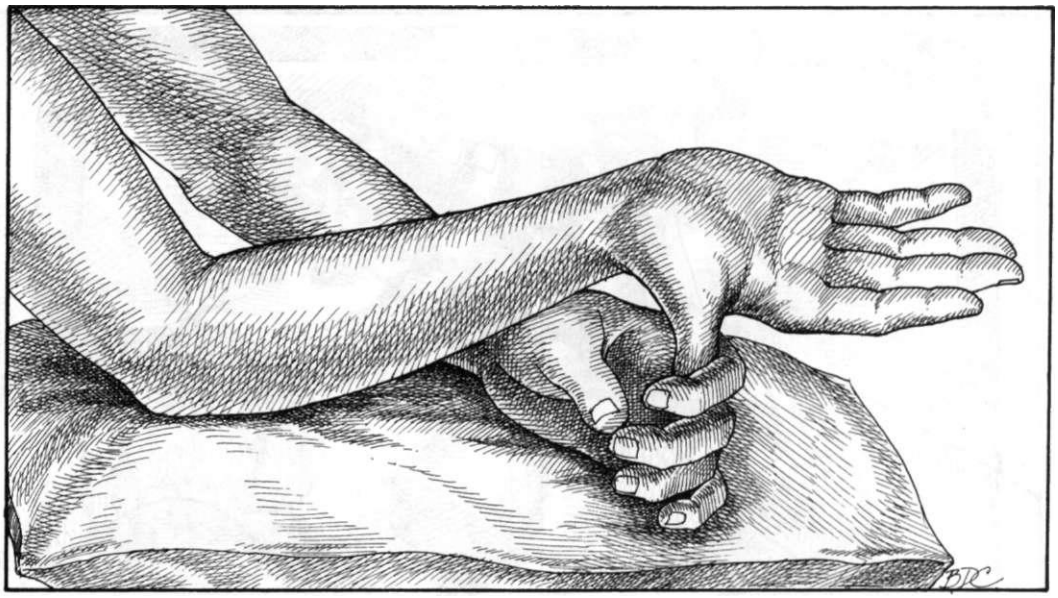


Figure 39.7. The Opponens Pollicis-stretch Exercise is accomplished with the fingers of the opposite hand by passively hyperadducting the extended thumb.

the sustained activity every 10 or 15 min by a stretch exercise, such as the Finger-extension Exercise (*see* Fig. 38.7). In the Finger-flutter Exercise (*see* Fig. 35.9), the patient drops the hands at the side with elbows straight and shakes the fingers loosely in a limp fluttery motion. This should relax the muscles and increase their circulation.

CASE REPORT

Bieber[8] described the diagnosis and treatment of distressing TrPs in the adductor pollicis and opponens pollicis muscles.

REFERENCES

1. Agur AM: ***Grant's Atlas of Anatomy.*** Ed. 9. Williams & Wilkins, Baltimore, 1991:443 (Fig. 6.129).
2. ***Ibid.*** p. 419 (Fig. 6.90).
3. ***Ibid.*** p. 422 (Fig. 6.95).
4. ***Ibid.*** p. 435 (Figs. 6.116B, 6.116C).
5. ***Ibid.*** pp. 414, 415, 420 (Figs. 6.83, 6.84, 6.91).
6. Basmajian JV, DeLuca CJ: ***Muscles Alive.*** Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 297, 306, 307).
7. ***Ibid.*** (pp. 299, 300).
8. Bieber B: The role of trigger point injections in the development of private practice. ***Phys Med Rehabil Clin North Am*** 8(1):197-205, 1997.
9. Brown M, Manktelow RT: A new cause of trigger thumb. ***J Hand Surg (Am)*** 217A:688- 690, 1992.
10. Carter BL, Morehead J, Wolpert SM, ***et al: Cross-Sectional Anatomy.*** Appleton-Century- Crofts, New York, 1977 (Sect. 60).
11. ***Ibid.*** (Sects. 59, 60).
12. Clemente CD: ***Gray's Anatomy.*** Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 550-552, Fig. 6-63).
13. ***Ibid.*** (Fig. 12-48).
14. ***Ibid.*** (Fig. 6-64).
15. Clemente CD: ***Anatomy.*** Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 61).
16. ***Ibid.*** (Figs. 107, 114).
17. ***Ibid.*** (Fig- 121).
18. ***Ibid.*** (Fig. 107).
19. ***Ibid.*** (Fig. 112).
20. ***Ibid.*** (Fig. 105).
21. Forrest WJ, Basmajian JV: Functions of human thenar and hypothenar muscles. *J* ***Bone Joint Surg*** 47A:1585-1594, 1965.
22. Jenkins DB: ***Hollinshead's Functional Anatomy of the Limbs and Back.*** Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 165, 166).
23. Kendall FP, McCreary EK, Provance PC: ***Muscles: Testing and Function.*** Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 19, 237, 239).
24. Lahey MD, Aulicino PL: Anomalous muscles associated with compression neuropathies. ***Orthop Rev*** 15(4):199-208, 1986.
25. Luethke R, Dellon AL: Accessory abductor digiti minimi muscle originating proximal to the wrist causing symptomatic ulnar nerve compression. ***Ann Plast Surg 28(3):307-308,*** 1992.
26. McMinn RM, Hutchings RT, Pegington J, et ***al: Color Atlas of Human Anatomy.*** Ed. 3. Mosby-Year Book, St Louis, 1993 (pp. 140A, 144A).
27. ***Ibid.*** (p. 150B).
28. ***Ibid.*** (p. 140A).

29. Mennell JM: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques.* Little, Brown & Company, Boston, 1964.
30. Pernkopf E. *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 92).
31. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (p. 354).
32. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981 (Table 1).
33. Salgeback S: Ulnar tunnel syndrome caused by anomalous muscles. *Scand J Plast Reconstr Surg* 21:255-258, 1977.
34. Schofield CB, Citron ND: The natural history of adult trigger thumb. *J Hand Surg* 18B:247-248, 1993.
35. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2, S. Hirzel, Leipzig, 1922 (p. 338).
36. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 334).
37. *Ibid.* (p. 335).
38. Tonkin MA, Lister GD: The palmaris brevis profundus. An anomalous muscle associated with ulnar nerve compression at the wrist. *J Hand Surg* 30/1:862-864, 1985.
39. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 31:425-434, 1952 (p. 428).
40. Weathersby HT, Sutton LR, Krusen UL: The kinesiology of muscles of the thumb: an electromyographic study. *Arch Phys Med Rehabil* 44:321-326, 1963.
41. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Little, Brown & Company, Boston, 1988 (p. 211, Fig. 12-2).

CHAPTER 40
Interosseous Muscles of the Hand, Lumbricals, and Abductor Digiti Minimi

HIGHLIGHTS: Heberden's nodes may be associated with trigger points (TrPs) in the interosseous musculature of the hand. **REFERRED PAIN** from either the dorsal or palmar interosseous muscles extends along the side of the finger to which that interosseous muscle attaches and, in the case of the first dorsal interosseous, may include the dorsum of the hand and ulnar side of the little finger. Pain from the lumbrical muscles is not distinguished from that referred by the interossei. The **FUNCTION** of each dorsal interosseous is to move a finger away from the midline of the middle finger (abduction). The abductor digiti minimi abducts the little finger. The palmar interossei adduct each of the other fingers toward the middle finger. A lumbrical muscle inhibits flexion of a distal finger phalanx selectively. **SYMPTOMS** caused by active TrPs in the interossei include pain, finger stiffness, and awkwardness. These TrPs are often associated with a tender nodule on the distal interphalangeal (IP) joint. This nodule, a Heberden's node, is closely associated with osteoarthritis of the distal IP joint. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the interossei are caused by prolonged or repetitive pincer grasp. **TRIGGER POINT EXAMINATION** reveals spot tenderness in the involved muscle; referred pain is rarely elicited and local twitch responses are not evident. **ENTRAPMENT** of digital nerves by the interossei is seen occasionally. **TRIGGER POINT INJECTION** is usually more effective than spray and stretch or trigger point pressure release in eliminating these TrPs. **CORRECTIVE ACTIONS** entail a change in daily activities and the interruption of sustained muscular contraction by the Finger-flutter, Finger-extension, Adductor Pollicis-stretch, and the Interosseous-stretch Exercises, as appropriate.

1. REFERRED PAIN (Fig. 40.1)

The first dorsal interosseous trigger points (TrPs) refer pain strongly down the same (radial) side of the index finger and deeply in the dorsum and through the palm of the hand (Fig. 40.1A). The referred pain also may extend along the dorsal and ulnar sides of the little finger.[56,58] Generally, patients experience the most intense pain at the distal interphalangeal (IP) joint where a Heberden's node may appear.

The first dorsal interosseous TrPs are the second most frequent source of referred pain in the palm, exceeded only by TrPs in the palmaris longus. Some patients have difficulty in deciding whether the pain referred from first dorsal interosseous TrPs is more severe on the palmar or on the dorsal aspect of the hand.

Myofascial TrPs in the remaining dorsal and palmar interossei refer pain along the side of the finger to which that interosseous muscle attaches (Fig. 40.1C). No distinction is made between the patterns of pain referred from the dorsal interossei, the palmar interossei, and the lumbrical muscles. Pain extends as far as the distal IP joint. The exact pain pattern varies somewhat, depending on the location of the TrP in the interosseous muscle. An active TrP in an interosseous muscle may be associated with a Heberden's node located within the TrP zone of referred pain and tenderness.

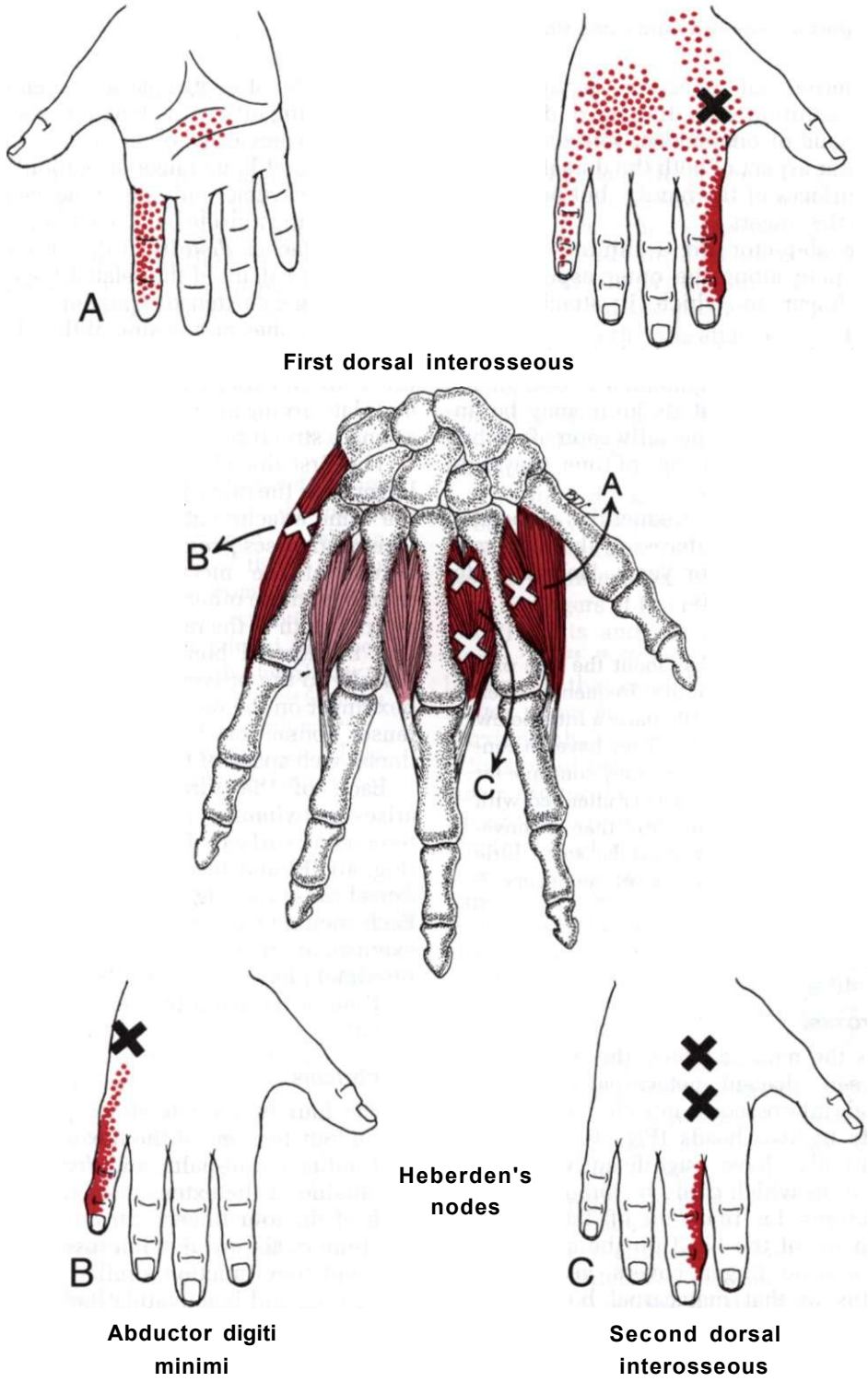


Figure 40.1. Referred pain patterns (*dark red)* and location of trigger points (**Xs**) for selected intrinsic muscles of the right hand. Essential zones are *solid red,* spillover zones are *stippled red.* **A,** the first dorsal interosseous *(medium red).* **B,** the abductor digiti minimi *(medium red).* **C**, the second dorsal interosseous *(medium red)* and the third and fourth dorsal interossei *(light red).* Trigger points may be found anywhere in the interossei, proximally or distally. This is to be expected since the two heads converge in a bipenniform manner and have endplate zones running in the shape of a horse-shoe the length of the muscles (see Fig. 2**.8B**). Note the small Heberden's nodes in the essential pain reference zones.

Experimental injection of hypertonic saline solution into the third dorsal interosseous of one subject referred pain to the ulnar aspect of both the dorsal and palmar surfaces of the hand,[27] but apparently not to the fingers.

The abductor digiti minimi similarly refers pain along the outer aspect of the little finger to which it attaches (Fig. 40.1B).

Heberden's nodes, which develop on the dorsolateral or dorsomedial aspect of the terminal phalanx at its joint, may be annoyingly tender, especially soon after they appear. With the passage of time, they tend to become pain-free.

The nodes are frequently associated with TrPs in the interossei; the TrPs may have been latent for years. Heberden described the nodes as

> "little hard knobs, about the size of a small pea, which are frequently seen upon the fingers particularly a little below the top, near the joint. They have no connection with the gout; ...they continue for life; and being hardly ever attended with pain, are rather unsightly than inconvenient, though they must be some little hindrance to the free use of the fingers."[25]

2. ANATOMY (Fig. 40.2)

Interossei

As the name denotes, the interossei lie between adjacent metacarpal bones. Each dorsal interosseous muscle arises ***proximally*** by two heads (Fig. 40.2A), which apparently have significantly different structures which could be important when examining for injection of TrPs. The attachment of the head on the side nearest the middle finger covers nearly three-fourths of that metacarpal bone,[6] which gives it a pennate structure as clearly illustrated for the first dorsal interosseous muscle.[36] The other head has a much shorter attachment to its metacarpal bone[6] and has a much more parallel arrangement of fibers.[36] Figure 2.9 illustrates the difference in these fiber arrangements. This indicates that the head on the side nearest the middle finger (designed for strength) has a long endplate zone running nearly the length of the muscle belly, whereas the other head (designed for speed and large range of motion) has a nearly transverse endplate zone near the middle of the muscle belly. Each bipennate muscle attaches ***distally*** at the base of the proximal phalanx of the related finger and to that finger's extensor aponeurosis. Each muscle attaches on the side of the phalanx away from the midline of the hand.[12] Figure 2.8B illustrates the horseshoe-shaped endplate arrangement in muscles with this pennate structure.

The first dorsal interosseous muscle is larger than the other interossei, but follows the same attachment pattern (Fig. 40.2A). One head arises ***proximally*** from the ulnar border of the metacarpal bone of the thumb, and the other head from almost the entire length of the radial border of the second metacarpal bone. Both heads attach ***distally*** to the proximal phalanx of the index finger on the radial side (and to the extensor aponeurosis). This muscle fills the dorsal web space of the thumb.

Each of the three palmar interossei arises ***proximally*** from the palmar interosseous surface of one metacarpal bone (Fig. 40.2B) and lies palmar to the related dorsal interosseous muscle (Fig. 40.2C). Each then attaches ***distally*** to that finger's extensor aponeurosis and to the base of the proximal phalanx on the side closest to the midline of the hand (center of the middle finger).

Lumbricals

The four lumbricals attach ***proximally*** to the four tendons of the flexor digitorum profundus in mid-palm, and ***distally*** to the radial side of the extensor aponeurosis on each of the four fingers. Strictly speaking, the lumbricals are not interosseous muscles, but they function similarly. In terms of locating and inactivating their TrPs, the first and second lumbricals lie palmar to the first and second dorsal interossei, but with the transverse head of the adductor pollicis interposed between these two lumbricals and the dorsal interossei. The third and fourth lumbricals lie palmar and adjacent to the second and third palmar interossei (Fig. 40.2C).

Abductor Digiti Minimi

This muscle provides half of what would be the next dorsal interosseous muscle, were there a 6th digit, and presents the parallel fiber arrangement[36] that has a transverse endplate zone in midmuscle. It abducts the 5th digit (*light red,* Fig. 40.2A and B). The muscle arises ***proximally*** from the pisiform bone, and attaches ***distally*** to the ulnar side of the base of the first phalanx of the little finger and to its associated extensor aponeurosis.

Supplemental References

Other authors have illustrated the interossei of the hand from the dorsal view[3,14,19,21,29,32,36,40,45,50,55] and in relation to arteries,[23] from the palmar view[2,12,21,29,32,38,42,49,53,54] from the lateral view,[13,22,39] and in cross section.[1,10,18,44]

The abductor digiti minimi has been similarly portrayed from the dorsal view,[40,51] from the palmar view,[15,20,37,43,54] from the lateral view,[5,30] and in cross section.[11,18,44]

The lumbricals are shown in palmar view without[15] and with adjacent nerves,[4] and in cross section.[17]

Heberden's Nodes

Heberden's nodes are often identified with osteoarthritis,[41, 48] particularly with the primary idiopathic form, rather than the traumatic secondary form.[9] The node is an enlargement of soft tissue, sometimes partly bony, on the dorsal surface on either side of the terminal phalanx at the distal IP joint (Fig. 40.2D). The patient may eventually develop a flexion deformity with lateral or medial deviation of the distal phalanx.[41] Similar nodes located at the proximal IP joints are called Bouchard's nodes, but they are seen in only 25% of individuals with Heberden's nodes.[34]

3. INNERVATION

All of the interosseous and the abductor digiti minimi muscles are supplied by branches of the ulnar nerve, through the medial cord and lower trunk from spinal nerves C_8 and T_1.[16] The first and second lumbrical muscles are supplied by the median nerve and the third and fourth by the ulnar nerve.

4. FUNCTION

Interossei and Lumbricals

To understand the actions of these intrinsic hand muscles, it is important to remember that the extensor digitorum strongly extends the first (proximal) phalanx of each finger, but only weakly extends the two distal phalanges. The flexor digitorum superficialis attaches to the middle of the second phalanx, flexing the proximal and middle phalanges. The flexor digitorum profundus attaches to the distal phalanx, flexing it and the more proximal phalanges.

The four dorsal and three palmar interossei have opposing actions in abduction, adduction and rotation, but both groups of interossei plus the lumbricals flex the fingers at the metacarpophalangeal (MCP) joints and extend the distal phalanges.[7,12,24,26,29] It is the interossei and lumbricals that extend the distal two phalanges when any degree of flexion of the proximal phalanx is present. The flexion or extension of the latter is controlled by the flexor digitorum superficialis and the extensor digitorum working as antagonists. The Dorsal interossei abduct (mnemonic—DAB), and the palmar interossei adduct (mnemonic—PAD) with reference to the midline of the middle finger.[7,12,24, 26, 29] Electromyographic studies have shown that the interosseous hand muscles act as flexors of the MCP joints only when this function does *not* conflict with their extensor function at the IP joints.[7]

The flexion-extension function of the interosseous muscles requires considerably less force than the lateral motions of abduction and adduction. Therefore, in disease, the lateral motions are lost earlier, and recover more slowly than flexion-extension. The abduction-adduction functions of the interossei must be tested with the fingers extended at the MCP joints. Spreading the fingers apart is normally severely limited when the fingers are flexed at the MCP joint.[24]

The first *dorsal* interosseous rotates the proximal phalanx to make the index finger pad face toward the ulnar side of the hand whereas the first *palmar* interosseous rotates it in the opposite direction. The first

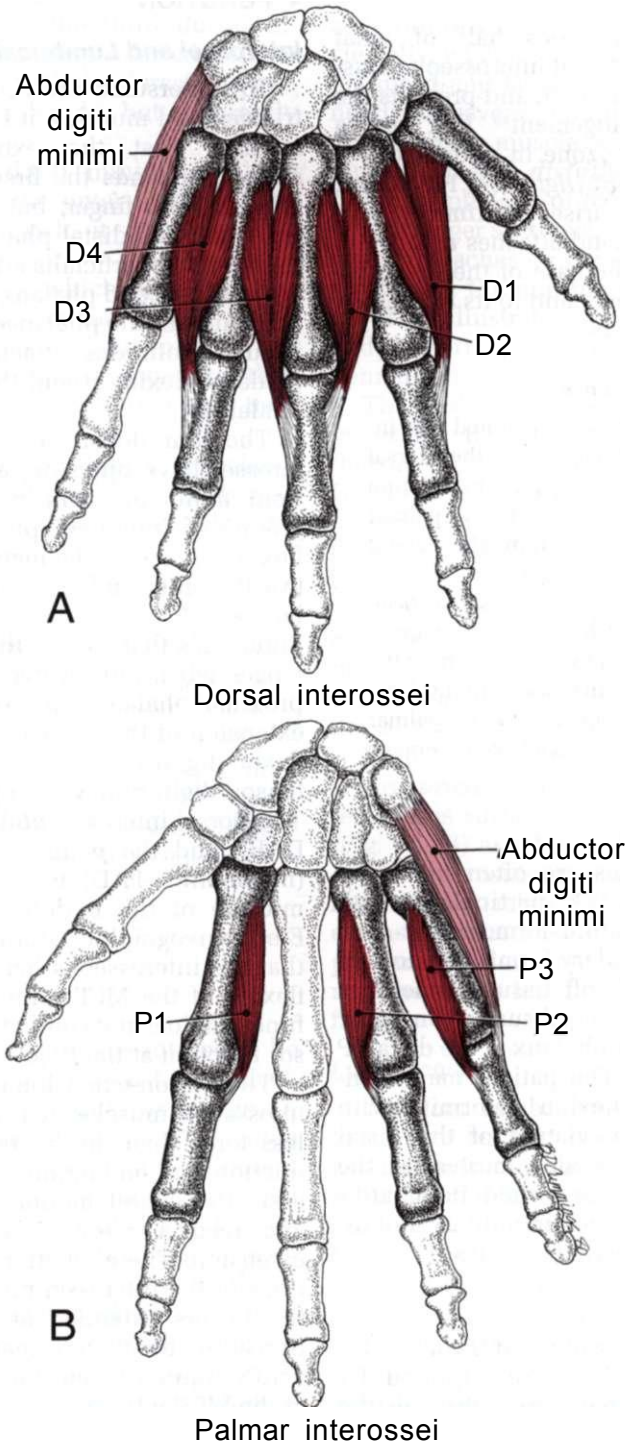


Figure 40.2. Attachments of the right interossei. **A,** dorsal view of the dorsal interosseous muscles *(dark red),* which move the fingers away from the midline of the middle finger, and of the abductor digiti minimi *(light red).* **B,** palmar view of all (the first, second and third) palmar interossei *(dark red).*

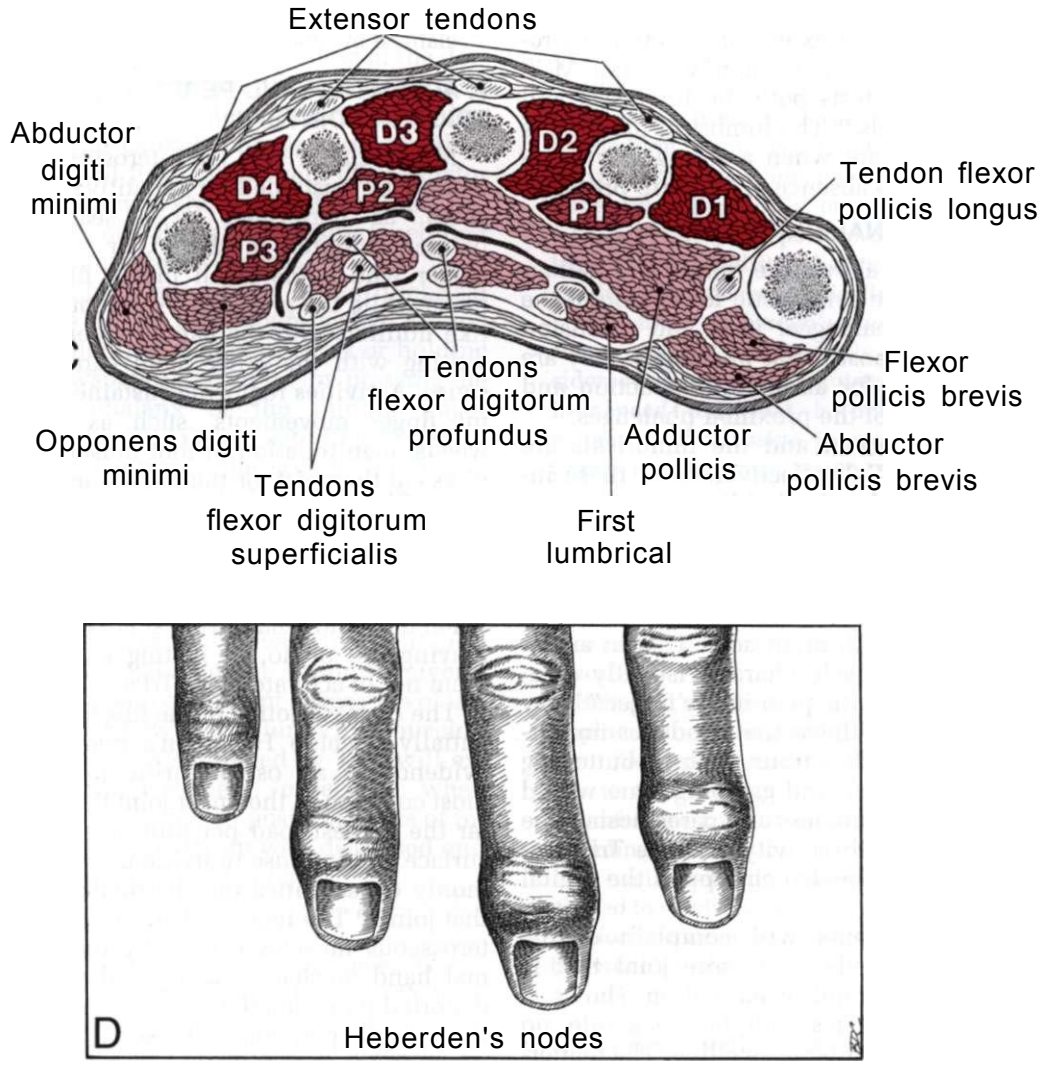


Figure 40.2—*continued.* **C,** cross-sectional view through the metacarpal bones showing the relationship between the dorsal (D1, D2, D3 and D4, *dark red)* and the palmar (P1, P2, and P3, *medium red)* interossei. The lumbricals are the *light red* muscle masses on the radial side of the four flexor digitorum profundus tendons. **D,** appearance of Heberden's nodes on the sides of the distal interphalangeal joints.

dorsal and first palmar interossei counterbalance their rotational movements while combining their flexion-extension actions. In precision handling of objects, the interossei function mainly as abductors and adductors of the fingers. In spherical grip, their rotational forces were found to position the proximal phalanges for best finger pad contact.[33]

The lumbricals are unusual in that they anchor not to bone but to the tendons of other muscles. Thus, the lumbricals function as the equivalent of an adjustable physiological tendon transplant. Contraction of these muscles converts the distal phalanx-flexion action of the flexor digitorum profundus to extension of the distal phalanges. The lumbricals specifically permit the flexor digitorum superficialis to strongly grip with the proximal two phalanges, yet release the distal phalanx grip in the presence of flexor digitorum profun-

dus activity. The usual test of the intrinsic muscles' flexion-extension function, by resisting IP joint extension with the MCP joint flexed, tests both the interossei and the lumbricals.[29] The lumbrical function is most important when a strong grip is required in the absence of fingertip pressure.

5. FUNCTIONAL UNIT

As noted above, the dorsal and palmar interossei are synergistic for flexion at the metacarpophalangeal joint and extension of the two most distal phalanges; they are antagonistic for adduction-abduction and for rotation of the proximal phalanges.

The interossei and the lumbricals are synergistic. Full effectiveness of these intrinsic muscles for holding and grasping objects also requires the assistance of the thumb muscles in the thenar eminence.

6. SYMPTOMS

Patients with myofascial TrPs in an interosseous muscle characteristically complain of "arthritis pain in my finger." They have finger stiffness that produces impairment of hand functions, such as, buttoning a shirt, writing, and grasping. One would not expect numbness and paresthesia to be associated with activity of these TrPs unless the muscle also entrapped the digital nerve.

Some patients will complain of the Heberden's node as a "sore joint that is swollen." Careful examination shows a tender Heberden's node but, as a rule, no true synovial or bony swelling. The tenderness may be *referred* to the joint. In time, the Heberden's node becomes less tender. Clinically it appears that myofascial TrPs in muscles can contribute to joint disease.[47]

> The arthritis literature dealing with Heberden's nodes describes symptoms of brief morning stiffness[34,41,57] due to increased viscosity of periarticular structures.[57] Subsequent loss of range of motion was ascribed to muscle spasm and contracture,[41] which are often simulated by the muscle shortening due to myofascial TrP activity. Heberden's nodes are sometimes, but not always, associated with local pain and tenderness.[34,41] A relationship of Heberden's nodes to osteoarthritis in other parts of the body is seriously questioned by some,[9,52] and claimed by others.[28,35]

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Myofascial TrPs in the interossei are activated by sustained or repetitive pincer grasp, as performed by a seamstress, painter, sculptor, mechanic, or a model-maker who holds small pieces firmly in place while the glue sets. A nervous habit like fiddling with the cap of a pen while writing with the other hand can be the cause. Activities requiring sustained forceful finger movements, such as pulling weeds, manipulation of foot muscles by a physical therapist, or the retraction of nail cuticles by a manicurist, have initiated interosseous TrPs. "Golf hands" have been found to be due to a constant tight grip on the handle of the golf club, especially when the handle has a very small diameter. Playing the piano, or batting a baseball, seem not to activate these TrPs.

The distal IP joints of the fingers preferentially develop Heberden's nodes. This evidence of an osteoarthritic process is most common in the finger joint that has by far the highest load per unit area of joint surface and in those individuals who commonly do activities that particularly load that joint.[46] The increased strain on the interosseous muscles caused by the abnormal hand mechanics associated with the distorted joint function of arthritis can activate and perpetuate these TrPs. *Vice versa,* it appears that the myofascial TrPs also can contribute to the arthritis.[47] Inactivating the related myofascial TrPs and the *elimination of their perpetuating factors* appear to be important parts of early therapy to delay or abort the progression of some kinds of osteoarthritis.

8. PATIENT EXAMINATION

Involvement of the palmar interossei restricts voluntary separation of adjacent fingers, and involvement of dorsal interossei restricts full closure of adjacent fingers.

Kendall, *et al.*[29] describe and illustrate clearly the effect of shortening of the interosseous and lumbrical muscles. TrP shortening of the palmar interossei which produce adduction of the fingers (PAD)

compromises the ability to fully spread the extended fingers. TrP shortening of the dorsal interossei which produce abduction of the fingers (DAB) interferes with the ability to bring the extended fingers close together. If the small finger sticks out, the abductor digiti minimi is shortened. If the index finger sticks out, it indicates a shortened first dorsal interosseous muscle.

Testing for shortening of the lumbrical muscles is a bit more complicated. For example, holding a hand of cards or holding up a newspaper to read it[30] by pressing the middle phalanx of the middle finger against the thumb but avoiding finger-tip pressure overloads the second lumbrical. When it becomes shortened (due to TrPs), it will tend to hyperextend the distal phalanx of the middle finger when the fingers are extended, and prevent full closure of the middle finger when attempting a claw position (fingers flexed with the MCP joint extended). The patient example given[30] also had a pain complaint suggestive of unrecognized TrPs in that lumbrical muscle.

The muscles shortened by TrPs will exhibit some weakness, especially when tested in a lengthened position. Tests of interosseous strength are well described and illustrated by Kendall, *et al.*[29]

The presence of Heberden's nodes is a common finding in patients with TrPs in the interossei. A node is palpable as an excrescence on the dorsal margin of the distal phalanx, or the distal end of the middle phalanx on either side, always near the distal IP joint (Fig. 40.2D). A Heberden's node also may appear on the thumb, usually on its ulnar side in conjunction with TrPs in the adductor pollicis muscle. Idiopathic Heberden's nodes are most commonly seen on the index and middle fingers.[26] They appear on the side of the finger to which the involved interosseous muscle attaches.

The mechanism by which TrPs in the interossei may lead to Heberden's nodes is speculative. Myofascial TrPs produce bands of taut muscle fibers, which could cause a sustained increase of tension on the tendon. The question also arises as to why, if trauma is a significant factor, distal joints of the fingers are involved, but not of the toes.[9] One possible answer is that fine manipulation with the fingers overloads the hand interossei, but we make no such use of the toes. The idiopathic form may be genetically governed. Early cases of idiopathic Heberden's nodes, radiographically, may show small islands of calcium deposit in the extensor tendons near the distal phalanx before the condition is apparent clinically.[52]

Idiopathic Heberden's nodes have sometimes, but not generally, been considered an inherited, autosomal, sex-influenced trait that is dominant in women and recessive in men, with a prevalence 10 times greater in women than in men.[23, 41] The nodes require a normal nerve supply to develop. Idiopathic Heberden's nodes have been closely related to menopause; nodes were first noted within 3 years of the last menstrual period in one-half of 99 cases.[52]

Heberden's nodes may be secondary to trophic changes induced by nerve entrapment (Section 10) or, more likely, may be due to an autonomic component within the reference zone of a TrP in the corresponding interosseous muscle.

A well-designed research study is needed to resolve to what extent there is a relation between myofascial TrPs and Heberden's nodes.

9. TRIGGER POINT EXAMINATION

Usually only one or two interosseous muscles contain active TrPs at one time; others may harbor latent TrPs. Myofascial TrPs in these muscles are difficult to palpate. Separating the fingers widely, which moves the metacarpal bones apart, permits pincer palpation between the bones. Meanwhile, counter-pressure is produced with a finger against the palm, beneath the muscle to be palpated. One can localize deep tenderness in the interossei and lumbricals but, except for the first dorsal interosseous, referred pain and local twitch responses are rarely induced until a needle impales the TrP.

When present, Heberden's nodes can serve as guides to TrPs in the interossei. They are identified as nodules located over

the distal IP joints, as seen in Figures 40.1 and 40.2D.[34] Nodes develop dorsally on that side of the finger to which the interosseous muscle attaches.

10. ENTRAPMENT

One may observe cutaneous hypoesthesia along one side of a finger where the patient reports a sensation of numbness when an active TrP lies in the corresponding interosseous muscle. This apparent neurological deficit disappears following inactivation of the TrP, suggesting that the median or ulnar digital nerve had been entrapped by the increased tension of the involved interosseous muscle. However, this could be sensory inhibition caused by the TrP. Electrodiagnostic testing would be required to establish an entrapment component.

On their way through the palm to the digits, the median and ulnar nerves lie next to the lumbrical and palmar interosseous muscles. The deep (motor) branch of the ulnar nerve pierces the opponens digiti minimi before supplying all interossei, the third and fourth lumbricals, the adductor pollicis, and the deep head of the flexor pollicis brevis.[16] Active TrPs in the opponens digiti minimi can be responsible for weakness of these ulnar-innervated muscles and, if weakness is present, the opponens should be examined for TrPs.

11. DIFFERENTIAL DIAGNOSIS

The diagnoses most likely to be confused with interosseous TrPs include C_6 radiculopathy, ulnar neuropathy, C_8 or T_1 radiculopathy, and, when the TrPs are primarily of the abductor digiti minimi muscle, a thoracic outlet syndrome. Rarely, one may see the pain misdiagnosed as an isolated digital nerve entrapment when, in fact, it is caused by TrPs in one of the dorsal interosseous muscles. When the TrP is inactivated, this finger pain resolves completely. Finger pain and numbness also may be due to nerve entrapment of the brachial plexus by taut scalene muscles, or compression as tbe plexus passes beneath the scapular attachment of a taut pectoralis minor muscle (see Fig. 43.4B).

Articular dysfunctions including the loss of joint play that are associated with interosseous TrPs can occur at either the level of the carpometacarpal joint or at the level of the metacarpophalangeal joint, and any of these joint dysfunctions need to be treated concurrently with the associated interosseous TrPs.

Related Trigger Points

When the interosseous muscles are involved, one should look for associated TrPs in the intrinsic thumb muscles. Other muscles that may refer myofascial pain into the fingers include the long flexors and extensors of the fingers, the latissimus dorsi, the pectoralis major, scalene muscles, and either the lateral or the medial head of the triceps brachii.

12. TRIGGER POINT RELEASE (Fig. 40.3)

With the exception of the first dorsal interosseous, spray and stretch are not generally effective for the management of interosseous trigger points (TrPs), since it is difficult to adequately stretch these muscles. Their TrPs may or may not be accessible for TrP pressure release or massage. In our experience, TrP injection usually provides the most rapid and sustained relief.

The first dorsal interosseous is stretched and sprayed by the operator abducting the thumb and adducting the index finger to the point of resistance while applying down-sweeps of the vapocoolant (Fig. 40.3). This is followed by three slow cycles of full active range of motion of the muscles that were treated.

Spray and stretch are more likely to be effective if the TrPs are superficial (dorsal interossei), if the fingers and their metacarpal bones can be separated widely, and if the down-sweep spray pattern is used over both the involved musculature and its pain pattern (Fig. 40.1A). Spray and stretch also are applied to these muscles immediately following injection of TrPs.

13. TRIGGER POINT INJECTION (Figs. 40.4 and 40.5)

Since the precise location of trigger points (TrPs) in the palmar interossei and

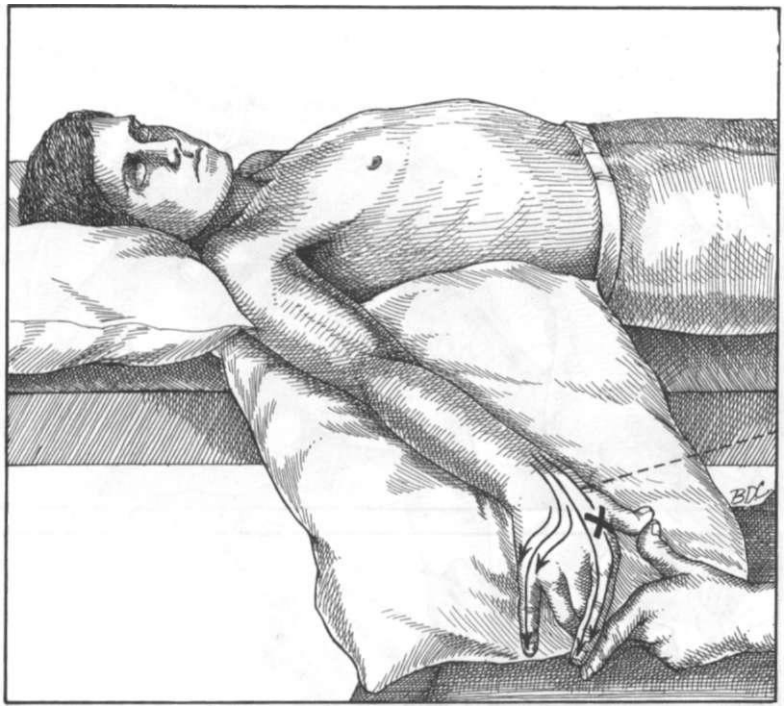


Figure 40.3. Stretch position and direction of the sweeps of spray (*arrows)* for a trigger point **(X)** in the first dorsal interosseous muscle. Spray should include the palmar aspect.

in the lumbricals is difficult to palpate, adequate exploration of the area with a 2.5-cm (1-in), 25-gauge needle is important.

Interossei

When the first dorsal interosseous harbors an active TrP, the patient's index finger is held between the operator's index and middle fingers (Fig. 40.4A), with the operator's middle finger pressed firmly into the web space beneath the first dorsal interosseous, so that the muscle is held firmly in a pincer grasp; this permits identification and fixation of the TrP for injection (Fig. 40.4A).

The dorsal interossei each have two heads. The one nearest the middle finger is pennate in structure, the other has a more parallel fiber arrangement the length of the muscle. The one nearest the middle finger will have a longer endplate zone approaching the length of the muscle. The other endplate zone will be more transverse at midmuscle. Both halves may need to be explored throughout for TrPs. For example, to inject the second dorsal interosseus, the needle is aligned with the side of the third metacarpal bone in the second interosseus space and is inserted into the center of the tender area (Fig. 40.5). If any tenderness remains, the needle is aligned with the second metacarpal bone on the other side of the space and the other head of the muscle probed for TrPs.

To inject the first palmar interosseous (Fig. 40.5A), the needle is directed away from the third metacarpal bone to reach the muscle, which lies beneath the ulnar side of the second metacarpal (Fig. 40.5B).

Following inactivation of TrPs in an interosseous muscle, soreness in the related distal IP joint and joint stiffness disappear. Tenderness of the Heberden's node usually disappears at once, whereas it diminishes in size with the passage of time.

Bieber[8] described a patient who required injection of TrPs in a first dorsal interosseous muscle for relief of symptoms.

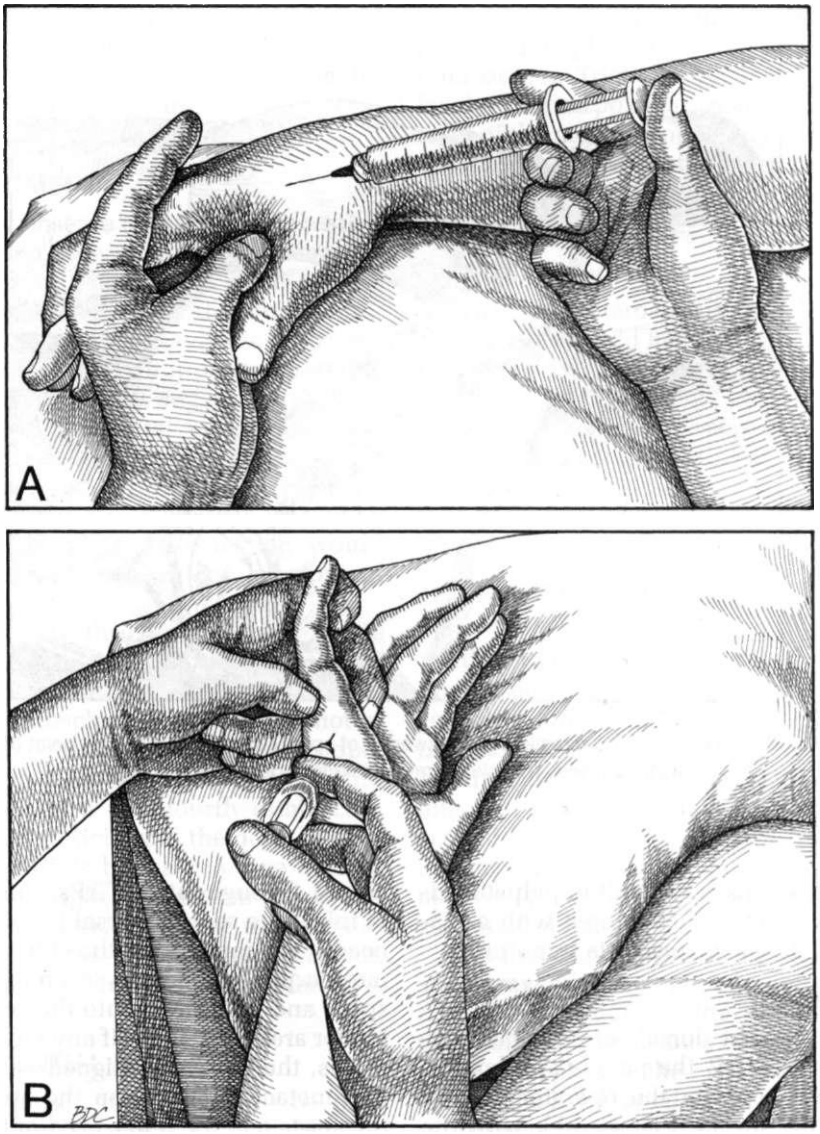


Figure 40.4. Injection technique for trigger points in the intrinsic hand muscles. **A,** first dorsal interosseous muscle approached from the dorsal aspect. **B,** the abductor digiti minimi, approached from the ulnar aspect of the hand.

Lumbricals

The four lumbricals, unlike the interossei, are injected from the palmar side of the hand because no major structure lies between them and the palmar skin. Each lumbrical muscle is found at the radial side of its corresponding metacarpal bone, in close association with a flexor digitorum profundus tendon (Fig. 40.2C).

Abductor Digiti Minimi

Either flat or pincer palpation may be used to locate TrPs in the abductor digiti minimi. To inject a TrP in this muscle, the patient turns the hand ulnar side up and

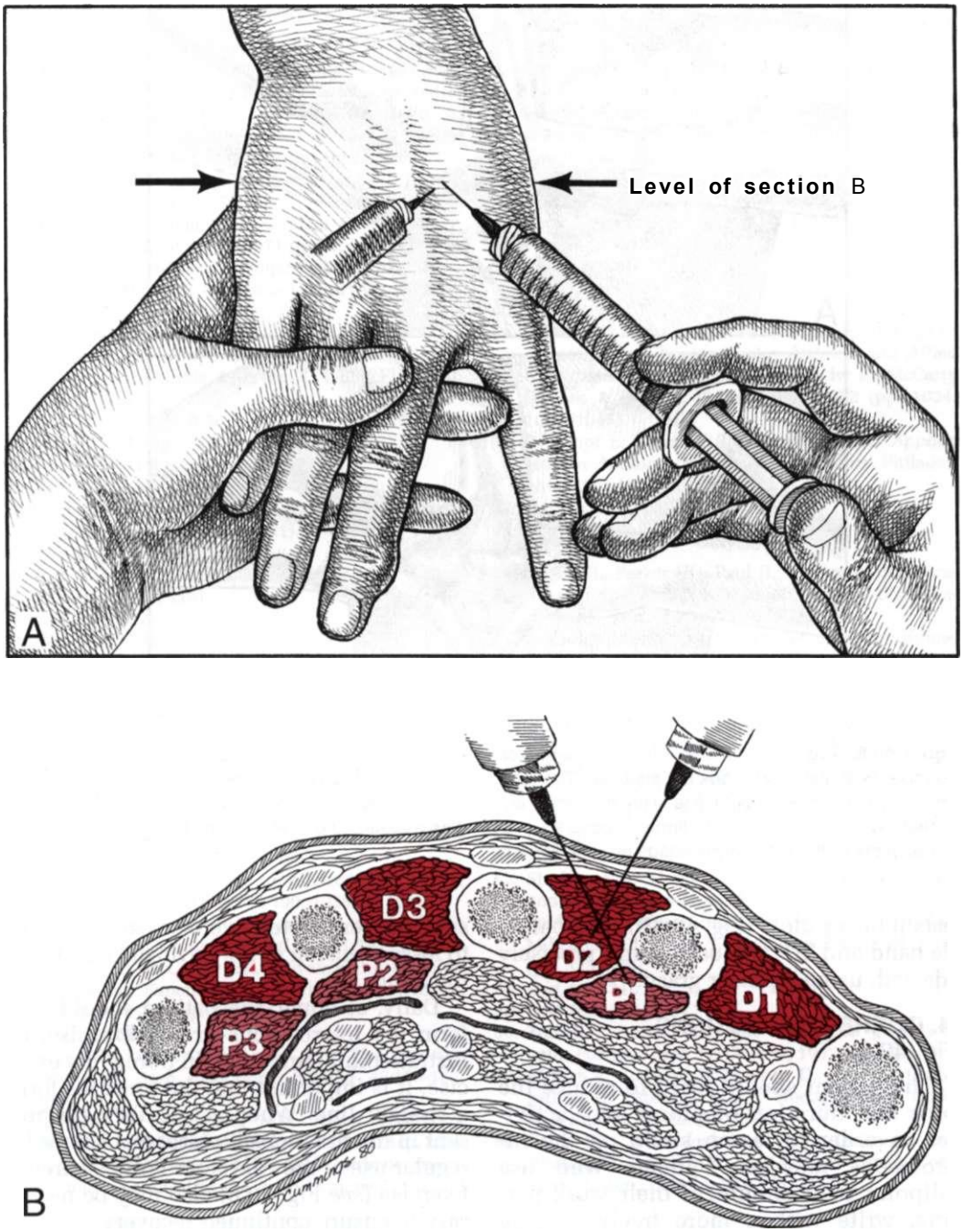


Figure 40.5. Injection technique for the interossei. **A,** the *complete syringe* is injecting a trigger point in the more ulnar penna of the second dorsal interosseous muscle; its corresponding Heberden's node is shown. The *incomplete syringe* is injecting the first palmar interosseous, which is reached as the needle penetrates deep to the second metacarpal bone. **B,** cross section of C showing relation of the needles to the muscles being injected (see also Fig. 40.2C for labels). *Dark red,* dorsal interossei; *light red,* palmar interossei.

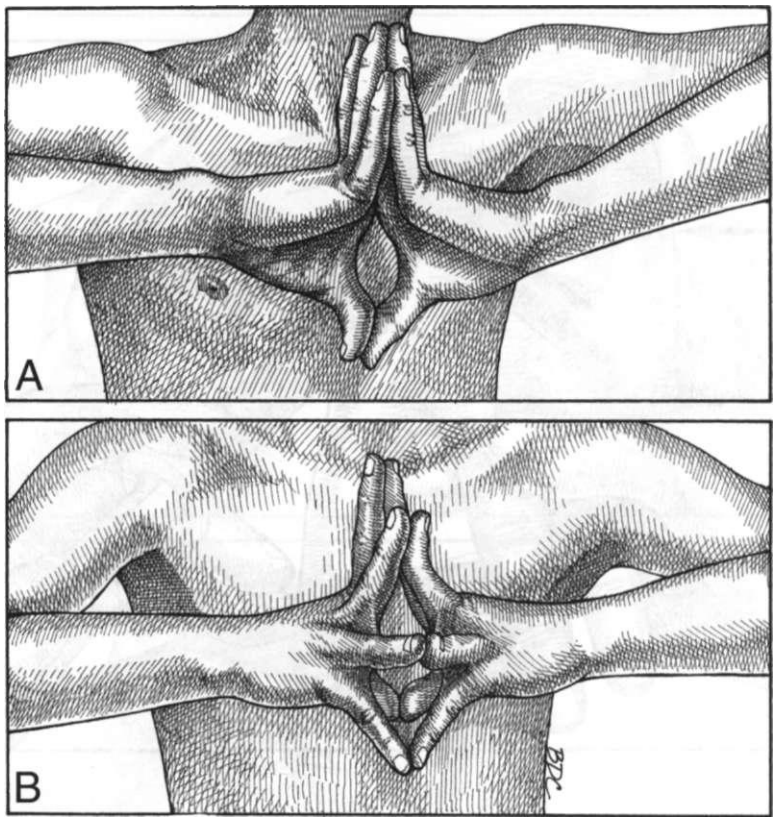


Figure 40.6. Two views of the Interosseous-stretch Exercise. Both hand positions are effective. The forearms are held in a straight line with the arms abducted. **A,** an effort is made to firmly oppose the palmar aspects of the metacarpal heads and the fingers, while the fingers and thumbs are spread apart. **B,** only the finger pads contact each other while the fingers and thumbs are spread apart with the uninvolved fingers assisting the stretch of the involved interossei.

rests it on a pillow (Fig. 40.4B). The palpable band and TrP are located and precisely injected, using a pincer grasp.

14. CORRECTIVE ACTIONS (Fig. 40.6)

The patient should learn to reduce the force and duration of pincer grip activities in order to lessen strain on the interosseous muscles. Patients who use ballpoint pens should, if their work permits, write with a more freely flowing felt-tip pen that needs a much lighter touch.

The patient should interrupt prolonged fine manual activity with the Finger-flutter Exercise *(see* Fig. 35.9), the Finger-extension Exercise *(see* Fig. 38.7), or the Artisan's Finger- stretch Exercise *(see* Fig. 35.8) to lessen tension of the intrinsic muscles of the hand.

Daily, at home, the patient should perform the Interosseous-stretch Exercise, illustrated in Figure 40.6. In doing this exercise, it is important that the forearms form a straight line. When active TrPs are present in the first dorsal interosseous muscle, regular use of the Adductor Pollicis-stretch Exercise *(see* Fig. 39.6) also may be necessary to ensure continued recovery.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:443 (Fig. 6.129A).
2. *Ibid,* p.424 (Fig. 6.99).

3. *Ibid.* p. 430 (Fig. 6.107).
4. *Ibid.* p. 420 (Fig. 6.91).
5. *Ibid.* p. 438 (Fig. 6.119B).
6. Bardeen CR: The musculature. Sect. 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (p. 444).
7. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 291, 292).
8. Bieber B: The role of trigger point injections in the development of private practice. *Phys Med Rehabil* 8(1):197-205, 1997 (p. 203).
9. Boyle JA, Buchanan WW: *Clinical Rheumatology.* F.A. Davis, Philadelphia, 1971 (pp. 5, 27, 32-34).
10. Carter BL, Morehead J, Wolpert SM, *et al: Cross-Sectional Anatomy.* Appleton- Century-Crofts, New York, 1977 (Sects. 60-63).
11. *Ibid.* (Sects. 59-62).
12. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 554- 556, Fig. 6-66).
13. *Ibid.* (p. 534, Fig. 6-53).
14. *Ibid.* (p. 539, Figs. 6-56, 6-65).
15. *Ibid.* (p. 553, Fig. 6-64).
16. *Ibid.* (pp. 1215-1219).
17. *Ibid.* (Fig. 6-51).
18. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 121).
19. *Ibid.* (Figs. 79, 103).
20. *Ibid.* (Figs. 107, 108).
21. *Ibid.* (Figs. 115, 116).
22. *Ibid.* (Fig. 112).
23. *Ibid.* (Fig. 104).
24. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (Fig. 25; pp. 128-130, 134-136, 153-154).
25. Heberden W: Digitorum nodi. Chapter 28. In: *Commentaries on the History and Cure of Diseases,* facsimile of the London 1802 Edition. Hafner, New York, 1962 (pp. 148- 149).
26. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 167, 168).
27. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3:175-190, 1938 (p. 183).
28. Kellgren JH, Moore R: Generalized osteoarthritis and Heberden's nodes. *Br Med J* 3:181-187, 1952.
29. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 248-251).
30. *Ibid.* (p. 252).
31. Kraft GH, Johnson EW, LeBan MM: The fibrositis syndrome. *Arch Phys Med Rehabil* 49:155-162, 1968.
32. Langman J, Woerdeman MW: *Atlas of Medical Anatomy.* W.B. Saunders, Philadelphia, 1978 (p. 253).
33. Long C, Conrad PW, Hall EW, *et al:* Intrinsic-extrinsic muscle control of the hand in power grip and precision handling. *J Bone Joint Surg* 52A:853-867, 1970.
34. Mannik M, Gilliland BC: Degenerative joint disease. Chapter 361. In: *Harrison's Principles of Internal Medicine.* Ed. 7. Edited by Wintrobe MM, *et al.* McGraw-Hill Book Co., New York, 1974 (p. 2006).
35. Marks JS, Stuart IM, Hardinge K: Primary osteoarthrosis of the hip and Heberden's nodes. Ann *Rheum Dis* 38:107-111, 1979.
36. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (pp. 35D, 147D).
37. *Ibid.* (pp. 140A, 142A).
38. *Ibid.* (p. 144B).
39. *Ibid.* (p. 150B).
40. *Ibid.* (p. 151C).
41. Moskowitz RW: Clinical and laboratory findings in osteoarthritis. Chapter 56. In: *Arthritis and Allied Conditions.* Ed. 8. Edited by Hollander JL, McCarty DJ. Lea & Febiger, Philadelphia, 1972 (pp. 1034, 1037, 1045).
42. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (p. 85).
43. *Ibid.* (p. 87).
44. *Ibid.* (p. 92).
45. *Ibid.* (p. 90).
46. Radin EL, Parker HG, Paul IL: Pattern of degenerative arthritis, preferential involvement of distal finger-joints. *Lancet 1*:377-379, 1971.
47. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62.111-114, 1981.
48. Sokoloff L: The pathology and pathogenesis of osteoarthritis. Chapter 55. In: *Arthritis and Allied Conditions.* Ed. 8. Edited by Hollander JL, McCarty DJ. Lea & Febiger, Philadelphia, 1972 (pp. 1018,1019).
49. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 340).
50. *Ibid.* (p. 341).
51. *Ibid.* (p. 334).
52. Stecher RM, Hersh AH, Hauser H: Heberden's nodes. *Am J Hum Genet* 5:46-60, 1953
53. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (pp. 335, 336).
54. *Ibid.* (p. 334).
55. ibid. (pp. 330, 331).
56. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med 1 1*:425-434, 1952 (p. 428).
57. Wright V, Goddard R, Dawson D, *et al.:* Articular gelling in osteoarthrosis-a bioengineering study. *Ann Rheum Dis* 29:339, 1970.
58. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little, Brown & Company, Boston, 1988 (p. 211, Fig. 12-2).

PART 5
TORSO PAIN

CHAPTER 41
Overview of Torso Region

INTRODUCTION TO PART 5

This fifth part of volume 1 of the TRIGGER POINT MANUAL includes those muscles of the chest, abdomen, and back that were not previously covered. Excluded are the muscles that attach to the scapula and those that cross the glenohumeral joint. This chapter is divided into three sections: A-the Pain Guide, B-the Enigma of Low Back Pain, and C-Postural Considerations: Static and Dynamic.

SECTION A
PAIN GUIDE TO INVOLVED MUSCLES

The Pain Guide of Section A lists the muscles that may be responsible for pain in the areas shown in Figure 41.1. Muscles covered in Volume 2 of the *Trigger Point Manual* are listed in *italics*. Volume 2 muscles are included because so many of them also cause low back pain. It is most important that these muscles also be considered as possible sources of the patient's pain complaint. The muscles most likely to refer pain to a given area are listed below under the name of that area. One uses this chart by locating (on the figure) the name of the area that hurts and then looking under that heading for all the muscles that are likely to cause the pain. Then, reference should be made to the individual muscle chapters; the number for each chapter follows in parentheses.

In a general way, the muscles are listed in the order of the frequency in which they are likely to cause pain in that area. This order is only an approximation. The selection process by which patients reach an examiner greatly influences which of their muscles are most likely to be involved. **Boldface** type indicates that the muscle refers an essential pain pattern to that pain area. Roman type indicates that the muscle refers a spillover pattern to that pain area.

GUIDE TO MUSCLES IN VOLUME 1	*GUIDE TO MUSCLES IN VOLUME 2*
LOW THORACIC BACK PAIN	***LOW THORACIC BACK PAIN***
Iliocostalis thoracis (48)	*Iliopsoas (5)*
Multifidi (48)	
Serratus posterior inferior (47)	
Rectus abdominis (49)	
Intercostals (45)	
Latissimus dorsi (24)	
LUMBAR PAIN	***LUMBAR PAIN***
Longissimus thoracis (48)	***Iliopsoas*** *(5)*
Iliocostalis lumborum (48)	*Gluteus medius (8)*
Iliocostalis thoracis (48)	
Multifidi (48)	
Rectus abdominis (49)	
SACRAL AND GLUTEAL PAIN	***SACRAL AND GLUTEAL PAIN***
Longissimus thoracis (48)	***Quadratus lumborum*** *(4)*
Iliocostalis lumborum (48)	***Piriformis*** *(10)*
Multifidi (48)	***Gluteus medius*** *(8)*
	Gluteus maximus *(7)*
	Levator ani *(6)*
	Obturator interims *(6)*
	Gluteus minimus *(9)*
	Sphincter ani *(6)*
	Coccygeus *(6)*
	Soleus (22)
SIDE-OF-CHEST PAIN	***SIDE-OF-CHEST PAIN***
Serratus anterior (46)	
Intercostals (45)	
Latissimus dorsi (24)	
Diaphragm (45)	
FRONT-OF-CHEST PAIN	***FRONT-OF-CHEST PAIN***
Pectoralis major (42)	
Pectoralis minor (43)	
Scaleni (20)	
Sternocleidomastoid (sternal) (7)	
Sternalis (44)	
Intercostals (45)	
Iliocostalis cervicis (48)	
Subclavius (42)	
External abdominal oblique (49)	
Diaphragm (45)	
ABDOMINAL PAIN	***ABDOMINAL PAIN***
Rectus abdominis (49)	*Quadratus lumborum (4)*
Abdominal obliques (49)	
Transversus abdominis (49)	
Iliocostalis thoracis (48)	
Multifidi (48)	
Pyramidalis (49)	

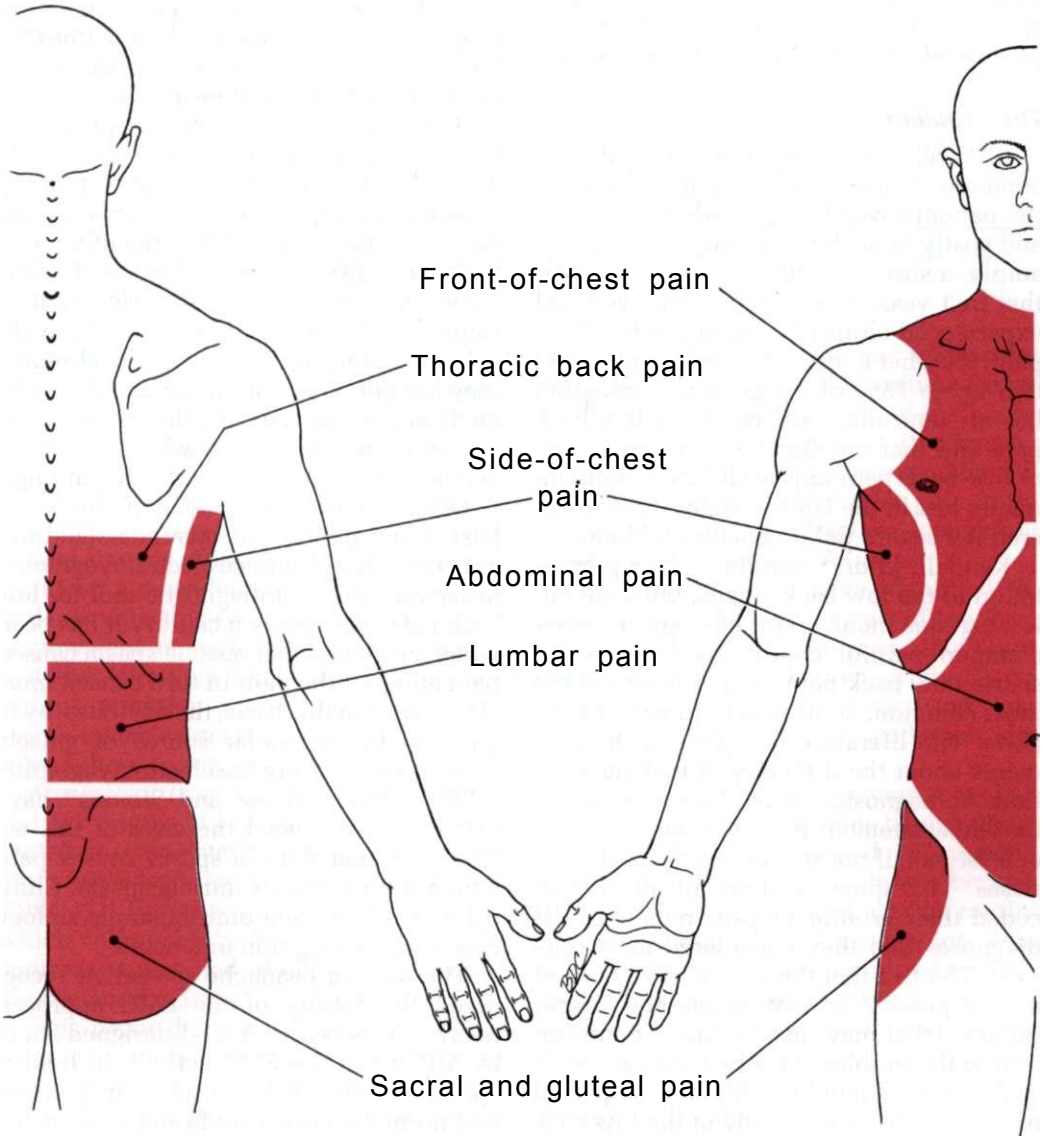


Figure 41.1. The designated areas within the torso region where the patient may describe pain referred there by myofascial trigger points.

SECTION B

ENIGMA OF LOW BACK PAIN (Figures 41.2 and 41.3)

This section considers commonly overlooked causes of low back pain that help to make it so enigmatic. Other commonly recognized but less common causes of low back pain are identified in Chapter **48,** Section **11.**

The Problem

It is well known that low back pain is a common affliction and that it is costly to the patient's well being, costly to industry, and costly to health care providers. For example, a survey of **306** employees[21] found that in **1** year, **41%** of those surveyed had experienced clinically significant low back pain. Another author, de Girolamo,[10] noted that up to **75%** of the general population has at some time suffered from low back pain and that the direct and indirect costs of low back pain approach **$24** billion annually just in the United States. He considered it a severe public health problem.

Kendall, *et al.*[24] introduce their presentation of the low back enigma with the following statement, "The etiology of many common painful conditions remains obscure. Low back pain, which is one of the most common, continues to puzzle the experts. The literature is replete with statements about the difficulty of making a definitive diagnosis." It is almost axiomatic that this statement is true because there is at least one, if not several overlooked diagnoses. This quote by Kendall, *et al.* preceded their erudite 11 page review of the diagnoses that they considered most relevant. The fact that the review gave no hint of the possibility that myofascial trigger points (TrPs) may make a major contribution to the problem may be relevant.

Similarly, another highly respected book[43] on physical therapy of the low back presented an outstanding review of the anatomy of back muscles (Chapter 4), and a classic chapter on functional imbalance of muscles and disturbed movement patterns (Chapter 10). No indication could be found in the book that the authors considered myofascial TrPs a possible contributing factor in low back pain and its associated muscular dysfunction. In the back-pain literature, this disregard of myofascial TrPs is the rule, not the exception.

Needless to say, there is much controversy within the medical profession concerning the appropriate management of low back pain. Recently, the American Academy of Physical Medicine and Rehabilitation declined to endorse the guidelines for low back pain developed by the Agency for Health Care Policy and Research.[3]

Part of the problem is the common position that, if an organic cause can not be demonstrated by a laboratory test or an imaging technique, there can be no organic cause for the pain. The frequently overlooked conditions that are discussed below have some common characteristics: they cannot be diagnosed by a currently available laboratory test or imaging technique, they are not apparent on the usual routine medical examination, and they require special skill and training in what to look for and how to examine for diagnostic findings.

Other sources of confusion are some false assumptions and misunderstandings as to the value of surface electromyographic measurements as a diagnostic tool for low back pain. For nearly a century it has been widely accepted that muscle spasm caused pain and that the pain in turn caused muscle spasm. On this basis, the obvious way to quantify the muscular source of muscle pain was to measure the electromyographic (EMG) activity. Mense and Simons[31] have extensively reviewed the error of this assumption that muscle spasm causes pain which in turn causes muscle spasm. Clinical research and neuromuscular physiology make that assumption untenable.

The tension headache specialists recognized the futility of the EMG approach nearly **10** years ago. A well-designed study by Miller[32] of the EMG activity in lumbar spinal muscles of low back pain patients and normal controls confirmed that such a reflex-spasm cycle was not present and was not the cause of pain. In another, more sophisticated study,[36] other authors concluded that the best their complex EMG equipment and analysis could do was to distinguish patients with low back pain from normal controls. They had nothing to say about what caused the pain or of what diagnostic value the results might be.

A common mistake is to equate palpably increased muscle tension with muscle spasm. Muscle spasm is, by definition,[31] caused by muscle contraction associated with motor unit action potentials that originate in the central nervous system. Muscle spasm is unambiguously identifiable by surface or needle EMG recordings. In addition, power spectral analysis of surface EMG detects muscle fatigue.[40] A *major* source of the muscle tension observed clinically in low back pain patients is due to endogenous contracture caused by myofascial TrPs (see Chapter 2, Sections B and D), which is *not* detected by surface EMG. Therefore, surface EMG is unable to detect a major source of the muscle tension associated with low back pain. At best, it provides an incomplete picture of the problem and by itself can be seriously misleading.

Low back pain deserves a fresh look at some old assumptions.

Likely Answers

The following are commonly overlooked sources of low back pain which account for a considerable percentage of the patients with this complaint. The problem is prevalent enough and serious enough that the following likely sources deserve serious attention by skilled clinical research investigators to clearly establish the role of these sources in low back pain.

Myofascial Trigger Points. The myofascial TrP origins of low back pain were presented in some detail and the pain patterns of 11 muscles were published in 1983.[41] Table 41.1 lists these muscles according to which volume of the *Trigger Point Manual* contains a detailed description of each muscle and its TrPs.

This paper did not call attention to the complex situation of many patients who present with the complaint of low back pain. Occasionally only one muscle will be responsible for the pain as presented,[41] but it is much more common for several muscles to contribute to overlapping pain patterns. The composite pattern resulting depends upon the extent of muscle involvement. No two patients present exactly the same picture.

Figure 41.2 illustrates an example of a composite pattern produced by TrPs in four of the muscles that refer pain to the lumbosacral region. The chapters concerning these TrPs are found in this volume except for the iliopsoas, which is found in Chapter 5 of volume 2. Figure 41.3 illustrates a comparable composite pattern produced by TrPs in four muscles that refer pain to the pelvic region. The chapters concerning these TrPs are found in Volume 2.

Table 41.1 *Muscles That May Harbor Trigger Points Which Can Cause or Contribute to Low Back Pahf*

Trigger Point Manual Volume 1	*Trigger Point Manual Volume 2*
Erector spinae	Quadratus lumborum
longissimus	Iliopsoas
iliocostalis	Gluteus Medius
Multifidi	Gluteus Maximus
Rotatores	Levator Ani
Rectus abdominis	Piriformis

From Simons DG, Travell JG: Myofascial origins of low back pain. Parts 1,2,3. Postgrad Med 73:66-108, 1983.

Other authors also have identified the importance of TrPs as a cause of low back pain.[15, 34] Dejung[11] observed that lumbosacral pain of unknown origin is frequently caused by TrPs. Bonica and Sola" illustrated 11 specific TrP syndromes that cause low back pain.

Enigmatic back pain is sometimes diagnosed as "chronic intractable benign pain." Rosomoff, *et al.*[38] examined 283 patients who qualified for this diagnosis because they had "no objective findings" on routine physical examination. Trigger points were found in 96.7% of those examined for TrPs; the authors concluded that the initial diagnosis was misleading, inappropriate, and probably nonexistent.

Among 18 patients with "*Hexenschuss*" (lumbago), Dejung[12] found that 14 had TrPs in the gluteal muscles, 13 had TrPs in the abdominal muscles, and 8 had TrPs in the paraspinal muscles, plus TrPs in 5 other muscles. Obviously, most of the patients had multiple TrPs. Within a day of TrP injection therapy, patients experienced a 75% reduction in symptoms suggesting, but not proving, a relationship. Controlled research studies critically examining the

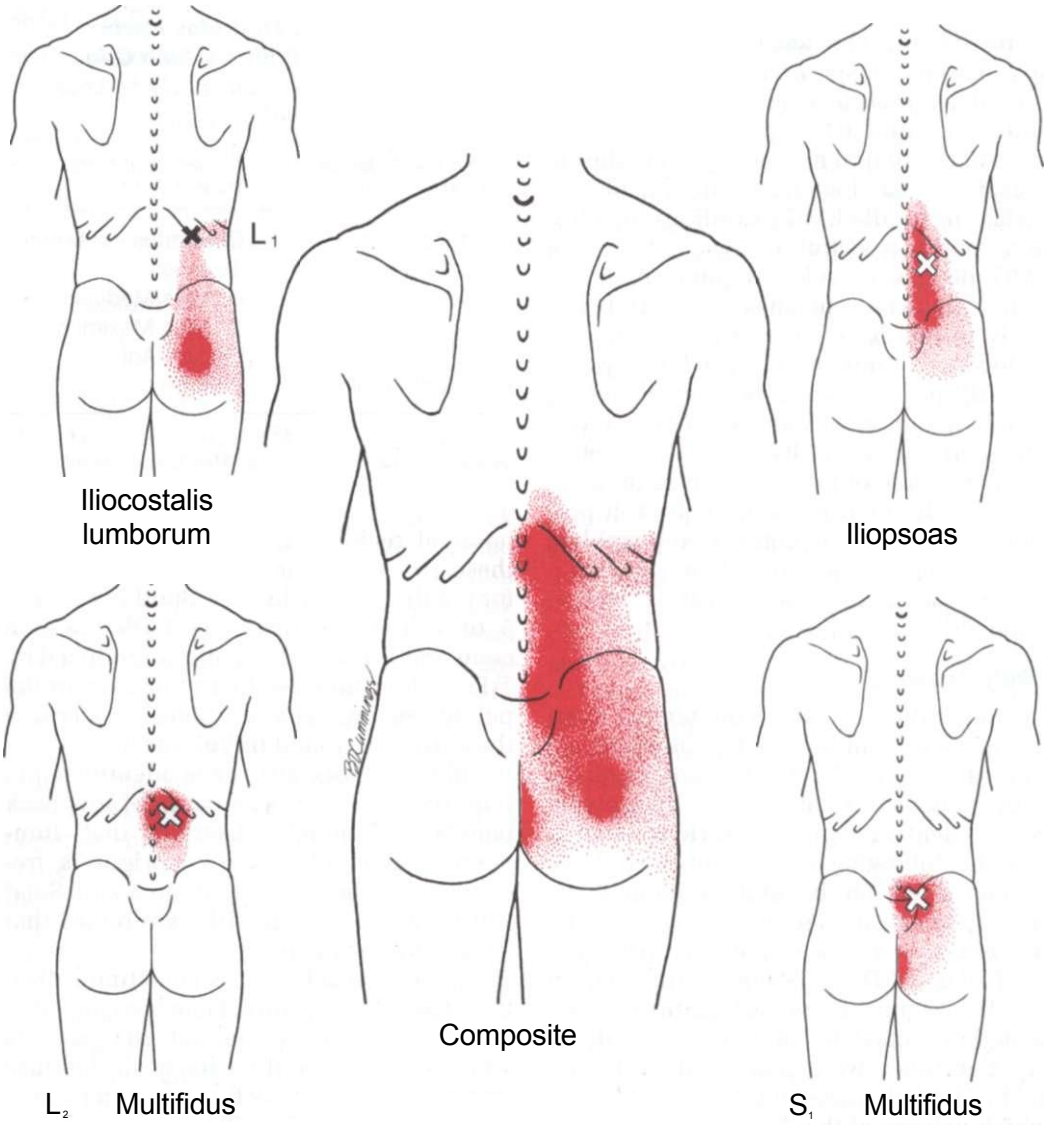


Figure 41.2. Individual pain patterns of several trigger points that refer pain to the lumbosacral region and that may superimpose on each other. The composite pain pattern in the central figure represents the summated pain (red) a patient can experience. It shows the sum of pain referred from trigger points **(Xs)** in the regional muscles illustrated: the iliocostalis lumborum, iliopsoas, L_2 multifidus, and the S_1 multifidus. Individual pain patterns are illustrated around the composite picture.

role of TrPs in low back pain are conspicuous for their absence and urgently needed.

Articular Dysfunctions. Articular dysfunction can be a source of pain from articulations throughout the body (including the low back) and is receiving increasing recognition. However, acceptance is impeded by the fact that the origin of the pain caused by articular dysfunctions that require mobilization for relief of the pain has not been satisfactorily explained. The zygapophysial joints of the spine as a potent source of referred pain is well documented.[5,6,30] However, this still leaves the pathophysiology responsible for the pain unresolved, and these joints were the

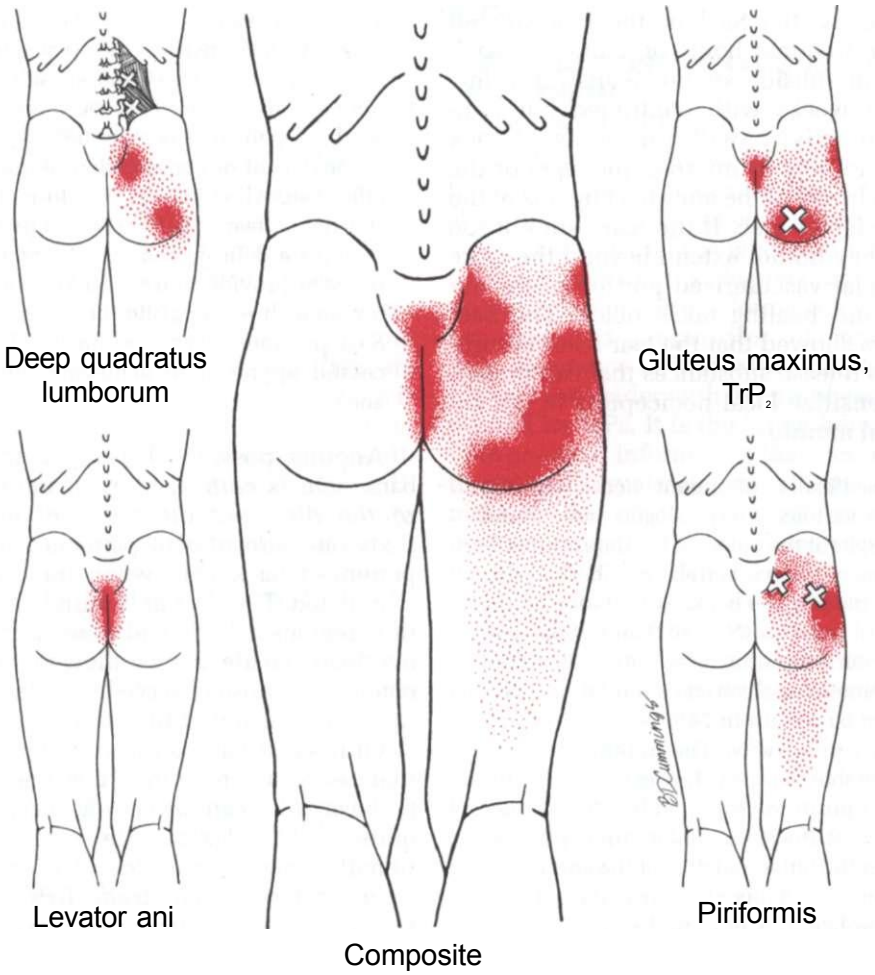


Figure 41.3. Individual pain patterns of several trigger points that refer pain to the pelvic region and that may superimpose on each other. The composite pain pattern in the middle of the figure represents the pain (red) that the patient can experience. It shows the sum of pain referred from trigger points **(Xs)** in specific regional muscles: the deep fibers of the quadratus lumborum, TrP_2 of the gluteus maximus, the levator ani, and the piriformis. The component pain patterns are placed around the composite figure.

source of the low back pain in less than 10% of 454 patients studied.[20]

As is also true for TrPs, the nature of the examination required to accurately diagnose these joint dysfunctions throughout the body requires a great deal of training and skill which is not generally found among medical practitioners. The professionals most likely to have these skills are osteopaths, physical therapists, and chiropractors, but the quality of training received and the level of skill achieved is highly variable among individuals. Articular dysfunctions are a source of musculoskeletal pain, including backache, that is often overlooked.

Intervertebral Discs: Surface Damage. The Saal brothers, JS and JA Saal, presented a convincing story at a conference that appears never to have been published in a similar coherent manner, at least not in totality. It is available as individual papers[14,37-39] and deserves serious consideration. Unfortunately, the condition isn't revealed by any routine laboratory test or imaging technique so it is not especially attractive to third party payers or to those needing objectively confirmed diagnoses.

Basically, the Saal brothers postulated that the frequent bouts of acute low back pain that subside spontaneously in a few days or weeks with continued but nonstressful activity (with care to avoid back strain) are due to microscopic tears of the surface layers of the annulus fibrosus of the intervertebral disc. If the tears are not too deep, they do not extend beyond the more superficial vascularized portion of the annulus and healing takes place. The Saal brothers showed that the tear itself is sufficient to release substances that can powerfully sensitize local nociceptors in the superficial annulus.

Specifically, abundant nerve endings with various morphologies are found throughout the outer half of the annulus fibrosus of lumbar vertebrae.[7] These nerves refer pain to the back. Stimulation of the central annulus (N = 183) and of the central lateral annulus (N = 144) with a blunt instrument or electrical stimulation[27] produced back pain in 74% and 71% of subjects, respectively. The annulus is most vulnerable to injury during rotation in the flexed position. Repeated insults produce lesions that are circumferential splits between the outer lamellae of the annulus fibrosus with avulsions, particularly in the posterolateral regions of the disc. With repeated injuries, progressively deeper laminae may be disrupted, eventually forming a radial fissure. When severe enough, these fissures permit disc protrusion, but protrusion is not necessary for a pain episode. A small initial tear apparently can be sufficient to produce pain.

Saal, *et al.*[39] demonstrated, in interarticular disc material excised at surgery for radiculopathy, high levels (up to 100 times normal) of phospholipase A_2. Phospholipase A_2 plays a critical role in the genesis of inflammatory mediators such as prostaglandins, leukotrienes and platelet activating factor. Its presence is characteristic of familiar inflammatory conditions characterized by pain and tenderness. A second paper reinforced these same observations.[14]

In summary, it is likely that a relatively minor tear of the annulus (or an extrusion) can cause release of phospholipase A_2 that sensitizes the nociceptors in the peripheral part of the annulus (and possibly also adjacent nerve roots) causing back pain that is extremely sensitive to movement or compression of that specific disc. Allowing the disc to repair itself (if the tear is sufficiently superficial) requires a delicate balance of enough activity to provide nutritional support, but not enough to aggravate the injury.[38] The Saal program[37] was one apparently successful approach to achieving this balance.

Another possible discogenic source of back pain is *enthesopathy at the junction of the disc and the vertebral endplate.* Sixty-one percent of 67 patients tested[27] experienced back pain when this area was stimulated. Horn, *et al.*[19] found that insertion regions which had been exposed to overload tensile forces showed the same changes as those observed in epicondylitis in another region of the body.

Of interest here is the strong possibility that discogenic pain from disc tears or disc enthesopathy can cause referred pain and quite likely reflex muscle spasm of functionally related muscles. The pain can mimic referred pain from TrPs, and the spasm is likely to be a major activator of TrPs in the muscles that are in spasm. Note, this does NOT assume that pain is coming from the muscle spasm, per se. Both the spasm and pain may be present, but for different reasons. It is very likely that most muscle spasm is of reflex origin outside of the muscle in spasm.

Multiple Interacting Conditions. It is not unusual for a patient presenting with musculoskeletal pain to have several organic dysfunctions contributing to the pain. Common combinations include fibromyalgia with myofascial TrPs, and articular dysfunctions with related myofascial TrPs. Incidence of the combination of surface damage to intervertebral discs or disc enthesopathy and myofascial TrPs is not as well established and needs to be determined by research studies.

A study of lumbar dorsal horn neurons in cats[17] showed that all 118 of the neurons

studied had receptive fields in deep somatic tissues, and/or regional skin. Seventy-two percent of the neurons were "hyperconvergent" in that they responded to stimulation of many different somatic tissues. Neurologically, the origin of pain can be far more complex than is generally appreciated. Myofascial TrPs provide a major source of nociceptive input from the muscles.

More than half (in one study it was three-quarters[16]) of patients with fibromyalgia ALSO have active myofascial TrPs contributing to their misery. These TrPs are more amenable to effective treatment than is the fibromyalgia. Apparently these two conditions can aggravate each other, and identifying them requires different and specific diagnostic procedures. Treatment approaches and prognosis also are quite different for the two conditions. Few clinicians are trained and skilled in the diagnosis of BOTH conditions. Each diagnosis tends to "belong" to separate medical specialties, and the educators of many specialties fail to train their students to recognize either condition.

Rarely do practitioners of manual medicine (who restore restricted movement of joints) relate the joint dysfunction being addressed to the specific muscle(s) associated with that dysfunction. Many practitioners usually relate to the muscles only in vague and general terms. One of the few exceptions is Dr. Karel Lewit, who for years has recognized the close relationship between the increased tension (due to TrPs) of specific muscles and the associated articular dysfunctions. In the case of thoracolumbar articular lesions[28] he identifies the iliopsoas muscle, the thoracolumbar portions of the erector spinae, the quadratus lumborum, and (less frequently) the rectus abdominis muscle.

A number of osteopathic physicians who were originally well trained in the identification and correction of articular dysfunctions have also become skillful in identifying and treating TrPs. Some physical therapists have taken the initiative to learn both skills following graduation. It is remarkable how much more effectively these dually trained clinicians (when skillful) can resolve common musculoskeletal pain problems.

SECTION C

POSTURAL CONSIDERATIONS: STATIC AND DYNAMIC (Figures 41.4-41.8)

Much is written in various books and articles about poor posture, but the knowledge about its effect is not always transmitted in a practical way to those who need it. As discussed in many of the chapters of this book, poor posture in various forms is a powerful activator and perpetuator of myofascial TrPs. Reference to individual chapters will provide the reader with details of each muscle and its TrPs. It is the intent here to provide practical information that can help patients with myofascial pain learn how to make needed changes. Joseph[23] noted that posture varies markedly among apparently healthy, normal individuals; however, if the muscles are causing pain, postural strain must be identified and resolved.

Excessive forward-head posture (anterior head positioning with posterior rotation of the occiput) and **"rounded" forward shoulders** frequently occur together, resulting in what is commonly referred to as round-shouldered, slumped, or slouched posture. This posture may be initiated from above or from below, that is, from the occiput and cervical spine downward or from the base of support upward. The pull of tense, shortened muscles (e.g., the rectus abdominis) can induce or perpetuate a forward-head posture. Trigger points in the upper rectus abdominis may need to be inactivated before postural correction can be maintained. Regardless of the initiating factor (including muscle weakness which must be considered in treatment), the resultant misalignment of body segments leads to increased strain on the muscles and supporting structures, causing vulnerability to injury and pain. If prolonged, it can result in overstretching and weakening of muscles and ligaments, adaptive shortening of muscles, activation of TrPs, stretching of some nerves and compression of others, increased pressure on discs, decreased lung capacity, and fatigue, as well as pain and many other problems (such as in the temporomandibular region, *see* Chapters 5 and 8-11).

Excessive forward-head posture limits cervical rotation, indirectly limits humeral range of motion (particularly elevation and rotation), and it activates and perpetuates TrPs in the posterior cervical muscles (*see* Chapters 15, 16, and 17). Other authors have emphasized the importance of recognizing this posture and improving it, especially if the patient has related symptoms.[9,25,29] Round-shouldered posture is an important mechanical activating and perpetuating factor for TrPs in the *pectoralis major* (*see* Chapter 42) and the *pectoralis minor* (*see* Chapter 43) muscles that then perpetuate the posture. Adaptive shortening from TrPs in the pectoralis minor may lead to "coracoid pressure syndrome,"[24] arm pain, and stretch weakness of posterior scapular muscles such as the lower trapezius. It is significant that what happens in one part of the body affects other areas.

It is important to remember that *what happens "from the ground up" influences head position.* This influence occurs whether a person is standing or sitting. The feet and the pelvis are significant supporting structures, but so are all the other segments between the base of support and the head. The position of the lumbar spine (lordotic or flattened) and tilt of the pelvis influence head position. In sitting, standing, walking, or reaching, *a flattened lumbar spine with loss of normal lordosis and excessive posterior pelvic tilt can induce a stressful forward-head posture (anterior head positioning) with posterior rotation of the occiput and rounding forward of the shoulders.* Muscle imbalances, pelvic asymmetry, lower limb-length inequality, and/or an excessively pronated foot are among the many factors that can influence posture, including anterior head positioning. An excessively pronated foot can cause genu valgum and medial rotation of the thigh, which can lead to postural imbalances in the pelvis, lumbar spine, and cervical spine. These imbalances can induce TrPs in the vastus medialis, gluteus medius, and other muscles. The base of support, whatever it is, needs to be as level as possible, and the spinal curvature needs to approach normal, for "good" posture.

Posture is **dynamic** more than static. Even in quiet "relaxed" standing, slight postural sway occurs.[33] When sitting or standing, a person has a need to shift position either for comfort or to maintain balance, or to do a task. *Change of position* is inevitable. Periodic movement is needed in order for ligaments, cartilage, intervertebral discs, and muscles to be nourished. These structures respond poorly to being in one position for a prolonged period. Daily living requires moving about from lying to sitting, sitting to standing, standing to walking, and the reverse.

The assessment of anterior head position is discussed in Chapter 5, Section C. *Normally, when the cheek bone is aligned in the same vertical plane as the sternal notch, the head and neck are in an erect position without muscular overload.* It is important for clinicians to help patients become *aware* of how they usually sit, stand, and move, and to help patients learn to *correct* bad postural habits and muscle imbalance in dynamic situations as well as in relatively static ones.

Patients with myofascial pain due to TrPs should learn to **change position often,** to reposition to nonstressful "erect" posture, and to perform appropriate stretching and other postural exercises. Repositioning to erect posture applies not only while sitting, (for example at a computer desk), but also while driving a car, while walking, while reaching and lifting, and at appropriate times while participating in sports activities. In a work setting, until good posture and habits are ingrained, these patients can use a timer or a clock alarm as a reminder to change position and to perform appropriate postural exercises.

Standing

When a person stands in a "slouched" posture, with the shoulders and head projected forward, the center of the body weight projects solidly on the back of the heels (Fig. 41.4A). When the subject is instructed (erroneously) to "Stand up straight!," posture may improve slightly, but the line of gravity remains on the heels (Fig. 41.4B). Maintaining this straightened position requires constant voluntary effort by the subject. The overloaded muscles soon fatigue and the person becomes discouraged.

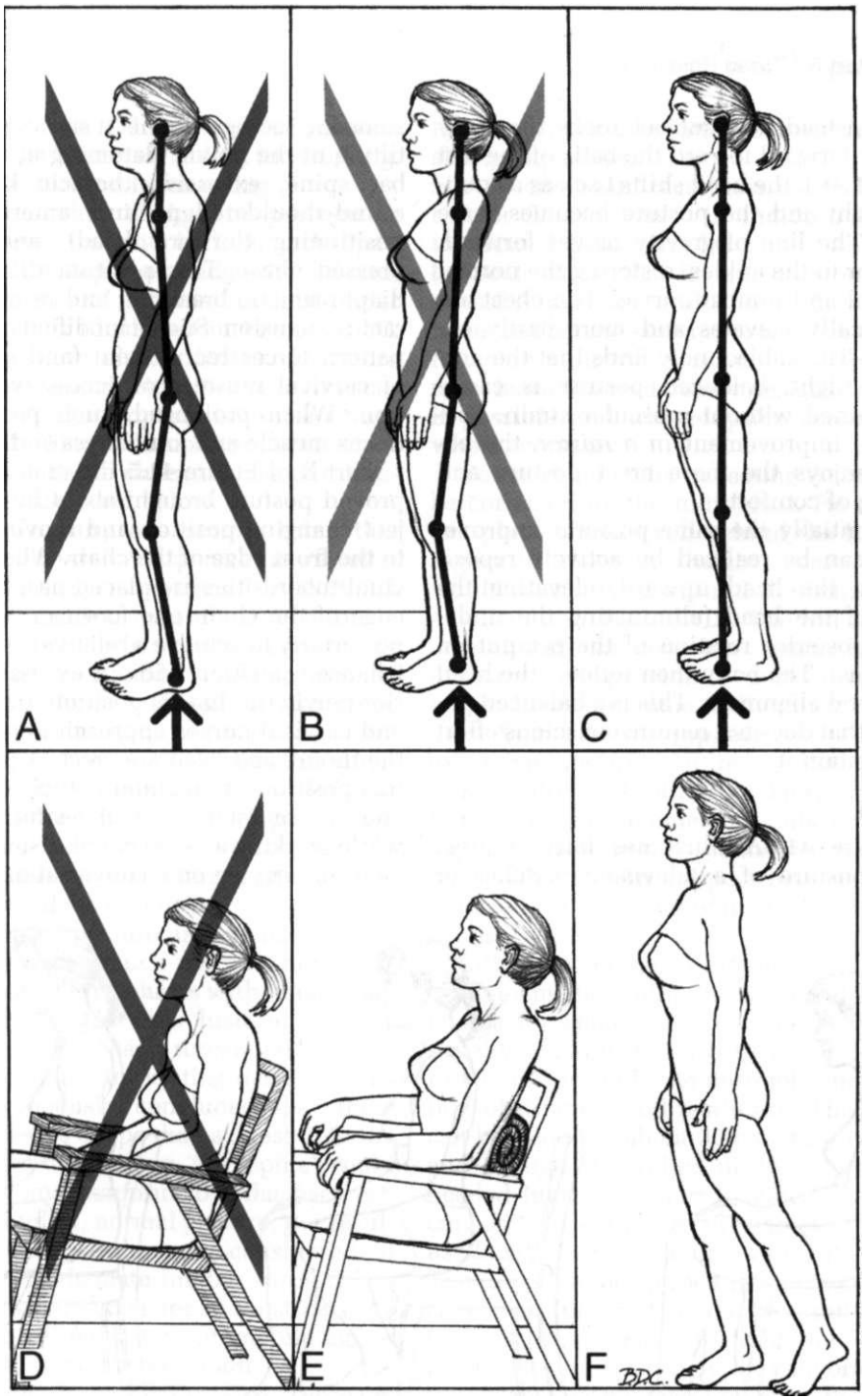


Figure 41.4. Improvement of standing and seated posture. **A,** this stooped, round-shouldered standing posture is aggravated by the increased tension that is caused by trigger points in the pectoralis major muscle. **B,** slight improvement in posture by having the patient "Stand up straight and hold the shoulders back!" (a position that is fatiguing and difficult to maintain). **C,** marked improvement when the patient shifts the body weight from the heels onto the balls of the feet, which moves the head backward over the shoulders as a counterweight and straightens the lines of weight bearing. The *arrows* mark the shift in the *center-of-gravity line*, through the feet. **D,** the stooped, round-shouldered seated posture that often results from pectoralis major trigger points and poorly designed chairs. **E,** addition of a lumbar pillow or roll maintains the normal lumbar curve (lordosis) and produces more erect posture. For a chair with this type of reclining back, **E** shows a relatively desirable posture. Some other chairs may be more adaptable. Refer to Figure 16.4D for an example of good sitting posture of a subject writing at a work table. **F,** forward-shifting posture that exaggerates the weight shift shown in panel **C** and can improve walking posture, particularly in fast walking.

If, instead, the subject rocks the body weight forward toward the balls of the feet (Fig. 41.4C), the head shifts back as a counterweight and the posture becomes more erect. The line of gravity moves forward, anterior to the ankles, restoring the normal cervical and lumbar curves. The chest automatically elevates and more easily expands. The subject now finds that the normal upright, balanced posture is easily maintained without muscular strain. Seeing the improvement *in a mirror,* the patient enjoys the more erect posture and feeling of comfort.

Essentially the same postural improvement can be realized by actively repositioning the head upward, elevating the back of the head (eliminating the undesired posterior rotation of the occiput on the atlas). The body then follows the head into good alignment. This is a balanced position that does not require conscious effort to maintain it.

Sitting

Figure 41.5A illustrates fairly typical poor posture of a television watcher or someone seeing a movie. It shows posterior tilting of the pelvis, flattening of the lumbar spine, excessive thoracic kyphosis, round-shouldered posture, anterior head positioning (forward head), and a depressed chest. This position discourages diaphragmatic breathing and restricts thoracic expansion. Such a modified breathing pattern forces recruitment (and overload) of cervical muscles of accessory respiration. When prolonged, such posture induces muscle and joint stress and pain.

Part B of Figure 41.5 illustrates the improved posture brought about by the subject changing position and **moving closer to the front edge of the chair.** When the ischial tuberosities are placed near the front edge of the chair, one foot can be placed posteriorly to achieve a relatively effortless balanced position without excessive anterior pelvic tilt. In this position, the lumbar and cervical curves approach normal, and the thorax and head are erect. A person in this position can maintain efficient airflow and can maintain a good restful position while working at a keyboard, listening to a lecture, carrying on a conversation, watch-

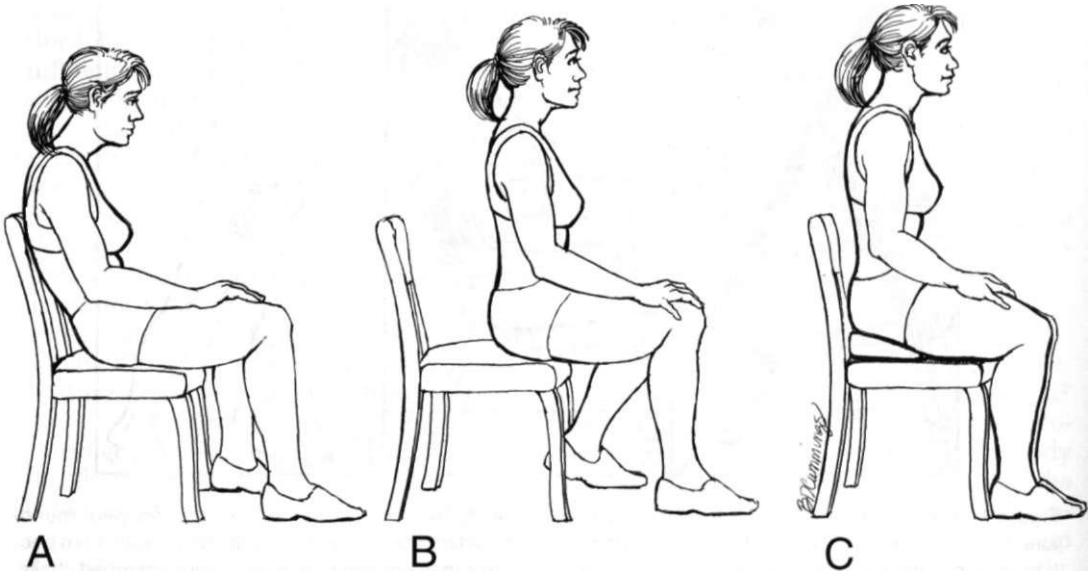


Figure 41.5. Slumped and desirable sitting postures. **A,** slumped, undesirable posture with posterior tilting of the pelvis, flattening of the lumbar spine, excessive thoracic kyphosis, round-shouldered posture, anterior head positioning (forward head), and a depressed chest. **B,** a desirable balanced posture accomplished by sliding forward on the chair so that the ischial tuberosities are closer to the front edge of the chair and one foot is placed more posteriorly. **C,** An alternative balanced erect posture accomplished by placing a small wedge under the ischial tuberosities.

ing a movie, etc. This change of position places the subject near a desk for working, or near a dining table for eating.

When another change in position is needed, one can sit with a small **pad (preferably a sloped or wedge-shaped pad) under the ischial tuberosities** *(not* under the thighs), as illustrated in Figure 41.5C. In this position also, a balanced posture can be maintained with little or no muscular effort. Another option is to use an appropriately placed lumbar support when sitting back in the chair; the use of a lumbar roll is discussed below.

If there is any one simple thing that makes it easier for a person to develop good posture and movement, it is to "*think tall*"—to **lengthen** oneself. If you move your head upward in relation to your body (away from your body, making yourself taller), your body will follow and come into good alignment. Although it is not practical for a person to perform this slight movement every minute of the day, the more often the movement is performed, with every daily movement activity, the easier it becomes to attain and maintain stress-free balanced posture.

Poor sitting posture unfortunately is encouraged by the much too common practice of constructing **chairs with inadequate lumbar support.**[42] This historic practice was reinforced by early research that established the model for seating design.[18] This study erroneously concluded that there was no need to shape the backrest to fit the lumbar curve because the spine could straighten and conform to a flat backrest. Neither comfort, normal posture, nor resultant muscular strain were considered in the study. Inadequate lumbar support is a major contributory factor in most patients for whom riding in an automobile aggravates back, chest, or neck pain.

Later on, a comprehensive approach provided more realistic data for the design of comfortable physiologic seating.[13] Selection of a pain-relieving chair requires that serious consideration be given to the needs of the muscles.[42] Figures 41.4**D** and **41.**5A show the result of sitting in a chair without lumbar support; the shoulders are "rounded" forward, the head is projected forward, the pelvis is tilted posteriorly, and the lumbar curve is flattened, resulting in a slumped posture.

Any one chair, regardless of its design, cannot fit everyone. An individual with myofascial pain must learn to distinguish between chairs that fit and promote nonstressful posture, and those chairs that have inadequate support and cause stressful pain-inducing posture. The **patient must learn to avoid poorly designed chairs if possible and use adaptive corrections when necessary.**

Some of the faults in seating design can be corrected by the individual. He or she can **place a small roll behind the lumbar region** at about the height where a belt would normally go around the waist (Fig. **41.4E** and *see* Fig. **16.4D).** The roll should comfortably support the thoracolumbar junction and provide a normal lumbar curve. However, some individuals benefit most from a **lower support** that prevents excessive posterior tilting of the pelvis. Since individuals **differ in body proportion,** available commercial rolls or other supports may not fit some people, and seats with built-in support may not provide adequate support of the body area that needs it.

Either a commercial *inflatable* pillow or a "homemade" support may be the best choice for some people. Foam rubber is usually too soft, but a bath towel, tightly rolled, can provide the desirable combination of firmness and resilience. The towel can be folded to about **30** cm **(12** in) wide, and enough of it rolled up to provide the needed lumbar support (usually 7.5 to **10** cm, or **3** to **4** inches, in diameter) when used with any chair or automobile seat. The towel can be slipped into an attractive cover with ties that can be put around the backrest of the chair to hold the roll in place. The roll also may be supported by two straps thrown over the top of the chair backrest, with enough lead weight sewn into the end of each strap to provide a counterweight and hold the roll in place. If a lumbar support slips out of place and does not remain in the correct position *for the individual* (as often happens when a person is driving a car or working at a desk and moving the limbs and body segments), the best solution may be to tie the support

around the person's waist rather than around the chair. Also, for the support to be effective, the individual must slide the pelvis and buttocks back to the posterior part of the seat.

The **chair seat** must also be designed with sufficient hollow or backward slope at the bottom of the backrest so that the buttocks are not pushed forward. For comfort during reading, talking and watching TV (but not while eating meals or working at a desk), the **chair-back should slope** 25-30° posteriorly from the vertical so that a person can be comfortably relaxed without the need to slide the hips forward on the seat.

The chair should have **armrests** at such a height that they support the person's elbows and/or forearms while allowing the shoulders to be relaxed in a neutral position (not hiked up). If a chair does not have armrests, or has armrests that are too low, a person who sits in it (particularly for a prolonged period) will likely cross the arms in front of the chest in order to be comfortable. This position induces round-shouldered posture and shortens the pectoral muscles and other anterior chest muscles, perpetuating their TrPs and tending to produce stretch weakness in the interscapular muscles. For prolonged sitting, an individual should avoid a chair without armrests. Arm rests that are too low can be made more effective by the addition of pads to increase the height.

For sitting at a desk or workstation, a chair needs to be adjustable and the worker must be able to move. A therapeutic exercise ball (appropriately sized) can be alternated with the usual desk chair. An **inclined board** can encourage healthy posture for a person who does a lot of writing at a desk or table (*see* Fig. 16.4D). When a desk or table is not available or desirable, erect sitting can be promoted by the use of a **lapboard** on a pillow or resting across the chair arms.

Unnecessary muscular tension should be reduced. To achieve relaxation, the patient should focus attention on *consciously* feeling the support provided by various parts of the chair (armrests, seat, backrest and perhaps headrest). The same method of relaxation applies in bed at night as the patient concentrates on detecting the texture of the sheet and mattress wherever it supports the body.

Movement Activities

F. Matthias Alexander deserves credit for his *principle of movement* that uses the conscious mind to change subconscious muscle patterns.[12] During his observations in the early part of the century, he found that instead of concerning himself with "doing," he needed to *prevent* doing things in his usual way (prevent movement habits that were causing physical and emotional stress). For example, in his personal experiments he found that instead of allowing tension and shortening of posterior neck muscles to move his head backward and down, compressing the spine, he needed to relax those muscles and let his head move *upward* when it moved forward (lengthening the spine instead of arching it) in daily movement activities such as rising to standing and walking. (The basic movement involves elevating the occiput with a slight anterior rotation of the occiput on the upper spine). Jones[22] stated that the significant aspect of the technique is not posture, but movement; the movement produces a kinesthetic effect of lightness that is pleasurable and thus reinforcing.

Although Alexander's method is an educational process that would best begin early in life with a sensory awareness of habitual movement patterns, it is never too late to use the conscious mind to adopt patterns of movement that lead to maximum balance and coordination with minimal expenditure of energy.

Walking Up Stairs. Figure 41.6 shows an undesirable method (part A) and a less stressful, more efficient method (seen in part B) of walking up stairs. The figure on the left shows a person who shifts weight to the forward foot and then with great effort extends the leg to lift the body weight to the next step. It also shows a posteriorly-rotated occiput, anterior head positioning, shortened pectorals, and a depressed chest. The figure on the right illustrates the improvement in movement and posture that occurs when the subject places his forward foot lightly on the step and deliberately elevates his occiput, let-

ting his head move upward and forward as he gradually extends the leg, with his body following the head. This method of movement provides good head/neck position and chest elevation, as well as balanced transfer of weight without excess energy expenditure.

Jogging. Figure 41.7 illustrates the change that occurs when the head moves upward (occiput is raised) as it moves forward, and the body follows the head, during jogging. In Figure 41.7A, the jogger's shoulders are hunched up and tense, and the tense posterior neck muscles are inducing a posteriorly-rotated occiput. This jogging style can activate and perpetuate TrPs in the suboccipital, splenius capitis, levator scapulae, and trapezius muscles. In Figure 41.7B, the body is lengthened by the upward and forward-rocking movement of the head, and the shoulders and arms appear more relaxed.

Figure 41.4F illustrates one way to attain an erect balanced posture when walking fast. Dr. Janet Travell recommended this method and referred to it as the "Indian Lope." The individual shifts the pelvis and hips forward, exaggerating the shift of weight to the balls of the feet, and rapidly advances the rear foot to keep from falling forward. The feet follow the body, and each step receives a vigorous push-off from the calf muscles, making it efficient to cover long distances quickly.

Letting the head move upward in line with the long axis of the body prior to weight shift would allow the body to follow with less muscular effort.

Turning, Rising, Eating, Reaching, Pulling, Pushing, Lifting. Any or all of these activities of daily living may be facilitated by using the basic principle of "thinking tall" or letting the head move upward and slightly forward (preventing posterior tilting of the head) while carrying out the movement or activity.

Of course, other well-known principles of body mechanics apply as well. In **rising from a chair,** for example, one should shift the hips toward the front edge of the chair

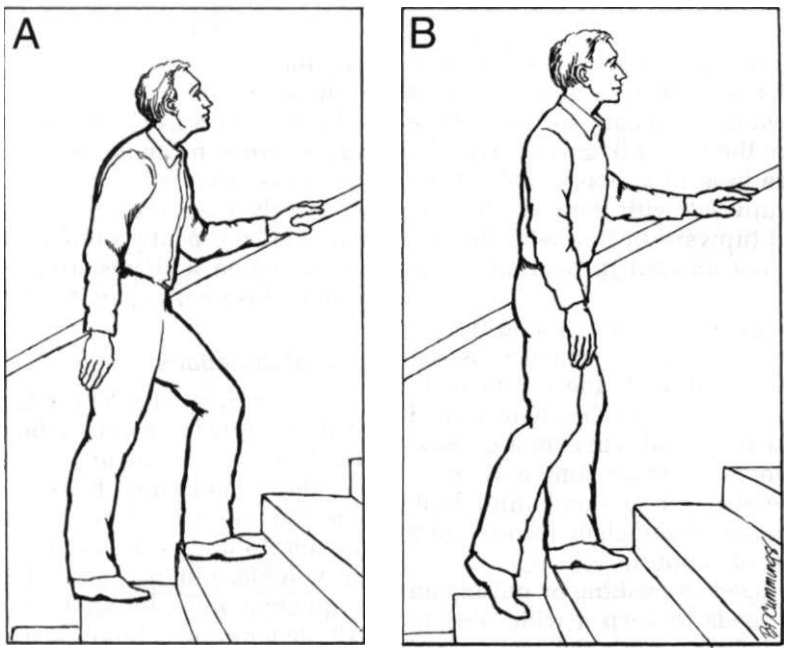


Figure 41.6. Undesirable and efficient ways of walking up stairs. **A,** undesirable transfer of weight with anterior head positioning, posteriorly-tilted occiput, shortened pectorals, and depressed chest. **B,** upward direction with improved head position, head leading the body upward, and a more natural elevated chest. (See text for details) (Redrawn from Barker S: *The Alexander Technique.* Bantam Books, New York, 1978.")

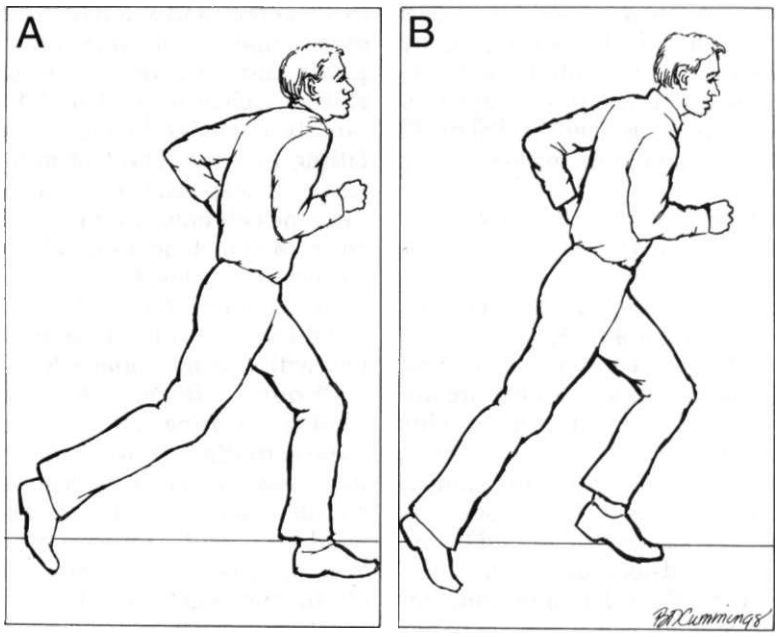


Figure 41.7. Strained and desirable ways of jogging. **A,** illustration of a compressed, stressful jogging style showing a subject with a posteriorly-rotated occiput and shoulders that are shrugged up and tense. **B**, an illustration of how lengthening and decompression from a head-upward movement can result in good head and shoulder position without excessive tension. See text for details. (Redrawn from Barker S: *The Alexander Technique.* Bantam Books, New York, 1978.[4])

and spread the feet apart with one foot placed posteriorly (to enlarge the base of support), then lean the body forward *at the hips* to move the center of gravity over the center of the base of support, and let the head move upward with the body following the head (upward in line with the forward torso, *not* upward in relation to the vertical).

An alternative method of rising from a chair involves the same sequence as described above, but with the entire body turned toward one side of the chair instead of facing straight ahead. This latter method is helpful when one lower limb is weak or painful. The stronger or nonpainful limb should be closer to the chair to provide a stronger base of support.

When engaged in pushing **or** pulling an object, one needs to keep a wide base of support for balance, with the feet spread apart in line with the direction of force. The force should be applied near or in line with the center of gravity of the object. When lifting an object, one should (in addition to maintaining a balanced stance) keep the object close to the body and avoid lifting above shoulder height.

Figure **41.8B** illustrates a way of **reaching** forward as recommended by Brugger.[9] If one hip is extended, placing one foot more posteriorly than the other, an individual may avoid the bent posture with a flattened lumbar spine and posteriorly-rotated occiput that is seen in part A of this figure.

Special Situations

For requirements and adaptations related to **sitting in an automobile** and **sitting at a desk** (car posture and office habits), as well as for additional body mechanics issues, the reader is referred to section **14** in Chapter **48** and section C in Chapter **5** of this volume, and to a comprehensive chapter on ergonomics by Khalil, *et.al.*[26]

In general, one should keep in mind:

- Any type of seating should allow for body symmetry and support of the normal spinal curves, in particular the cervical lordosis and lumbar lordosis,

with a minimum of muscular energy on the part of the person.

- A chair must be adjusted to the individual. No single chair fits everyone, nor does it fit every situation, even for the same person.
- The feet should rest solidly on the floor or on some type of footrest and not dangle.
- Flexibility and comfort are important. A subject at a work station should be able to change position as needed for specific tasks and, as needed, to provide adequate circulation and nutrition to body structures.
- A headset will be beneficial to anyone who spends a significant amount of time talking or listening on the telephone.
- To avoid twisting stresses when looking toward an object or toward another person, one should turn the entire body, and not just the head.
- When a person types from copy, it is important to place the copy at eye level and as near as possible to the center of the line of vision.
- It is important to avoid a twisting movement while bending forward, especially when lifting, pushing, or pulling.
- One should take frequent breaks to stand momentarily, walk a few feet, or change the activity.

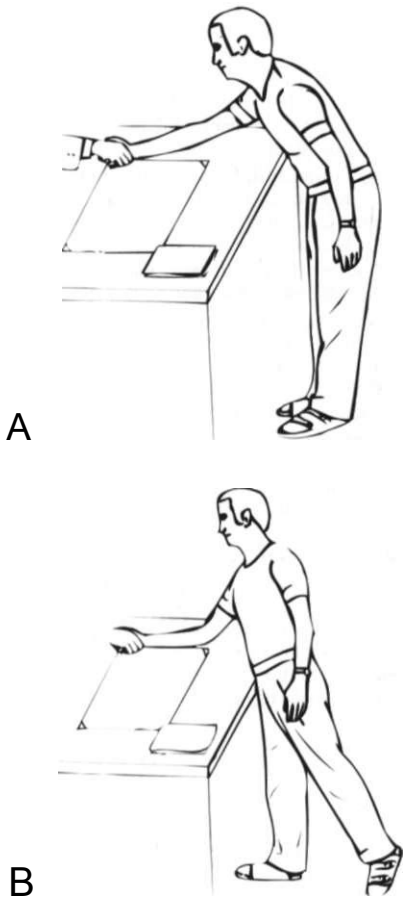


Figure 41.8 Undesirable and desirable ways of reaching forward. A, reaching forward over a desk in a strained manner that produces a flattened lumbar spine, upper trunk and hip flexion, and a posteriorly-rotated occiput. **B,** the improved posture when the subject extends one hip, placing that foot more posteriorly than the other. (Adapted from Briigger A: ***Die Erkrankungen des Bewegungsapparates und seines Nervensystems.*** Gustav Fischer Verlag, Stuttgart, New York, 1980. [Fig. 337].)

REFERENCES

1. **Alexander FM:** *Man's Supreme Inheritance.* **Reissued, Centerline Press, Long Beach, 1988.**
2. **Alexander FM:** *The Use of Self.* **Methuen Co. Ltd, Great Britain, 1932. Reprinted, Victor Gollancz, London, 1996.**
3. **American Academy of Physical Medicine and Rehabilitation: Academy declines to endorse guideline for low back pain.** *Arch Phys Med Rehabil* **76:294, 1995.**
4. **Barker S:** *The Alexander Technique.* **Bantam Books, New York, 1978.**
5. **Bogduk N: Lumbar dorsal ramus syndrome.** *Med J Aust* **2:537-541, 1980.**
6. **Bogduk N, Simons DG: Neck pain: joint pain or trigger points? Chapter 20. In:** *Progress in Fibromyalgia and Myofascial Pain,* **Vol. 6 of** *Pain research and Clinical Management.* **Edited by Vaer0y H, Mersky H. Elsevier, Amsterdam, 1993 (pp. 267-273).**
7. **Bogduk N, Twomey LT:** *Clinical Anatomy of the Lumbar Spine.* **Churchill Livingstone, New York, 1987.**
8. **Bonica JJ, Sola AE: Other painful disorders of the low back. Chapter 72. In:** *The Management of Pain.* **Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR,** *et al.* **Lea & Febiger, Philadelphia, 1990 (pp. 1490-1498).**
9. **Briigger A:** *Die Erkrankungen des Bewegungsapparates und seines Nervensystems.* **Gustav Fischer Verlag, Stuttgart, New York, 1980.**
10. **de Girolamo G: Epidemiology and social costs of low back pain and fibromyalgia.** *Clin J Pain 7(Suppl. 1):S1-S7,* **1991.**
11. **Dejung B: Manuelle Triggerpunktbehandlung bei chronischer Lumbosakralgie.** *Schweiz Med Wochenschr 124(Suppl* **62).82-87, 1994.**
12. **Dejung B: Die Behandlung des akuten Hexenschusses.** *Der informierte Arzt-Gazette Medicale* **9:619-622, 1996.**

13. Diffrient N, Tilley AR, Bardagjy JC: *Humanscale 1/2/3.* The MIT Press, Cambridge, Mass., 1974.
14. Franson RC, Saal JS, Saal JA: Human disc phospholipase A_2 is inflammatory. *Spine 17(6 Suppl.):S129-*S132, 1992.
15. Gerwin RD: Myofascial aspects of low back pain. *Neurosurg Clin North Am 2(4):761-784,* 1991.
16. Gerwin R: A study of 96 subjects examined both for fibromyalgia and myofascial pain [Abstract]. */Musculoske Pain 3(Suppl 1):*\2\, 1995.
17. Gillette RG, Kramis RC, Roberts WJ: Characterization of spinal somatosensory neurons having receptive fields in lumbar tissues of cats. *Pain* 54:85-98, 1993.
18. Hooten EA: *A Survey in Seating.* Heywood-Wakefield Co., Gardner, Mass., 1945. Reprinted by Greenwood Press, Westport, Conn., 1970.
19. Horn V, Vlach O, Messner P: Enthesopathy in the vertebral disc region. *Arch Orthop Traum Surg 110(4):187-189,* 1991.
20. Jackson RP, Jacobs RR, Montesano PX: 1988 Volvo award in clinical sciences. Facet joint injection in low-back pain. A prospective statistical study. *Spine 13(9):996-971,* 1988.
21. Jefferson JR, McGrath PJ: Back pain and peripheral joint pain in an industrial setting. *Arch Phys Med Rehabil* 77:385-390, 1996.
22. Jones FP: *Body Awareness in Action: A Study of the Alexander Technique.* Schocken Books, New York, 1976.
23. Joseph J: *Man's Posture.* Charles C Thomas, Springfield, 1960.
24. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 349-361).
25. Kendall HO, Kendall FP, Boynton DA: *Posture and Pain.* Williams & Wilkins, Baltimore, 1952. Reprinted by Robert E. Krieger, Melbourne, FL, 1971.
26. Khalil TM, Abdel-Moty E, Steele-Rosomoff R, *et al:* The role of ergonomics in the prevention and treatment of myofascial pain. Chapter 16. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 487-523.
27. Kuslich SD, Ulstrom CL, Michael CJ: The tissue origin of low back pain and sciatica: a report of pain response to tissue stimulation during operations on the lumbar spine using local anesthesia. *Orthop Clin North Am 22(2):181-187,* 1991.
28. Lewit K: Muscular pattern in thoraco-lumbar lesions. *Manual Med* 2.105-107, 1986.
29. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Butterworth- Heinemann, Oxford, 1991.
30. McCall IW, Park WM, O'Brien JP: Induced pain referral from posterior lumbar elements in normal subjects. Spine 4:441-446, 1979.
31. Mense S, Simons DG: *Muscle Pain: Understanding Its Nature, Diagnosis, and Treatment.* Williams & Wilkins, Baltimore, in press.
32. Miller DJ: Comparison of electromyographic activity in the lumbar paraspinal muscles of subjects with and without chronic low back pain. *Phys Ther* 65(9j:1347-1354, 1985.
33. Murray MP, Seireg AA, Sepic SB: Normal Postural Stability and Steadiness: Quantitative Assessment. *J* Bone *Joint Surg* 57A:510-516, 1975.
34. Rosen NB: The myofascial pain syndromes. *Phys Med Rehabil Clin North Am 4(Feb)Al-63,* 1993.
35. Rosomoff HL, Fishbain D, Goldberg M, *et al:* Myofascial findings in patients with "chronic intractable benign pain" of the back and neck. *Pain Manage* 3(2):114-118, 1990.
36. Roy SH, De Luca CJ, Casavant DA: Lumbar muscle fatigue and chronic lower back pain. *Spine 14(9)* :992-1001, 1989.
37. Saal JA: Dynamic muscular stabilization in the nonoperative treatment of lumbar pain syndromes. *Orthop Rev 19(8)*:691-700, 1990.
38. Saal JA, Saal JS, Herzog RJ: The natural history of lumbar intervertebral disc extrusions treated nonoperatively. Spine *15(7)*-683-686, 1990.
39. Saal JS, Franson RC, Dobrow R, *et al.:* High levels of inflammatory phospholipase A_2 activity in lumbar disc herniations. Spine 15(7):674-678, 1990.
40. Seidel H, Beyer H, Brauer D: Electromyographic evaluation of back muscle fatigue with repeated sustained contractions of different strengths. *Eur J Appl Physiol* 56:592-602, 1987.
41. Simons DG, Travell JG: Myofascial origins of low back pain. Parts 1,2,3. *Postgrad Med* 73:66-108, 1983.
42. Travell J: Chairs are a personal thing. *House Beautiful* (Oct):.190-193, 1955.
43. Twomey LT, Taylor JR: *Physical Therapy of the Low Back.* Churchill Livingstone, New York, 1987.

CHAPTER 42
Pectoralis Major Muscle (and the Subclavius)

HIGHLIGHTS: **REFERRED PAIN** from pectoralis major trigger points (TrPs) may localize substernally, may include the anterior chest and breast, and may extend down the ulnar aspect of the arm to the fourth and fifth fingers. The region of the costal section of the pectoralis major muscle can have a somatovisceral TrP that is located medially on the *right* side. The inactivation of this TrP terminates episodes of cardiac arrhythmia. When on the *left* side, pectoralis major TrPs refer pain in patterns that are easily mistaken for the pain of ischemic heart disease. **ANATOMY** of the pectoralis major muscle is complex. It is rarely mentioned that this muscle consists of multiple overlapping laminae in a playing-card arrangement. The muscle is divided into clavicular, sternal, costal, and abdominal sections. Several caudal laminae wrap around the lateral border of the muscle. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the pectoralis major may be caused by stress overload of the muscle or by referred phenomena associated with a myocardial infarction. **PATIENT EXAMINATION** reveals shortening of the pectoralis major muscle by active or latent TrPs, which often pulls the shoulders forward to produce a stooped, round-shouldered, head-forward posture. **TRIGGER POINT EXAMINATION** is performed by palpating the clavicular, sternal and costal sections of the muscle for tender nodules within firm bands, which often react with highly visible twitch responses. **TRIGGER POINT RELEASE** by spray and stretch is performed by abducting and flexing the arm at the shoulder while applying the vapocoolant cephalad over the stretched muscle fibers and distally over the arm. Other manual techniques can also be useful. **TRIGGER POINT INJECTION** requires care when injecting deeper pectoral musculature over the thoracic cage. Pincer palpation is used whenever possible. **CORRECTIVE ACTIONS** start with convincing the patient (when true) that the myofascial chest pain is a treatable pain of skeletal muscle, rather than of cardiac origin. Correction of poor standing and sitting posture, avoiding mechanical overload of this muscle, and using the In-doorway Stretch Exercise help to ensure continued freedom from this source of myofascial TrP pain.

1. REFERRED PAIN
(Figs. 42.1 and 42.2)

Edeiken and Wolferth,[19] in 1936, identified the "trigger zone" as a hypersensitive spot in the skeletal musculature of the chest. The "trigger zone" was responsible for referred chest pain that persisted following an acute myocardial infarction.[19,52] Subsequent authors noted that tender spots in the left pectoralis major muscle ("pectoral myalgia")[30] referred pain to the chest in a manner that confusingly simulated the pain of coronary insufficiency in persons with no history or evidence of cardiac disease.[31,34,50,79,96] Other authors recognized the noncardiac nature of this pain, but were unaware of its trigger point (TrP) origin.[13,21,71] Lange[54] emphasized the shoulder and arm components of pain arising from the pectoralis major muscle.

This muscle is likely to develop TrPs in five areas, each with a distinctive pain reference pattern. Pain and tenderness are referred unilaterally.

The TrPs located in the *clavicular section* (Fig. 42.1A) refer pain over the anterior deltoid muscle and locally to the clavicular section of the pectoralis major itself.

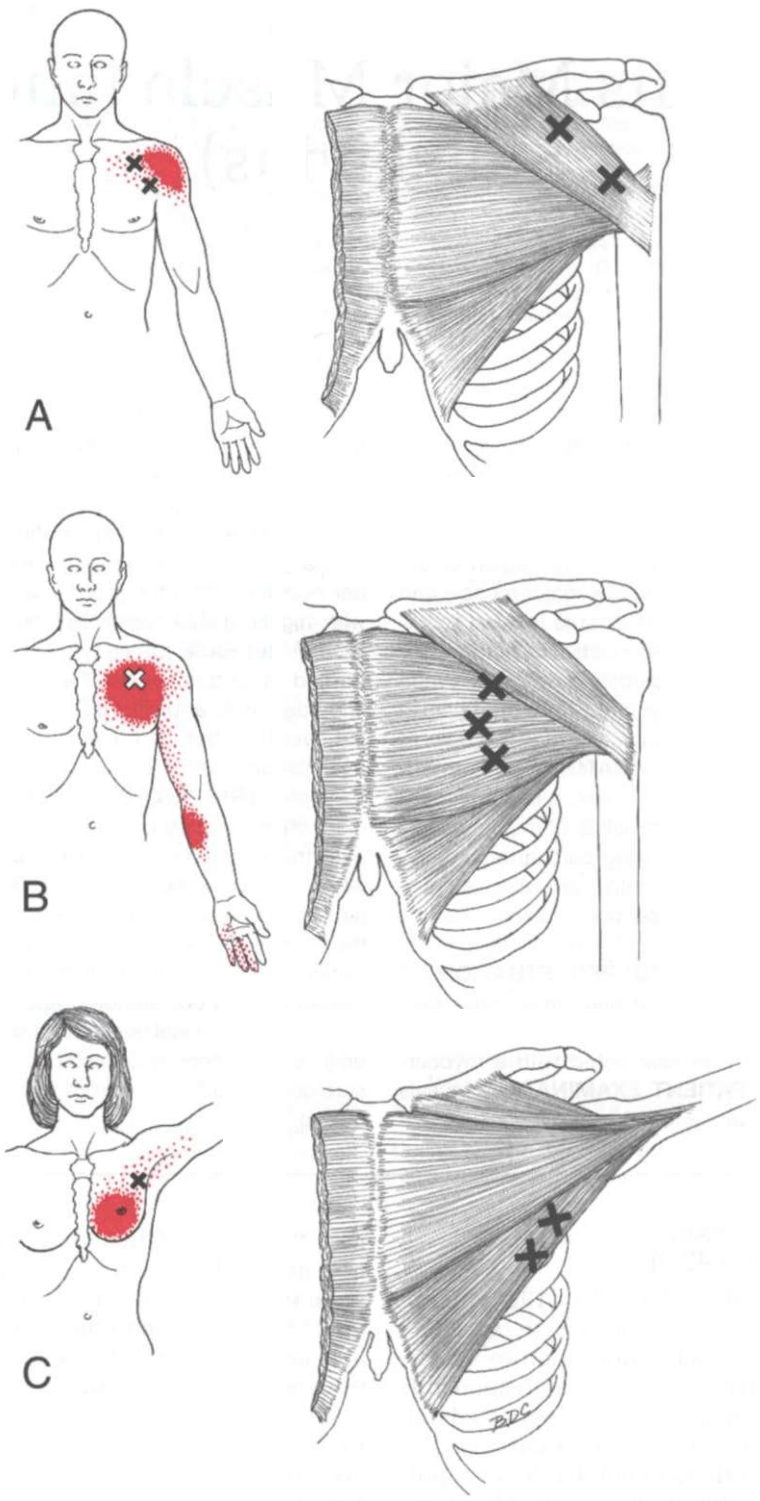


Figure 42.1. Referred pain patterns *(red)* and trigger points (**Xs**) in the left pectoralis major muscle. *Solid red* shows essential areas of referred pain, and *stippled red* shows the spillover pain areas. **A,** the clavicular section. **B,** three central trigger point locations of the intermediate sternal section. **C,** two central trigger point locations in the lateral free margin of the pectoralis major muscle, which includes fibers of the costal and abdominal sections that form the anterior axillary fold.

Active TrPs in the *intermediate sternal section* of the pectoralis major (Fig. 42.1**B**) are likely to refer intense pain to the anterior chest[49,50,63,98] (to the precordium, if on the left side) and down the inner aspect of the arm. The arm pain accents the medial epicondyle. If sufficiently active, these TrPs refer pain also to the volar aspect of the forearm and ulnar side of the hand. The hand pain includes the last two, or two and one half, digits (more than the one and one half digits usually innervated by the sensory fibers of the ulnar nerve).[95] The uppermost of these sternal-section TrPs (Fig. 42.1**B**) lies at the three-way overlap of the clavicular and manubrial sections of the pectoralis major and the underlying pectoralis minor muscle. This TrP location lies in the midfiber region of both sections of the major and of the minor muscle, and TrPs occur frequently in both muscles. The three central TrP locations illustrated in Figure **42.IB** for the sternal section of the muscle demonstrate the principle that central TrPs (CTrPs) may be found anywhere in the midmuscle endplate zone from one side of the muscle to the other.

Active TrPs located in the *medial sternal section* of the pectoralis major refer pain locally and over the sternum without crossing the midline[49,63,92] (Fig. 42.2**A**). At times, when injecting TrPs located over the sternum in the area of a sternalis muscle *(see* Chapter **44),** one may encounter TrPs in a second, deeper layer of muscle, **1.5-2** cm (1/2-3/4 in) beneath the surface. These TrPs are probably located in pectoralis major fibers close to their musculotendinous junctions, occasionally beneath a sternalis muscle.

In the *costal and abdominal section* of the pectoralis major, TrPs develop in two pectoral regions. One of these regions lies along the lateral border of the muscle. These border TrPs (Fig. 42.1**C**) cause breast tenderness with hypersensitivity of the nipple, intolerance to clothing, and often breast pain.[89] Complaints of this distressing syndrome are made by both women and men, but more often by women.

More medially, a TrP associated with somatovisceral cardiac arrhythmias[90] is located on the right side between the fifth and sixth ribs, just below the point where the lower border of the fifth rib crosses a vertical line that lies midway between the margin of the sternum and the nipple (Fig. 42.2**B**). This TrP has been observed only on the right side, except in *situs inversus.* The spot tenderness of this TrP is associated with ectopic cardiac rhythms, but not with any pain complaint. There may be nearby tender points over or between adjacent ribs that are not pertinent to cardiac arrhythmia.

Subclavius
(Fig. 42.3)

The subclavius muscle can develop active TrPs that refer pain into the upper extremity on the same side (Fig. 42.3). The pain travels across the front of the shoulder, and down the front of the arm and along the radial side of the forearm, but skips the elbow and wrist to reappear on the radial half of the hand. In addition, the dorsal and volar aspects of the thumb, the index finger, and the middle finger also may hurt.

2. ANATOMY
(Figs. 42.4 and 42.5)

Anatomy books contradict each other in their descriptions of the arrangement of the lowest fibers of the pectoralis major muscle. They generally agree that the fibers of the entire muscle attach ***medially*** as four separate sections: **(1)** clavicular fibers (Fig. 42.4) to the clavicle, **(2)** sternal fibers to the sternum, **(3)** costal fibers to the cartilages of the second to sixth or seventh ribs, and **(4)** abdominal fibers (Fig. 42.5) to the superficial aponeuroses of the obliquus externus abdominis, and occasionally to the rectus abdominis muscle.[3,10,20,22,39,45,61,63,81] These are the components of the muscle that authors illustrate in very nearly all anatomical atlases.[1,20,22,39,45,62,73,81,85] The abdominal section of the pectoralis major is the portion most likely to be omitted and occasionally fails to develop.[13]

Anatomists, except Eisler[20] who identified three layers, agree that the ***lateral*** termination of the muscle on the humerus comprises two layers, a ventral and a dorsal. All are attached to the crest of the greater tubercle of the humerus (along the lateral lip of the groove for the bicipital tendon)[3,10,22,39,45,62,69,76,81,85]

In **1912,** Eisler[20] described the bulk of the muscle as strips of fibers that overlap

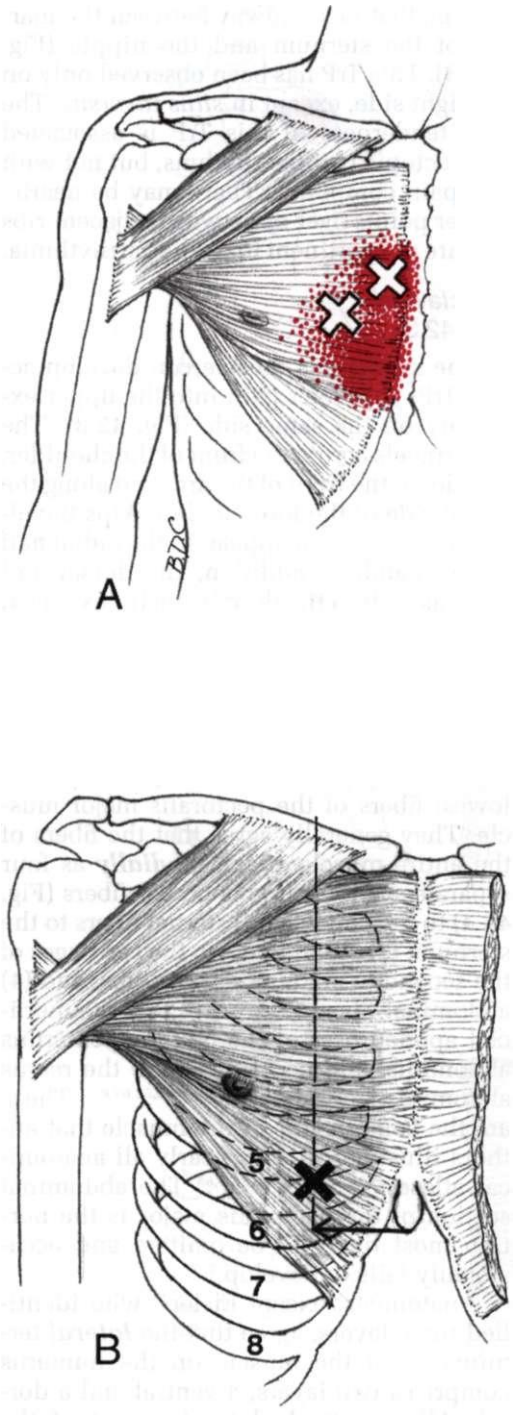


Figure 42.2. Right pectoralis major muscle trigger-point phenomena. **A,** overlapping referred pain patterns (red) of two parasternal attachment trigger points (**Xs**) located in the medial sternal section of the muscle. **B,** location of the "cardiac arrhythmia" trigger point **(X)** below the lower border of the fifth rib in the vertical line that lies midway between the sternal margin and the nipple line. On this line, the sixth rib is found at the level of the tip of the xiphoid process *(arrow).*

each other like the shingles on a roof or the leaves of a fan. Hollinshead[39] clearly described this relationship between the clavicular and sternocostal sections. A few other authors recognized the overlap of these sections.[3,10,39,62,76,81] Many illustrations of the muscle show a variable degree of this overlap,[1,3,39,62,68,73,81,85] while others do not.[1,10,22]

Eisler[20] described the lower sternocostal fibers and the abdominal section as folding upward beneath the rest of the muscle at its lateral end; because of this folding, the lowermost fibers had the most proximal attachment to the humerus. Hollinshead[39] also described this folding process and illustrated it diagrammatically.[39] Some illustrations of the muscle also portray this feature,[1,10,20,22,39,62,68,81,85] but others do not.[1,3,73] Figures of the muscle with and without this fold sometimes appear in the same volume.

Frustrated by these inconsistencies, Ashley[2] dissected 60 adult cadavers and 8 fetuses to establish the facts. He presented clear schematics of his findings. The arrangement of most of the pectoralis major fibers can be seen clearly ONLY from the dorsal (under) side of the muscle, a view not found in anatomy texts. Ashley's drawings[2] were followed closely in the preparation of Figure 42.5, which is a semischematic presentation of the muscle's fiber arrangement. However, his terminology has been modified to clarify the description.

Ashley[2] found that the tendinous pectoralis major attachment ***laterally*** to the humerus has two layers (Fig. 42.5), each of which is made up of laminae. The ventral layer (named by its attachment at the humerus) was described by Eisler,[20] and is composed of six or more overlapping laminae splayed in the manner of playing cards. These six laminae attach ***medially*** to the clavicle, sternum, and ribs. The lower sternal and costal laminae of this ventral (superficial) layer at the humerus attach ***medially*** as underlying, but unfolded, *deep* fibers.

As seen from the usual ventral view, however, these deep lower laminae are hidden by a more superficial lamina of lower sternal, costal, and abdominal fibers that wrap or fold around the caudal end of the deeper laminae to attach on the humerus and to comprise most, if not all, of the dorsal (deep) layer at that location.

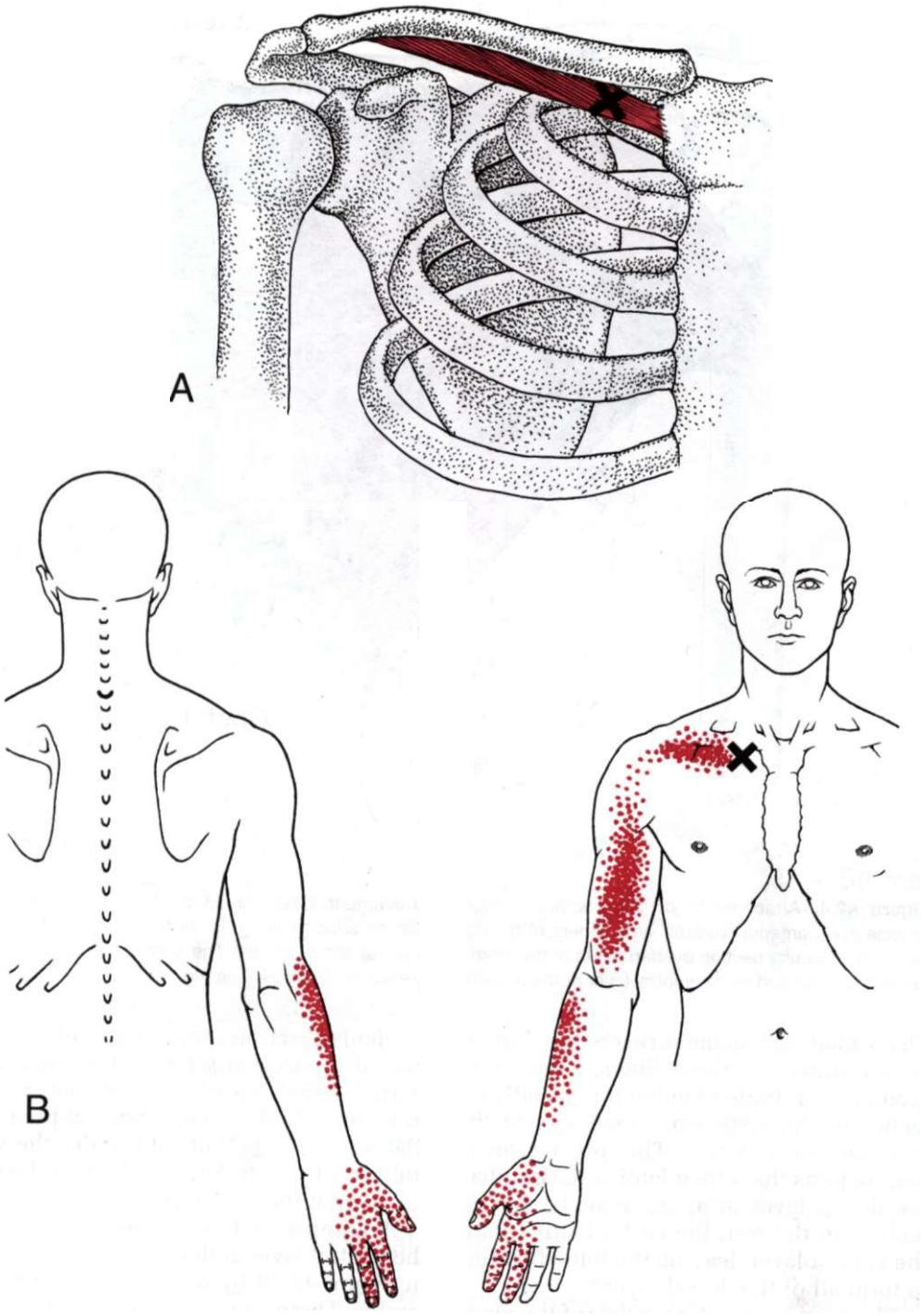


Figure 42.3. Subclavius muscle. **A,** attachments of the muscle *(medium red)* and the location (**X**) of a trigger point in this muscle. **B,** Referred pain pattern *(dark red)* of a subclavius trigger point (**X**).

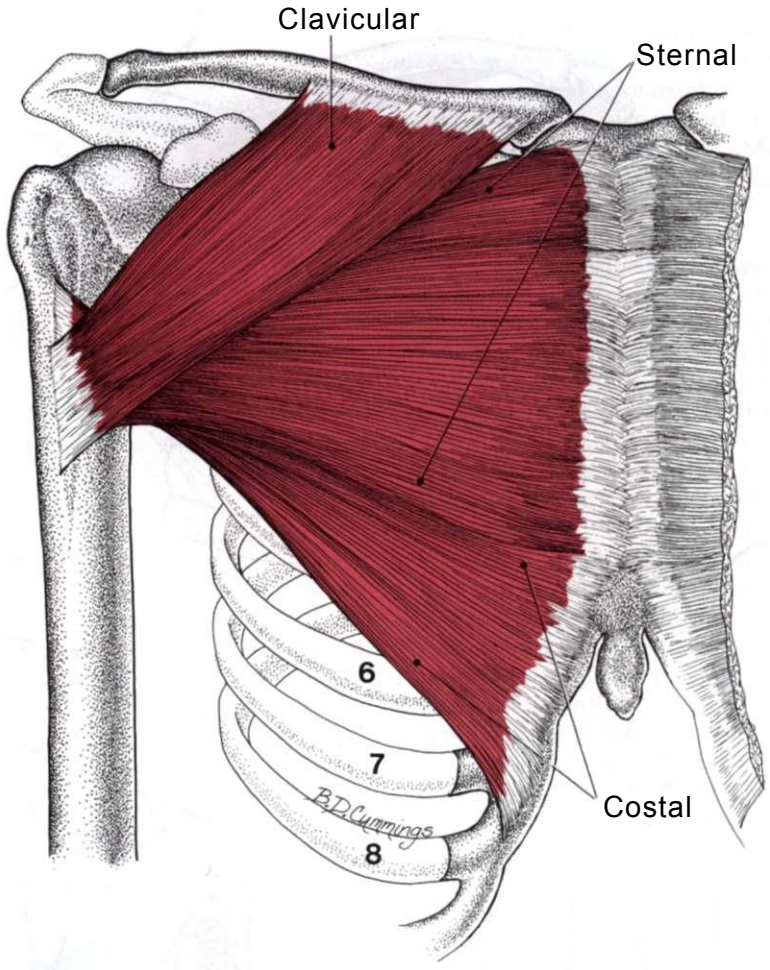


Figure 42.4. Attachments of the pectoralis major muscle (red), anterior (ventral) view. Fibers of the uppermost clavicular section overlap fibers of the sternal section to form part of the ventral layer at the humeral attachment. Costal fibers curl around the lateral border (anterior axillary fold) to form most of the dorsal layer at the humerus. The variable abdominal fibers are not shown here (see Fig. 42.5).

The folded arrangement reverses the order of attachment of these fibers. They wrap around an unfolded lamina that usually attaches to the sixth rib, sometimes to the fifth and seventh ribs. This pivotal costal lamina joins the folded lamina to complete the dorsal layer in approximately 9 of 10 bodies. In the rest, the costal lamina joins the ventral layer, leaving the folded lamina to form all of the dorsal layer.[2]

The semi-schematic version of the usual anterior view of the undisturbed pectoralis major muscle (Fig. 42.5A) clearly shows the first two overlapping laminae of the ventral layer, which are the fibers of the clavicular section and the manubrial portion of the sternal section. The remaining sternal, costal, and abdominal fibers visible in Figure 42.5A are superficial at their medial attachments, but fold under the ventral layer fibers to form the bulk of the dorsal layer at the humerus.

A glimpse of the remaining laminae of the ventral layer at the humerus is revealed in Figure 42.5B by retraction of the folded lamina. These remaining laminae are clearly seen in the reflected dorsal view of the fibers in Figure 42.5C. These ventral layer fibers attach ***medially*** to the sternum and ribs, deep to the more superficial folded lamina.

Knowledge of this arrangement is important in order to interpret accurately the direction of the fibers palpated for TrPs and the direction of contraction when a local twitch response is elicited. Each lamina very likely has its own nerve branch and midfiber endplate zone.

Rarely, all or a portion of the pectoralis major muscle may be congenitally absent; the sternocostal sections are more likely to be absent than the clavicular section.[65] An axillary arch variant of the pectoralis major has been illustrated.[39] This and other variations have been well described.[3,20] The

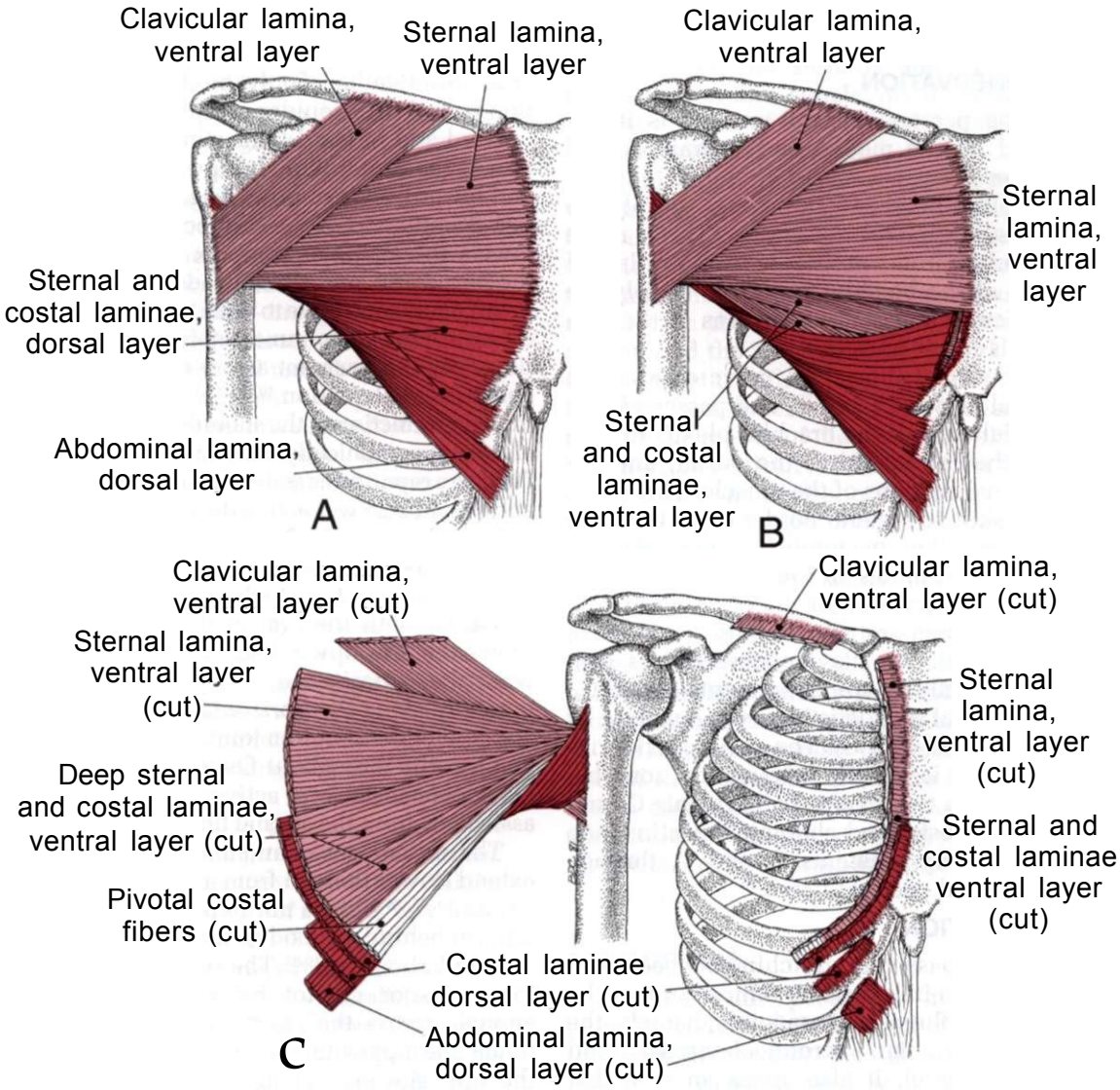


Figure 42.5. Semischematic drawings of the fiber arrangement in the pectoralis major muscle. A, usual ventral view. **B,** ventral view with the superficial dorsal-layer fibers retracted to show the seldom seen deep lamina of the ventral layer *(light red).* **C,** muscle reflected laterally to show the seldom seen dorsal aspect that reveals the playing card arrangement of the deep lamina of the ventral layer. The dorsal layer *(dark red)* swings around the other fibers to attach on the humerus dorsal to them. (Adapted from Ashley GT: The manner of insertion of the pectoralis major muscle in man. *Anat Rec 11*3:301-307, 1952.)

degree of congenital absence can be imaged using computed tomography.[14]

Subclavius Muscle

The subclavius muscle (Fig. 42.3A) lies beneath the clavicle over the first rib and attaches ***medially*** by a short thick tendon to the junction of the first rib with its cartilage. The muscle attaches ***laterally*** in a groove on the under side of the middle third of the clavicle.[11] A number of authors have clearly illustrated its attachments.[11,12,69]

3. INNERVATION

The pectoralis major muscle is innervated by the medial and lateral pectoral nerves.

Spinal nerves C_5 through C_7 supply the *lateral* pectoral nerve.[39] This nerve branches from, or just above, the lateral cord of the brachial plexus to supply the clavicular and sternal sections of the pectoralis major muscle.[10]

The *medial* pectoral nerve arises from spinal nerves C_8 and T_1 and passes *via* the medial cord of the brachial plexus to supply the caudal third, the costal, and abdominal sections of the muscle. This nerve may skirt the lateral border of, but usually pierces, the pectoralis minor muscle, which it supplies en route.[45]

The innervation of the pectoralis major fibers progresses segmentally from above downward. The clavicular section is supplied chiefly by spinal segments C_5 and C_6. The sternal section is innervated mainly by segments C_6 and C_7. The costal section innervation is usually a transition zone between the two nerves by segments C_7 and C_8. The costal and abdominal sections are supplied by segments C_8 and T_8 through the medial pectoral nerve.[20]

4. FUNCTION

When passively stretching the pectoralis major, it is important to remember that the muscle influences three joints, namely, the sternoclavicular, acromioclavicular, and glenohumeral. It also spans an area that functions like a joint to provide the gliding movement of the scapula over the ribs.

When the thorax is fixed, the **pectoralis major** as a whole acts to adduct and medially rotate the humerus. In addition, the upper fibers flex the humerus and the lower fibers can depress the shoulder girdle through their pull on the humerus. On the other hand, when the humerus is fixed or stabilized, the pectoralis major can function to move the sternal and clavicular attachments toward the humerus. In its pull on the thorax, it may assist in forced inhalation. It also can assist in supporting the body weight for crutch-walking and parallel-bar work.[51]

Electrical stimulation of the entire pectoralis major muscle medially rotates the arm.[17] Stimulation of only the clavicular section moves the shoulder complex obliquely upward and forward. This stimulation also moves the arm obliquely upward, forward and inward, so as to press it against the thorax. Stimulation of the sternocostal section lowers the shoulder complex and extends the flexed arm, and strongly adducts it.[17]

All fibers contribute to three movements of the arm at the glenohumeral joint: (1) adduction,[10,45] (2) movement across the chest,[45,76] and (3) medial rotation.[10,39,76] All fibers assist forced protraction of the shoulder.[4] However, electromyographically, only the sternocostal fibers are reported as active during adduction and the muscle was active during medial rotation only against resistance.

The *clavicular section* assists flexion of the glenohumeral joint when the movement is started with the arm at the side,[41,42,76] draws the arm upward across the chest toward the opposite ear,[10,45] moves the arm medially along the horizontal,[72] and medially rotates the shoulder joint.[51] Electromyographically, throughout flexion, chiefly the clavicular fibers were active,[41] with some assistance from the sternal fibers.[41]

The *sternal, costal* and *abdominal* fibers extend (lower the arm from an elevated position),[10,45,76] but do not hyperextend[45] the arm (to behind the body); they depress the arm and shoulder.[10,65] The unassisted pectoralis major cannot bring the arm far enough across the chest for the hand to touch the opposite ear, but only to reach the opposite side of the chest; the unassisted anterior deltoid, however, can complete the former movement.[4,17]

Bilateral, surface-electrode electromyographic (EMG) activity in the clavicular and sternal sections of the pectoralis ma-

jor muscles was reported for the right-handed execution of sport skills in four underhand patterns, three overhand patterns, four sidearm patterns and two kinds of 1-ft jumps.[8] Generally, both right and left muscles were slightly-to-moderately active and were most active when the subject batted a baseball. Generally, the left clavicular section responded most vigorously and showed a prolonged or double burst of activity. The bursts of activity seen in the left pectoralis major muscle appeared to counterbalance rotary movement imparted to the body by acceleration of the right hand; this effect was noteworthy in all but the underhand throwing patterns.[8]

Among 13 professional right-handed golfers,[46] the greatest pectoralis major activity occurred during the acceleration and early follow-through phases of the swing. The left side showed more activity than the right and men showed more activity than women. The power in the shoulder for the drive came first from the latissimus dorsi and then the pectoralis major which showed more activity than any of the other seven muscles tested. This activity provided the powerful arm adduction and internal rotation required.[74]

Fifteen skilled players pitching a baseball with an unstable shoulder were compared to 12 normal players.[27] The painful shoulders showed only 68% of the EMG amplitude of normal players during late cocking and only 40% during follow-through. Whether the inhibition was due to pain or was of some other reflex origin was not determined. Trigger points can powerfully inhibit functional activity of muscles very selectively.

During freestyle swimming,[70] the clavicular section of the pectoralis major in normal subjects was active during the pull-through phase with peaks of activity during early and late pull-through as medial rotation of the arm progressed. During simulated driving,[48] the clavicular section showed more activity bilaterally during left turns than during right turns and that section showd more activity than the sternocostal section.

The **subclavius** muscle assists protraction of the shoulder indirectly by approximating the clavicle and the first rib.[11]

5. FUNCTIONAL UNIT

All sections of the pectoralis major muscle contract together during strong adduction of the arm, assisted by the teres major and minor, the anterior and posterior deltoid, the subscapularis, and the long head of the triceps muscles. For protraction of the shoulder, the serratus anterior, pectoralis minor, and subclavius muscles assist those parts of the pectoralis major below its clavicular section.

Agonist muscles in parallel and in series, which may assist the *clavicular* section of the pectoralis major, include the anterior deltoid, coracobrachialis, subclavius, scalenus anterior and sternocleidomastoid muscles on the same side. The clavicular section and the anterior deltoid work very closely together. They lie side-by-side with adjacent attachments and are separated only by the groove of the cephalic vein.

The more vertically oriented, lower fibers of the *costal* and *abdominal sections* of the pectoralis major depress the shoulder with the help of corresponding fibers of the latissimus dorsi, lower trapezius, and lower serratus anterior. These lower pectoralis major fibers are also assisted by the subclavius and the pectoralis minor muscles. The pectoralis major muscle contracts bilaterally during forceful adduction of both arms together.

The major antagonists to the sternal section of the pectoralis major are the rhomboidei and middle trapezius muscles. For adduction of the arm at the shoulder, antagonists are the supraspinatus and deltoid muscles.

6. SYMPTOMS

Patients with pectoralis major TrPs (which produce shortening that protracts the shoulder girdle) are likely to be as aware of their secondary interscapular back pain as they are of the pain referred by their pectoral TrPs. In fact, the pectoral muscle TrPs may be painlessly latent, but potent as the cause of pain-producing overload of scapular adductors including the middle trapezius and rhomboid muscles.

Trigger point shortening of the clavicular head of the pectoralis major can produce a downward and forward pull on the medial part of the clavicle, putting tension on the clavicular portion of the sternocleidomastoid, activating or perpetuating TrPs in that muscle which, in turn, cause other problems including autonomic phenomena.

In addition to pain in the front of the shoulder and in the subclavicular region (Fig. 42.1**A**), patients with active TrPs in the clavicular section of the pectoralis major muscle may be aware of restricted abduction, particularly horizontal abduction, of the arm at the glenohumeral joint.

Active TrPs in the central part of the pectoralis major refer pain widely over the precordium (if on the left side) and down the ulnar aspect of the arm to the fourth and fifth fingers (Fig. 42.1B), and also may cause a sense of chest constriction that is readily confused with angina pectoris. The patient with TrPs in the intermediate fibers of the left sternal section is likely to complain of intermittent, intense chest pain (Fig. 42.2**A**) that appears in the precordial region with upper limb activity, and if the TrPs are severe, also at rest. Nocturnally this pain can disturb sleep.

Breast pain and diffuse soreness are a feature of TrPs in the free margin of the costal section, laterally (Fig. 42.1**C**). The nipple may be hypersensitive making it difficult to wear a bra or shirt.

Subclavius shortening by TrPs can contribute to symptoms of a vascular thoracic outlet syndrome.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

A posture or activity that activates a TrP, if not corrected or if continued, can also perpetuate it. In addition, many structural and systemic factors *(see* Chapter 4) perpetuate TrPs that have been activated by an acute or chronic overload.

Pectoralis major TrPs are activated and perpetuated by a round-shouldered posture because it produces sustained shortening of the pectoral muscles. This activation is likely to occur during prolonged sitting, when reading and writing, and when standing with a slouched, flat-chested posture. Conversely, TrP shortening in this muscle can induce such posture.

Pectoralis major TrPs may be initiated or re-activated in many ways: by heavy lifting (especially when reaching out in front), by overuse of arm adduction (use of manual hedge clippers), by sustained lifting in a fixed position (use of a power saw), by immobilization of the arm in the adducted position (arm in a sling or cast), by sustained high levels of anxiety, or by exposure of fatigued muscles to cold air (while sitting in the shade in a wet suit after a swim, or when exposed to the draft from an air conditioner).

In acute myocardial infarction, pain is commonly referred from the heart to the midregion of the pectoralis major and minor muscles. The injury to heart muscle initiates a viscerosomatic process that activates TrPs in the pectoral muscles. Following recovery from the acute infarction, these self-perpetuating TrPs tend to persist in the chest wall unless wiped away like dust collected on a shelf.

8. PATIENT EXAMINATION

The patient should be observed initially for a stooped, round-shouldered, head-forward posture and weak interscapular muscles that are typical of patients with shortened pectoral muscles. The pectoralis major is widely recognized as being prone to shortening due to hypertonicity and reflex facilitation.[33,43,57] The taut bands and reflex effects of latent TrPs may be responsible (see Chapter **2,** Section C). Observing the patient from the rear, the examiner may see abducted scapulae.

After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram representing the pain described by the patient. The drawing should be in the style of the pain patterns in this volume using a copy of an appropriate body form found in Figures 3.2-3.4.

The TrPs in the pectoralis major, when it is involved alone, cause minimal restriction of motion at the shoulder, as shown by the Hand-to-shoulder-blade Test *(see* Fig. **22.3).** Pectoralis TrPs do not cause restriction of the Finger-flexion Test *(see* Fig. **20.6)** unlike the situation where

the similar upper limb pain pattern is caused by TrPs in the scalene muscles. The myofascial TrPs of the pectoral muscles do restrict scapular adduction, which can be tested by having the patient place the back of the ipsilateral hand on the hip and move the elbow posteriorly for range of backward movement. Bilateral comparison is the most sensitive indicator of restriction if muscle involvement is unilateral (which it seldom is in the pectorals). Production of interscapular pain is another indicator of restriction.

Weakness can be tested as described and illustrated by Kendall, *et al.*[51] by testing the clavicular and sternal portions for adduction at the glenohumeral joint with the patient supine, the arm held straight up in the air, and the opposite shoulder stabilized against the table. The costal and abdominal sections can be similarly tested by resisting the patient's attempt to adduct the elevated arm obliquely downward toward the contralateral iliac crest.

If this supine patient position is used to test stretch range of motion fan-wise in the various directions of the pectoralis major muscle fibers (moving the arm into horizontal abduction, lateral rotation, and elevation similar to Figure 42.7), a sensitive operator can feel the restriction of the involved taut band and the patient can feel the increased tension, often as local pain, in the involved part of the muscle.

The patient with chest pain due to pectoralis major TrPs is likely to suffer additional referred pain and restriction of movement at the shoulder due to associated myofascial TrPs in functionally related shoulder-girdle muscles and these need to be considered.

The diagnosis of angina pectoris sometimes is made clinically when there is no definite evidence that the chest pain is due to myocardial ischemia. In many such patients, one can demonstrate that the pain is referred from TrPs in the pectoralis major muscle.[21] The patient with the diagnosis of angina pectoris is naturally *fearful* of any activity that produces the pain. This fear inhibits full movement, which accelerates both physical and psychological deterioration and perpetuates the myofascial TrPs.

When a patient complains of breast soreness (referred tenderness), she or he also may describe a feeling of congestion in that breast. When compared with the other side, the breast may be slightly enlarged and feel doughy. These signs of impaired lymph drainage, possibly due to entrapment or reflex inhibition of peristalsis, soon disappear after inactivation of the responsible TrPs in the lateral border of the tense pectoralis major muscle (Fig. 42.1C).

The symptom of sudden acute pain in the muscle during strenuous effort may be due to rupture of the muscle belly. The tear is usually easily recognized by the visible and palpable discontinuity of the muscle belly when compared with the normal side.[65,101]

9. TRIGGER POINT EXAMINATION (Fig. 42.6)

Pectoralis Major

Most of the TrPs found in the clavicular section, and all of the TrPs in the parasternal section of the muscle are identified by flat palpation. The TrPs in the intermediate and lateral parts of the sternal and costal sections are best located by pincer palpation (Fig. 42.6). The muscle is placed on moderate tension by abducting the arm to approximately 90° in order to maximize the spot tenderness found at a *palpable nodule* in a *taut band.* Pressure on the tender spot should produce sensations *recognized* by the patient as recently experienced symptoms. Local twitch responses may be elicited. The lateral part of the pectoralis major muscle is one of the easier muscles in which to identify nodules and taut bands by pincer palpation.

Gerwin, *et al.*[25] established that, of the criteria tested, the most reliable criteria for making the diagnosis of myofascial TrPs were the detection of a taut band, the presence of spot tenderness, the presence of referred pain, and reproduction of the patient's symptomatic pain. For several muscles, agreement on the presence of a local twitch response was lower, but it was high for the latissimus dorsi muscle, and the pectoralis major should be similarly accessible for reliable examination.

To find the "cardiac arrhythmia" TrP (Fig. 42.2B), the tip of the xiphoid process is first located. Then, at this level on the right

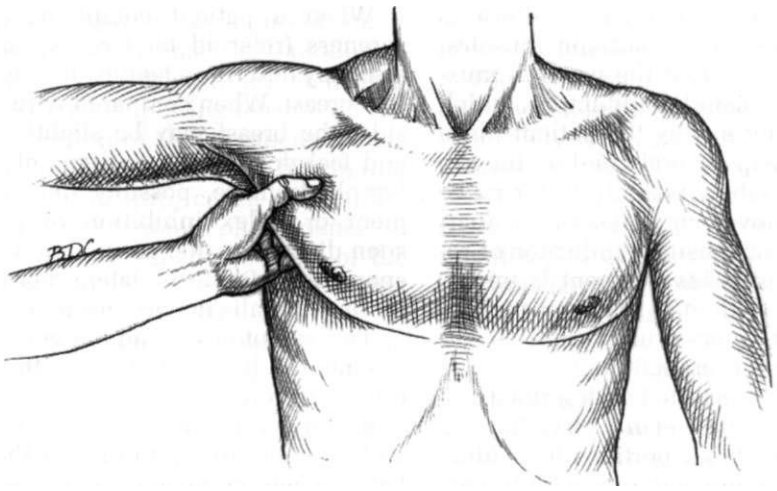


Figure 42.6. Pincer palpation used to examine the sternal division of the pectoralis major for TrPs. Local twitch responses are best elicited when the muscle is placed on a moderate stretch by abducting the arm.

side, in a vertical line midway between the sternal border and the nipple line, the region of the hollow between the fifth and sixth ribs is examined for a tender spot. This TrP is found by pressing upward against the inferior edge of the fifth rib and exploring for spot tenderness. This may be an intercostal rather than pectoralis major TrP.

Subclavius Muscle

Since the subclavius muscle must be palpated through the clavicular division of the pectoralis major, localization of its TrPs is best achieved with the pectoralis major placed on slack. To do this, the relaxed patient's arm is placed in adduction, and medial rotation. The examiner can palpate subclavius central TrPs at the lateral portion of the medial third of the clavicle by rolling the thumb underneath the clavicle, deep into the recess and across the tense fibers. Palpation of the nodule or taut band of a TrP is not reliable through the pectoral muscle (although a different angle). One should distinguish the attachment TrP (ATrP) tenderness just lateral to and below the costoclavicular joint from the CTrP tenderness that is found closer to midclavicle.

10. ENTRAPMENT

No direct nerve entrapments by the pectoralis major have been confirmed.

Shortening of the subclavius muscle because of TrPs will draw the clavicle down toward the subclavian artery and vein as they pass over the first rib. In some patients this pressure can at least contribute to, if not cause, entrapment and the symptoms of a vascular thoracic outlet syndrome.

Lymphatic drainage from the breast usually travels in front of, and around, the pectoralis major muscle to the axillary lymph nodes. A lymph vessel from the cephalad portion of the breast may pierce the pectoralis major muscle and terminate in the subclavicular lymph nodes.[10] Entrapment of this lymph duct by passage between tense fibers of an involved pectoralis major muscle, may cause edema of the breast. In these patients with TrPs, the signs of entrapped lymphatic drainage and breast tenderness are relieved by inactivation of the related pectoralis major TrPs.

11. DIFFERENTIAL DIAGNOSIS

Diagnoses based on tenderness and pain in the chest that need to be considered in addition to TrPs—and which are commonly made mistakenly when TrPs are the cause of the pain—include angina pectoris, tear of the muscle belly,[101] bicipital tendinitis, supraspinatus tendinitis, subacromial bursitis, medial epicondylitis, lateral epicondylitis, C_5-C_6 radiculopathy, C_7

and/or C_8 radiculopathy, intercostal neuritis or radiculopathy, irritation of the bronchi, pleura, or esophagus; hiatal hernia with reflux, distension of the stomach by gas, mediastinal emphysema,[80] gaseous distension of the splenic flexure of the colon,[18] and lung cancer.

Pectoral tension can be associated with lack of mobility in the midthoracic region. A form of self-treatment is described in the following section.

When active TrPs occur in the left pectoralis major muscle, the referred pain is easily confused with that due to coronary insufficiency.[21,53,94] Chest pain that persists long after an acute myocardial infarction is often due to myofascial TrPs.[35,78,79,93]

Sometimes tender points consistent with the fibromyalgia syndrome occur directly over the sternocostal junction of the second rib. The latter diagnosis can also be confused with costochondritis or can be mistaken for an enthesopathy resulting from a pectoralis major TrP located in the specific taut band fibers that attach at that sternocostal junction. Conversely, active TrPs in the subclavius muscle may be mistakenly diagnosed as overlying pectoralis major TrPs.

Some of the less common noncardiac skeletal syndromes that cause pain and tenderness in the chest include the chest wall syndrome,[21] Tietze's syndrome,[44,55,84] costochondritis, the hypersensitive xiphoid syndrome, the precordial catch syndrome,[9,82] the slipping rib syndrome,[38] and the rib-tip syndrome.[66] Each patient should be carefully examined to determine if the symptoms are partially or entirely due to myofascial referred pain and tenderness, especially from TrPs in the pectoralis major muscle. Of the above conditions, each has been reported as sometimes relieved by injection of the tender area with a local anesthetic without reference to examination for TrPs. Relief by injection is characteristic of TrPs.

In a study exploring the origin of chest-wall pain of noncardiac origin in 100 patients, Wise, *et al.*" found that 69 had chest-wall tenderness. Apparently, the patients were not examined specifically for myofascial TrPs and a diagnosis of fibromyalgia was made in **5** patients, two of whom had their pain symptoms reproduced when pressure was applied to the tender point. However, recognition of elicited pain was observed in a minority of patients, most of whom one would expect to have had TrPs. To reproduce the TrP pain for recognition, it is necessary to press precisely on the TrP causing the pain. Without appropriate physical examination for the TrPs, the TrPs could easily have been missed.

A similar study[56] of 62 adults referred for coronary angiography included an examination to identify musculoskeletal sources of the pain. Among the **7** patients (11%) in whom the chest pain was reproduced on physical examination, **5** had normal angiograms and were diagnosed as *nonanginal chest pain.* The muscles of these patients were not examined for palpable TrP characteristics, and that diagnosis was apparently not considered. A third group[13] expressed concern that roughly 20% of the patients evaluated by angiography for chest pain causing major functional impairment had normal coronary arteries. The cause of their pain was enigmatic and had no explanation. The authors were unaware of TrPs.

The patient who presents with a painful or tender breast, often with hypersensitivity of the nipple to light contact, may harbor responsible TrPs in the lateral margin of the pectoralis major muscle[89, 95] (Fig. 42.1C). Cancer may be a serious, but *unexpressed* fear in patients who express enormous relief when they realize that the pain has a benign treatable myofascial cause.

Distinguishing Pain of Cardiac Origin

Other authors have noted that pectoralis major TrPs can simulate the symptoms of angina pectoris[36] and have illustrated similar referred pain patterns for pectoralis major TrPs in the clavicular and costal divisions,[6] and in the sternal division and medial and lateral margins.[7] The intensity, quality and distribution of true cardiac pain can be reproduced in every detail by the pain referred from active TrPs in the anterior chest muscles.[53, 77, 94] Although these patterns strongly mimic cardiac pain, myofascial TrP pain shows a much wider

variability in its response to activity from day-to-day than does the more consistent exercise response of angina pectoris.

A definite diagnosis of active myofascial TrPs based on their characteristic signs and symptoms and a dramatic response to local treatment does NOT exclude cardiac disease. Adding to this diagnostic challenge is the fact that noncardiac pain may induce transient T-wave changes in the electrocardiogram.[28] A disorder of the heart may coexist and must be ruled out by appropriate tests of cardiac function.[86]

Complaints of circumscribed areas of unilateral parasternal pain should arouse suspicion of parasternal TrPs in the pectoralis major muscle (Fig. 42.2A). However, one should be aware that pain clearly of cardiac origin can be abolished temporarily or permanently by the application of a vapocoolant spray to,[79,86,93] or by infiltration of procaine subcutaneously into, the area of referred cardiac pain[32, 61]; these measures also eliminate pain solely of myofascial origin. Hence, relief of pain by a vapocoolant spray or by local injection cannot be used diagnostically to exclude myocardial ischemia as a cause of the pain.[77]

On the other hand, relief of pain by nitrites does not ensure that the pain is due to coronary artery insufficiency, because a placebo sometimes is equally effective in angina pectoris.[29] Furthermore, nitrites dilate the peripheral, as well as the coronary arteries and occasionally have relieved skeletal muscle pain.[16,32,53] Foley and colleagues[23,24] showed that, in the patient with Raynaud's disease or the patient who had an absent radial pulse due to vasospasm, sublingual nitroglycerin promptly restored the pulsation of the radial artery.

The pectoralis minor muscle (see Chapter 43) has a similar referred pain pattern and a close anatomical relationship to the pectoralis major. Active TrPs in the scaleni (see Chapter 20) also refer pain to the pectoral region.[95] Tender spots in the deep paraspinal muscles to the left of the second to the sixth thoracic vertebrae,[100] and in the region of the left upper rectus abdominis muscle, induce chest pain that strongly mimics cardiac disease.[49] Experimentally, Lewis and Kellgren[60] accurately reproduced the pain of effort angina by injecting hypertonic saline to the left of the interspinous ligaments below both the C_7 and T_1 spinous processes.

Somatovisceral Effects. A common example of a somatovisceral response is found in the patient who experiences episodes of supraventricular tachycardia, supraventricular premature contractions, or ventricular premature contractions without other evidence of heart disease. The patient with such an ectopic rhythm should be checked for an active TrP in the right pectoral region between the fifth and sixth ribs at the specific site[90] (Fig. 42.2B). Although this TrP is tender to palpation, it is not a source of spontaneous pain. Inactivation of the TrP promptly restores normal sinus rhythm when the TrP is helping to cause an ectopic supraventricular rhythm and also can eliminate recurrences of the paroxysmal arrhythmia or frequent premature contractions for a long period of time.

A comparable somatovisceral effect is the well known onset of angina pectoris appearing when an anginal patient suddenly breathes cold air through the nose.[26] Another effect is the slowing of the heart rate that occurs when the face is placed in cold water, known as the diving reflex.

The somatic area of referred pain exerts a strong influence on the perceived pain originating in an ischemic myocardium. The pain of angina pectoris was relieved in three patients by infiltrating the painful area subcutaneously with 2% procaine.[97] Even the application of only vapocoolant spray to the area of chest pain referred from a myocardial infarct relieved the pain at once.[90] Chest pain that persisted in 12 patients following a myocardial infarct, or angina pectoris that developed shortly after a myocardial infarct, was relieved by procaine injection or vapocoolant spray of the TrPs in the chest wall muscles.[79]

Another example of somatic modulation of visceral cardiac pain was observed using the intravenous ergonovine test, which induced sufficient myocardial ischemia to cause anginal pain and depression of the S-T segment in the normal resting electrocardiograms of patients subject to effort angina, but not in pain-free controls. This pain and electrocardiographic response to

intravenous ergonovine is quickly reversed by sublingual nitroglycerin, but persists for more than 10 minutes when untreated.

Patients who responded to the ergonovine test in this manner were sprayed with vapocoolant over the somatic areas of anginal pain that developed on effort and after intravenous ergonovine injection.[90] In no case did the vapocoolant delay or modify the electrocardiographic ischemic response. However, 10 of 12 patients whose pain areas were sprayed immediately following injection obtained complete relief of pain, and two patients obtained partial relief. More surprisingly, when the spray was applied to the areas that were known to become painful just *before* the ergonovine injection, 9 of 15 patients experienced no pain at all, although the electrocardiographic effects of coronary ischemia developed as before. The other 6 patients, who had been presprayed, experienced a delayed onset or attenuation of anginal pain after intravenous ergonovine.

Viscerosomatic Effects. An example of a myofascial viscerosomatic interaction begins with coronary artery insufficiency, or other intrathoracic disease, that refers pain from these visceral structures to the anterior chest wall. As a result, satellite TrPs develop in the somatic pectoral muscles. Kennard and Haugen[52] related the presence of palpably tender TrPs in the chest muscles to chest and arm pain, and to the disease process responsible for the pain. They found that 61% of 72 patients with cardiac disease, 48% of 35 patients with other visceral chest disease, and only 20% of 46 patients with pelvic and lower extremity disease, had tender TrPs in the chest muscles. In the patients with chest and arm pain due to cardiac and other unilateral intrathoracic disease, tender TrPs were strongly lateralized to the affected side.

Additional examples of somatovisceral and viscerosomatic effects in relation to the abdominal viscera[83] are presented in Chapter 49, Section 6, where the neurophysiology of referred visceral pain is summarized.

Related Trigger Points

The pectoralis major is one of the four muscles comprising the quadrad that can produce the myofascial pseudothoracic outlet syndrome *(see* Chapter 18, Section B). This muscle and the latissimus dorsi, teres major, and subscapularis muscles individually, and especially in combination, produce referred pain that confusingly mimics a thoracic outlet syndrome. The patient may, however, have a true entrapment or compressive thoracic outlet syndrome with similar symptoms, but referred from scalene TrPs.

Parallel functional unit muscles, the anterior deltoid and coracobrachialis, are synergists that substitute in part for impaired function of the pectoralis major. The anterior deltoid is especially likely to develop satellite TrPs because it also lies within the pain reference zone of the pectoralis major. Before long, the subscapularis and latissimus muscles, which are also part of the synergistic functional unit, may develop active TrPs.

Involvement of the serratus anterior, the rhomboid muscle, and middle trapezius antagonists often follows, especially in the patient with a round-shouldered posture. The infraspinatus, teres minor and posterior deltoid antagonistic muscles also may develop active TrPs, with the end result of a "frozen" shoulder.

12. TRIGGER POINT RELEASE (Fig. 42.7)

Correction of round-shouldered posture and maintenance of good dynamic posture are essential for lasting relief of pectoral trigger points (TrPs) *(see* Chapter 41).

In addition to the spray-and-stretch technique described here, other techniques including trigger point pressure release, postisometric relaxation and contract-relax as described in Chapter 3, Section 12, are also effective for release of *central* TrPs in the pectoralis major muscle. The primary therapeutic approach to *attachment* TrPs is to inactivate the *central* TrPs that are causing them. Some clinicians find indirect techniques[47] to be effective for treating what usually are myofascial attachment TrPs but have not been recognized as such.

For spray and stretch, *all sections* of the pectoralis major are usually more effectively stretched with the patient seated

than supine. The former position permits greater motion of both the scapula and arm. The freedom is important because this muscle must effectively be stretched across three articulations (Section 4). Therefore, traction is applied to the arm as part of the stretch. The object is not only to increase the range of motion at the glenohumeral joint, but also to adduct the scapula over the chest wall to fully elongate the pectoral muscle.

In any of the three stretch positions described below, but particularly when stretching the most caudal fibers, tightness of the subscapularis due to TrPs can limit stretch of the pectoralis major. If the subscapularis is also involved, its spray pattern (see Fig. 26.5C) should be included alternately with the pectoralis pattern to release both muscles together.

For passive stretch of the *clavicular section* (Fig. 42.7A), the arm is laterally rotated and horizontally extended (abducted) slightly below 90° at the shoulder to fully take up the slack in the clavicular fibers. The vapocoolant spray is swept laterally from the clavicle across the muscle and then over the shoulder and upper limb to cover the referred pain pattern before and while taking up any slack that develops.

To stretch the *intermediate sternal fibers,* the arm is placed at approximately 90° of abduction, then laterally rotated and then moved slowly toward the back into extension. Just before and while this position is achieved, parallel sweeps of the vapocoolant are directed laterally and upward across the sternal portion of the muscle, starting at the sternum and continuing over the upper limb to cover all of its referred pain patterns, including the fingers (Figs. 42.1B and 42.7B). TrPs in the anterior deltoid muscle will also restrict this movement, but their spray pattern is already included in that of the pectoralis major muscle.

When inactivating *parasternal* TrPs, the stretch position of Figure 42.7B is used. However, the spray is swept medially following the sternal section of the muscle from its lateral border, over the TrPs and pain reference zone to the midline.

The pectoralis major muscle can be stretched and sprayed with the patient in the supine position if care is taken not to fix the scapula against the table. Figure 42.7C illustrates and describes pectoral release in the supine position for the *lower sternal and costal sections.*

For stretch of the *lowest costal section,* with the patient either seated or supine, the arm is flexed at the shoulder while held in lateral rotation (like the stretch used for the pectoralis minor muscle). When there is no more slack in the muscle, sweeps of spray or icing are directed *downward* and medially from the humerus over the passively stretched fibers, also covering the tender breast. The clinician takes up any slack that develops.

It is always advisable to check the contralateral pectoralis major since both are frequently involved in round-shouldered posture. If there is bilateral involvement, both sides should be sprayed and released.

Three *slow* cycles of *full* active range of motion are followed immediately by application of moist heat. Afterward any residual TrPs (and TrPs in the *subclavius)* usually can be inactivated by **trigger point pressure release** (see Chapter 3, Section 12) or by injection with 0.5% procaine solution, followed again by brief stretch and spray and then moist heat.

Latent TrPs of the antagonistic rhomboid and middle trapezius muscles can be activated by unaccustomed shortening during stretch of the pectoralis major. Also, the tense pectoral muscles overload these posterior muscles causing painful stretch weakness. In either case, these interscapular muscles should be released by vapocooling and non-stretch procedures (such as trigger point pressure release, taut band massage, or indirect techniques) followed by strengthening exercises.

Vapocooling the skin over the pectoralis major muscle in the pattern shown (Fig. 42.7A and B) may relieve the pain of true cardiac ischemia, as well as pain arising from active myofascial TrPs.[87,95] Thus, the cardiac status should be known in every patient who experiences relief of chest pain by these simple measures.

Myofascial TrP tightness of the clavicular portion of the pectoralis major can exert forward and downward traction on the clavicle, increasing tension on the clavicu-

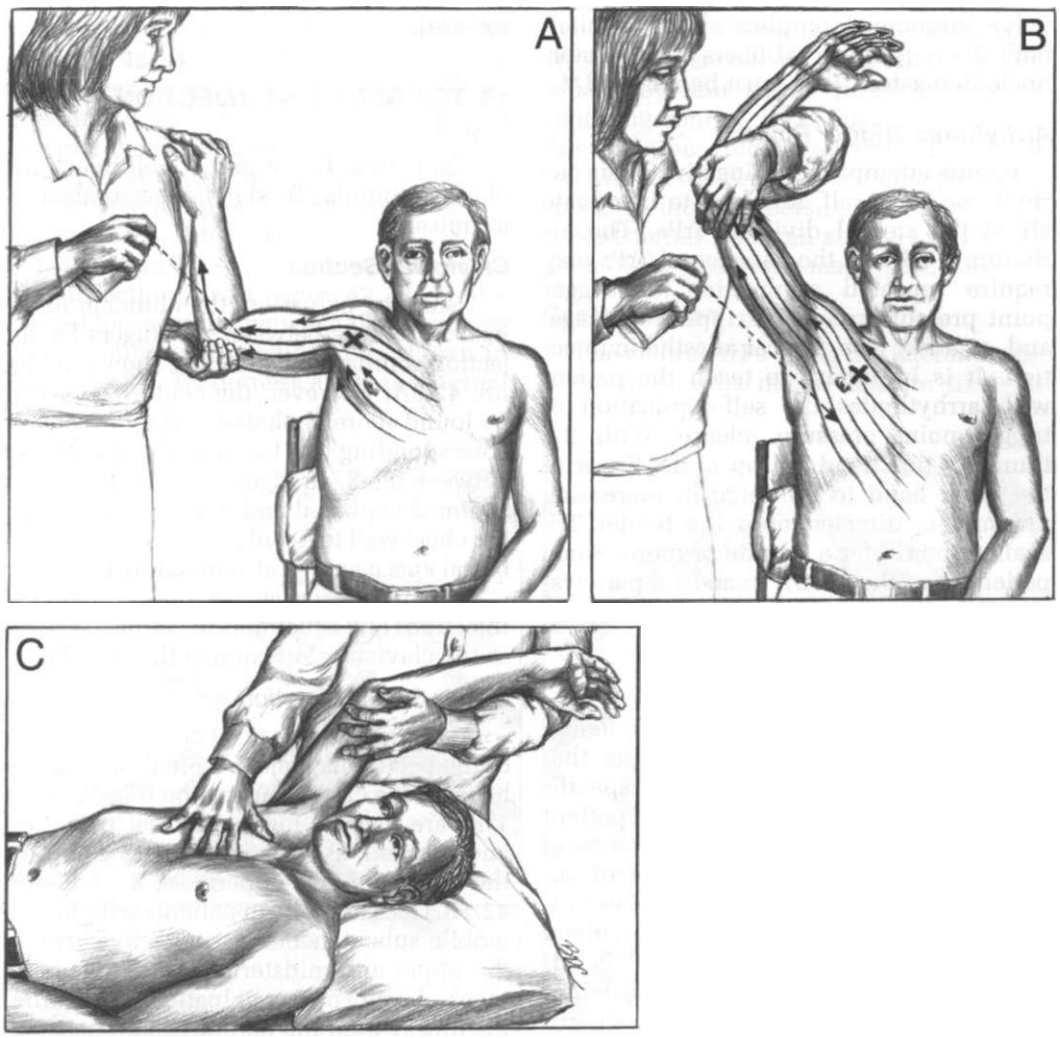


Figure 42.7. Positions for muscle release and patterns for application of intermittent cold *(arrows)* for trigger points (**Xs**) in the pectoralis major muscle. **A,** vapocoolant and stretch for the clavicular section, patient seated, relaxed and comfortably leaning against the back support. As the patient slowly exhales, the operator applies vapocoolant (or ice) in the pattern indicated while gently horizontally abducting the arm. Application of cold is resumed after the patient has slowly taken another deep breath. This cycle continues rhythmically until maximum available range is achieved. **B,** vapocoolant and stretch for the sternal and costal sections of the muscle may be applied with a sequence and rhythm similar to that in A, above, except that the arm is placed in flexion above the horizontal. **C**, Using another method, intermittent cold can be applied in the same pattern as in **B** to a supine patient *prior* to muscle lengthening. Then the trigger points of the sternal division may be inactivated and their tension relieved by this pain-free and effective manual release technique. The operator uses one hand to stabilize the sternum and lower part of the muscle while slowly exerting countertraction with the other hand at the distal humerus, slowly releasing the tissues to the point of resistance (barrier). Another pectoral release is shown in Figure **12.8A.**

lar head of the sternocleidomastoid muscle, which can induce TrPs in it and involve autonomic complications. For relief, both the tense pectoral fibers and the sternocleidomastoid TrPs must be released.[64]

Arrhythmia Trigger Points

Before attempting to inactivate the arrhythmia TrP itself, it is best to inactivate all of the sternal division TrPs. The arrhythmia TrP and the parasternal TrPs may require repeated application of trigger point pressure release, stripping massage, and, as a last resort, local anesthetic injection. It is important to teach the patient with arrhythmias the self-application of trigger point pressure release. With the thumb of one hand on top of the finger of the other hand to reinforce it, increasing pressure is directed onto the tender TrP against the rib for a minute or more. Some patients can thus learn to abort a paroxysmal ectopic tachycardia as soon as the attack is recognized.

Other Release Techniques

For many patients, *Postisometric Relaxation* (PIR) is a valuable technique that Lewit illustrated and described specifically for this muscle with the patient supine.[58] He identified the importance of the patient locating the direction of abduction of the arm that places uncomfortable tension on specifically the involved (taut) muscle fibers. When PIR is attempted with the patient sitting, it is not as effective in some patients who have difficulty relaxing during exhalation with the arm raised overhead. The patient must be warned not to contract with too much force.[64]

For patients who have pectoralis major TrPs associated with lack of mobility in the midthoracic spine, a self-treatment similar to that described (for the lower thoracic spine) by Lewit[59] can be beneficial. The patient sits facing a wall, knees touching the wall, with hands behind the head, elbows back, and bending forward at the hips so that the forehead touches the wall. The patient is then instructed to breathe in deeply, bringing the air in to a point in the midthoracic region (which produces a slight kyphosis and pectoral contraction) and to breathe out slowly, relaxing completely (which encourages mobilization of the midthoracic area and also pectoral release).

13. TRIGGER POINT INJECTION (Fig. 42.8)

The patient lies supine for all injections of trigger points (TrPs) in the pectoralis major muscle.

Clavicular Section

Using flat palpation, the clinician localizes these TrPs between the fingers for injection, similar to the manner shown in Figure 42.8A. However, the central TrPs will be found more cephalad and more lateral, corresponding to a location about half way between the Xs in Figure 42.1A. The needle is aimed cephalad and nearly tangent with the chest wall to avoid penetrating an intercostal space as was also noted by Rinzler.[78]

Rachlin[75] describes pectoralis major TrP injections and illustrates injection of a CTrP in the clavicular division of the muscle.

Upper Sternal Section

About half of this most cephalad portion of the pectoralis major's sternal section lies beneath the clavicular section (Fig 42.4). Its TrPs are usually located by flat palpation and injected, as shown in Figure 42.8A, in the region of the uppermost **X** of Figure 42.1B. Occasionally, in patients with highly mobile subcutaneous tissue, active TrPs in the upper and midsternal sections may be reached using pincer palpation by inserting the fingers (and the patient's skin) between the underside of the pectoralis major and the chest wall. Pincer palpation permits more accurate positioning of the needle. Fingernails must be short to permit a pincer grasp, as in Figure 42.8C, but with more of the muscle between the fingers. To do this, the clinician must slacken the muscle by bringing the arm close to the patient's side. Finger pressure adjusts muscle tension.

Which layer of muscle contains the TrPs may be inferred by the depth of the needle on contact with a TrP and the fiber direction of the local twitch response.

Mid- and Lower-sternal Sections

Figure 42.8A illustrates a technique for injecting the central TrPs that frequently occur in the midfiber region of the midster-

nal and lower-sternal sections of the pectoralis major. These locations correspond to the locations of the middle and lower **Xs** of Figure 42.1**B.** The TrPs in these regions are injected with a 37mm (1.5 in) needle by directing it upward toward the coracoid process, nearly parallel to the thoracic cage. The Hong technique for holding the syringe *(see* Chapter 3, Section 13) is recommended in this location.

Parasternal TrPs in the medial part of the sternal section (**Xs** in Figure 42.2A) are localized for injection between the fingers by flat palpation and positioned as shown in Figure 42.8B. Tenderness along the sternal border in the region of the musculotendinous attachments of the pectoralis major is likely to be enthesopathy of attachment TrPs that are secondary to sustained tension caused by taut bands running to corresponding central TrPs. Sustained relief of this tenderness requires inactivation of central TrPs in the central portion of the corresponding muscle fibers.

Midsternal TrPs can strongly induce all parts of the pectoralis major pain pattern.

Costal Section

The central TrPs along the lateral border of the pectoralis major that are likely to

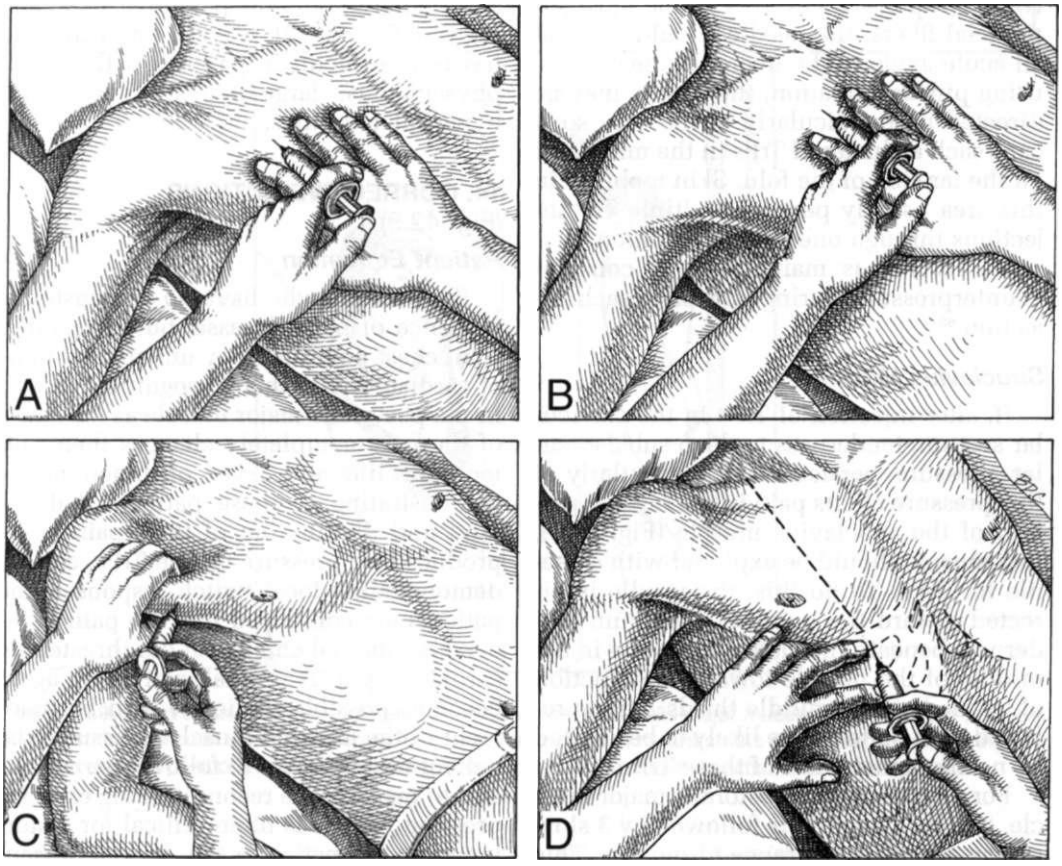


Figure 42.8. Injection of trigger points in the pectoralis major muscle. **A,** the trigger points are localized and fixed by flat palpation for injection in the central portion of the midsternal section of the muscle. If the needle is not directed nearly tangent to the chest wall, beware of entering the pleura. **B,** similar technique for injection of parasternal attachment trigger points in the midsternal section of the muscle. **C,** pincer grasp illustrated for injection of fibers in the lateral margin of the lower costal and abdominal sections of the muscle. **D,** flat palpation for injection of the cardiac arrhythmia trigger point by directing the needle upward toward the lower margin of the fifth rib, into the spot of maximal tenderness.

cause breast pain and nipple hypersensitivity (Fig. 42.1C) usually occur in the fibers of the costal section of the muscle.

To inject central TrPs in the lateral margin of the costal and abdominal sections of the pectoralis major, palpate the midfiber portion of the muscle for tender nodules in taut bands where the lower **X** is located in Figure 42.1C. The muscle is grasped between the thumb and fingers of one hand, as in Figure 42.8C so that the TrPs can be precisely injected by palpating and localizing the TrP between the fingers. For this TrP, appropriate muscle tension is usually attained by abducting the arm to approximately 90°. The TrPs in this location can usually be verified by their vigorous local twitch responses. For TrPs in the most superficial fibers, the needle should enter at an acute angle to the fibers; for deep TrPs, using pincer palpation, the needle may be directed perpendicularly to the skin so it can reach a cluster of TrPs in the middle or on the far side of the fold. Skin mobility in this area usually permits multiple TrP injections through one skin penetration.

Hemostasis is maintained by constant counterpressure during and after each injection.[89]

Subclavius Muscle

If, after injection of TrPs in the clavicular section, tenderness to deep subclavicular pressure persists, and particularly if this pressure elicits pain in the referral pattern of the subclavius muscle (Fig. 42.3), that muscle should be explored with a needle for TrPs. To do this, the needle is directed toward the point of maximum tenderness beneath the clavicle, usually in the middle of the muscle toward the junction of its medial and middle thirds. Strong referred pain patterns are likely to be elicited by needle penetration of these TrPs.

For all parts of the pectoralis major muscle, the TrP injection is followed by 3 slow cycles of active full range of motion. This activity "re-educates" the muscle in its normal range of motion.[91] If desired, application of moist heat can be used also. Any residual TrPs may be inactivated by trigger point pressure release and/or by stretch and spray. Both procedures seem to be more effective during, rather than before or after, the duration of the local procaine analgesia (about 15 minutes).

Arrhythmia Trigger Point

After locating the precise spot tenderness of the arrhythmia TrP by flat palpation, the needle is directed cephalad toward the fifth rib (Fig. 42.8D). The needle is aimed nearly tangential to the skin, since the TrP lies no deeper than the anterior surface of the lower border of the rib. This TrP is located close to the depth of the external intercostal muscles. During and after treatment, the patient breathes in a manner that keeps the chest diameter small, using normal, coordinated respiration and *not* the surprisingly common paradoxical breathing (see Fig. 20.15). Resolution of this TrP has been difficult in patients with an emphysematous, large-diameter chest with hyperinflated lungs.

14. CORRECTIVE ACTIONS (Fig. 42.9)

Patient Education

For patients who have *no* demonstrable evidence of heart disease, but who suffer from chest pain that they understood to be of cardiac origin, their recognition of TrPs in the pectoralis major muscle as the cause of the pain completely changes their outlook on life and level of function. By demonstrating to these patients that the kind and distribution of their pain is reproduced by pressure on the TrPs, and by demonstrating local twitch responses, the patients are convinced that the pain is indeed myofascial and not of life-threatening cardiac origin. A normal, active life again becomes possible. Relief of pain by treatment of the afflicted muscles reassures the patient that it is safe to follow instructions and to perform the reconditioning exercise program, which is often critical for restoring normal function of the skeletal musculature and the quality of life.

When coronary artery disease and pectoralis major TrPs coexist, relief of the TrP-induced pain is important for more than comfort. Pain itself may reflexly diminish the caliber of the coronary arteries and

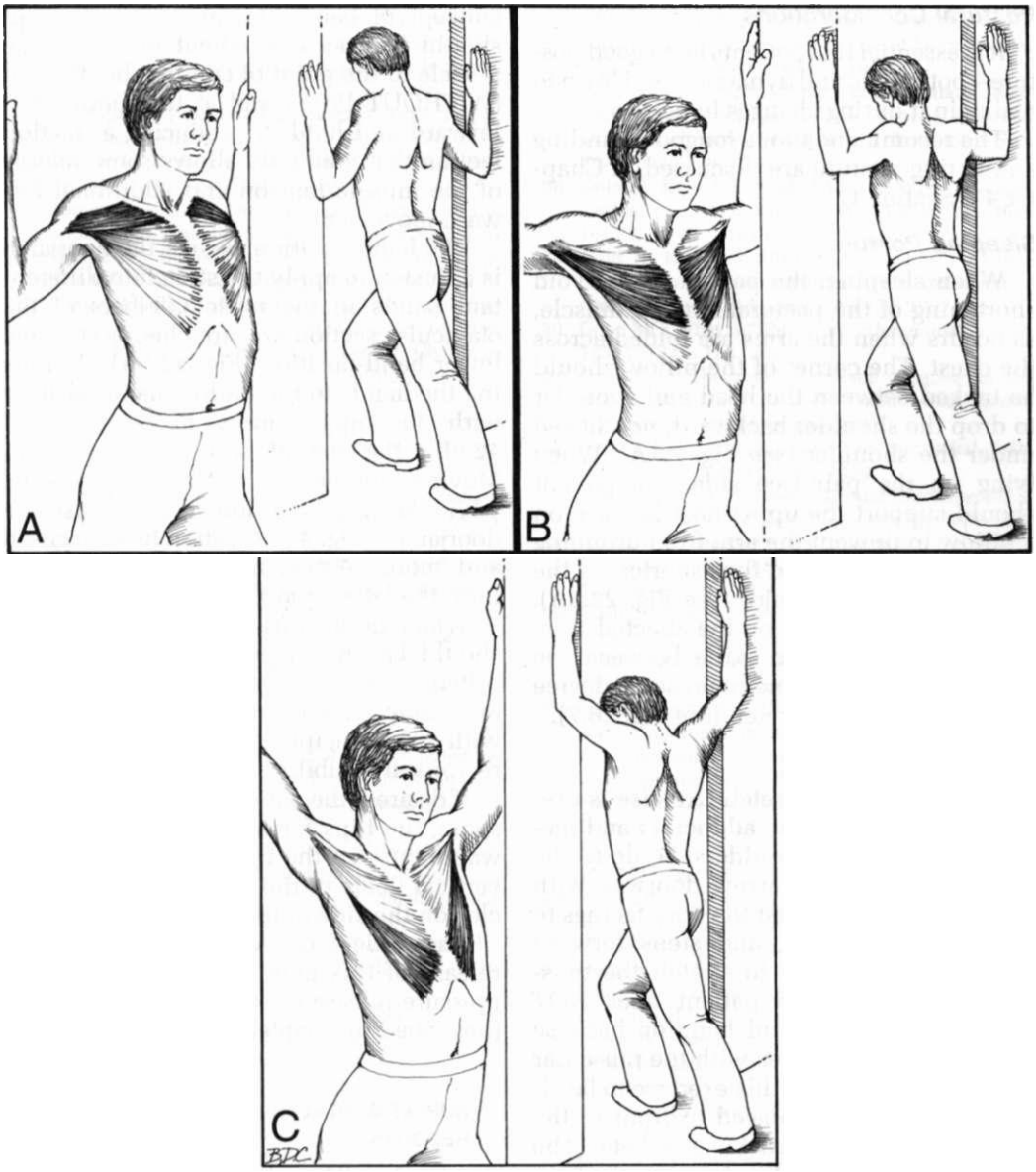


Figure 42.9. Effect of the In-doorway Stretch Exercise on the pectoralis major muscle. **A,** lower hand-position to stretch the clavicular section bilaterally. **B,** middle hand-position to stretch the sternal section bilaterally. **C,** upper hand-position to stretch the fibers of the costal and abdominal sections. See text for details.

thereby even further increase myocardial ischemia.[28,61,67]

Patients with large heavy breasts not only suffer from compression of the tissues across the shoulders by tight bra straps, but they commonly have bras that exert constricting pressure around the chest that makes deep indentations in the skin. This can aggravate and perpetuate pectoralis major TrPs. In this case, the tension around the chest must be eased either by adding a bra extender between the hooks or by releasing some of the elasticity built into the bra by using a hot iron.

Postural Considerations

It is essential that patients learn good posture—both static and dynamic—and learn to maintain it during changes in position.

The recommendations for good standing and sitting posture are discussed in Chapter **41,** Section C.

Sleeping Posture

When sleeping, the patient must avoid shortening of the pectoralis major muscle, as occurs when the arms are folded across the chest. The corner of the pillow should be tucked between the head and shoulder to drop the shoulder backward, nof tucked under the shoulder (*see* Fig. *7.7A).* When lying on the pain-free side, the patient should support the uppermost forearm on a pillow to prevent the arm from dropping forward to the bed and thus shortening the affected pectoralis major (*see* Fig. 22.6**A).** When the patient lies on the affected side, the pillow fits in the axilla between the arm and the chest to maintain some degree of pectoralis major stretch (*see* Fig. 26.7).

Stretch Exercises

The In-doorway Stretch Exercise is useful to stretch all of the adductors and medial rotators at the shoulders. To do it, the patient stands in a narrow doorway with the forearms flat against the door facings to anchor the forearms, and steps forward through the doorway to stretch the muscles (Fig. 42.9). The patient does *NOT* grasp the doorjamb and hang on because that seriously interferes with the muscular relaxation needed for this exercise to be effective. One foot is placed in front of the other, and the forward knee is bent. The patient holds the head erect looking straight ahead, neither craning the neck forward, nor looking down at the floor. As the forward knee bends and the patient shifts the body through the doorway, a slow, gentle, passive stretch is exerted bilaterally on the pectoralis major muscle and on its synergistic muscles. The stretch is held briefly, for only a few seconds. The patient pauses, relaxes, and takes a few slow abdominal breaths between each cycle to enhance relaxation.

For full effectiveness of this stretch technique, the patient needs to learn the concept of barrier release. The clinician should instruct the patient to stretch the muscle to the point of comfortable tension (WITHOUT PAIN) and at that point shift forward and hold it, producing a traction feeling that gradually allows some release of the muscle tension and additional forward movement.

The hand position against the doorjamb is adjusted to apply the stretch to different taut bands in the muscle. Fibers of the clavicular section are stretched best in the lower hand-position (Fig. **42.9A).** By raising the hands to the middle hand-position with the upper arms horizontal (Fig. 42.9**B),** the sternal section is stretched. Moving the hands as high as possible, while keeping the forearms against the doorjambs (Fig. 42.9**C),** stretches the costal and more vertical abdominal fibers that form the lateral margin of the muscle.

When doing this exercise, the patient should be encouraged to distinguish the different feelings of stretch for each section of muscle. This exercise can be combined with the principles of contract-relax and reciprocal inhibition to good advantage.

If desired, the patient may be told also to swing the hips forward through the doorway to stretch the iliopsoas and the more vertical fibers of the latissimus dorsi muscles on the side of the rear leg.

The patient can be instructed in self-release of this muscle using trigger point pressure release or stripping-type massage (described in Chapter **3,** Section **12).**

SUPPLEMENTAL REFERENCES, CASE REPORTS

Dr. Travell has presented case reports of patients with myofascial pain and myocardial infarction or effort angina,[79, 86] with pseudoangina,[94] and with breast pain and soreness due to TrPs in the lateral border of the pectoralis major.[89]

REFERENCES

1. **Agur AM:** ***Grant's Atlas of Anatomy.*** **Ed. 9. Williams & Wilkins, Baltimore, 1991:3, 81, 83 (Figs. 1.2, 2.5, 2.7).**
2. **Ashley GT: The manner of insertion of the pectoralis major muscle in man.** ***Anat Rec 113:301-*** **307, 1952.**

3. Bardeen CR: The musculature. Sect. 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (pp. 405, 406).
4. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 266, 267).
5. Blumer I: Chest pain and intercostal spasm [Letter]. *Hasp Pract 24(5A):13,* 1989.
6. Bonica JJ, Sola AE: Other painful disorders of the upper limb. Chapter 52. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, 1990, pp. 947-958.
7. Bonica JJ, Sola AE: Chest pain caused by other disorders. Chapter 58. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990, pp. 1114-1145.
8. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sports Skills.* Charles C Thomas, Springfield, Ill., 1967.
9. Calabro JJ, Jeghers H, Miller KA, *et al:* Classification of anterior chest wall syndromes. *fAMA* 243.1420-1421, 1980.
10. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 518, 519,890,891).
11. *Ibid.* (pp. 520, 521, Fig. 6-45).
12. Clemente CD: *Anatomy.* Ed. 3.Urban & Schwarzenberg, Baltimore, 1987 (Figs. 151, 233).
13. DeMaria AN, Lee G, Amsterdam EA, *et al.:* The anginal syndrome with normal coronary arteries. *JAMA* 244:826-828, 1980.
14. Demos TC, Johnson C, Love L, *et al:* Computed tomography of partial unilateral agenesis of the pectoralis muscles. *J Comput Assist Tomog 9(3):558-*559, 1985.
15. Diffrient N, Tilley AR, Bardagjy JC: *Humanscale 1/2/3.* The MIT Press, Cambridge, MA, 1974.
16. Dixon RH: Cure or relief of cases misdiagnosed "angina of effort." *Br Med J 2:891,* 1938.
17. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 71-74).
18. Dworken HJ, Fructuoso JB, Machella TE: Supradiaphragmatic reference of pain from the colon. *Gastroenterology* 22:222-228, 1952.
19. Edeiken J, Wolferth CC: Persistent pain in the shoulder region following myocardial infarction. *Am J Med Sci 191:*201-210, 1936.
20. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (pp. 456-464).
21. Epstein SE, Gerber LH, Borer JS: Chest wall syndrome, a common cause of unexplained cardiac pain. *JAMA 241:2793-2797,* 1979.
22. Ferner H, Staubesand J: *Sobotta Atlas of Human Anatomy.* Ed. 10, Vol. 2, *Thorax, Abdomen, Pelvis, Lower Extremities, Skin.* Urban & Schwarzenberg, Baltimore, 1983 (pp. 3, 4).
23. Foley WT, McDevitt E, Tulloch JA, *et al.:* Studies of vasopasm: 1, The use of glycerol trinitrite as a diagnostic test of peripheral pulses. *Circulation* 7:847-854, 1953.
24. Foley WT, Wright IS: *Color Atlas and Management of Vascular Disease.* Appleton- Century-Crofts, New York, 1959 (p. 86).
25. Gerwin RD, Shannon S, Hong CZ, *et al.:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
26. Gilbert NC: Influence of extrinsic factors on the coronary flow and clinical course of heart disease. *Bull NY Acad Med 18:83-92,* 1942.
27. Glousman R, Jobe F, Tibone J, *et al.:* Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg 70A(2):220-226,* 1988.
28. Gold H, Kwit NT, Modell W: The effect of extracardiac pain on the heart. *Proc A Res Nerv Ment Dis* 23:345-357, 1943.
29. Gold H, Kwit NT, Otto H: The xanthines (theobromine and aminophylline) in the treatment of cardiac pain. *JAMA* 108:2173-2179, 1937.
30. Good MC: What is "fibrositis?" *Rheumatism 5:117-*123, 1949 (p. 121,. Fig. 7).
31. Good MC: The role of skeletal muscles in the pathogenesis of diseases. *Acta Med Scand* 338:285-292, 1950 (pp. 286, 287).
32. Gorrell RL: Local anesthetic in precordial pain. *Clin Med Surg* 46:441-442, 1939.
33. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 146,147).
34. Gustein M: Diagnosis and treatment of muscular rheumatism. *Br J Phys Med* 3:302-321, 1938 (p. 309, Case IX; p. 311, Case 52).
35. Gustein-Good M: Idiopathic myalgia simulating visceral and other diseases. *Lancet* 2:326-328,1940.
36. Harman JB, Young RH: Muscle lesions simulating visceral disease. *Lancet 238(1):1111-* 1113, 1940.
37. Harms-Ringdahl K, Ekholm J: Intensity and character of pain and muscular activity levels elicited by maintained extreme flexion position of the lower-cervical-upper-thoracic spine. *Scand J Rehabil Med 18(3):117-126,* 1986.
38. Heinz GJ, Zavala DC: Slipping rib syndrome; diagnosis using the "hooking maneuver." *JAMA* 237:794-795, 1977.
39. Hollinshead WH: *Anatomy for Surgeons.* Ed 3. Harper & Row, Hagerstown, 1982 (pp. 279-281, Figs. 4-18, 4-19).
40. Hooten EA: *A Survey in Seating.* Heywood-Wakefield Co., Gardner, Mass., 1945. Reprinted by Greenwood Press, Westport, Conn., 1970.
41. Inman VT, Saunders JB, Abbott LC: Observations of the function on the shoulder joint. *J Bone Joint Surg* 26:1-30, 1944.
42. Ito N: Electromyographic study of shoulder joint. *J Jpn Orthop Assoc* 54:1529-1540, 1980.
43. Janda V: Evaluation of muscular imbalance. Chapter 6. In: *Rehabilitation of the Spine: A Practitioner's Guide.* Edited by Liebenson C. Williams & Wilkins, Baltimore, 1996 (pp. 97-112).
44. Jelenko C III: Tietze's syndrome at the xiphisternal joint. *South Med J* 67:818-819, 1974.
45. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Rack.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 78, 79).
46. Jobe FW, Perry J, Pink M: Electromyographic shoulder activity in men and women professional golfers. *Am J Sports Med 17(6):782-787,* 1989.
47. Jones LH: *Strain and Counterstrain.* American Academy of Osteopathy, Colorado Springs, 1981(pp. 56-59).
48. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving. IV. The pectoralis major, serratus anterior and latissimus dorsi muscles. *Ergonomics 18(6):*643- 649, 1975.

49. Kelly M: The treatment of fibrositis and allied disorders by local anaesthesia. *Med J Aust* 3:294-298, 1941. (p. 296).
50. Kelly M: Pain in the Chest: Observations on the use of local anaesthesia in its investigation and treatment. *Med J Aust 1:4-7,* 1944. (pp. 5, 6; Cases V, VII, IX).
51. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 276, 277).
52. Kennard MA, Haugen FP: The relation of subcutaneous focal sensitivity to referred pain of cardiac origin. *J Am Soc Anesthesiologists* 36:297-311, 1955.
53. Landmann HR: "Trigger areas" as cause of persistent chest and shoulder pain in myocardial infarction or angina pectoris. *J Kans Med Soc* 50:69-71, 1949.
54. Lange M: *Die Muskelhaerten (Myogelosen); Ihre Entstehung und Heilung.* J.G. Lehmanns, Munchen, 1931 (pp. 118-135, Fig. 40A, Examples 14, 20, 21, 22).
55. Levey GS, Calabro JJ: Tietze's syndrome: report of two cases and review of the literature. *Arthritis Rheum* 5:261-269, 1962.
56. Levine PR, Mascette AM: Musculoskeletal chest pain in patients with "angina": a prospective study. *South Med J* 82(5):580-585, 1989.
57. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (p. 24).
58. *Ibid.* (p. 198).
59. *Ibid.* (p. 165).
60. Lewis T, Kellgran JH: Observations relating to referred pain, viscero-motor reflexes and other associated phenomena. *Clin Sci* 4:47-71, 1939 (p. 48).
61. Lindgren I: Cutaneous precordial anaesthesia in angina pectoris and coronary occlusion (an experimental study). *NordMed Cardiologia* 33:207-218, 1946.
62. Lockhart RD, Hamilton GF, Fyfe FW: *Anatomy of the Human Body.* Ed. 2. J.B. Lippincott, Philadelphia, 1969 (pp. 200-203, Fig. 322).
63. Long C II: Myofascial pain syndromes, part III-some syndromes of the trunk and thigh. *Henry Ford Hosp Med Bull* 4:102-106, 1956.
64. Maloney M: Personal communication, 1995.
65. Marmor L, Bechtol CO, Hall CB: Pectoralis major muscle: function of sternal portion and mechanism of rupture of normal muscle: case reports. *J Bone Joint Surg* 43A.-81-87, 1961.
66. McBeath AA, Keene JS: The rib-tip syndrome. *J Bone Joint Surg* 57A.795-797, 1975.
67. McEachern CG, Manning GW, Hall GE: Sudden occlusion of coronary arteries following removal of cardiosensory pathways. *Arch Intern Med* 65:661-670, 1940.
68. McMinn RM, Hutchings RT, Pegington J, *et al: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 116).
69. *Ibid.* (p. 117).
70. Nuber GW, Jobe FW, Perry J, *et al.:* Fine wire electromyography analysis of muscles of the shoulder during swimming. *Am J Sports Med 14(1)17-11,*1986.
71. Pasternak RC, Thibault GE, Savoia M, *et al.:* Chest pain with angiographically insignificant coronary arterial obstruction. *Am J Med* 66:813-817, 1980.
72. Pearl ML, Perry J, Torburn L, *et al:* An electromyographic analysis of the shoulder during cones and planes of arm motion. *Clin Orthop* 284:116-127, 1992.
73. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (p. 47, Fig. 38).
74. Pink M, Jobe FW, Perry J: Electromyographic analysis of the shoulder during the golf swing. *Am J Sports Med* 18(2);137-140, 1990.
75. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (p. 218).
76. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 164, 165).
77. Reeves TJ, Harrison TR: Diagnostic and therapeutic value of the reproduction of chest pain. *Arch Intern Med* 91:8-25, 1953 (p. 15).
78. Rinzler SH: *Cardiac Pain.* Charles C Thomas, Springfield, Ill., 1951 (pp. 82, 84).
79. Rinzler SH, Travell J: Therapy directed at the somatic component of cardiac pain. *Am Heart J* 35:248-268, 1948 (pp. 249, 256; Cases 1 and 3).
80. Smith JR: Thoracic pain. *Clinics* 2:1427-1459, 1944.
81. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (pp. 280, 281).
82. Stegman D, Mead BT: The chest wall twinge syndrome. *Nebr Med J 55(9):528-533,* 1970.
83. Theobald GW: The relief and prevention of referred pain. *J Obstet Gynaecol Br Com* 56:447-460, 1949 (pp. 451-452).
84. Tietze A: Ueber eine eigenartige Haufung von Fallen mit Dystrophie der Rippenknorpel. *Berl Klin Wochenschr* 58:829-831, 1921.
85. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 274).
86. Travell J: Early relief of chest pain by ethyl chloride spray in acute coronary thrombosis, case report. *Circulation* 3:120-124, 1951.
87. Travell J: Introductory remarks. In: *Connective Tissues. Transactions of the Fifth Conference, 1954.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1954 (p. 18).
88. Travell J: Chairs are a personal thing. *House Beautiful,* Oct. 1955 (pp. 190-193).
89. Travell J: Referred pain from skeletal muscle: the pectoralis major syndrome of breast pain and soreness, and the sternomastoid syndrome of headache and dizziness. *NY State J Med* 55:331-339,1955 (p. 332, Fig. 1A, Cases 1 and 2).
90. Travell J: *Office Hours: Day and Night.* The World Publishing Company, New York, 1968 (pp. 261, 263, 264).
91. Travell J: Myofascial trigger points: clinical view. In: *Advances in Pain Research and Therapy.* Edited by Bonica JJ, Albe-Fessard D. Raven Press, New York, 1976 (pp. 919- 926).
92. Travell J, Bigelow NH: Role of somatic trigger areas in the patterns of hysteria. *Psychosom Med* 9:353-363, 1947.
93. Travell J, Rinzler SH: Relief of cardiac pain by local block of somatic trigger areas. *Proc Soc Exp Biol Med* 63:480-482, 1946.

94. Travell J, Rinzler SH: Pain syndromes of the chest muscles: resemblance to effort angina and myocardial infarction, and relief by local block. *Can Med Assoc J* 59:333-338, 1948 (Case 1).
95. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 11:425-434, 1952.
96. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J Am Osteopath Assoc* 72:697-710, 1973.
97. Weiss S, Davis D: The significance of the afferent impulses from the skin in the mechanism of visceral pain. Skin infiltration as a useful therapeutic measure. *Am J Med Sci* 176:517-536, 1928.
98. Winter Z: Referred pain in fibrositis. *Med Rec* 157:34-37, 1944 (pp. 4, 5).
99. Wise CM, Semble EL, Dalton CB: Musculoskeletal chest wall syndromes in patients with noncardiac chest pain: a study of 10 patients. *Arch Phys Med Rehabil* 73(2):147-149, 1992.
100. Young D: The effects of novocaine injections on simulated visceral pain. *Ann Intern Med* 19:749-756, 1943 (pp. 751, Cases 1 and 2).
101. Zeman SC, Rosenfeld RT, Lipscomb PR: Tears of the pectoralis major muscles. *Am J Sports Med* 7(6):343-347, 1979.

CHAPTER 43
Pectoralis Minor Muscle

HIGHLIGHTS: When the pectoralis minor muscle harbors trigger points (TrPs), its taut fibers are likely to entrap the axillary artery and the brachial plexus and frequently mimic cervical radiculopathy. **REFERRED PAIN** from a left-sided muscle, pectoralis major or minor, may refer pain to the precordium that mimics the angina of myocardial ischemia. The pectoralis minor refers pain over the front of the chest, primarily to the front of the shoulder, and sometimes down the ulnar side of the arm, forearm and fingers. The **ANATOMY** of this muscle differs from that of the pectoralis major by connecting the anterior rib cage to the coracoid process rather than to the humerus. **FUNCTION** of the pectoralis minor, therefore, includes pulling the scapula and shoulder region down and forward, and assisting the upper chest muscles in forced inhalation. **PATIENT EXAMINATION** discloses a round-shouldered posture. Shoulder motion is somewhat restricted when reaching forward and upward, and more restricted when reaching backward at shoulder level. **TRIGGER POINT EXAMINATION** proceeds by palpating the pectoralis minor indirectly through the pectoralis major, or directly, reaching it by sliding the thumb beneath the pectoralis major, using pincer palpation. **ENTRAPMENT** symptoms due to compression of the brachial plexus (medial and lateral cords) and of the axillary artery by an abnormally taut pectoralis minor muscle are accentuated when the arm is fully abducted. **TRIGGER POINT RELEASE** is initiated by applying vapocoolant in upsweeps over the anterior chest and shoulder and down the ulnar surface of the arm, followed by application of a manual release technique. **TRIGGER POINT INJECTION** is performed by directing the needle nearly parallel to the chest wall and not toward the ribs, using pincer palpation wherever the patient's anatomy permits. **CORRECTIVE ACTIONS** for long-term relief require that a stooped posture or other stress overload on the muscle be eliminated.

1. REFERRED PAIN
(Fig. 43.1)

The trigger points (TrPs) in the pectoralis minor muscle refer pain most strongly over the anterior deltoid area. With very active TrPs, the pain may extend upward over the subclavicular area, and sometimes covers the entire pectoral region on the same side. Spillover referred pain extends along the ulnar side of the arm, elbow, forearm, and palmar hand to include the last three fingers (Fig. 43.1). At this point, no distinction is drawn between the pain originating from an upper attachment TrP (ATrP) or the lower central TrP (CTrP) in the pectoralis minor.

Essentially the same pattern also is referred from adjacent clavicular division TrPs of the pectoralis major muscle *(see* Fig. 42.1A).[43]

Pain from either pectoral muscle[25,34] and specifically the pectoralis minor,[28] can closely mimic the pain of cardiac ischemia.

2. ANATOMY
(Fig. 43.2)

The pectoralis minor muscle attaches ***above*** to the medial aspect of the tip of the coracoid process of the scapula and ***below*** to the third, fourth and fifth ribs near their costal cartilages[9] (Fig. 43.2). It also may attach as low as the sixth rib, or as high as the first rib.[5]

The tip of the coracoid process also provides a site of attachment for the tendons of the coracobrachialis and short head of the biceps brachii muscles. A slip of the pectoralis minor may extend beyond the coracoid process in about 15% of bodies to

attach to tendons of adjacent muscles, or to the greater tuberosity of the humerus.[4,5]

Two other, relatively infrequent, anatomical variations are described.[16] The pectoralis *minimus* connects the first rib cartilage to the coracoid process, effectively extending the thoracic cage influence of the pectoralis minor muscle cephalad.[33] The pectoralis *intermedius* may attach more medially than the pectoralis minor onto the third, fourth and fifth rib cartilages and attach above to the fascia covering the coracobrachialis and biceps brachii muscles. This arrangement sandwiches the intermedius between the pectoralis major and minor muscles.[16]

Supplemental References

Other authors have clearly illustrated the pectoralis minor muscle as seen from the front,[1,10,15,27,33,38,41] from in front with neurovascular structures,[2] from the side,[30] from the side with neurovascular structures,[12] from below with neurovascular structures,[31] and in cross section.[14,17] In a common variation, fibers extend over the coracoid process to reinforce the coracohumeral ligament.[4]

Approximately 40% of pectoralis minor fibers are type II, decreasing slightly after age 60. The volume of type II fibers is significantly decreased after that age.[37]

3. INNERVATION

The pectoralis minor is innervated by the medial pectoral nerve from the medial cord, and by fibers of roots C_8 and T_1.[9]

4. FUNCTION

The pectoralis minor draws the scapula forward, downward and inward at nearly equal angles.[33] Depression of the shoulder by this muscle[9,13,21] stabilizes the scapula when the arm exerts downward pressure against resistance.[33] Since the inward force component is blocked by the clavicle when this muscle contracts, the resultant force draws the glenoid fossa of the scapula

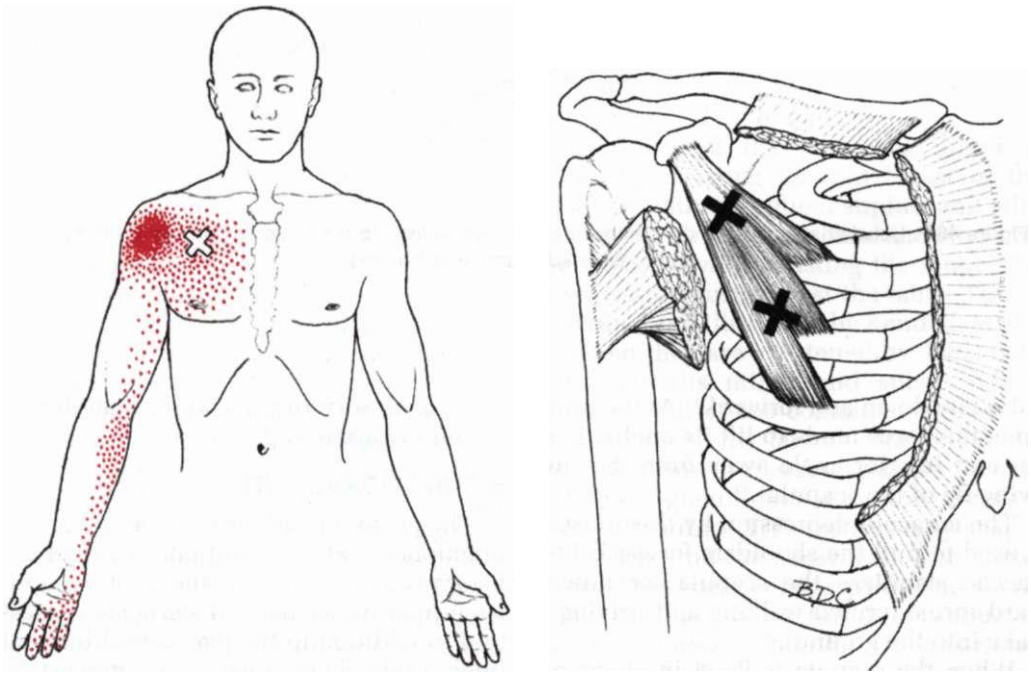


Figure 43.1. Referred pain pattern (*solid red* is the essential portion, *stippled red* shows the spillover portion), and trigger point locations (**Xs**) in the right pectoralis minor muscle. The upper **X** identifies the location of an attachment trigger point and the lower **X** a central trigger point location in this muscle.

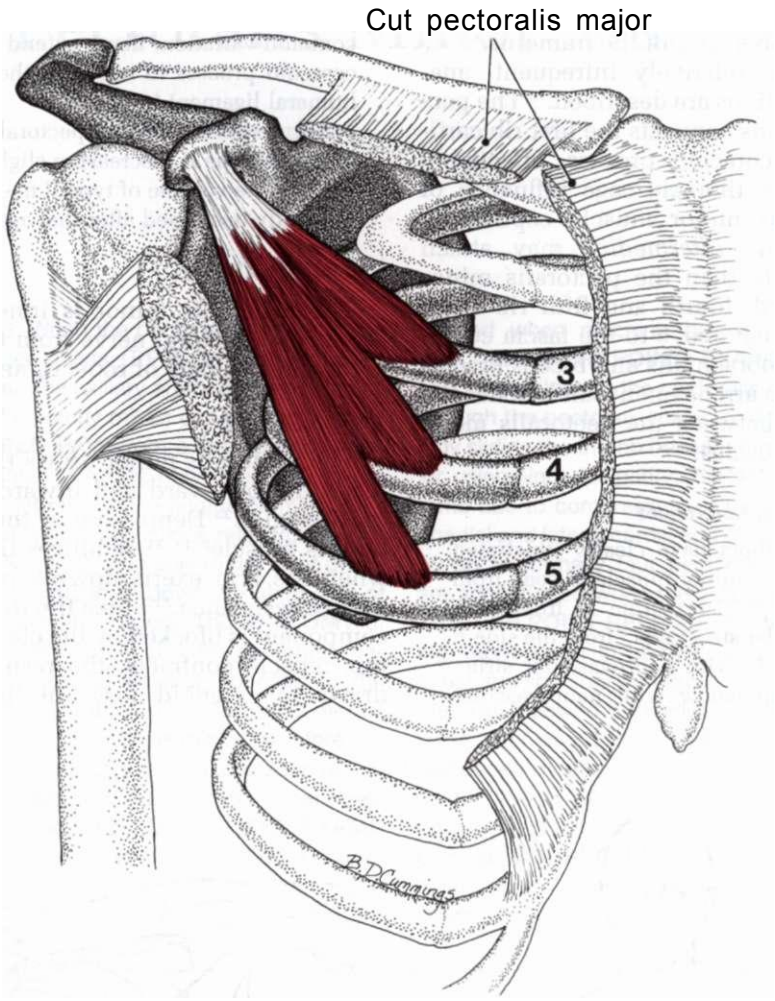


Figure 43.2. Usual attachments of the pectoralis minor muscle (red) to the coracoid process of the scapula and to the third, fourth, and fifth ribs.

obliquely down and forward.[13] At the same time, this force tends to lift its medial border and inferior angle away from the ribs (winging of the scapula).[33]

The coracoid depression by this muscle is used to pull the shoulders forward. The muscle stabilizes the scapula for downward thrust (crutch walking and driving a stake into the ground).[39]

When the scapula is fixed in elevation by the upper trapezius and levator scapulae muscles, the pectoralis minor becomes active during strong inhalation efforts that involve the upper chest.[13] It thus can serve as an accessory respiratory muscle during forced inspiration.[6,9,33]

5. FUNCTIONAL UNIT

The pectoralis minor forms a synergistic functional unit for additional support for vigorous inhalation from the levator scapulae, upper trapezius, and sternocleidomastoid in addition to the parasternal internal intercostals, lateral external intercostals, the diaphragm, and the scalene muscles. Electromyographically, the pectoralis minor is active in forced inspiration, but not in quiet breathing.[6] The pectoralis minor

assists the pectoralis major in depression of the shoulder, forward pull of the scapula, and downward rotation of its lateral angle (glenoid fossa). It also assists the latissimus dorsi in depression of the shoulder.

The lower trapezius muscle acts as an antagonist to the pectoralis minor in scapular rotation and protraction.

6. SYMPTOMS

The patient's chief complaint is pain with no sharp distinction being made between the pain referred from TrPs in the pectoralis minor and from TrPs in the overlying and adjacent portions of the pectoralis major. The intensity and quality, as well as the distribution, of cardiac pain may be reproduced by this pectoral muscle's referred pain.[34]

The patient may be aware of difficulty in reaching forward and up, or reaching backward with the arm at shoulder level.

The shortened pectoralis minor may cause distinctive neurovascular symptoms through entrapment of the neurovascular bundle to the upper extremity[36] (see Section 10, Fig. 43.4).

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

A posture or activity that activates a TrP, if not corrected or if continued, can also perpetuate it. In addition, many structural and systemic factors (see Chapter 4) will perpetuate a TrP that has been activated by an acute or chronic overload.

Pectoralis minor TrPs may be activated as satellite TrPs due to their presence within the zone of pain induced by myocardial ischemia, as satellites of scalene or pectoralis major TrPs,[19] by trauma (a gunshot wound through the upper chest, or fracture of upper ribs), by a whiplash type motor vehicle accident,[20] by strain through overuse as a shoulder depressor (unaccustomed crutch-walking), by strain as an accessory muscle of inspiration (during paroxysms of severe coughing, or to assist paradoxical breathing), by poor seated posture (keeping the muscle chronically shortened because of a poorly designed chair or work environment), or by prolonged compression of the muscle (knapsack with a tight strap over the front of the shoulder).

Weakness of the lower trapezius can allow the scapula to ride up and tilt down anteriorly, and may lead to adaptive shortening of the pectoralis minor, activating or perpetuating TrPs in it.

8. PATIENT EXAMINATION

After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram representing the pain described by the patient. The drawing should be in the style of the pain patterns in this volume using a copy of an appropriate body form found in Figures 3.2-3.4.

A patient with significant TrP shortening of the pectoralis minor will usually demonstrate forward (rounded) shoulders because of the forward and downward tilt of the coracoid process by the pectoralis minor.

The increased tension due to TrPs in the pectoralis minor prevents the patient from reaching fully behind the back at shoulder level. The anterior depression of the caracoid and downward rotation of the glenoid fossa that are caused by pectoralis minor tension limits full flexion of the arm at the shoulder joint.[23] Shortening of this muscle is observable as elevation (forward position) of the involved shoulder away from the table in the supine patient, as illustrated by Kendall, er a7.[23]

Weakness of the pectoralis minor is tested by resisting forward thrust of the shoulder with the patient supine, and with the subject elevating the hand and elbow off the table to avoid assisting the motion by downward thrust against the table. This is described and illustrated by Kendall, *et al.*[23]

When they are shortened by TrPs, both the pectoralis minor and subscapularis muscles restrict the combined movement of abduction and lateral rotation at the shoulder. However, subscapularis TrPs restrict only glenohumeral motion, whereas pectoralis minor TrPs restrict only scapular mobility on the chest wall. The movement of the scapula is palpable and sometimes visible. With the arm abducted to 90°, lateral rotation is restricted markedly by both muscles; with the arm at the side, only the subscapularis seriously restricts lateral rotation. Also, when abduction of the arm at the shoulder is restricted by pectoralis minor taut-

ness, the patient may be aware of pulling *on the ribs at the limit of abduction.* These observations are of confirmatory value. The subscapularis and pectoralis minor muscles have different referred pain patterns, which are not likely to be confused.

9. TRIGGER POINT EXAMINATION (Fig. 43.3)

First, the pectoralis major should be examined for active TrPs that might obscure and confuse the localization of TrPs in the underlying pectoralis minor.

If the examiner is unsure of the position of the pectoralis minor muscle under the pectoralis major, it can be located by palpation when the patient tenses the pectoralis minor. To do this, the supine patient raises the shoulder away from the examining table, while relaxing the arm and carefully avoiding downward pressure against the table with the hand.[23] In the sitting position, the patient holds the arm close to the side, a little to the rear to inhibit the pectoralis major, strongly protracts the shoulder, and then inhales deeply with the chest.[33] Both maneuvers activate the pectoralis minor so that it can be identified.

In both the supine and seated positions, pectoralis minor TrPs can be localized either by flat palpation through the pectoralis major against the chest wall (Fig. 43.3A) as also illustrated by Webber,[44] or by pincer palpation (Fig. 43.3B). With either approach, the pectoralis major is slackened by keeping the patient's arm toward the front of the body and the forearm on the abdomen, and the pectoralis minor may be placed on the desired degree of stretch by adducting the scapula toward the military-brace position. The two pectoral muscles may be distinguished by noting the muscle fiber direction of palpable bands and of local twitch responses.

Although the patient achieves better relaxation in the supine than in the seated position, it is often convenient and informative to screen both pectoral muscles for TrPs using flat palpation with the patient seated. The seated position simplifies range-of-motion testing and the Irving S. Wright hyperabduction maneuver.[45]

In the supine position, and in non-obese *patients with relatively loose skin, the* pectoralis minor can usually be palpated directly by pincer palpation (Fig. 43.3B). The pectoralis major may be further slackened by placing the arm in the position described above, and, if additional relief is necessary, the shoulder is protracted by padding placed under it. The operator places the thumb (with a well-trimmed fingernail) in the apex of the axilla and slides it against the chest wall beneath the pectoralis major toward the midline, until it encounters the muscle mass of the pectoralis minor. That muscle (and the pectoralis major above it) are then encompassed by a pincer grasp between the thumb and fingers (Fig. 43.3B) partially separating it from the chest wall. The fibers of the pectoralis minor can then be palpated directly through the skin for a tender nodule in a taut band. Identification of TrPs in the pectoralis minor may be enhanced by elevating the shoulder cephalad to tauten the pectoralis minor, which increases the sensitivity of its TrPs without tightening the pectoralis major.

10. ENTRAPMENT (Fig. 43.4)

The pectoralis minor is the landmark for anatomically dividing the axillary artery into three parts; the second part of the artery lies deep to the muscle. Likewise, the distal portion of the brachial plexus passes deep to the pectoralis minor muscle where the muscle attaches to the coracoid process. When the arm is abducted and laterally rotated at the shoulder, the artery, vein, and nerves are bent and stretched around the pectoralis minor muscle close to its attachment, and are likely to be compressed if the muscle is firm and tightened by myofascial TrPs (Fig. 43.4B). The pectoralis minor tension increases the entrapment potential of the C_7 and C_8 roots that hook over the first rib. Kendall, *et al.*[24] have described in detail this entrapment of muscular origin noting that pectoralis minor shortening is the most likely cause, aggravated by tension in the biceps brachii and coracobrachialis, and by weakness (or inhibition) of the lower trapezius muscle.

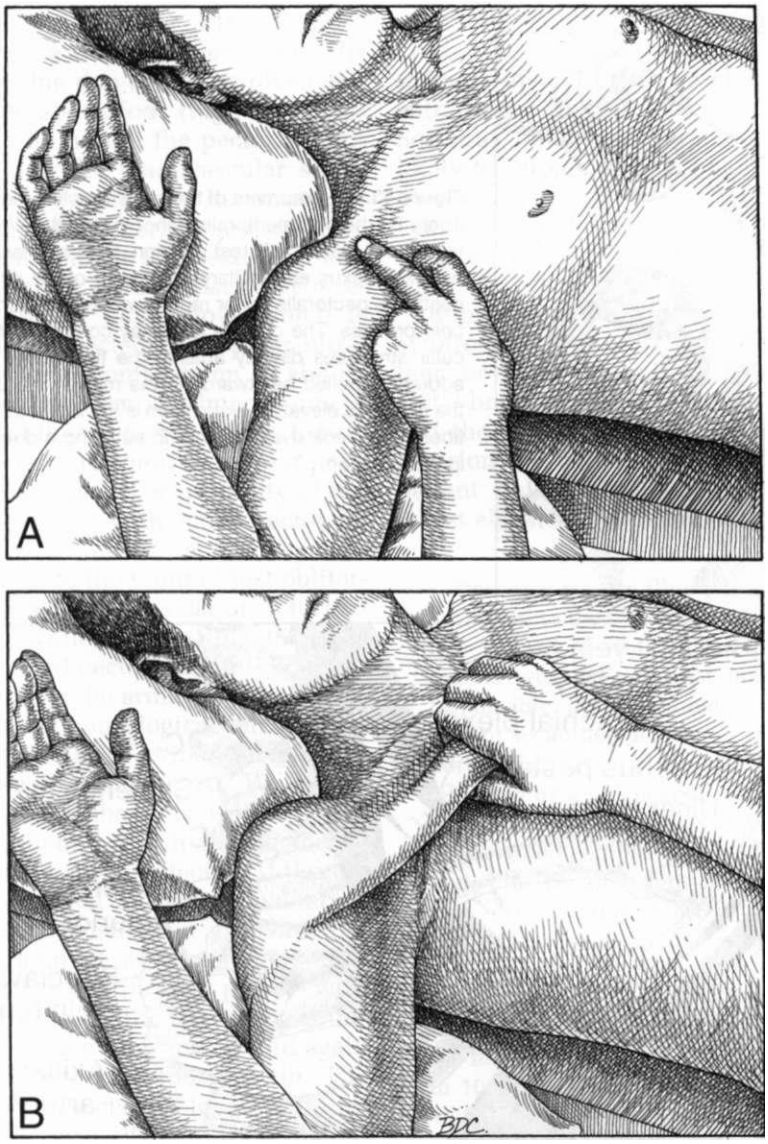


Figure 43.3. Palpation of trigger points in the pectoralis minor muscle. The overlying pectoralis major is slackened by supporting the arm as shown, or by placing the forearm on the abdomen. **A,** flat palpation of the pectoralis minor through the pectoralis major. **B,** pincer palpation around the pectoralis major. The thumb contacts the pectoralis minor, through only the skin. The fingers grasp it through the pectoralis major. Together they can partially separate it from the chest wall. The pectoralis minor may be tautened for better identification of its trigger points by elevating the shoulder.

The entrapment of the axillary artery can be demonstrated by the Wright maneuver,[45] which places the arm in lateral rotation and abduction at the shoulder (Fig. 43.4A) while the radial pulse is palpated. The test is more effective if the patient is not allowed to elevate the scapula and relieve tension on the neurovascular structures. This position can produce compression of the neurovascular structures by the pectoralis minor,[7,8] and by closure of the costoclavicular space if the scapula also is adducted. Entrapment symptoms and obliteration of the radial pulse by abduc-

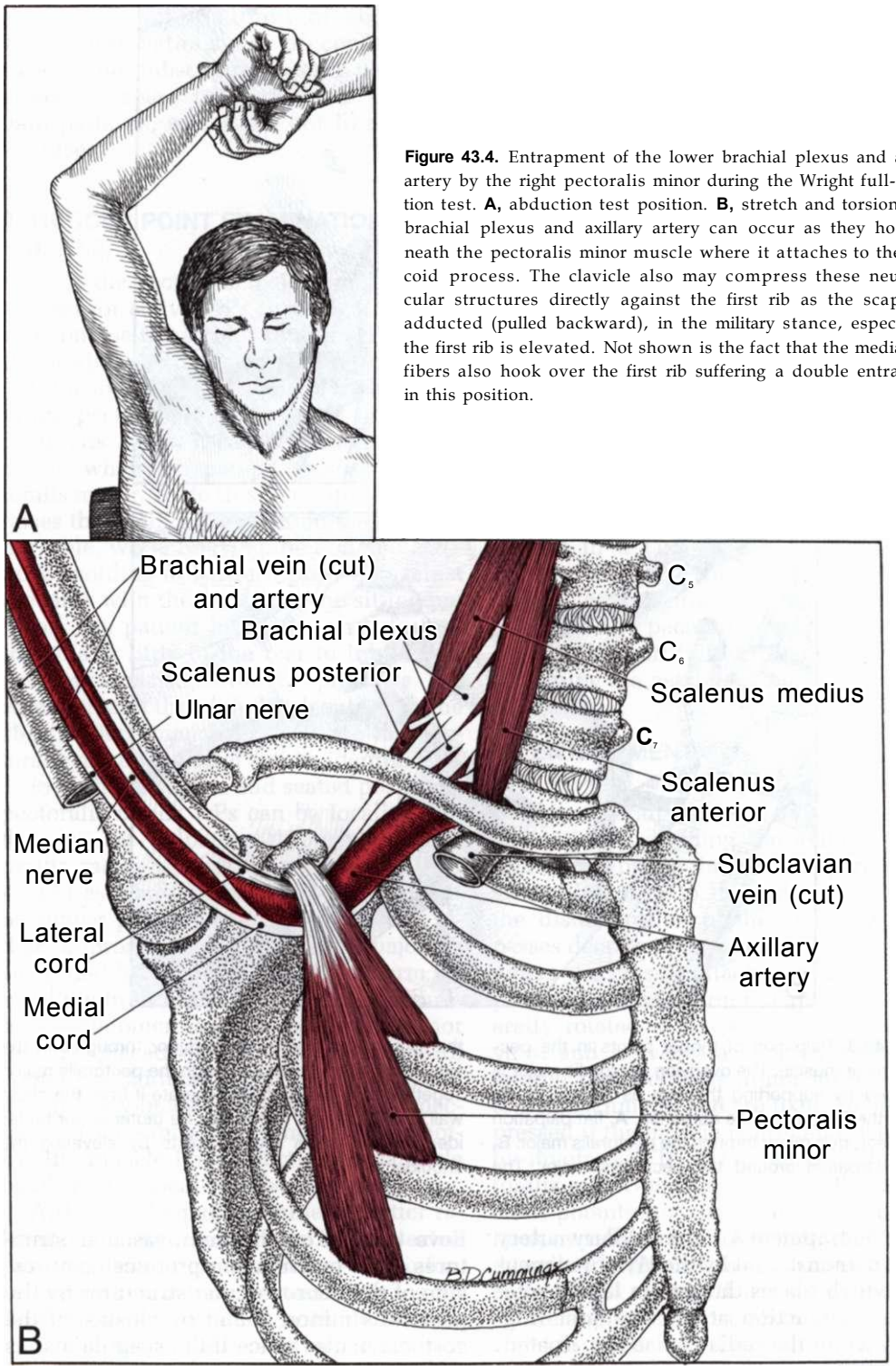


Figure 43.4. Entrapment of the lower brachial plexus and axillary artery by the right pectoralis minor during the Wright full-abduction test. **A,** abduction test position. **B,** stretch and torsion of the brachial plexus and axillary artery can occur as they hook beneath the pectoralis minor muscle where it attaches to the coracoid process. The clavicle also may compress these neurovascular structures directly against the first rib as the scapula is adducted (pulled backward), in the military stance, especially if the first rib is elevated. Not shown is the fact that the medial cord fibers also hook over the first rib suffering a double entrapment in this position.

tion of the arm to only 90° at the shoulder demonstrates the effect of severe pectoralis minor shortening due to TrP involvement. Further hyperabduction (Fig. 43.4) increases tension on both the pectoralis minor muscle and the neurovascular structures, which may produce demonstrable compression in some normal subjects.

Arterial entrapment is detected by loss of the radial pulse at the wrist or by a reduction of arterial blood flow, which is more precisely detected by Doppler ultrasound.[32] If arterial compression in abduction or hyperabduction is primarily due to TrP activity of the pectoralis minor, the radial pulse may be restored in the test position by eliminating hyperirritability of the TrPs. When patients with active pectoralis minor TrPs were placed in the hyperabducted position to the point of just obliterating the radial pulse, pulsation returned immediately while vapocooling the skin over the stretched pectoralis minor muscle, without changing the arm position.

Symptoms of neurological entrapment are similar to those described for the scalene muscles in Chapter 20. When the Wright maneuver (above) is used to detect nerve entrapment beneath the pectoralis minor, the test is more effective if the patient is not allowed to elevate the shoulder to relieve tension on the brachial plexus. Entrapment of the medial cord (Fig. 43.4B) occurs in two places with this arm position, as the nerve hooks under the pectoralis minor tendon and again as its fibers hook over the first rib. The medial cord connects the lower trunk to the ulnar nerve.[3, 11] This entrapment causes numbness and paraesthesias of the fourth and fifth digits, but usually not of the thumb and other fingers. The lateral cord is more directly compressed (Fig. 43.4B) than the medial cord and connects with the upper and middle trunks proximally, and the musculocutaneous and median nerves distally.[3, 11] This entrapment disturbs sensation over the dorsum and radial aspects of the forearm and over the palmar side of the first three and one-half digits.[9] Compression of both cords disturbs much of the sensation below the elbow.

Entrapment by the taut pectoralis minor does not produce the hand edema and stiffness of the fingers so characteristic of entrapment by the scalenus anterior. Scalenus anterior entrapment is more likely to impair venous, than arterial, circulation by compression of the subclavian vein between the clavicle and first rib. This occurs because the first rib is elevated by shortening of the scalenus anterior muscle.

Entrapment due to the costoclavicular syndrome is caused by compression of either, or both, the axillary artery and the distal brachial plexus between the clavicle and the first rib. The effects of this compression are demonstrated by having the patient hold the military brace position (chest elevated and scapulae adducted).

Two case reports illustrate entrapment attributed to the pectoralis minor muscle. In both cases, the findings were fully compatible with TrPs in that muscle being responsible, but in neither case was the patient examined for them. Hewitt[18] reported obstruction of the axillary vein established by a phlebogram that, when explored surgically, revealed no thrombus, but revealed compression by a tense tendon of the pectoralis minor. Surgical division of the tendon relieved the patient's entrapment symptoms. Pasquariello, *et al.*[29] reported a patient with chest pain and signs of venous and lower trunk entrapment and signs of costochondritis of the first to sixth ribs. Symptoms resolved in 10 days with application of local heat and oral salicylates and were attributed to spasm of the pectoralis minor muscle secondary to the costochondritis.

11. DIFFERENTIAL DIAGNOSIS

Differential diagnosis of symptoms caused by TrPs in the pectoralis minor muscle includes thoracic outlet syndrome, C_7 and C_8 radiculopathy, supraspinatus tendinitis, bicipital tendinitis, and medial epicondylitis.

Articular dysfunctions that are likely to be associated with pectoralis minor TrPs include elevation of the third, fourth, and fifth ribs.

Related Trigger Points

One rarely, if ever, finds active TrPs in the pectoralis minor without active TrPs in the pectoralis major. Therefore, the same muscles that are commonly associated with pectoralis major involvement are likely to harbor active TrPs when the pectoralis minor is involved: the anterior deltoid, scalene, and the sternocleidomastoid muscles.

On the other hand, one may find TrPs in the pectoralis major without involvement of the pectoralis minor, especially when the TrPs are located in the parasternal section and lower lateral border of the costal section of the pectoralis major muscle.

Connective tissue TrPs have been found in posttraumatic scar tissue in the region of the coracoid attachment of the pectoralis minor. These TrPs have referred tenderness, hot burning pain, prickling, and lightening-like jabs to the pectoral region and olecranon process on the same side. Injection of these connective tissue TrPs caused brilliant momentary flashes of local and referred pain, followed by relief.

12. TRIGGER POINT RELEASE (Fig. 43.5)

Of primary importance is the correction of faulty posture, particularly round-shouldered posture, and instructions to the patient for maintenance of correct posture and movement. Refer to Chapter 41, Section C for a discussion of static and dynamic postural considerations.

Instead of the usual spray-and-stretch technique, which can be used effectively by following the same principles applied in other chapters, this chapter presents the same initial spray followed by manual release of the tense muscle. Other techniques that are effective include postisometric relaxation and contract-relax as described in Chapter 3, Section 12. These techniques are primarily effective for release of *central* trigger points (CTrPs). The primary therapeutic approach to *attachment* TrPs (ATrPs) is to inactivate the CTrPs that are causing them.

The prespray technique for the pectoralis minor muscle is described and illustrated in Figure 43.5A and the manual stretch that follows is illustrated and described in Figure 43.5B. A similar myofascial release technique is illustrated in Figure 12.8A that includes release of the clavicular and upper sternal portions of the pectoralis major muscle.

Lewit[26] describes the tenderness of enthesopathy at the rib attachments of the pectoralis minor muscle and relieves it by applying postisometric relaxation for that muscle. The gentleness of his technique is critical for this application because excessive stretch force would tend to irritate the enthesopathy and compromise effective release of the central TrPs causing it. Hard stretch of an already tense muscle can also aggravate nerve entrapment syndromes.

13. TRIGGER POINT INJECTION (Fig. 43.6)

Injection of pectoralis minor trigger points (TrPs) should be done with the patient supine, *not* seated, to avoid psychologically induced syncope and only after TrPs in the pectoralis major have been inactivated to avoid recurrence. The upper *X* in Figure 43.1, an ATrP, is in the region of the musculotendinous junction near the coracoid process and is reached by directing the needle toward the coracoid process,[34] as illustrated in Figures 43.6A and C. Whenever possible, the hand of the operator locates the pectoralis minor underneath the pectoralis major. This requires pincer palpation as described in Section 9, with the fingers (or thumb) contacting the pectoralis minor directly (Figs. 43.6B and C). The needle is directed parallel to the rib cage toward the coracoid process.

The lower *X* in Figure 43.1 is close to the midregion of muscle fibers in the fourth rib digitation, where central TrPs are found in that digitation. Generally, these midfiber CTrPs are approached with the needle directed caudad, as tangential to the plane of the chest wall as possible (Fig. 43.6B), so as to preclude the needle's entering an intercostal space. The Hong technique (*see* Chapter 3, Section 13) is recommended for injection here.

After injection of TrPs, the patient should move the arm and shoulder slowly three times through *full* range of motion for the pectoralis major, followed by moist heat over the pectoral region.

A study of the response of whiplash-injury patients to repeated injections of the pectoralis minor muscle[20] demonstrated clearly that the longer the interval between injury and the start of appropriate TrP therapy, the greater the number of repeat injections that were required. Also the effect of individual injections did not last as long in those with delayed onset of treatment. The same principle very likely applies to other muscles as well.

14. CORRECTIVE ACTIONS

TrPs should be inactivated in any muscles, such as the scalene group and pectoralis major, that refer pain to the region of

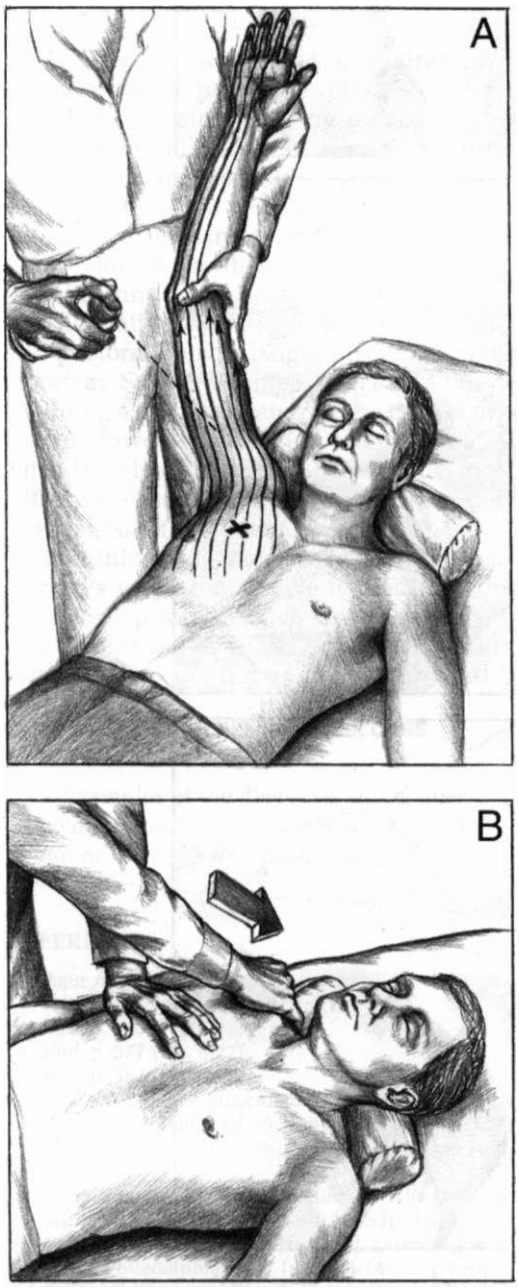


Figure 43.5. Vapocoolant spray and stretch release of the right pectoralis minor muscle. **A,** Application of vapocoolant spray *(arrows)* for a trigger point (**X**) in the pectoralis minor muscle. The arm is raised diagonally overhead (slightly abducted and laterally rotated) just to the onset of resistance or discomfort. Upsweeps of the spray cover over the pectoralis minor muscle and its pain pattern, which extends distally to include the ulnar aspect of the forearm and the three ulnar fingers. This position of the arm also lengthens the pectoralis major which should be sprayed at the same time to avoid aggravating its trigger points. Frequently the two pectoral muscles are involved together. **B,** release of right pectoralis minor trigger point tightness by applying pressure *(arrow)* on the shoulder to move the upper part of the scapula posteriorly with one hand while stabilizing the costal attachments of the muscle with the other. If the lower trapezius is weak, it should be strengthened in order to provide scapular stabilization.

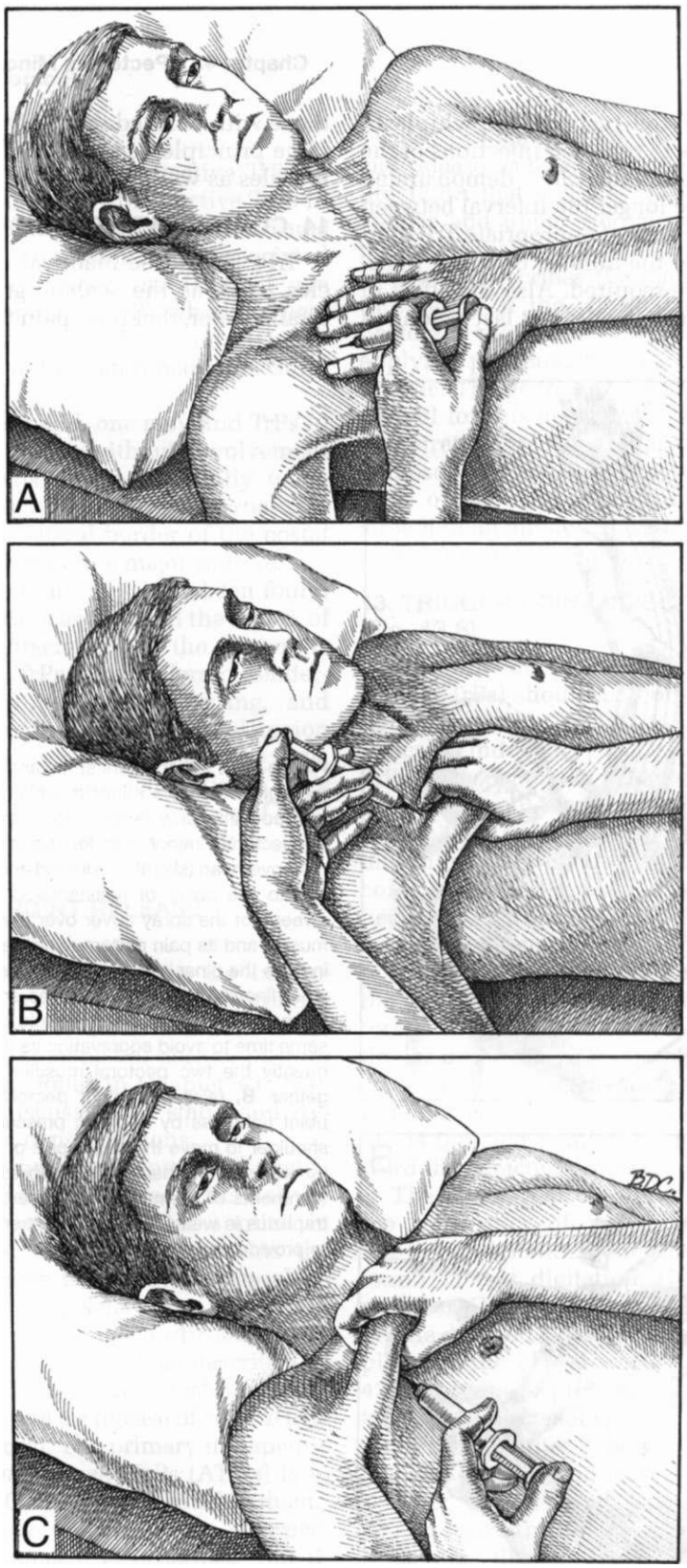


Figure 43.6. Injection of the pectoralis minor muscle by a left handed operator. **A,** injection of the upper, attachment area trigger point after its localization by flat palpation. **B,** injection of a midfiber, central trigger point from above with the trigger point localized between the digits by pincer palpation. **C,** injection of the upper trigger point from below with the trigger point localized by the finger tips in a pincer grasp.

the pectoralis minor and thus are likely to induce satellite TrPs in it.

Activity stress due to overuse must be avoided by identifying and limiting the offending activity, such as gardening, working at a desk, and crutch walking. Paradoxical breathing *(see* Fig. 20.15A) needs correction, as described in Chapter 20.

Standing and seated posture should be improved (Chapter 41, Section C). A weak lower trapezius should be strengthened.

A heavily loaded brassiere strap that compresses the pectoralis minor should be avoided. The strap may be placed on the acromion to relieve pressure on the muscle or padded to distribute the load more widely. An elastic vest-type support is also effective in helping to hold the shoulders back without the discomfort of straps under the arms.[22]

The patient should learn to maintain full pectoral muscle length by using the Indoorway Stretch Exercise *(see* Fig. 42.9) or by doing a similar stretch in the corner of a room. However, a manual stretch that rotates the shoulder girdle back is the most effective stretch but requires another person to assist the patient.

To minimize aggravation of pectoralis minor TrPs when the muscle is placed in the shortened position when sleeping, the patient avoids sleeping "curled up" on the side with the shoulder forced strongly forward.[36]

SUPPLEMENTAL REFERENCES, CASE REPORTS

Examples of the diagnosis and management of patients with active pectoralis minor TrPs were presented by Dr. Travell.[35,42]

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991:16 (Fig. 1.15).
2. *Ibid.* p. 373 (Fig. 6.22).
3. *Ibid.* p. 377 (Fig. 6.27).
4. *Ibid.* p. 383 (Fig. 6.35).
5. Bardeen CR: The musculature, Sect. 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (pp. 406, 407).
6. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (p. 426).
7. Cailliet R: *Soft Tissue Pain and Disability,* FA. Davis, Philadelphia, 1977 (pp. 144-146, Fig. 116).
8. Cailliet R: *Neck and Arm Pain.* FA. Davis, Philadelphia, 1964 (pp. 95, 96).
9. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 520, 521).
10. *Ibid.* (Fig. 6-45).
11. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 18).
12. *Ibid.* (Fig. 20).
13. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 19, 479, 481).
14. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (Fig. 68).
15. *Ibid.* (Fig. 69).
16. *Ibid.* (Fig. 73, pp. 477-479).
17. Ellis H, Logan B, Dixon A: *Human Cross-Sectional Anatomy: Atlas of Body Sections and CT Images.* Butterworth Heinemann, Boston, 1991 (Sects. 32, 33, 35).
18. Hewitt RL: Acute axillary-vein obstruction by the pectoralis-minormuscle. *N Engl J Med279(ll):595,*1968.
19. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(1):29-59, 1994.
20. Hong CZ, Simons DG: Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. *J Musculoske Pain* 1(1):89-131, 1993.
21. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (p. 80).
22. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 68).
23. *Ibid.* (p. 278).
24. *Ibid.* (p. 343.)
25. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (p. 98).
26. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 198, 199).
27. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 117).
28. Mendlowitz M: Strain of the pectoralis minor, an important cause of precordial pain in soldiers. *Am Heart J* 30.123-125, 1945.
29. Pasquariello PS Jr., Sherk HH, Miller JE: The thoracic outlet syndrome produced by costochondritis. *Clin Pediatr* 20(9);602-603, 1981.
30. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 38).
31. *Ibid.* (Fig. 39).
32. Pisko-Dubienski ZA, Hollingsworth J: Clinical application of doppler ultrasonography in the thoracic outlet syndrome. *Can J Surg* 21:145-150, 1978.
33. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 154, 155, 164).
34. Rinzler SH: *Cardiac Pain.* Charles C Thomas, Springfield, Ill. 1951 (pp. 37, 85).
35. Rinzler SH, Travell J: Therapy directed at the somatic component of cardiac pain. *Am Heart]* 35:248-268, 1948 (pp. 261-263, Case 3).
36. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Rehabil* 62:107-110, 1981.
37. Sato T, Akatsuka H, Kito K, *et al.:* Age changes in size and number of muscle fibers in human minor pectoral muscle. *Mech Ageing Dev* 28(lJ:99-109, 1984.

38. Spalteholz W: *Handatlas der Anatomie des Men-schen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 282).
39. Steindler A: *Kinesiology of the Human Body.* Charles C Thomas, Springfield, Ill, 1955 (pp. 468, 469).
40. Sucher BM: Thoracic outlet syndrome—a myofascial variant: Part 1. Pathology and diagnosis. *J Am Osteopath Assoc* 90(8):686-704, 1990.
41. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 274).
42. Travell J, Rinzler SH: Pain syndromes of the chest muscles. Resemblance to effort angina and myocardial infarction, and relief by local block. *Can Med Assoc J* 59:333-338, 1948 (pp. 333, 334; Case 1).
43. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med 11*:425-434, 1952.
44. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J Am Osteopath Assoc* 72:697-710, 1973 (pp. 10, 11; Fig. 29).
45. Wright IS: The neurovascular syndrome produced by hyperabduction of the arms. *Am Heart J* 29:1-19, 1945.

CHAPTER 44
Sternalis Muscle

HIGHLIGHTS: **REFERRED PAIN** from active trigger points (TrPs) in the anomalous sternalis muscle produces a deep substernal ache that is unrelated to movement. **ANATOMY** of the sternalis muscle is highly variable. The fibers are superficial to the pectoralis major and generally lie parallel to the margins of the sternum. The muscle may be located on one or both sides, running at right angles to, and overlying the sternal end of the pectoralis major muscle. It is reported to be present in approximately 1 of 20 black or white adults. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in this muscle are associated with pain referred to the sternum from the heart during myocardial ischemia or from the lower end of the sternocleidomastoid muscle. **TRIGGER POINT EXAMINATION** for sternalis TrPs is by flat palpation of the muscle against underlying bone to locate exquisite spot tenderness at a nodule in a taut band and to elicit pain that the patient recognizes as familiar. **TRIGGER POINT INJECTION** is directed precisely into a TrP, with the needle aimed toward bone. The TrPs also are readily accessible and responsive to trigger point pressure release. **CORRECTIVE ACTIONS** include primarily self-application of trigger point pressure release by the patient to ensure sustained relief.

1. REFERRED PAIN (Fig. 44.1)

The referred pain pattern of the sternalis usually includes the entire sternal and substernal region, and may extend on the same side across the upper pectoral area and front of the shoulder to the underarm and to the ulnar aspect of the elbow (Fig. 44.1).[2,18,20] This pattern closely mimics the substernal ache of myocardial infarction or angina pectoris. The chest pain referred from this muscle has a terrifying quality that is remarkably independent of body movement. The left-sided pattern of the sternalis differs from the referred pain of the left pectoralis major muscle in that the latter is more likely to extend beyond the elbow into the ulnar aspect of the left forearm and hand. Both muscles may contribute simultaneously to the pain reported by the patient; this is illustrated in case reports.[15,17,18]

Trigger points (TrPs) may be located anywhere within the sternalis muscle: as high as the manubrium, as low as the xiphoid process, and on either or both sides, including the midline of the sternum when the muscle fuses across the sternum. Sternalis TrPs usually occur over the upper two-thirds of the sternum and are most likely to be found as central TrPs slightly to the left of the midline at the mid-sternal level. Anatomically, a unilateral muscle is as common on the right as on the left, but active TrPs appear to be more common on the left side, probably because of their activation as satellite TrPs within the zone of referred pain from the heart.

Albeit the sternalis may be only a small remnant of muscle, the intensity of pain arising from TrPs in it (or any other muscle) is not related to the size of the muscle, but to the degree of irritability and size of the TrP.

At times, a TrP located at the confluence of the sternalis, pectoralis major and sternal division of the sternocleidomastoid muscles can be the source of a dry, hacking cough. Penetration of this TrP with a needle, in whichever muscle it lies, activates the cough momentarily, and then relieves it.

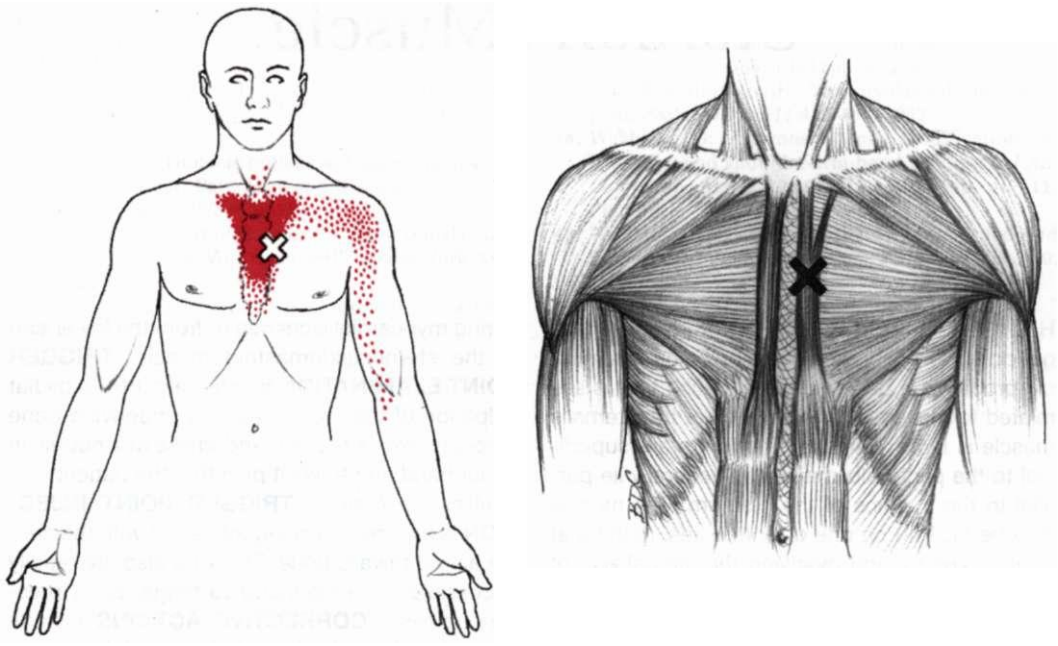


Figure 44.1. A trigger point (X) in the left sternalis muscle gives rise to the referred pain pattern shown in *red*.

2. ANATOMY
(Fig. 44.2)

The anomalous sternalis muscle is highly variable in presence, symmetry, length, bulk, attachments and innervation. It may occur bilaterally (Fig. 44.2), or more often unilaterally, on either side of the sternum or, rarely, the two muscles may fuse across the sternum. It may attach ***above*** to the sternum, to the fascia over either the pectoralis major or sternocleidomastoid muscle, or it may form a continuation of those muscles. ***Below*** it may attach to the third through seventh costal cartilages, the fascia covering the pectoralis major, and/or to the sheath of the rectus abdominis muscle.

The sternalis was found in 1.7% to 14.3% (median 4.4%) of cases in 13 studies of at least 10,200 bodies;[4] at most, in 48% of anencephalic specimens;[4] in 4.3% of 2,062 cadavers as summarized by Christian;[3] and in 6% of 535 cadavers according to Barlow.[1] Eisler,[4] Hollinshead,[8] Grant[7] and Toldt[13] each have illustrated the sternalis muscle. Christian[3] illustrated two bilateral muscles; Shen *et al.* reported one pair.[12] Barlow[1] reported no significant difference in the incidence of the sternalis muscle in white and black Americans. The muscle may be as thick as 2 cm (3/4 in) over the sternum, a depth of sternalis muscle through which it is difficult to palpate the features of pectoralis major TrPs (Fig. 44.2).

3. INNERVATION

Based on the innervation patterns of 26 sternalis muscles in 20 cadavers,[3] the sternalis muscle was considered a variant of either the pectoralis major or the rectus abdominis muscle. Sixteen of 26 sternalis muscles (62%) received their innervation from intercostal nerves (anterior primary divisions of thoracic spinal nerves), and were considered homologous to the rectus abdominis. The remaining 38% received their innervation from the cervical plexus, usually *via* the medial pectoral nerve, which is derived from spinal nerves C_n and T_1, so that these muscles were considered homologous with the sternal portion of the pectoralis major. Two muscles received a dual innervation.[3] Whether the sternalis muscle has an exact analogue in other species, it has been the subject of unresolved controversy. Its diverse innervation suggests that it may represent variable remnants of several muscles.

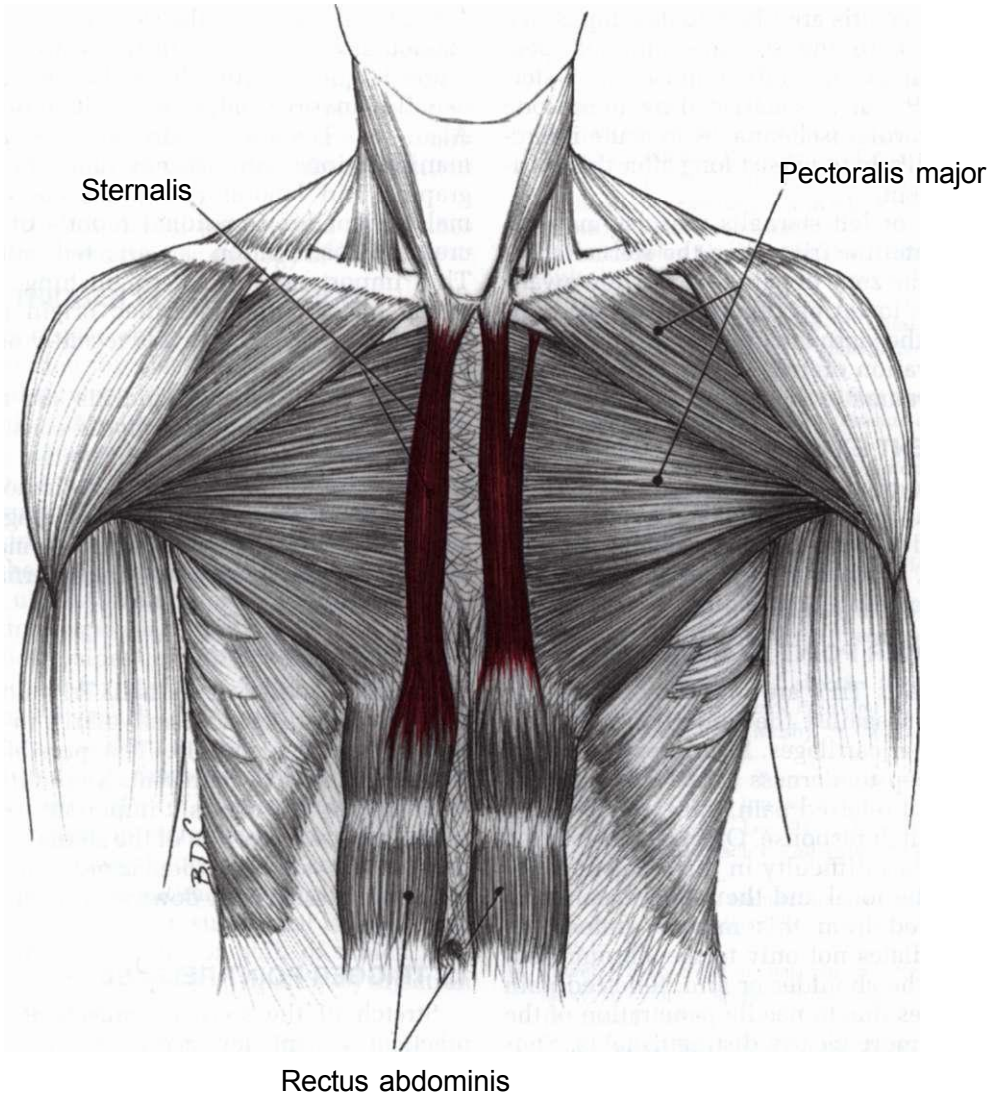


Figure 44.2. Commonly seen attachments of the anatomically variable sternalis muscle (red). It is twice as likely to be unilateral as bilateral and equally likely to occur on the right or left sides.

4. FUNCTION

No skeletal movement is attributed to this muscle. No electromyographic data or clinical reports of muscular contraction of the sternalis were located; thus, if, when, or why it contracts is unresolved.

5. FUNCTIONAL UNIT

The functional relation of the sternalis to other muscles must await determination of its function.

6. SYMPTOMS

The symptoms associated with TrPs in this muscle are intense deep substernal pain and occasionally, soreness over the sternum. Since the pain arising from this muscle is not aggravated by movement, its musculoskeletal origin is easily overlooked.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

It is important to realize that patients with either acute myocardial infarction or

angina pectoris are likely to develop active TrPs in both the sternalis and left pectoralis major and minor muscles. A sternalis TrP that was activated by an episode of myocardial ischemia, as in acute infarction, is likely to persist long after this initiating event.

Right or left sternalis muscles may develop satellite TrPs when the sternalis lies within the zone of pain referred downward from the lower portion of the sternal division of the sternocleidomastoid muscle.

Activation of TrPs also may result from direct trauma to the costosternal area.

8. PATIENT EXAMINATION

Range-of-motion tests are negative, since the pain is neither relieved nor aggravated by any musculoskeletal activity, such as, movement of the shoulder girdle, deep breathing, or stooping.

9. TRIGGER POINT EXAMINATION

Sternalis TrPs are found by systematic palpation against the underlying sternum and costal cartilages. Firm pressure elicits focal deep tenderness at the TrP and projection of referred pain, but rarely elicits a local twitch response. On examination, the patient has difficulty in distinguishing between the local and the referred pain that is elicited from this muscle, unless the pain radiates not only to the sternum, but also to the shoulder or arm. Referred pain responses due to needle penetration of the TrP are more clearly distinguishable. Sternalis central TrPs are most commonly found to the left of the midline at the midsternal level.[18, 19] Attachment TrPs sometimes also are found close to the attachment region at an end of the muscle belly.

10. ENTRAPMENT

None are attributed to this muscle.

11. DIFFERENTIAL DIAGNOSIS

When multiple areas of spot tenderness are found over the costochondral junctions without the referred pain feature of sternalis TrPs, the examiner should consider costochondritis or Tietze's syndrome.[9] This syndrome is identified by upper anterior chest pain with tender, nonsuppurative swelling in the area of the costal cartilages or the sternoclavicular junctions. Multiple lesions are more frequent than single lesions and usually involve adjacent articulations. Also, in Tietze's syndrome, systemic manifestations are absent and radiographic and laboratory studies are normal, except for occasional reports of increased calcification at affected sites.[9] The importance of distinguishing between chest pain of cardiac origin and that of chest wall origin has recently been emphasized.[8]

In addition to costochondritis and cardiac disease, the clinician should consider gastroesophageal reflux, esophagitis, and an anginal presentation of a C_7 radiculopathy. On the other hand, a mistaken diagnosis of one of these conditions is made when the symptoms arise from sternalis TrPs.

Related Trigger Points

One rarely observes sternalis TrPs alone, without the presence of active TrPs in the pectoralis major muscle. The possibility that a sternalis TrP represents a satellite of a distant key TrP makes it important to examine the lower portion of the sternal division of the sternocleidomastoid muscle, which may refer pain downward over the sternum.

12. TRIGGER POINT RELEASE

Stretch of the sternalis muscle is not practical except for myofascial release, however, application of vapocoolant spray is occasionally effective in the treatment of these myofascial trigger points (TrPs). Application in a crisscross pattern while the patient holds a deep breath[17] has been the most successful spray technique for TrPs in this muscle. The sternalis TrPs are responsive, however, to **trigger point pressure release** against the underlying bone, and the TrPs are easily injected. Deep friction massage applied to the muscle fibers in the region of the TrP is also beneficial.

Local treatment of the sternalis myofascial pain syndrome is not complete until active TrPs in the pectoralis major, or in the lower end of the sternal division of the sternocleidomastoid muscle, have been inactivated often by trigger point release (*see*

Chapters 42 and 7, respectively). The patient is less likely to experience recurrence of pain due to TrPs in the sternalis muscle if these other two muscles are released prophylactically, even though they contain only latent TrPs which are clinically silent with respect to pain.

Relief of sternal pain by the spray does not rule out a cardiac etiology of the pain.[11,14]

13. TRIGGER POINT INJECTION

A trigger point (TrP) in the sternalis is identified by flat palpation and is then fixed between two fingers, probed, and precisely infiltrated. When a sternalis TrP is encountered by the tip of the needle, the patient reports projection of pain under the sternum and sometimes across the upper pectoral region and down the ulnar aspect of the arm as far as the elbow. Injection has not usually been observed to induce a local twitch response in this muscle.

Both sides of the sternum must be checked for sternalis TrPs. During injection, TrPs on the front of the sternum may be found as deep as 2 cm (¾ in) beneath the skin surface. Such deep TrPs may be attachment TrPs of the pectoralis major, rather than in sternalis fibers. This possibility is strengthened by the sensation that the needle sometimes penetrates two layers of muscle, a superficial and then a deeper one, either or both of which may contain TrPs. Rachlin[10] illustrated injection of this muscle.

Moist heat is applied promptly after injection of TrPs. This muscle cannot be stretched except by massage.

14. CORRECTIVE ACTIONS

The patient should learn to perform trigger point pressure release on his or her own sternalis TrPs, followed by application of moist heat. The patient selects a tender spot and presses on it steadily with one finger to the point of discomfort and holds it until it fully releases. This release is assisted by slow relaxed exhalation. When the previously tender spot of muscle at the TrP becomes normosensitive, it is no longer a source of referred pain. It may remain quiescent indefinitely, unless the TrP is reactivated, as by recurring angina pectoris.[16]

REFERENCES

1. Barlow RN: The sternalis muscle in American whites and Negroes. *AnatRec* 61:413-426, 1935.
2. Bonica JJ, Sola AE: Chest pain caused by other disorders. Chapter 58. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990, pp. 1114-1145.
3. Christian HA: Two instances in which the musculus sternalis existed—one associated with other anomalies. *Bull Johns Hopkins Hosp* 9:235-240, 1898.
4. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer Verlag, Jena, 1912 (pp. 470-475, Figs. 70, 72).
5. Epstein SE, Gerber LH, Borer JS: Chest wall syndrome, a common cause of unexplained cardiac pain. *JAMA* 241:2793-2797, 1979.
6. Gasser HS, Erlanger J: The role of fiber size in the establishment of a nerve block by pressure or cocaine. *Am J Physiol* 88:581-591, 1929.
7. Grant JC: *An Atlas of Human Anatomy.* Ed. 7. Edited by Anderson JE. Williams & Wilkins, Baltimore, 1978 (Fig. 6-120B).
8. Hollinshead WH: *Anatomy for Surgeons.* Ed. 3, Vol. 1, *The Head and Neck.* Harper & Row, Hagerstown, 1982 (p. 281, Fig. 4-19).
9. Levey GS, Calabro JJ: Tietze's Syndrome: Report of two cases and review of the literature. *Arthritis Rheum* 5:261-269, 1962.
10. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES Mosby, St. Louis, 1994, pp. 197-360 (p. 221).
11. Rinzler SH: *Cardiac Pain.* Charles C. Thomas, Springfield, Ill., 1951 (pp. 80, 81).
12. Shen CL, Chien CH, Lee SH: A Taiwanese with a pair of sternalis muscles. *Kaibogaku Zasshi. J Anat* 67(5):652-654, 1992.
13. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul, Vol. 1. Macmillan Company, New York, 1919 (p. 282).
14. Travell J: Early relief of chest pain by ethyl chloride spray in acute coronary thrombosis. *Circulation* 111.120-124, 1951.
15. Travell J: Pain mechanisms in connective tissue. In: *Connective Tissues, Transactions of the Second Conference, 1951.* Edited by Ragan C. Josiah Macy, Jr. Foundation, New York, 1952 (pp. 86-125).
16. Travell J, Rinzler SH: Therapy directed at the somatic component of cardiac pain. *Am Heart J* 35:248-268, 1958.
17. Travell J, Rinzler SH: Pain syndromes of the chest muscles: Resemblance to effort angina and myocardial infarction, and relief by local block. *Can Med Assoc J* 59:333-338, 1948 (Cases 2 and 3).
18. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 11:425-434, 1952 (p. 429).
19. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J Am Osteopath Assoc* 72:697-710, 1973 (pp. 10, 12; Fig. 32).
20. Zohn DA, Mennell JM: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little, Brown & Co., Boston, 1988 (p. 212, Fig. 12-4).

CHAPTER 45
Intercostal Muscles and the Diaphragm

HIGHLIGHTS: **REFERRED PAIN** from myofascial trigger points (TrPs) in the *intercostal muscles* is primarily local in the region of the TrP, tending to extend anteriorly when severe. Pain is referred from the *diaphragm* in two different patterns mediated by two neural pathways. It is referred to the upper border of the ipsilateral shoulder near the angle of the neck, or to the region of the costal margin. **ANATOMY:** the external and internal intercostal muscles are located between adjacent ribs and form a crisscross pattern. The central tendon of the dome-shaped diaphragm separates the thoracic and abdominal cavities. Its central tendon is surrounded by muscle fibers that are attached to the inferior thoracic outlet peripherally. **FUNCTION** of the *diaphragm* is inhalation. Function of the *intercostal muscles* is both postural and respiratory. The interosseous intercostal muscles are mechanically well suited for, and are electrically active during rotation of the thoracic spine. During normal *quiet respiration* the activity of the interosseous intercostals is minimal during exhalation. The driving force is supplied primarily by the elasticity of the lungs and chest. During quiet inhalation, the diaphragm, the scalene muscles, some of the upper and more lateral external intercostals, and the parasternal internal intercostals become active. With increasingly forced inhalation, successively more caudal external intercostals are recruited and for a longer period. During forced exhalation, when intercostal activity is present, the recruitment is progressively upward from the lowest intercostals to the highest. **SYMPTOMS** of intercostal TrPs are restricted rotation of the thoracic spine when twisting to look behind and chest pain that is increased by deep respiration, especially coughing or sneezing. Shortness of breath can be a symptom of diaphragmatic TrPs. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS:** Trauma, surgery, or coughing can activate TrPs in the intercostal muscles. Chronic cough and paradoxical breathing will perpetuate TrPs in the diaphragm and the intercostal muscles. **PATIENT EXAMINATION** begins by testing for restricted rotation of the thoracic spine and for painful deep inhalation caused by intercostal TrPs, and proceeds by testing for painful full exhalation caused by diaphragmatic TrPs. Thoracic side bending to the side away from the intercostal TrPs tends to be painful. **TRIGGER POINT EXAMINATION** for *intercostal* TrPs starts with examination of the painful segment for narrowed rib space and continues with palpation along the full length of a suspected rib space for tenderness. *Diaphragmatic* TrPs are not directly palpable and tenderness of attachment TrPs inside the costal margin is hard to distinguish from transversus abdominis muscle tenderness. **DIFFERENTIAL DIAGNOSIS** of lower rib articular dysfunctions, intercostal muscle spasm, and costochondritis should include consideration of TrPs as another cause of the symptoms. On the other hand, myocardial infarction, tumor, pleural effusion and pyothorax need to be ruled out. Patients with herpes zoster are prone to develop intercostal TrPs that can contribute significantly to pain and are treatable. Presumptive diagnoses of diaphragmatic spasm, undiagnosed atypical chest pain, and negative studies for symptoms of peptic ulcer or gallbladder disease should include myofascial TrPs of the diaphragm in the differential diagnosis. **TRIGGER POINT RELEASE** of *intercostal muscles* can be achieved by direct manual methods that involve digital contact with the TrPs, by methods which stretch the tense muscles, or by indirect techniques using a position of ease. Release of *diaphragmatic* TrPs requires stretching the diaphragm, which occurs at the end of exhalation and is enhanced by voluntarily contracting the abdominal muscles and/or by pressing on the abdomen. **TRIGGER POINT**

INJECTION of *intercostal muscles* can be effective with proper precautions to prevent pneumothorax, but should be attempted only by those who have already become very skillful in the injection of TrPs. Injection of the *diaphragm* is usually unnecessary, extremely dangerous, requires an unusual combination of skills, and probably is not very effective because only attachment TrPs are within reach. **CORRECTIVE ACTIONS** start with identifying and correcting paradoxical respiration which is very common in this group of patients and can be responsible for TrPs. It is unlikely that lasting relief from TrPs of primary respiratory muscles, and from TrPs in any active accessory muscles of respiration, can be realized until normal coordinated respiration has been restored. Correction of a head-forward, slumped posture is necessary.

1. REFERRED PAIN (Fig. 45.1)

Intercostal Muscles

Trigger points (TrPs) in intercostal muscles refer pain locally in the region of the TrP and tend to refer pain along that interspace around toward the front, away from the vertebral column rather than toward the back (Fig. 45.1). The more posteriorly the TrP is located, the stronger is its tendency to refer pain toward the front. More severe TrPs may refer pain that includes interspaces above and below the TrP.

Bonica and Sola[8] illustrated a similar local intercostal pain pattern around the TrP.

Diaphragm

During vigorous exercise, diaphragmatic TrPs can produce the pain commonly described as a "stitch in the side" that is felt deep anterolaterally in the region of the lower border of the rib cage. The pain tends to be aggravated by continued exercise and relieved by rest.

Pain arising from stimulation of the central dome portion of the diaphragm can be referred to the upper border of the ipsilateral shoulder. Stimulation of the peripheral part is referred as an aching pain to the region of the adjacent costal margin. The difference in pain distribution depends on the innervation of the stimulated site.[25]

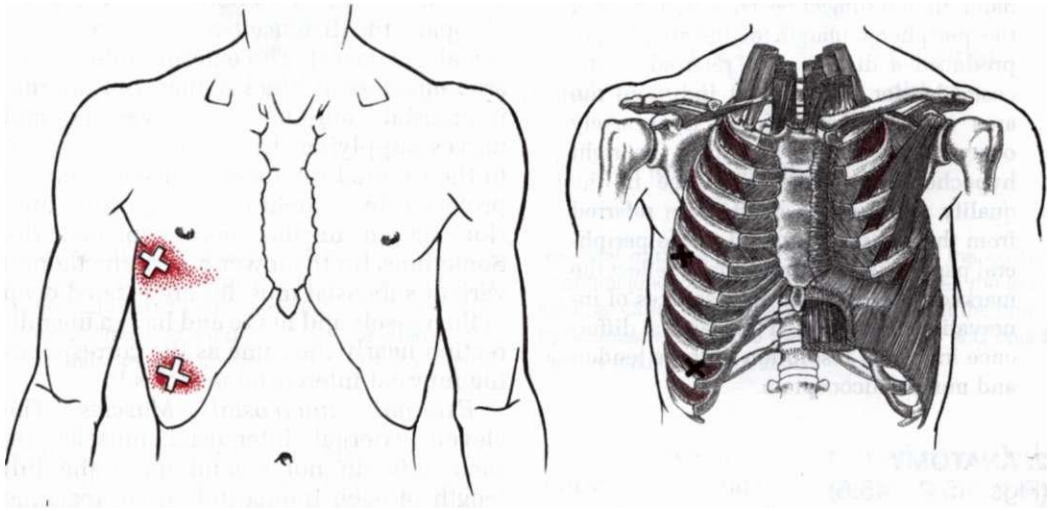


Figure 45.1. Examples of referred pain patterns (*dark red)* of myofascial trigger points (**Xs**) in intercostal muscles *(light red).* The center of the ***X*** locates the trigger point, which can occur in any intercostal muscle. The more dorsally the trigger point is located, the farther the pain pattern tends to extend toward the sternum. Patterns tend to follow the curvature of the ribs. When TrPs are very active, the pain may spread over several segments.

Among a series of 17 patients complaining of chest pain and dyspnea attributed to spasm of the diaphragm,[51] 9 complained of pain in the substernal region and 8 located their pain in or near the right hypochondrial region, which suggests that the location of the pain also identified the nerve supply and identified from which part of the diaphragm the pain originated. This principle may also apply to pain referred from diaphragmatic TrPs.

> Fields[25] called attention to experiments of Capps[12] that involved direct stimulation of the peritoneal (caudal) surface of the diaphragm with a smooth bead or the rough end of a wire. In 3 subjects, stimulation of the central portion of the diaphragm with the bead caused a sharply localized pain that was illustrated as referring to the middle of the region of the anterior border of the upper trapezius muscle about half way between the acromion and the base of the neck. Stimulation with the rough end of the wire produced pain of great intensity at the same location. One subject described the sensation as, "the wire sticking into my neck," and could point to the precise spot with a fingertip. When pressed, that spot was abnormally tender. On the other hand, in one subject tested, stimulation of the peripheral margin of the diaphragm produced a diffuse pain referred to the costal border. The patient indicated the area with his hand placed transversely over the lower ribs and over the right hypochondrium. The difference in the quality and location of the pain referred from the central compared to the peripheral parts of the diaphragm may reflect the marked differences in their sources of innervation *(see* Section 3)., and a difference in spatial resolution of these tendon and muscle nicoceptors.

2. ANATOMY
(Figs. 45.2 - 45.6)

The highly complex nature of mammalian motor nerve terminals and endplates is well illustrated for the diaphragm muscle of the rat.[42] The size and complexity of both the nerve terminals and endplates increase progressively from type I fibers through type IIa to type IIb fibers as they depend more strongly on oxidative metabolism for their energy supply. In one study, the distribution of fiber types in the human diaphragm was 42% type I, slow twitch, fibers and 58% type II, fast twitch, fibers.[9]

The number of muscle spindles per gram of respiratory muscle corresponds strongly to muscles characterized by sustained tonic (postural) activity rather than intermittent phasic (respiratory) activity and to muscles composed largely of type I fibers rather than type II fibers.[22] The diaphragm of the cat has practically no muscle spindles and the interchondral muscles have very few. The external intercostals have more spindles than the internal intercostal muscles, and those muscles in the first seven spaces have a higher density of spindles than those in the intercostal muscles in the last five spaces.[22]

Intercostal Muscles
(Figs.45.2 - 45.5)

The external and internal intercostal muscles have a crisscross arrangement, crossing each other at nearly a right angle, similar to the external and internal abdominal oblique muscles *(see* Chapter 49), and in the same directions. This is the type of arrangement that has been used in successive plies of an automobile tire. Each muscle spans the distance between two ribs (or costal cartilages). The external intercostals are considerably thicker than the internal intercostal muscles. The vessels and nerves supplying these muscles run deep to the internal intercostal muscles and are protected by a slight overhang of the inferior margin of the more cephalad rib. Sometimes (in the lower part of the thorax) variant subcostal muscles are located deep to the vessels and nerve and have a fiber direction nearly the same as the corresponding internal intercostal muscles.[15]

External Intercostal Muscles. The eleven external intercostal muscles on each side do not extend quite the full length of each intercostal space, reaching only to the costal cartilage anteriorly, except between the lowest ribs (Fig. 45.2). They do reach the end of the rib posteriorly at the tubercle (Fig. 45.3). Anteriorly, the external intercostal has only a fascial ex-

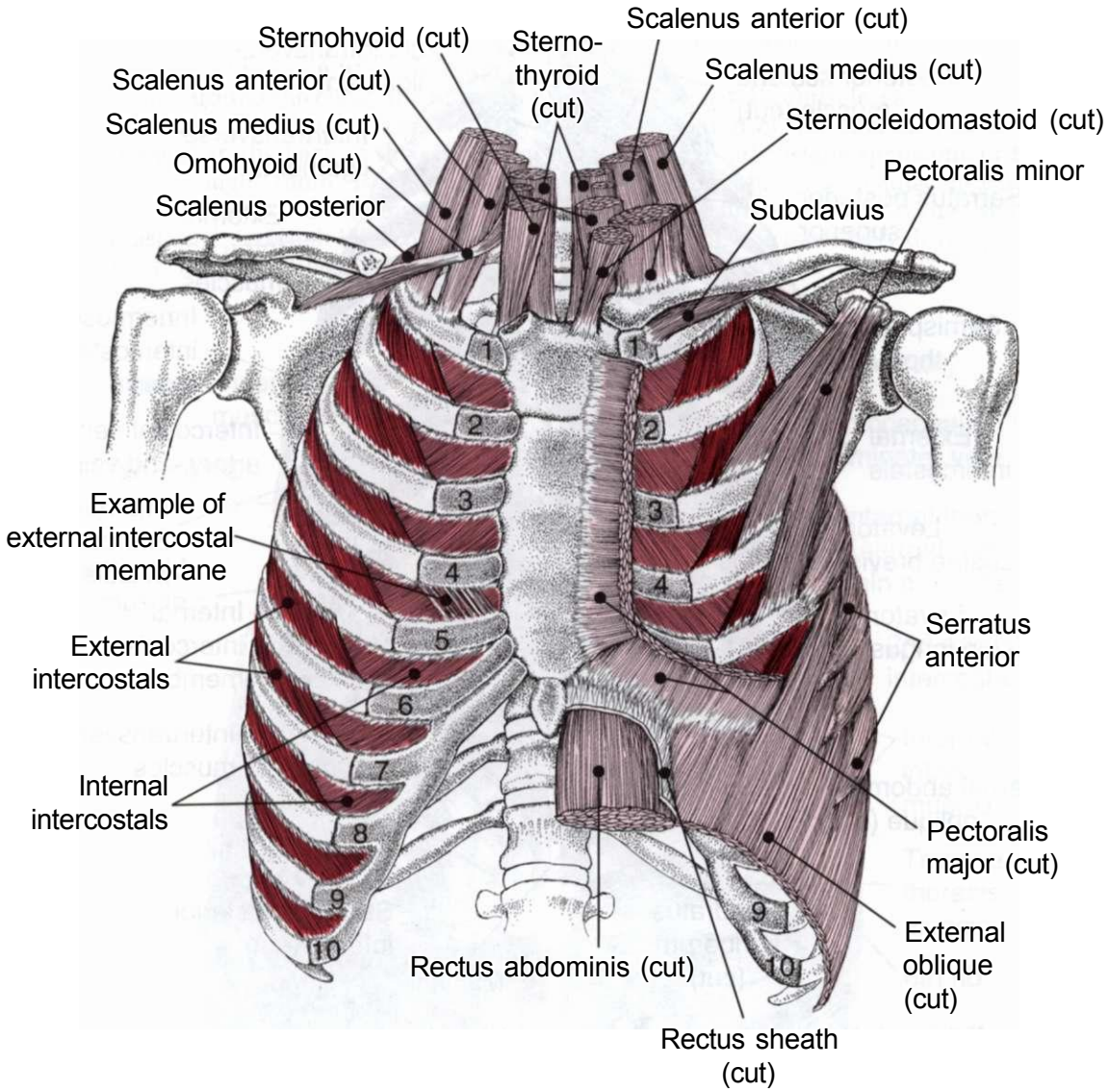


Figure 45.2. Exterior of anterior thoracic wall, showing the anatomical relations and attachments of intercostal and related respiratory muscles. The external intercostal muscles are *darkest red*, the internal intercostal muscles are *intermediate red.* The external intercostal muscles do not extend beyond the costochondral junctions medially, except between the lowest ribs. Other muscles are *light red.* All but the omohyoid muscle attach to the thoracic cage and could directly influence respiration.

tension, the external intercostal membrane, that reaches to the sternum. The external fibers are angled obliquely inferomedially as seen from in front (see Fig. 45.2 and 45.9) and obliquely inferolaterally as seen from behind (Fig. 45.3). Figure 49.3 provides a convenient way of remembering the direction of each muscle.[15]

The twelve posterior, extra-thoracic **levator costae (costarum)** muscles can be considered an extrathoracic nonintercostal version of the external intercostal muscles (Fig.45.3 *left side).* They attach **above** to the ends of transverse processes and attach **below** and more laterally to the adjacent rib (levator costae brevis) between the rib's

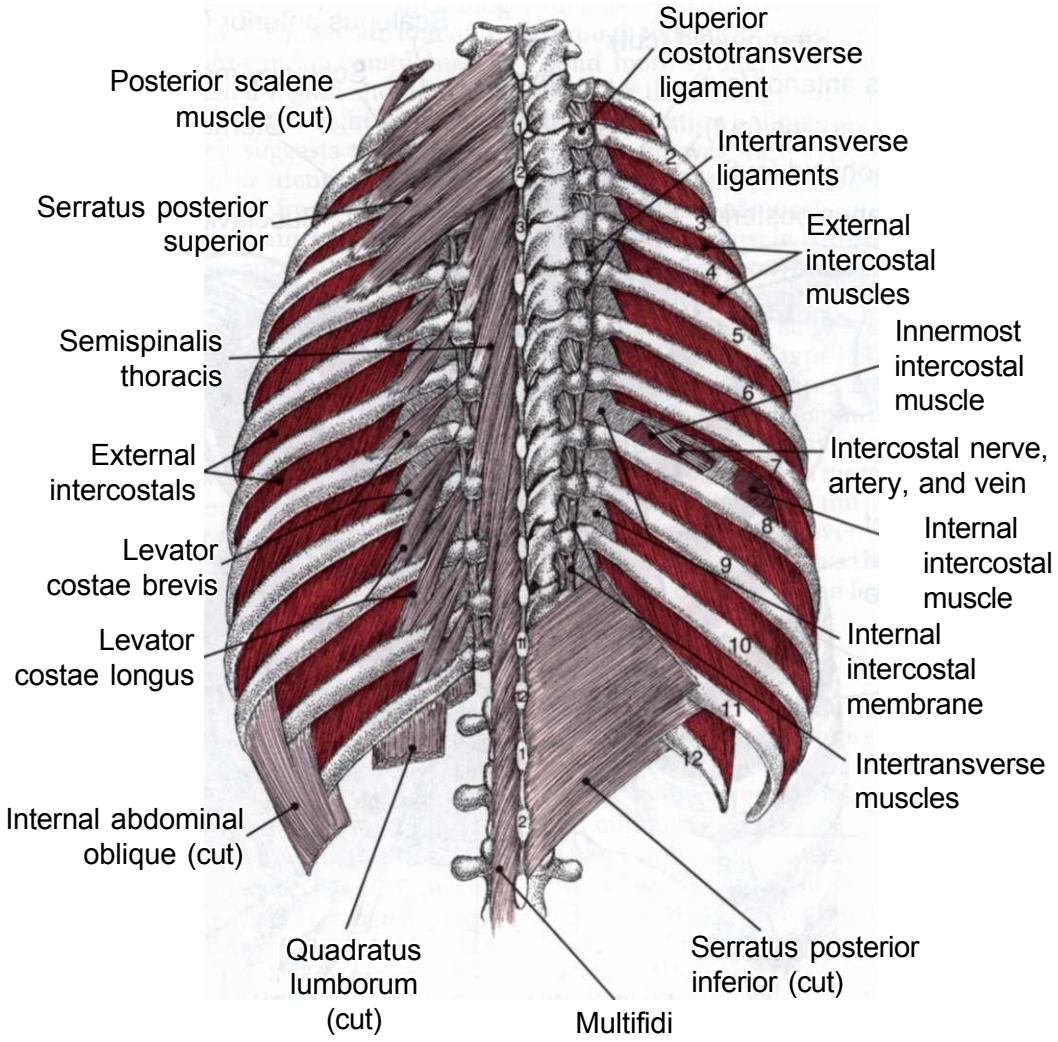


Figure 45.3. Exterior of posterior thoracic wall, showing anatomical relations and attachments of intercostal and related respiratory muscles. The external intercostal muscles are *dark red*, the internal intercostal muscles are *intermediate red*. Other muscles are *light red*. The posterior scalene (cut), external intercostal, and the levator costae longus and brevis are primary muscles for inhalation that appear in this figure. The serratus posterior superior muscles help to elevate the ribs during forced inhalation. The serratus posterior inferior (cut), quadratus lumborum (cut), and internal abdominal oblique (cut) muscles shown here may assist exhalation. The detailed drawing between ribs 7 and 8 on the right side shows that the internal intercostal muscles are absent medial to the region of the angle of the ribs, but are represented medially as the internal intercostal membrane. The neurovascular bundle runs between the internal intercostal muscle or membrane, which lies superficial to it, and the innermost intercostal muscle or membrane which lies deep to it. The internal intercostal and innermost intercostal muscles have an almost identical fiber direction and are usually referred to collectively as the internal intercostal muscle. The intercostal neurovascular bundle actually lies deep to the lower border of the cephalad rib and might not be visible from this view.

tubercle and its angle, or span one rib (levator costae longus).

Internal Intercostal Muscles. The eleven internal intercostal muscles on each side are incomplete posteriorly (Fig. 45.5) extending from near the sternum anteriorly to only the angles of the ribs posteriorly, where thin aponeuroses, the internal intercostal membranes, extend to the vertebral column. The internal intercostal fiber direction is the reverse of the direction of the external fibers; the internal fibers are an-

gled obliquely inferolaterally in the front of the chest (Figs. 45.2, 45.4 and 45.9). Since the muscle has the same fiber direction as it continues around the chest, the fibers appear to be angled obliquely inferomedially when viewed in the back of the chest (Fig 45.5).[15] Although there is no obvious anatomical difference in the muscle itself, the parasternal internal intercostal fibers that attach to the cartilaginous part of the ribs have a distinctly different function than the interosseous internal intercostal muscles.

The **subcostalis** muscle can be considered a variation of the internal intercostal muscles. The subcostalis spans one or two ribs instead of attaching to adjacent ribs. It has the same fiber direction as the internal intercostals, and is most fully developed in the lower part of the thorax.[15] The sub-

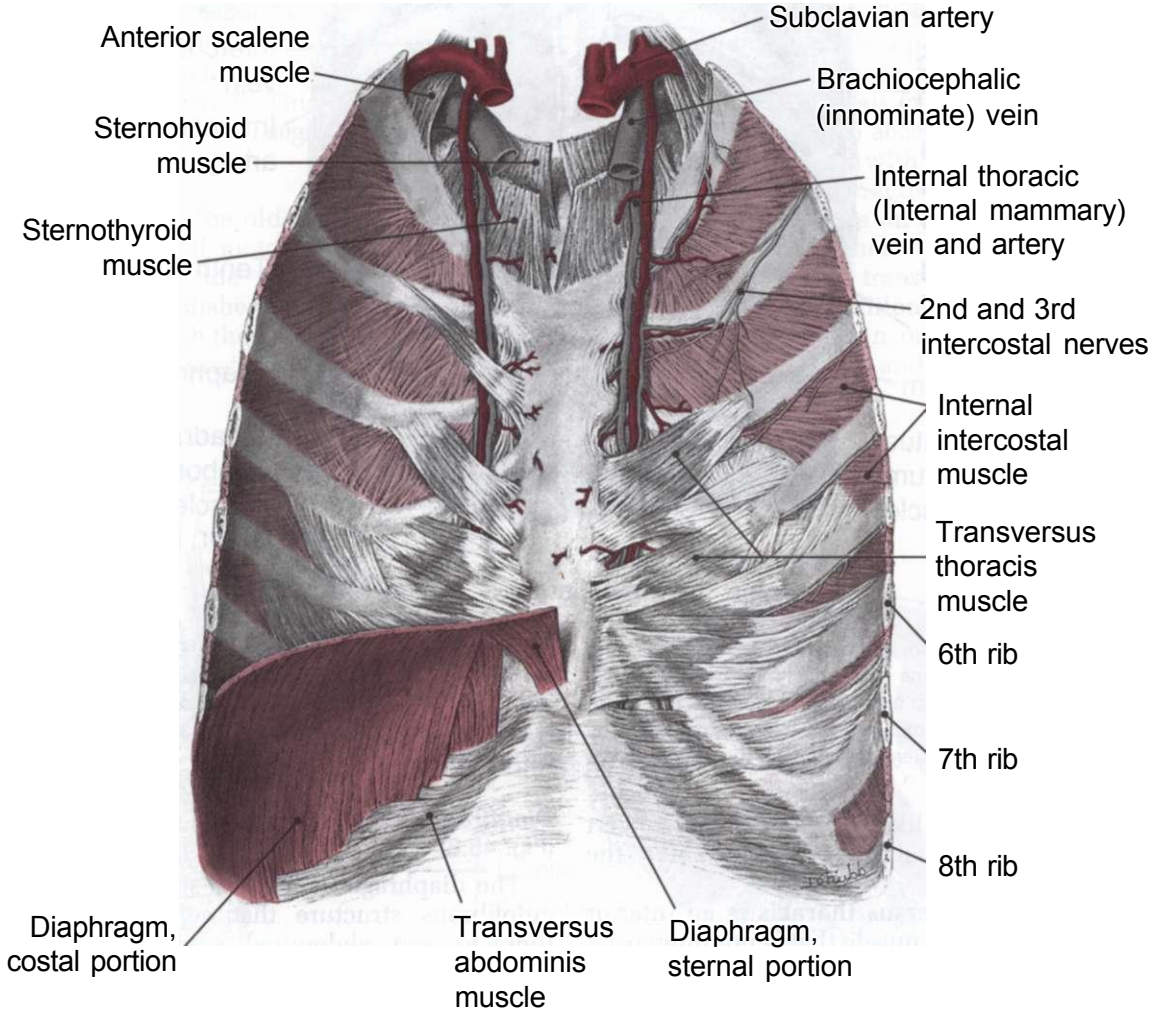


Figure 45.4. Interior of anterior thoracic wall. The subclavian and internal thoracic arteries are *darkest red*, the diaphragm (shown in part and only on the left side) is *dark red*, the internal intercostal muscles are *intermediate red*, and the remaining muscles are *light red*. Note that, generally, only the internal intercostal muscles continue anteriorly as far medially as the sternum (completing coverage of the anterior costal interspaces). The external intercostal muscles (not seen in this view) stop short at the costochondral junctions. The diaphragm is a primary muscle for inhalation. Note how it extends downward to lie against the lowest rib. (Reproduced and adapted with permission from Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991.)

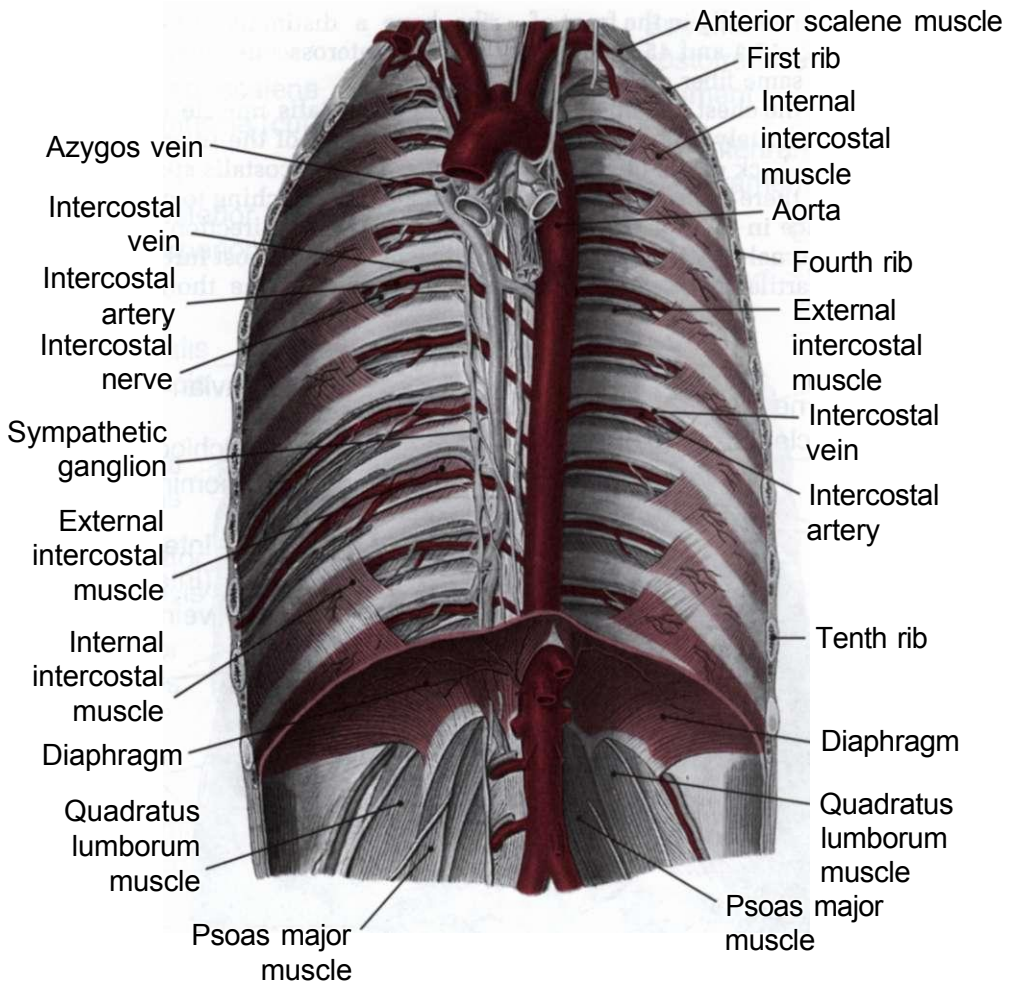


Figure 45.5. Interior of posterior chest wall showing anatomical relations and attachments of the intercostal muscles and also major blood vessels. The internal intercostal muscles are *intermediate red.* The diaphragm and external intercostal muscles are *dark red*, and the arteries are *darkest red.* Other muscles are *light red.* (Reproduced and adapted with permission from Ferner H, Staubesand J: *Sobotta Atlas of Human Anatomy*, Vol. 2. Urban & Schwarzenberg, Munich 1983.)

costalis very likely functions in concert with the internal intercostal muscles of the lower thorax.

The **transversus thoracis** is an interior anterior chest muscle that is not intercostal (Figure 45.4). It lies deep to the sternum and the parasternal intercostal muscles and is composed of tendinous and muscular fibers that attach in a fan-like arrangement. The upper digitations of the muscle reach from the inner surface of the lower sternum and xiphoid process upward (craniad) to the costal cartilages of the second to sixth ribs. The lowest fibers are essentially horizontal.[15]

Diaphragm
(Fig. 45.6)

The diaphragm is a dome-shaped musculofibrous structure that separates the thoracic and abdominal cavities (Figs. 45.4-45.6). The dome of the diaphragm is a central tendon surrounded by muscle fibers that form an extended "skirt" which attaches peripherally to the circumference of the inferior thoracic outlet.[16] The muscle is divided into a sternal portion anteriorly that attaches to the sternum, a costal portion laterally that attaches to the costal margin, and a lumbar portion posteriorly that attaches by two muscular crura to the bod-

ies of the upper lumbar vertebrae. The lumbar portion also attaches to two bilateral arcuate ligaments which span from the vertebrae to the transverse processes and from those processes to the 12th rib (Fig. 45.6).

The diaphragm is penetrated by the aorta, vena cava, and esophagus. The arcuate ligaments provide passage posteriorly for the psoas major and quadratus lumborum muscles (Figs. 45.5 and 45.6).

3. INNERVATION

Intercostal Muscles

Each intercostal muscle is supplied by several branches of the corresponding intercostal nerve.[15] This arrangement is a classic example of segmental innervation.

Diaphragm

Although the older literature suggested an intercostal motor innervation of some portions of the diaphragm,[3] it is now clearly established that its only *motor* supply is through the phrenic nerves, which in humans originate in the third, fourth and fifth cervical segments.[19] The older literature is probably correct with regard to sensory innervation.

4. FUNCTION (Figs. 45.7-45.9)

Recent studies have helped clarify much of the controversy associated with the activity and role of several respiratory muscles. To understand their function it is helpful to remember basic respiratory mechanics. Inhalation is an active process requiring muscular effort. Exhalation during quiet breathing is largely a passive process performed by the elastic recoil of the lungs.[37] In that sense, all expiratory muscles are to some degree accessory muscles of respiration recruited with increased respiratory demand. The function of the intercostal muscles depends on their internal-external position, on their anteroposterior position, and on their transverse location on the rib cage. In addition, the muscle's superior-inferior position on the rib cage affects the relative order and magnitude of

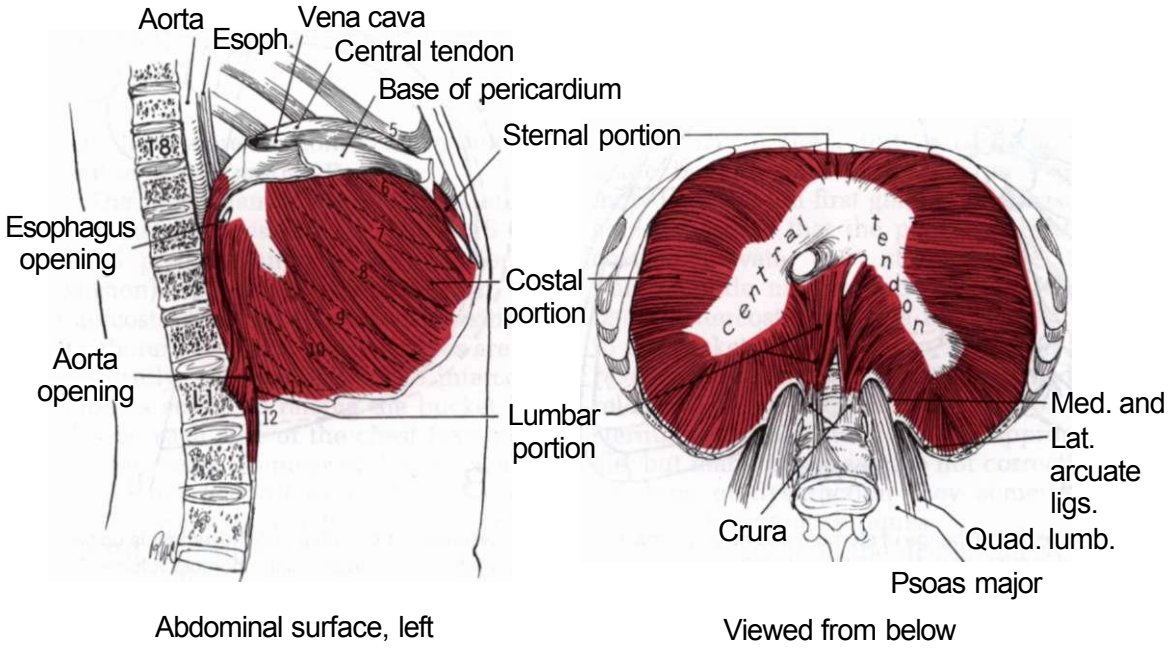


Figure 45.6. Caudal (abdominal) surface of diaphragm muscle *(red)*, which is the most important muscle for inhalation. **A,** internal aspect of left hemidiaphragm as seen from the right side of body; **B,** diaphragm viewed from below showing its attachment to the caudal margins of the thoracic cage. (Reproduced with permission from Kendall FR McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993.)

recruitment. It is understandable that there has been much confusion produced by numerous contradictory reports that did not consider these distinctions. The accessory muscles of respiration that participate in forced breathing are considered in Section 5 of this chapter.

It is of fundamental importance that the intercostal muscles are tailor-made for rotating the thoracic spine, a function that is frequently overlooked. It is true, however, that respiratory demand takes precedence over postural activity.

Respiratory Mechanics (Figs 45.7-45.9)

Movement of the chest wall during inhalation is a complex integrated process that requires sophisticated coordination of numerous muscles. Lung volume is controlled by three basic movements. Figure 45.7 illustrates two of the movements: (1) *elevation of the sternum* (Fig. 45.7A) that increases the anteroposterior diameter by rotating the ribs around the spinal attachments, and (2) *spreading of the lower ribs* (Fig. 45.7B) that increases the lateral diameter of the thorax by rotating the ribs around their sternal attachments.[35] The downward piston-like *motion of the diaphragm* provides the third (Fig. 45.8). The sternal elevation movement is often compared to that of an old-fashioned pump handle, and the lateral rib movement to that of a bucket handle (one on each side).

The axis of rotation of a rib is defined by its articulations with the vertebral body and the transverse process. Since most ribs are inclined obliquely approximately 45° to the horizontal, when the rib rotates upward it increases the volume within the rib cage, which is associated with inhalation. The upper ribs that are attached to the sternum with short costal cartilages tend to move in unison, whereas the lower ribs that are attached with longer costal carti-

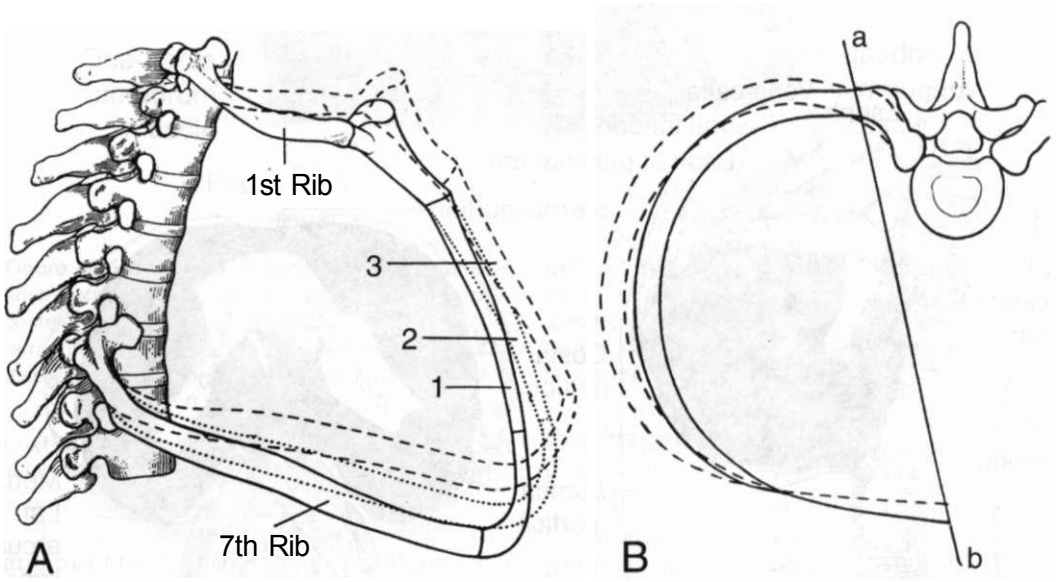


Figure 45.7. Change of sternum and rib positions with inhalation. **A,** lateral view of chest showing the upward and outward (forward) movement of the anterior rib cage during inhalation, which increases intrathoracic volume. This can be compared to a "pump-handle" movement. *Position 1*, ordinary exhalation; *position 2 (dotted lines),quiet inhalation; position 3 (dashed lines)* deep inhalation. **B,** View from above showing how, for ribs attaching to the costal cartilages below the sternum (vertebrochondral ribs), the movement is upward and lateral, which increases intrathoracic volume. The *dashed lines* represent the position of the rib during inhalation. The line labeled *a-b* represents the axis of movement. This upward and lateral rib movement can be compared on each side to the movement of a bucket handle. (Reprinted with permission from Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985.)

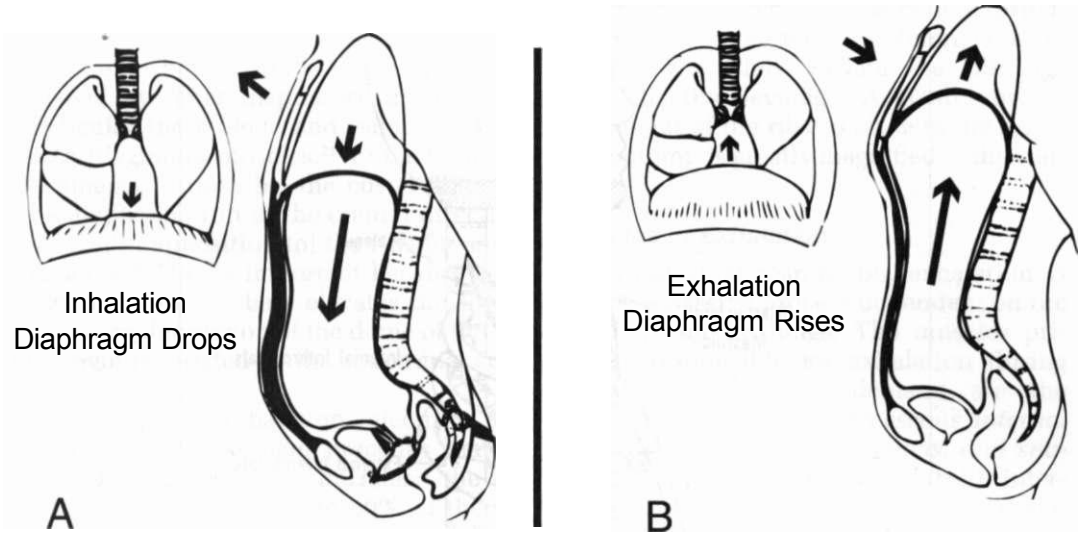


Figure 45.8. Schematic of respiration dynamics. **A. Inhalation.** The sagittal section *(right figure* of *A)* shows how the combination of depression (contraction) of the diaphragm *(long down arrow)* that is displacing the abdominal contents downward and the simultaneous expansion of the thoracic cage *(diagonal up arrow)* reduce intrathoracic pressure. This sucks air into the lungs *(short down arrow)*, inflating them. The frontal section *(left figure* in *A)* shows the depressed diaphragm and inflated lungs. **B. Exhalation.** The sagittal section *(right figure* in **B)** shows how depression of the thoracic cage *(diagonal down arrow)* and elevation (relaxation) of the diaphragm *(long up arrow)* tend to increase intrathoracic pressure. During quiet respiration the elastic recoil of the lungs and chest forces air out of the lungs *(short up arrow)*, deflating them. The frontal section *(left figure)* shows the elevated diaphragm and deflated lungs. In forced exhalation, the abdominal muscles displace the abdominal contents inward and upward and pull the thoracic cage downward and inward, accelerating airflow out of the lungs.

lages have more freedom to move independent of sternal motion.[18]

The pump-handle movement of inhalation that elevates the sternum (and produces predominantly anteroposterior expansion) depends primarily on the intercostal muscles located at the *sides* of the thorax for which these muscles are mechanically well situated.[35] The intercostal muscles suited to raising the bucket handles on each side of the chest (expanding the transverse diameter of the rib cage) are located *nearly midline* close to the sternum and the spine, especially the parasternally located internal intercostals and the paraspinally located levator costae. These relationships were identified by calculations using three dimensional finite element analysis of the human rib cage[35] and were confirmed experimentally in dogs.[30]

The discrepancy between the location of the muscles and their effect on rib motion is contrary to the intuitive assumptions made by clinicians and sometimes promoted as fact. On first glance, it is reasonable to assume that the parasternal intercostals elevate the sternum in the pump-handle motion and that the lower lateral intercostals elevate the lateral ribs in the bucket-handle motion. However, *the reverse is true.* The techniques described to release tight muscles in the region of the sternum or lower lateral ribs are appropriate, but many clinicians are not correcting the type of dysfunction they sometimes claim with these techniques.

The depression of the diaphragm by its activity during inhalation and its passive elevation during exhalation are illustrated in the sagittal sections in Figure 45.8. The corresponding effect on lung volume is shown in the frontal sections in Figure 45.8. Contraction of the diaphragm tends to elevate and spread the lower costal margin

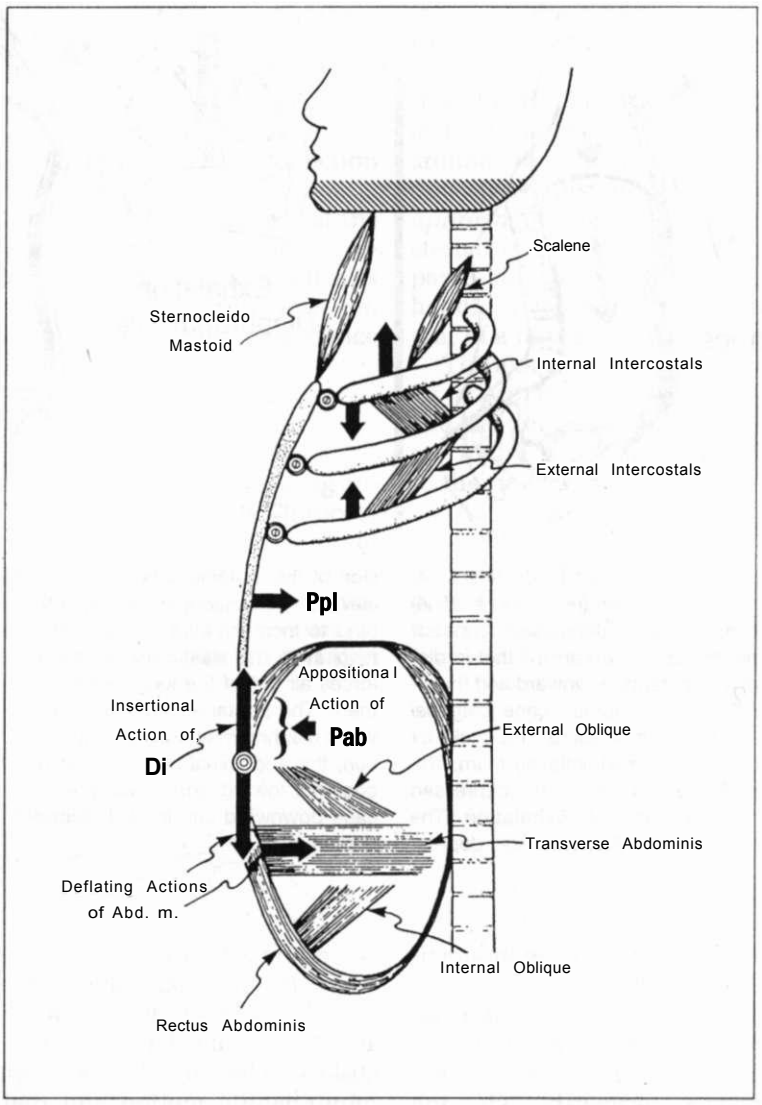


Figure 45.9. Schematic of respiratory mechanics illustrating some of the most important respiratory muscles and their actions, *(thick black arrows). Abd. m.* = abdominal muscles, *Di* = diaphragm, *Pab* = abdominal pressure, *Ppl* = pleural pressure. (Reprinted with permission from Roussos C: Function and fatigue of the respiratory muscle. *Chest* 88(Suppl):124s-132s, 1985.)

and lower ribs when support and resistance are supplied to the central tendon by the abdominal contents.[39]

The muscles largely responsible for these movements are illustrated in Figure 45.9 in greatly simplified form with arrows that indicate the force vector produced by contraction of the muscle.

Muscles of Inhalation

The muscles primarily responsible for inhalation are the *diaphragm, parasternal (intercartilaginous) internal intercostals, scaleni, upper and more lateral external intercostals, and the levator costae* muscles. The diaphragm, which is the main respiratory muscle in humans, does not ex-

pand the entire chest wall, but just the abdomen and lower rib cage. Expansion of the cranial half of the rib cage is accomplished by other inspiratory muscles, in particular the scalene and parasternal intercartilaginous muscles.[21] From their attachments to the ribs, the costal fibers of the diaphragm run in the cranial direction next to (in apposition to) the ribs for some distance.[10] This is important because contraction of these fibers elevates the lower ribs when depression of the dome of the diaphragm is resisted by the abdominal contents.[17, 20]

During quiet inhalation, electrical activity of the diaphragm precedes that of the external intercostal muscles;[4] the diaphragm produces 70% to 80% of the inhalation force.[37] This is why paradoxical breathing is such a serious dysfunction. Hyperinflation of the lungs due to obstructive lung disease puts the diaphragm at a serious disadvantage, and under some circumstances the flattened diaphragm can reverse its effect by pulling the costal margin in rather than lifting it up and out.[30]

During quiet breathing, the first external intercostals (between the first and second ribs) are always active, the second pair of muscles are usually active, and the third, occasionally. With increasingly more forced respiration, successively more caudal external intercostal muscles are recruited during inhalation.[4]

The scalene muscles (anterior scalene monitored) are always active in quiet inhalation (Fig. 45.9) and are likely to activate shortly before the parasternal internal intercostal (chondral, not interosseous) muscles start to contract.[4] The activity of the scalene muscles is needed during inhalation to prevent the downward suction produced by the piston action of the diaphragm from pulling the sternum down and in. A downward motion of the sternum tends to reduce intrathoracic volume rather than increase it. The scalene muscles respond increasingly vigorously to increasing respiratory effort.[4]

The posteriorly located levator costarum muscles (Fig. 45.3), which also show some activity in quiet respiration,[4] become increasingly active with increasing ventilatory demand.[30] They are anchored proximally to the vertebral column, not to another rib. They elevate the rib cage with effective leverage. A small upward movement of the ribs so close to the vertebral column is greatly magnified at the sternum.

Muscles of Exhalation

During quiet respiration, exhalation is largely a passive process dependent on the elasticity of the lungs. The muscles primarily responsible for exhalation during periods of increased demand are the *abdominal muscles, interosseous internal intercostal, transversus thoracis, and subcostal muscles.* The lowest (11th) intercostal pair is most important for exhalation, and an EMG study showed that, as intercostal activity developed during forced exhalation, recruitment progressed upward from the 11th pair of muscles. Electrical activity of the transversus thoracis appeared only during exhalation.[4]

When functioning as expiratory muscles, the abdominal muscles squeeze the abdominal contents upward and pull the chest cage downward, thus accelerating expiratory airflow and emptying the lungs more than would occur with passive expiration. In this way, these muscles regulate end-expiratory lung volume and breathing efficiency.[5] The detailed role of the abdominal muscles is covered in the next section.

Postural Functions

Experimental evidence[22] supports the view that intercostal muscles, particularly the laterally located external intercostal muscles in the more cephalic spaces, are mainly involved in postural functions. The opposite appears to be the case for the intercartilaginous muscles (ventrally located) and the levator costae muscles (dorsally located), which, in all circumstances, exhibit phasic inspiratory activity quite similar to the diaphragm.[22]

A recently verified postural role of the intercostal muscles[38,50] that has not been emphasized[15,33] is rotation of the thorax. Respiration is executed with bilaterally synchronized intercostal activity. The crisscross pattern of these muscles makes

them admirably suited to a rotation function if the internal intercostals on one side contract with the external intercostals on the opposite side, and *vice versa.* Whitelaw, *et al.*[50] reported that the *right external* intercostals were strongly activated by rotation of the trunk to the left and that the *right internal* intercostal muscles were strongly activated by rotation of the trunk to the right. Rimmer, *et al.*[38] showed that the tonic discharge of internal and external intercostal muscles induced by holding a rotated position is modulated by respiration. When the respiration and rotation functions are compatible, they reinforce the EMG activity. When they are incompatible, respiration takes precedence and inhibits the rotation function.[38]

5. FUNCTIONAL UNIT

The interosseous intercostal muscles serve two major roles: postural and respiratory.

Postural Functions

The external intercostals on the left side and the internal intercostals on the right side both rotate the trunk to the right. Conversely, the internal intercostals on the left side and external intercostals on the right side rotate the trunk to the left. The corresponding internal and external abdominal obliques would augment these rotations and the iliocostalis lumborum augments rotation toward the side on which that muscle lies. The multifidi and rotatores on the right can help to rotate the trunk to the left. The lateral interosseous intercostals, the lateral abdominals, and the quadratus lumborum help to side bend the trunk toward the same ipsilateral side.

The scalene muscles, which are primary in respiration, also serve an important postural role. They stabilize the neck against lateral movement; unilaterally, they laterally flex the neck and bilaterally, they forward flex the neck. Other muscles that are accessory for respiration (such as the sternocleidomastoid and the upper trapezius) also flex the neck and rotate the head.

Inhalation

The diaphragm initiates quiet inhalation quickly followed by activity of other primary muscles of respiration including the scalene muscles, the parasternal internal intercostals, the levator costorum muscles, and the upper and more lateral external intercostals

As the vigor of *forced* respiration increases, additional (accessory) muscles of inhalation are recruited. The total list of muscles that can contribute to labored inhalation is long. Which muscles are activated and how much they are activated depend strongly on the circumstances. Therefore, there is considerable diversity of opinion as to the relative roles of muscles that may serve as accessory muscles of respiration.

Although the scalene muscles have been classified in the past as accessory muscles of respiration, they serve as *primary* muscles of inhalation. With increased ventilatory demand, the sternocleidomastoid muscle also becomes active bilaterally and rapidly increases its level of activity. The sternocleidomastoid appears to be the most important *accessory* muscle.[11] Other muscles which may be recruited include the upper trapezius, serratus anterior and serratus posterior superior, pectoralis major and minor, latissimus dorsi, thoracic erector spinae, subclavius,[11, 33] and the omohyoid.

With *paradoxical respiration (see* Fig. 20.15A), accessory muscles of inhalation must carry a major part of the load because the respiratory effects of the intercostal muscles and the diaphragm largely cancel each other.

Exhalation

When the abdominal muscles are used during exhalation, they increase intra-abdominal pressure, which elevates the diaphragm and assists the outflow of air that is normally accomplished primarily by the elastic recoil of the lungs.

During *forced* exhalation, the abdominal muscles are the prime movers assisted by the internal intercostals (with the exception of the parasternal internal intercostals, which support inhalation). With increased

ventilatory demand the latissimus dorsi, serratus posterior inferior, quadratus lumborum, and iliocostalis lumborum may also be recruited.[33]

Special Functions

Many complex special functions including coughing, sneezing, vomiting, gasping, running, and speech depend on the abdominal muscles.

Both **coughing and sneezing** are protective reflexes that defend the airways against inhaled particles and noxious substances and remove mucus by inducing high airflow velocities during forced exhalation. A **cough** has three phases: inhalation, compression, and expulsion. Following reflex inhalation, the brief compressive phase involves continued activity of the diaphragm and activation of ribcage and abdominal expiratory muscles against a closed glottis. The expulsive phase begins with opening of the glottis as relaxation of the diaphragm and vigorous reflex expiratory muscle activity produce high airflow velocities.[41] Repeated coughing can induce enthesopathy in the attachments of, and activate TrPs in, the expiratory muscles (especially the abdominals). A coughing spell can become excruciatingly painful for this reason.

The neurogenesis for **sneezing** is somewhat different than for coughing. During this reflex, there are often intermittent pauses during the inspiratory effort, and expired air is diverted through the nose in addition to the mouth.[41] Since a prolonged series of sneezes is much less likely to occur than a protracted period of coughing, sneezing is less likely to produce muscular distress.

The inhalations and exhalations of **gasping,** which are induced by severe hypoxia, are more sudden in beginning and ending compared to the rhythmic respirations of eupnea (normal breathing). This unique pattern of autonomic ventilatory activity differs fundamentally from eupnea because the neurogenesis of gasping depends on a specific region of the medulla.[45]

Another reflex respiratory activity, **vomiting,** involves violent contraction of expiratory muscles. Vomiting can be induced by reverse peristalsis of the duodenum, by motion sickness, or by pregnancy. It is such a primitive reflex that it is preserved in decerebrate animal preparations and is produced by the thoracoabdominal respiratory muscles. Expulsion of the gastric bolus by vomiting is usually preceded by retching, which involves successive waves of reflex cocontraction of the diaphragm and abdominal muscles that override the respiratory cycle. Recurrent attacks of retching are feared by clinicians and patients because of the severe fatigue they can induce in respiratory muscles, and the attacks have occasionally led to rib fractures.[29] Again, this muscle overload can produce severely painful enthesopathy and can activate TrPs that persist after an attack.

Most experienced conditioned **runners** show a tight locomotor-respiratory coupling which is established during the first four or five strides of the run. The ratio is usually 2 strides to one respiratory cycle. Inexperienced runners show little or no tendency for such coupling.[48] During prolonged maximal exercise, the blood flow requirements of respiratory muscles are comparable to those of propulsive limb muscles.[2]

6. SYMPTOMS

Intercostal Muscles

The patient complains of aching pain as described in Section 1 of this chapter and often is unable to lie in the position that places body weight onto the TrP. The TrP pain is increased by deep inhalation (for example, during demanding exercise) and either coughing or sneezing may be extremely painful.

Cardiac arrhythmia including auricular fibrillation can depend on the arrhythmia TrP considered in detail in Chapter 42. It sometimes seems to be located in intercostal muscles on the right side. When this occurs, cardiac arrhythmia is one symptom of intercostal TrPs.

Diaphragm

Seventeen patients diagnosed with episodic spasms of the diaphragm[51] complained of chest pain, dyspnea and inability to get a full breath. Sometimes attacks were precipitated by anxiety-producing

situations. Patients sometimes had so much difficulty breathing that they feared they might die. This demonstrates the importance of the diaphragm. The author[53] apparently did not consider TrPs, which could account for the symptoms and, if present, would have been a treatable cause.

When patients have diaphragmatic TrPs, they are prone to develop a "stitch in the side" when doing exercise that requires rapid deep breaths. The pain is likely to be most intense at the end of a full exhalation when the diaphragm fibers are stretched. Coughing can be excruciatingly painful.

Hiccup represents a reflex contraction of the diaphragm; the anatomy, physiology, and clinical aspects of hiccup were thoroughly reviewed by Travell.[46] Often hiccup can be relieved by mechanical (and cold) stimulation of the uvula, suggesting that a trigger area in the mucosa or musculature of the uvula can be a major factor in causing hiccup.[46] Also, TrPs of the diaphragm muscle are suggested by the observation that exhalation tends to relieve the hiccups and deep inhalation (shortening the muscle fibers) tends to aggravate them. However, this respiratory effect may also be an example of respiratory synkinesis.[34]

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Intercostal Muscles

A posture or activity that activates a TrP, if not corrected or if continued, can also perpetuate it. In addition, many structural and systemic factors *(see* Chapter 4) will perpetuate a TrP that has been activated by an acute or chronic overload. For the intercostal muscles, postural considerations are important.

Intercostal TrPs may be activated by gross or local impact trauma, excessive coughing, and chest surgery.[8] Chest retractors used during surgery were found likely to leave painful clusters of intercostal TrPs.[44] Open heart surgery that employed incision of the sternum rather than ribs was more likely to result in TrPs in the pectoralis major and minor muscles than in the anterior intercostal muscles.[44] Other causes for activation include an attack of herpes zoster,[13] fracture of a rib to which the muscle attaches, and possibly a breast implant.

Intercostal TrPs also may become active in association with intrathoracic lesions, such as pneumothorax, pyothorax, and pleural effusion (secondary to a tumor). These associated TrPs are likely to involve the last three intercostal muscles and a complaint of posterolateral low chest pain.

Significant perpetuating factors can be a chronic cough, key TrPs in the overlying pectoralis major muscle, and paradoxical breathing. It is not always clear which comes first as the abnormal respiratory pattern and the TrPs seem to reinforce each other.

Diaphragm

Diaphragmatic TrPs may be activated by exercise, such as rapid walking or running, or by a persistent cough. It is likely they could appear following gastrectomy. To date, relatively few physicians have considered the possibility that diaphragmatic TrPs may be the cause of unexpectedly persistent symptoms related to muscular activity.

8. PATIENT EXAMINATION

After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram of the pain pattern described by the patient. More than intercostal and/or diaphragmatic TrPs may be involved. The drawing should be in the style of the pain patterns in this volume using a copy of an appropriate body form found in Chapter 3, Section 1, Figures 3.2-3.4.

The clinician should be sure to examine the patient for paradoxical breathing *(see* Section 4 of this chapter and Section 14 of Chapter 20). If paradoxical breathing is present, high priority should be given to effective correction of this abnormal breathing pattern, both during initial therapy and at follow-up visits.

Intercostal Muscles

Even with normal coordinated breathing, the vital capacity of the patients with intercostal TrPs is likely to be reduced because the TrPs often painfully restrict deep inhalation or full exhalation.

Rotation of the thoracic spine may be restricted in one or both directions by inter-

costal TrPs. Trigger Points in the diagonal abdominal muscles, the serratus posterior inferior, and the iliocostalis lumborum can also restrict trunk rotation.

The patient with intercostal TrPs is unable to raise the arm straight up on the affected side because of painfully restricted rib mobility. With intercostal TrPs, pain is usually aggravated by bending the trunk toward the side opposite the TrPs, and may be somewhat relieved by bending toward the painful (TrP) side. Careful visual inspection of the chest from in front and/or palpation of the chest for symmetry of expansion during respiration is likely to show a restricted excursion and narrowed rib spaces on the side of interosseous intercostal TrPs.

Diaphragm

Patients with diaphragmatic TrPs are likely to experience pain at the end of a maximal exhalation. To increase the sensitivity of testing, the patient can increase stretch tension on the diaphragm near full exhalation by vigorous contraction of reasonably strong abdominal muscles. If the abdominal musculature is weak, the patient can apply external pressure to the abdomen to increase intra-abdominal pressure forcing the diaphragm upward and stretching it. The effectiveness of this effort is blocked if the patient closes the glottis, which is what patients normally do when contracting the abdominal muscles to increase intra-abdominal pressure. Performing this abdominal maneuver during *continued exhalation* ensures an open glottis. Vigorous coughing at nearly complete exhalation can also induce pain from diaphragmatic TrPs. If the TrPs have caused appreciable enthesopathy, any vigorous cough is likely to be painful.

Among 17 patients with episodes that were diagnosed as diaphragmatic spasm, the author[51] was able to induce an attack in 12 of them during fluoroscopic examination. As the patient had increasing difficulty taking in a full breath, the diaphragm became progressively contracted until it was essentially flat across the abdomen and the patient was in serious respiratory distress because of inability to inhale adequately. Episodes were usually precipitated by engaging the patients in discussion of topics known to be emotionally very stressful to them. The diaphragmatic spasm (or contracture) eliminated diaphragmatic function and was blocking both the pump-handle and bucket-handle movements of the thorax.[51] Increased diaphragmatic muscle tension caused by TrPs would produce the same effect to a lesser degree and also would be aggravated by emotional stress.

9. TRIGGER POINT EXAMINATION

Intercostal Muscles

To locate intercostal TrPs, the clinician should examine the rib cage for abnormally narrow rib interspaces that could indicate tense intercostal muscles. The patient usually describes pain along a narrowed interspace if active intercostal TrPs are responsible. The region of increased muscle tension and of TrP tenderness can be found by running the palpating finger between the ribs for the full length of the suspected segment. Intercostal TrPs are usually located anterolaterally or posterolaterally and less commonly in the extreme anterior and posterior portions of the muscle. The parasternal internal intercostal muscles are an exception to this finding and should be carefully investigated in cases of suspected costochondritis and Tietze syndrome. These syndromes may be caused by TrPs in these work-horse respiratory muscles. This possibility needs clinical research investigation.

Dr. Travell observed that a TrP in the intercostal muscle located posteriorly between ribs 4 and 5, close to the rhomboid minor muscle, initiated a hiccup when pressed before TrP injection, but not following injection.

Diaphragm

The midfiber central TrPs of the diaphragm are obviously not accessible to palpation. However, the tenderness of attachment TrPs in the costal portion of the diaphragm is detectable just inside the lower border of the thoracic cage. Tenderness detected in this region could originate in the diaphragm, the external oblique, internal oblique, or transversus abdominis muscles. The oblique abdominal muscles

attach to the ribs externally above the costal margin (Fig. 49.4), whereas the transversus abdominis muscle attaches to the costal margin and interdigitates with angulated diaphragm fibers (Fig. 45.4 and *see* Fig. 49.5). Palpation of the patient's active abdominal contractions and identification of appropriate fiber directions can help the examiner to distinguish taut bands and TrP tenderness in the more superficial abdominal muscles from tenderness of the deeper muscles.[3,6]

The ambiguity of distinguishing between the attachment tenderness of the transversus abdominis and the diaphragm at the costal margin can be resolved by testing sensitivity to stretch. The examiner can test whether pain and tenderness are increased by stretching the abdominal muscles (protruding the abdomen) or by stretching the diaphragm (compressing the abdomen near the end of exhalation).

10. ENTRAPMENT

No nerve entrapment has been attributed to the intercostal or diaphragm muscles, nor is one likely, considering their anatomy.

11. DIFFERENTIAL DIAGNOSIS

with contributions by
Roberta Shapiro, D.O.

Intercostal Muscles

Differential diagnoses of intercostal TrPs include herpes zoster, rib articular dysfunctions, fibromyalgia (when pain is widespread), cardiac diseases (in cases of left unilateral intercostal TrPs), painful rib syndrome (which Dyer[23] identified as often being a misnomer for abdominal myofascial TrPs), Tietze syndrome or costochondritis (diagnoses which Calabro *et al.*[10] clearly differentiated), thoracic radiculopathy, and intercostal muscle spasm (which Blumer[6] considered one of the most common, generally unrecognized, benign causes of chest pain in daily practice). The muscle tension caused by TrPs is commonly mistakenly identified as spasm,[43] which may be the case in the Blumer[6] paper.

Serious intrathoracic disease that can mimic the symptoms of intercostal TrPs include myocardial infarction, tumor, pleural effusion, and pyothorax. These conditions must be ruled out; when present they also can induce and perpetuate TrP activity. Thus, if intercostal TrPs responded poorly to treatment, imaging of the chest and a search for other conditions is strongly indicated.

Intercostal TrPs commonly develop in conjunction with an attack of herpes zoster.[13] In this study, the neurogenic pain of herpes was often described as a shooting pain which was generally responsive to Tegretal therapy. Pain from TrPs was described as a localized ache that, in these cases, persisted despite Tegretal therapy but responded to TrP therapy.[13] The TrP pain is most likely to be prominent in the chronic stage of a herpes attack, and may be the only remaining source of chest pain. The intercostal TrP pain tends to be well localized, most commonly in the posterolateral part of the chest.

Related Trigger Points. Spot tenderness of the chest wall in locations where the serratus anterior attaches to ribs may appear to be intercostal TrPs, but in fact, usually represents enthesopathy secondary to TrPs of the serratus anterior muscle. A taut band palpable between the spot tenderness of the chest wall and a serratus anterior central TrP helps to identify the tenderness as belonging to an attachment TrP of the serratus anterior muscle.

Full elevation of an upper limb opens up the intercostal spaces on the same side and stretches fascial tissues over the chest wall. This movement is painful to patients who have intercostal TrPs, who are recovering from thoracotomy, or who have herpes zoster with or without intercostal TrPs. Patients with these conditions are vulnerable to developing a painful myofascial "frozen" shoulder because of the pain-induced restricted range of motion at the shoulder that encourages the development and perpetuation of subscapularis TrPs as described in Chapter 26, Section 11.

At times, the cardiac arrhythmia TrP associated with the pectoralis major muscle *(see* Chapter 42) appears just as likely to be located in an intercostal muscle.

Articular Dysfunction. Articular dysfunction associated with intercostal TrPs is usually isolated to one or two rib levels and presents as an exhalation or depressed

rib lesion. This dysfunction is best treated by inactivating the TrPs, by rib-mobilizing muscle stretch using respiration to augment relaxation, or by functional (indirect) techniques.

Diaphragm

With regard to diaphragmatic TrPs, differential diagnoses include diaphragmatic spasm,[51] peptic ulcer, gastroesophageal reflux, and gallbladder diseases (in cases of right-side unilateral diaphragmatic TrPs).

Atypical chest pain (which, when in the lower sternal area, has also been called slipping rib syndrome, xiphoidalgia, or precordial catch syndrome) was shown in one characteristic example to be due to a TrP in the diaphragm muscle.[52] Clinical research studies are needed to clarify the relation between these syndromes and TrPs.

When chest pain is closely associated with increased tension of the diaphragm it must not be assumed that the tension is caused by spasm. Increased muscle tension and pain in the absence of spasm are cardinal features of TrPs.

In one study, the 17 patients diagnosed as suffering from episodes of diaphragmatic spasm[51] were not monitored for EMG activity, so it was not conclusively shown that they all had spasm of the diaphragm; some of them may have had increased muscle tension from TrPs, especially some of the less typical cases. In some cases, the diaphragmatic tension could be released by an effort at full exhalation, which helps to inactivate diaphragmatic TrPs. One of the case reports demonstrated that after the diaphragm went into spasm in response to psychic distress, it relaxed upon return of emotional equanimity. The patient learned to identify and avoid buildup of the emotional tension that precipitated attacks. The critical question remained unanswered as to why that patient's diaphragm was so prone to spasm. Here TrPs may well have been playing a critically important role, especially if other muscles had TrPs that were prone to increase markedly the excitability of the motor neuron pool serving the diaphragm.

Diaphragmatic TrPs can be satellites to TrPs in the upper portion of the rectus abdominis muscle on the same side. Athletes who overexercise the rectus abdominis muscle by concentrating on situps (which overload the rectus muscle in a shortened position) are likely to develop rectus abdominis TrPs. In addition, the overuse of heavy resistive exercises to develop the pectoral and biceps muscles makes demands of the abdominal muscles for stabilization. On examination, the tenderness of rectus abdominis TrPs is increased by stretching that muscle or by asking the supine patient to raise the feet off of the examining table. If these movements do not increase the TrP sensitivity, then tenderness to pressure applied inside the lower border of this region of the rib cage most likely indicates diaphragmatic TrP involvement.

Confusion may arise between pain from TrPs in the diaphragm and pain from TrPs in the interdigitating transversus abdominis muscle. Pain experienced on full inhalation (abdomen protruded, transversus stretched) is more likely to come from transversus TrPs; pain experienced on full exhalation (abdomen pulled in, diaphragm stretched) is more likely to come from diaphragmatic TrPs.

Diaphragmatic TrPs have no recognized articular dysfunctions related to them.

12. TRIGGER POINT RELEASE (Figs. 45.10-45.12)

For lasting release of trigger points (TrPs) in these respiratory muscles, and for lasting relief of pain, the patient must be instructed to correct paradoxical breathing, if it is present *[see* Figs. 20.15 and 20.16). Good posture is also essential for maintenance of muscle length and effective respiratory patterns *[see* Chapter 41, Section C).

Intercostal Muscles

Inactivation of TrPs in these muscles can be approached by direct techniques (treatment directed against the barrier) or by indirect techniques (utilizing a position of ease). Application of direct manual techniques specifically to the muscle,

such as TrP pressure release and deep stripping massage, is effective for essentially all intercostal muscles. Spray and stretch, and stretch using postisometric relaxation enhanced by coordinated respiration are also effective. When muscle energy techniques are used, as pointed out by Goodridge and Kuchera,[27] the technique for releasing the first rib is different from that for ribs **2-10,** which is different from that for ribs **11** and **12,** because of the differences in their articulations.

The use of vapocoolant enhances release of these TrPs and relief of pain. The spray is applied over the involved muscles, fully covering the TrP area and the entire zone of pain and tenderness.

Upper Thorax. The discrepancy between the location of the muscles and their effect on rib motion is contrary to the intuitive assumptions that are made by many clinicians and then promoted as fact. It has been reasonably assumed by many that the parasternal intercostals elevate the sternum in the pump-handle motion and that the lower lateral intercostals elevate the ribs laterally in the bucket-handle motion. The *reverse* is true (see Section **4** of this chapter). However, the manual release techniques described for myofascial tension in the region of the sternum or lower lateral ribs are appropriate for releasing TrPs in muscles of these regions.

An approach for release of upper intercostal muscle tension is illustrated and described in Figure **45.10.** Goodridge and Kuchera[27] describe and illustrate several additional applications of muscle energy. Greenman[28] describes functional (indirect) techniques that use a position of ease to release tension in this region. Upledger and Vredevoogd[47] approach the resultant sternal elevation directly by depressing it between the hands, assisted by respiration.

In addition to releasing the specific pain-producing TrPs, it is helpful to release all tense myofascial tissues in that region.

Lower Thorax. An effective approach to release TrPs in these lower intercostal muscles is illustrated and described in Figure **45.11.** Goodridge and Kuchera[27] as well as Greenman[28] also describe and illustrate the application of other techniques to the lower rib cage.

Diaphragm

The muscle fibers of the diaphragm are placed on stretch by maximum exhalation, which moves the dome of the diaphragm up into the chest cavity. The fibers are also stretched by any compression of the abdomen at full exhalation. The diaphragm is inaccessible to direct manual therapy techniques, such as TrP release. However, it and the lower intercostal TrPs can be released by the technique illustrated and described in Figure **45.12.** This manual release can be preceded with application of vapocoolant spray to cover the margin of the lower rib cage where the costal diaphragm fibers attach.

An increase in intra-abdominal pressure for added stretch to the diaphragm on full exhalation can be accomplished in various ways, such as voluntary contraction of the abdominal muscles, application of hand or arm pressure to the abdomen, and bending the body forward on exhalation.

Ingber[32] identified one cause of the common problem of atypical chest pain to be diaphragmatic TrPs. Leaning forward and inhaling aggravated the pain. Application of TrP therapy including TrP pressure release applied to the right diaphragm, upper thoracic extension using a postisometric relaxation technique, and home corrective exercises rendered the patient pain free for at least **1** year.

Upledger and coworker[47] described and illustrated anteroposterior compression of the upper abdomen and lower rib cage by placing one hand on the epigastrium and the other hand under the upper lumbar spine. No respiratory maneuver was described. However, the principle of postisometric relaxation can be applied effectively with the hands in this position to stretch and release tense fibers of the diaphragm muscle. The patient should take quiet gentle breaths with the lungs kept as empty as possible. This can be accomplished by the operator gently assisting exhalation with pressure applied between the hands, asking the patient to hold the exhalation for several seconds, and then gently resisting inhalation. This encourages successively smaller lung volumes with each breath, which means progressive lengthening of diaphragm fibers.

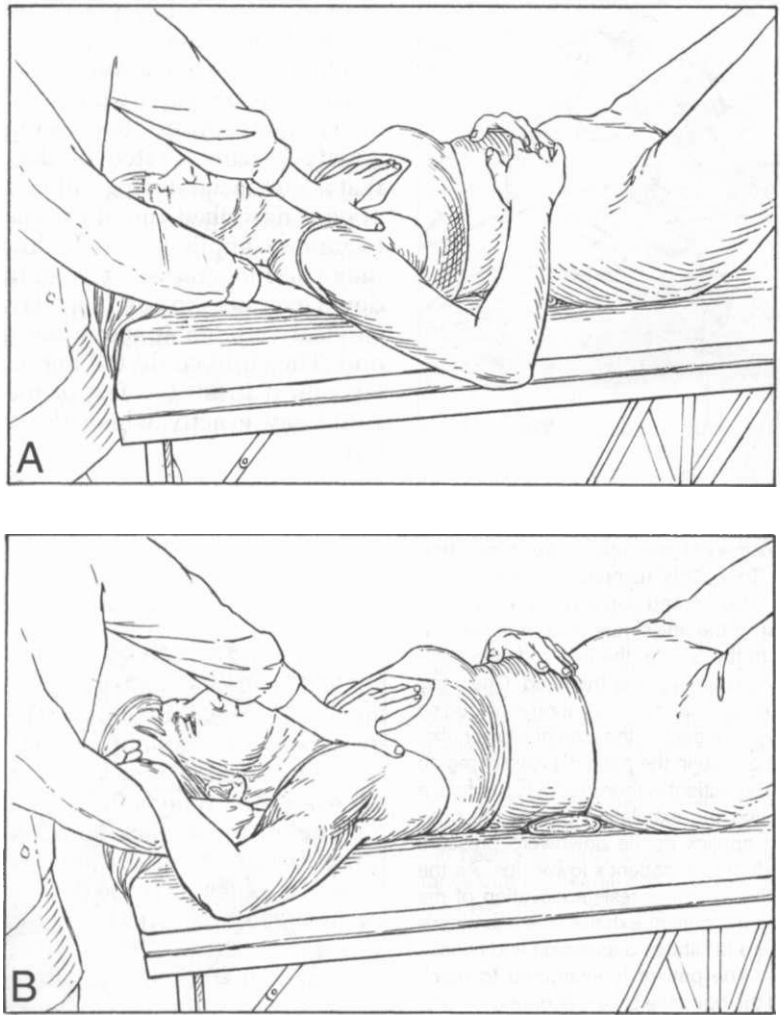


Figure 45.10. Two positions for release of upper intercostal muscle tension caused by TrPs. This release procedure is also referred to as an upper rib release of ribs 2-4 or 5. **A,** the clinician places one hand medial to the vertebral border of the scapula, contacting the appropriate affected ribs posteriorly, and applies pressure in a cephalad direction with the fingers. The clinician's other hand is positioned anteriorly over the affected ribs, applying downward pressure that resists rib elevation when the patient inhales and then assists rib depression when the patient exhales (utilizing respiration to augment release of the tense intercostal muscles). **B,** alternate position for the same "upper rib" release, with the patient's arm positioned in elevation.

With a variation in the position of the hand applying pressure posteriorly on the scapula, this procedure can then be effective for releasing trigger point tension in the upper and middle portions of the serratus anterior muscle.

Goodridge and Kuchera[27] describe a muscle energy technique for releasing the diaphragm that concentrates on correcting asymmetry of the lower rib cage without including any respiratory instructions in the therapeutic protocol. This approach may be as effective at releasing TrP tension in intercostal muscles of the lower rib cage as for releasing tension in the diaphragm.

Although there is no specific evidence that hiccups are directly related to diaphragmatic TrPs, it is interesting that breathing while in the position of as full exhalation as possible (which stretches the

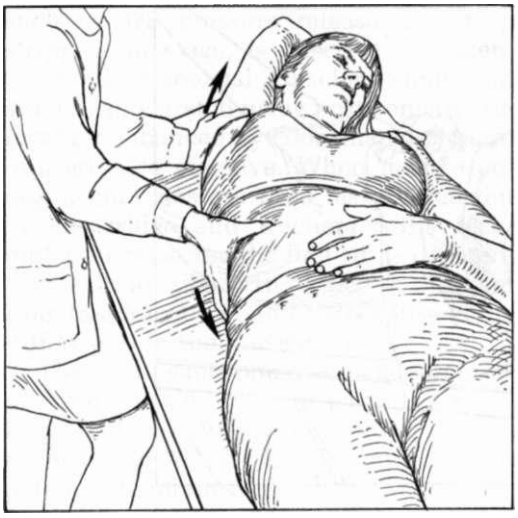


Figure 45.11. Release of lower intercostal muscle tension caused by TrPs. This technique is sometimes called "lower rib release" and can also be used to release TrP tension of the latissimus dorsi muscle. Patient is supine with the arm on the affected side positioned upward and reaching over the head. One hand of the clinician (the right hand in this figure) is placed so as to span the lateral aspect of the patient's lower ribs; the other hand is placed in the patient's axillary region for stabilization. The patient is then instructed to take a deep breath. During the exhalation phase, the clinician's right hand applies gentle downward pressure (caudally directed) on the patient's lower ribs. As the patient inhales, the examiner resists elevation of the lower ribs, and as the patient exhales, the examiner's downward pressure facilitates depression and release of the lower ribs. The patient is instructed to reach overhead toward the opposite shoulder during exhalation, which accentuates the stretch of the intercostal muscles and the latissimus dorsi muscle. The stretch cycle is repeated until release is satisfactory.

diaphragm) tends to reduce hiccup activity and discourage its return, while taking a deep inhalation (which shortens diaphragm muscle fibers) can reactivate hiccups.[46] The fact that severance of both phrenic nerves may not terminate hiccups suggests that hiccups can be produced by reflex activity of the inspiratory chest muscles without diaphragmatic contraction. Dr. Travell spent many years exploring ways to end persistent hiccups in challenging cases, and in 1977[46] summarized some of the techniques she had found to be most effective. All of them affected the position of the uvula.

When a person's body is in the head-down position, gravity pushes the abdominal contents toward the chest, producing a gravity-assisted stretch of the diaphragm that is enhanced during full exhalation. Dr. Travell described the effectiveness of this technique applied to her children when they had hiccups. She turned them upside down over her lap and tapped over the fifth cervical vertebra at about one tap per second. The child could tell her to correct the tap rate if it felt too fast or too slow, and could say exactly where the tapping felt best.

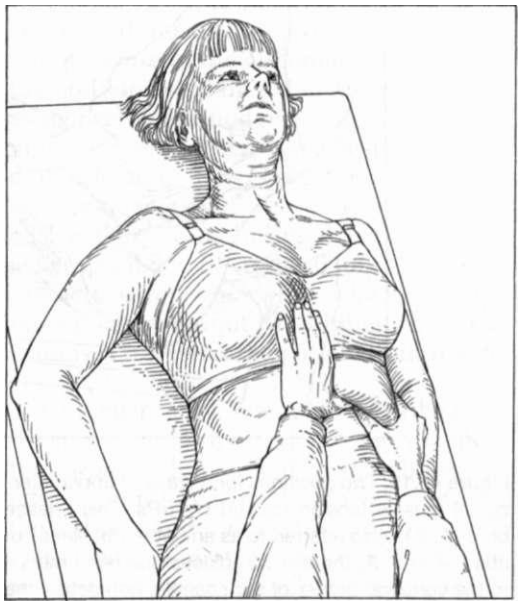


Figure 45.12. Diaphragm release with the patient supine. The clinician stands at the patient's side that is opposite to the muscle to be released (i.e., at the patient's right side for release of the left part of the diaphragm), and places both hands anteriorly at the lower border of the patient's rib cage. The patient is instructed to breathe in normally in a relaxed manner and then breathe out slowly. During exhalation, the clinician's thumbs follow the diaphragm inward under the rib cage and then lift the rib cage anteriorly, which is the actual release phase of the procedure. Some additional release occurs on subsequent respiratory cycles. This procedure is also helpful for releasing lower intercostal muscle trigger points.

13. TRIGGER POINT INJECTION (Fig. 45.13)

Injection of intercostal and diaphragmatic trigger points (TrPs) should be attempted only by those with much experience and skill in needling TrPs, and who have a highly sensitive "feel" for the tissue being penetrated by the needle. Injection of these muscles is not for beginners. Injection should NOT be considered until noninvasive techniques (see Section 12) have been exhaustively explored and proven to be unsuccessful in the hands of *skilled* clinicians. Injection of these muscles should not be considered until systemic and mechanical perpetuating factors (including a chronic cough) have been fully investigated and corrected.

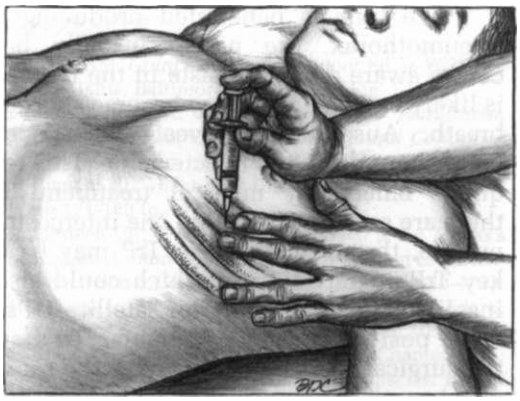


Figure 45.13. Injection of intercostal muscles using Hong's safer method of holding the syringe as compared to the traditional method. The Hong technique[31] provides additional protection from unintended advancement of the needle into the pleura due to a sudden unexpected movement by the patient such as a sneeze, cough or startle reaction. The syringe is held in such a way that the wrist and ulnar side of the hand rest solidly on the patient's rib cage (See text for details). When the location of TrP tenderness has been identified, the spot is marked by placing a finger on the rib on each side of the TrP. No attempt should be made to inject an intercostal muscle until the clinician has had considerable experience injecting TrPs in other muscles and has developed a fine feel for the different textures of the tissues and the depth to which the needle is penetrating. It is of paramount importance that the needle **not** penetrate the epimysium on the deep side of the muscle.

Intercostal Muscles

For injection, the patient should be placed in the supine or sidelying position depending on the location of the TrP. Pillows are used as necessary to ensure that the patient is COMFORTABLE. The clinician doing the injection should be seated comfortably, should be aware of the patient's respiratory rhythm, and should encourage the patient to take quiet shallow breaths and not to hold the breath. Holding the breath could result in a sudden unexpected deep breath.

Palpation of the taut band of an intercostal TrP may not be possible because of overlying muscles. When it is necessary to palpate through an overlying muscle, such as the pectoralis major, the examiner should be sure that overlying muscle does not have a TrP that has been overlooked and needs to be inactivated.

The guide hand is placed solidly on the patient's chest with two fingers straddling the TrP by placing them on the adjacent ribs (Fig. 45.13). A 27-gauge needle on a 5 ml syringe loaded with 0.5 % procaine or lidocaine is used for injection. The needle should be angled close to the chest wall, no more than 45° from the skin surface. This increases control of the needle depth. The needle is slowly advanced toward the spot of maximum TrP tenderness between the fingers. The syringe is held as shown in Figure 45.13 with the ulnar side of the injecting hand resting solidly on the patient, so that any unexpected movement by the patient would move that hand with the body and prevent unintended advancement of the needle into the pleura.

When injecting, the operator notes the resistance encountered by the needle when it reaches the fascial covering of the intercostal muscle. If there is doubt about the depth of that muscle, the needle can be angled to one side until it gently encounters the rib, and then be redirected. The muscle should be carefully monitored for palpable evidence of a local twitch response, which is important to insure clinical effectiveness of the injection. Special care is taken *not* to proceed beyond a second barrier of fascial resistance that would represent the inner fascial covering of the muscle (usually less than 5 mm of muscle depth beyond the first barrier).

If the lung is penetrated producing a pneumothorax, the patient usually becomes aware of a salty taste in the mouth, is likely to cough, and may become short of breath. Auscultation reveals a lack of breath sounds on the injected side. This requires emergency medical treatment. If there are several TrPs along one intercostal muscle, the most posterior TrP may be a key TrP, inactivation of which could also inactivate any more anterior satellite TrPs.

In post-thoracotomy patients, TrPs in the surgical scar tissue may have the same effect as a key intercostal muscle TrP. Elimination of key *scar tissue* TrPs, which usually requires injection, can then also inactivate any satellite muscular TrPs.

Chen, *et al*[33] reported that patients who developed active intercostal TrPs in association with herpes zoster responded to injection of the TrPs with 0.5% Lidocaine with immediate relief of chest pain following injection. Pain relief usually lasted 1 or 2 weeks following the first injection, with progressively longer periods of relief (up to 2 months) with repeated injections.

Diaphragm

Needle penetration in this region is hazardous because of the great danger of producing a pneumothorax. If subcostal spot tenderness is clearly not from abdominal TrPs and if mobilization maneuvers are not helpful, then one who is sufficiently skilled and properly equipped can consider injection of *attachment* TrPs of the diaphragm. However, accurate localization of a TrP in this muscle is difficult because of its location. The technique for injecting attachment TrPs at the costal margin is similar to that used for making needle EMG recordings of diaphragm motor unit activity, which was well illustrated and described by Saadeh, *et al.*[40] and also was reported by Bolton, *et al*[7] In fact, the safest TrP injection technique in this location is to use an EMG hypodermic needle that is sold to confirm a muscular site by EMG monitoring for injecting Botulinum Toxin A. Only the type of needle is recommended, NOT the toxin. This EMG ensures injection of procaine or lidocaine into the diaphragm muscle, which is identified by its activity only during inhalation.

It is important to remember that injection techniques like this can only reach attachment TrPs of the diaphragm. The endplate zone, where the central TrPs are located, is a horseshoe-shaped line running midway between the peripheral end of each fiber and its attachment onto the central tendon of the diaphragm *(see* Fig. 2.10D). For that reason, subcostal injection of Botulinum Toxin A would be essentially useless and is seriously hazardous.

14. CORRECTIVE ACTIONS (Fig. 45.14)

Patients with intercostal TrPs and especially those with diaphragmatic TrPs are likely to exhibit paradoxical respiration that may be both a contributing factor to, and the result of, the TrPs. It is important for full recovery of normal function to retrain these patients to use normal coordinated respiration (see Section 4 above, Chapter 20, Section 14, and Figs. 20.15 and 20.16). One study showed that surface EMG feedback from *only* inspiratory muscles of the upper thorax was not significantly helpful for this training.[26] Training is usually effective when a skilled clinician combines tactile monitoring with appropriate verbal feedback to facilitate normal breathing patterns. The clinician then should help the patient to become aware of normal lateral lower rib movements.

Erect posture facilitates good respiratory patterns. Head-forward, slumped posture needs to be corrected. The patient should be instructed in practical ways to attain and maintain optimal posture (see Chapter 41, Section C).

When lower thoracic intercostal and/or diaphragmatic TrPs are identified on one side, the release technique illustrated and described in Figure 45.14 can be used as a self-applied release. Maximum elevation of the diaphragm is achieved in the supine position by letting the breath out completely and then contracting the abdominal muscles. This places the diaphragm on maximum passive stretch with some additional help from reciprocal inhibition supplied by the voluntary contraction of the abdominal muscles. The importance of po-

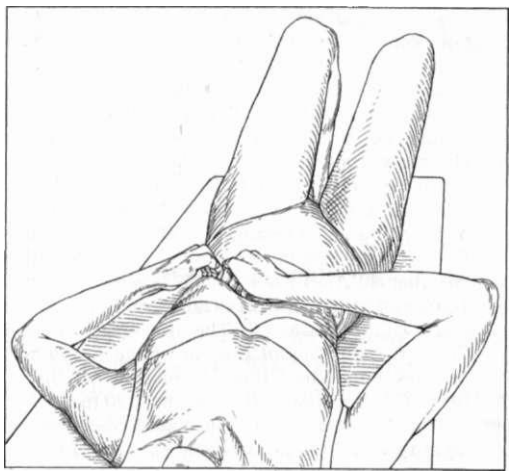


Figure 45.14. Self-release of diaphragm. This self-stretch procedure is done in the supine position with the hips and knees flexed to relax the abdominal musculature. The patient hooks his or her fingers under the lower ribs of the affected side and then inhales deeply in a slow, relaxed manner. During slow exhalation, the patient's fingers follow the diaphragm in and under the ribs and then apply upward traction on the ribs for the actual release. This self-stretch procedure also helps to release lower intercostal muscle trigger points.

sition is reinforced by the study of Wanke, *et al.*[49] which showed electromyographically that inhalation required more inspiratory muscle activity when the subject was supine than when the subject was upright. This helps to explain the usual clinical experience that patients in respiratory distress are more comfortable in a nearly upright position.

When the patient has a chronic cough, it must be controlled before one can obtain lasting relief from TrPs in these respiratory muscles. If the source of the cough cannot be eliminated, the patient can learn how to suppress a cough and raise the sputum by clearing the throat, assisted by a cough suppressant, if necessary.

Intercostal TrPs are aggravated and perpetuated by restriction of rib movement. One should avoid using a chest binder any longer than really necessary. If possible, the chest binder should be removed for 5 minutes or so approximately every 3 hours to reestablish intercostal muscle function.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 17, Fig. 1.16).
2. Ainsworth DM: Respiratory muscle recruitment during exercise. Chapter 14. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.171-180 (p. 178).
3. Bardeen CR: The musculature, Sect. 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (pp. 458-471).
4. Basmajian JV, Deluca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp.409-426).
5. Bishop BP: The abdominal muscles. Chapter 4. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.35-46 (pp. 35-37).
6. Blumer I: Chest pain and intercostal spasm [Letter]. *Hasp Pract 24(5A):13,* 1989.
7. Bolton CF, Grand'Maison F, Parkes A, *et al:* Needle electromyography of the diaphragm. *Muscle Nerve* 25:678-681, 1992.
8. Bonica JJ, Sola AE: Chest pain caused by other disorders. Chapter 58. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990, pp. 1114-1145, (p. 1133).
9. Brondbo K, Dahl HA, Teig E, *et al.:* The human posterior cricoarytenoid (PCA) muscle and diaphragm. *Acta Otolaryngol* 302:474-481, 1986.
10. Calabro, JJ, Jeghers H, Miller KA, *et al.:* Classification of anterior chest wall syndromes. *JAMA* 243(14):1420-1421, 1980.
11. Campbell EJ: Accessory muscles. Chapter 9. In: *The Respiratory Muscles.* Ed. 2. Edited by Campbell EJ, Agostoni E, Davis JN. W.B. Saunders, Philadelphia, 1970 (pp. 181-195).
12. Capps JA: *An Experimental and Clinical Study of Pain in the Pleura, Pericardium and Peritoneum.* The MacMillan Company, New York, 1932 (see pp. 69-99).
13. Chen SM, Chen JT, Wu YC, er *al.:* Myofascial trigger points in intercostal muscles secondary to herpes zoster infection to the intercostal nerve [Abstract]. *Arch Phys Med Rehabil* 77:961, 1996.
14. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (p. 357, Fig.5- 25 and p. 358, Fig. 5-27).
15. Ibid. (pp. 476-477)
16. Ibid, (pp 478-482).
17. De Troyer A: Actions of the respiratory muscles or how the chest wall moves in upright man. *Clin RespirRes* 20(5):409-413, 1984.
18. De Troyer A: Mechanics of the chest wall muscles. Chapter 6. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.59-73 (pp. 60).
19. *Ibid.* (Figs. 1 and 3; p. 61)
20. *Ibid.* (p. 63)
21. *Ibid.* (Fig. 4; p. 71)
22. Duron B, Rose D: The intercostal muscles. Chapter 3. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.21-33 (pp. 24, 28).
23. Dyer NH: Painful rib syndrome [Letter]. *Gut 35(3):429,* 1994.

24. Ferner H, Staubesand J: *Sobotta Atlas of Human Anatomy,* Vol. 2. Urban & Schwarzenberg, Munich 1983 (p 62, Fig.89).
25. Fields HL: *Pain.* McGraw-Hill Book Co., New York, 1987.
26. Gallego J, de la S0ta AP, Vardon G, ef *al.:* Electromyographic feedback for learning to activate thoracic inspiratory muscles. *Am J Phys Med Rehabil 70(4) :186-190,* 1991.
27. Goodridge JP, Kuchera WA: Muscle energy treatment technique for specific areas. Chapter 54. In: *Foundations for Osteopathic Medicine.* Edited by Ward RC. Williams & Wilkins, Baltimore, 1997 (pp. 697-761, see pp. 710-715 and 756-759).
28. Greenman PE: *Principles of Manual Medicine.* Ed.2. Williams & Wilkins, Baltimore, 1996 (pp. 105-108, 123-128).
29. Gre'lot L, Miller AD: Neural control of respiratory muscle activation during vomiting, Chapter 20. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.239-248 (pp. 241, 242).
30. Han JN, Gayan-Ramirez G, Dekhuijzen R, *et al.:* Respiratory function of the rib cage muscles. *Eur Resp J* 6(5);722-728, 1993.
31. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain 2(l):29-59,* 1994.
32. Ingber RS: Atypical chest pain due to myofascial dysfunction of the diaphragm muscle: a case report. *Arch Phys Med Rehabil* 69:729, 1988.
33. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 322-330).
34. Lewit K, Berger M, Holzmuller G, Lechner-Steinleitner S: Breathing movements: the synkinesis of respiration with looking up and down. *J Musculoske Pain 5(4):57-69,* 1997.
35. Loring SH: Action of human respiratory muscles inferred from finite element analysis of rib cage. *J Appl Physiol* 72(4):l461-1465, 1992.
36. Maloney M: Personal Communication, 1995.
37. Reid WD, Dechman G: Considerations when testing and training the respiratory muscles. *Phys Ther 75(11):971-982,* 1995.
38. Rimmer KP, Ford GT, Whitelaw WA: Interaction between postural and respiratory control of human intercostal muscles. *J Appl Physiol* 79(5):1556-1561, 1995.
39. Roussos C: Function and fatigue of the respiratory muscle. *Chest* 88(Suppl):124s-132s, 1985.
40. Saadeh PB, Crisafulli CF, Sosner J, *et al.:* Needle electromyography of the diaphragm: a new technique. *Muscle Nerve* 36:15-20, 1993.
41. Shannon R, Bolser DC, Lindsey BG: Neural control of coughing and sneezing. Chapter 18. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.213- 222 (pp. 214, 220).
42. Sieck GC, Prakash YS: The diaphragm muscle. Chapter 2. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.7-20 (p. 17).
43. Simons DG, Mense S: Understanding the measurement of muscle tone as related to clinical muscle pain. *Pain 75:1-17,* 1999.
44. Sola A: Personal Communication, 1986
45. St. John WM: Gasping. Chapter 16. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.195-202 (p. 200).
46. Travell JG: A trigger point for hiccup. *J Am Osteopath Assoc* 77:308-312, 1977.
47. Upledger JE, Vredevoogd JD: *Craniosacral Therapy.* Eastland Press, Chicago, 1983, (pp. 47-49).
48. Viala D: Coordination of locomotion and respiration. Chapter 24. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997, pp.285-296 (pp. 286-287).
49. Wanke T, Lahrmann H, Formanek D, *et al.:* Effect of posture on inspiratory muscle electromyogram response to hypercapnia. *Eur J Appl Physiol Occup Physiol 64(3):266-* 271, 1992.
50. Whitelaw WA, Ford GT, Rimmer KP, *et al.:* Intercostal muscles are used during rotation of the thorax in humans. *J Appl Physiol* 72(5):1940-1944, 1992.
51. Wolf SG: Diaphragmatic spasm: a neglected cause of dyspnoea and chest pain. *Integr Physiol Behav Sci 29(l):74-76,* 1994.

CHAPTER 46
Serratus Anterior Muscle

HIGHLIGHTS: **REFERRED PAIN** from the serratus anterior muscle is projected to the side and back of the chest and sometimes down the ulnar aspect of the arm. **ANATOMY:** this muscle has three distinct fiber arrangements that run from the upper 8 or 9 ribs to the costal surface of the vertebral border of the scapula. **INNERVATION** of the serratus anterior muscle is by the long thoracic nerve. **FUNCTION** of the muscle includes rotation of the scapula to turn the glenoid fossa upward, abduction and elevation of the scapula, and prevention of winging of the scapula. **SYMPTOMS** of trigger points (TrPs) in this muscle are pain and sometimes a sense of air hunger with short panting respiration. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** can be caused by stressful running, coughing, and by psychogenic factors. **PATIENT EXAMINATION** may reveal reduced chest expansion, winging of the scapula due to TrP inhibition, and some limitation of scapular adduction. **TRIGGER POINT EXAMINATION** locates TrPs along the upper two-thirds of the midaxillary line often around the fifth or sixth rib. **TRIGGER POINT RELEASE** by spray and stretch requires adduction of the scapula with the spray directed first posteriorly, then anteriorly, to cover the muscle and all of its pain pattern. Other manual techniques can also be effective. For **TRIGGER POINT INJECTION** the needle is directed at the TrP fixed between the fingers against a rib. **CORRECTIVE ACTIONS** include modification of patient activities to reduce and eliminate overuse of the muscle, as by coughing, paradoxical breathing, push-ups, and body-lift exercises. Appropriate self-stretch exercises for the home program include the Seated Serratus Anterior Stretch and the Indoorway Stretch Exercises.

1. REFERRED PAIN
(Fig. 46.1)

Referred pain from trigger points (TrPs) concentrates anterolaterally at midchest level,[29] and in a separate posterior area medial to the inferior angle of the scapula (Fig. 46.1). Pain may also be projected down the medial aspect of the arm, extending to the palm and ring finger.[10,41, 51-53] Respiratory symptoms[49] are described under Section 6.

The interscapular pain caused by serratus anterior TrPs can be particularly annoying and distressing, partly because it can be so persistently intense, partly because it is so refractory to positional relief, and partly because few practitioners adequately examine the serratus anterior for TrPs based on this pain complaint. As a result, the patient becomes saddled with the diagnosis of an enigmatic, behavioral pain. That misdiagnosis and the failure to identify and treat the TrP cause of the pain often lead to the end of the patient's normal way of life.

In some patients, TrPs in the serratus anterior contribute to abnormal breast sensitivity, in addition to the TrPs in the pectoralis major muscle[48] (Fig. 42.1C) that are usually responsible for this breast symptom.[49]

Central TrPs can occur in the midfiber region of any digitation in this muscle. Secondary attachment TrPs can be found anteriorly where each digitation attaches to a rib. Posteriorly these attachment TrPs are located along the underside of the vertebral border of the scapula and may be one reason why interscapular pain caused by this muscle is so enigmatic and pernicious.

2. ANATOMY
(Fig. 46.2)

The serratus anterior muscle is composed structurally of three groups of fibers. The most superior serration, which attaches ***anteriorly*** to the first (and sometimes the second) rib, connects ***posteriorly***

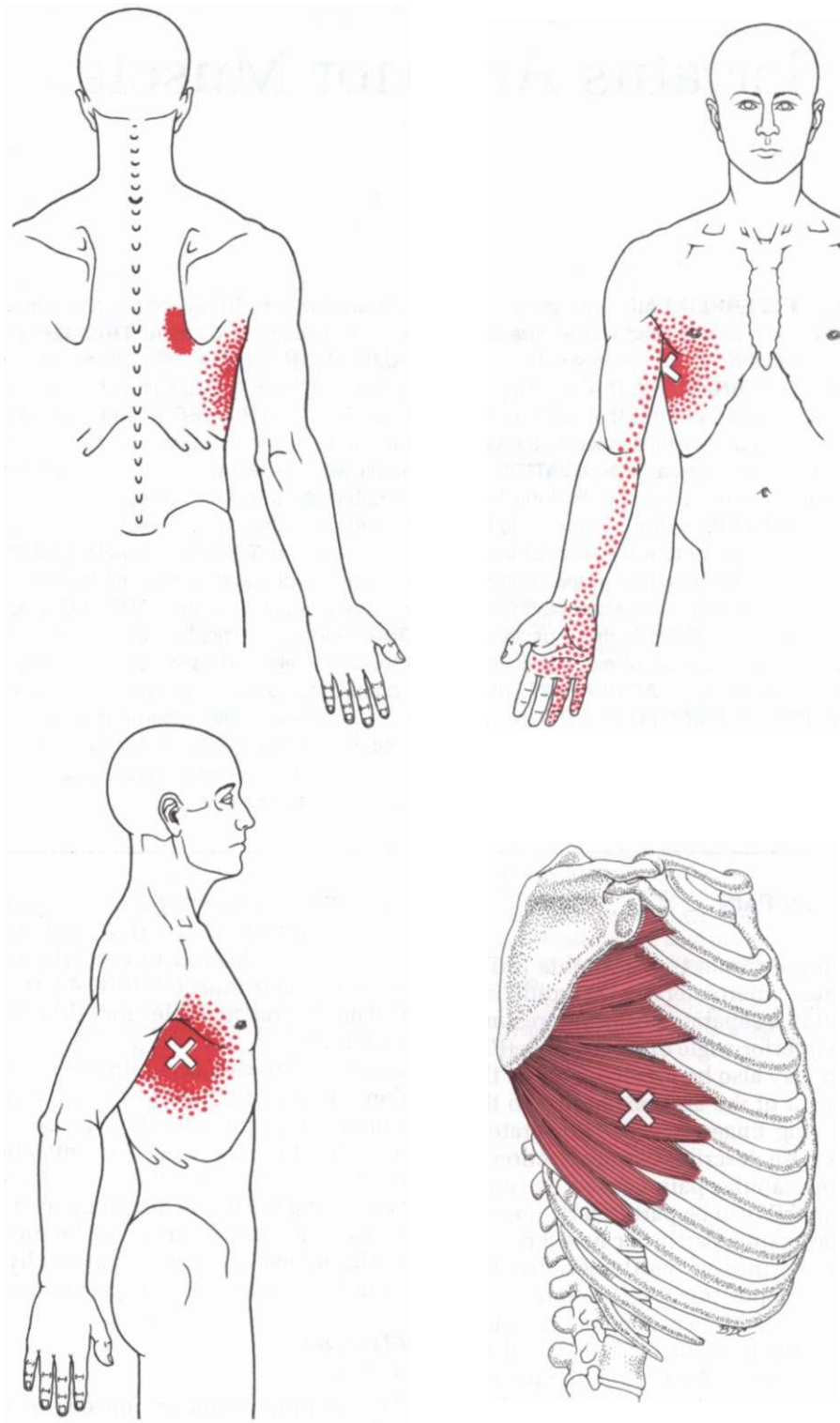


Figure 46.1. Referred pain pattern (essential areas *solid dark red*, spillover areas *stippled dark red)* from a trigger point (**X**) in the right serratus anterior muscle *(medium red)*, as seen from the back, front, and side view. Central trigger points can occur in the midfiber region of any digitation. Trigger points in fibers covering the first two ribs can be difficult or nearly impossible to reach for examination.

to the superior angle of the scapula.[8] This bundle of fibers lies nearly parallel to the underlying ribs (Fig. 46.2).

The next two serrations connect ***anteriorly*** to nearly half the length of the second and third ribs to form a flat sheet of nearly parallel fibers, which attach ***posteriorly*** to the length of the vertebral border of the scapula. These fibers lie snugly against the ribs, angling across them at nearly 45°.

The inferior five or six serrations attach ***anteriorly*** to the next five or six ribs (rib 4 to rib 8 or 9). This third group is the strongest part of the muscle and forms a quarter-circle fan, which converges ***posteriorly*** on the inferior angle of the scapula[14]

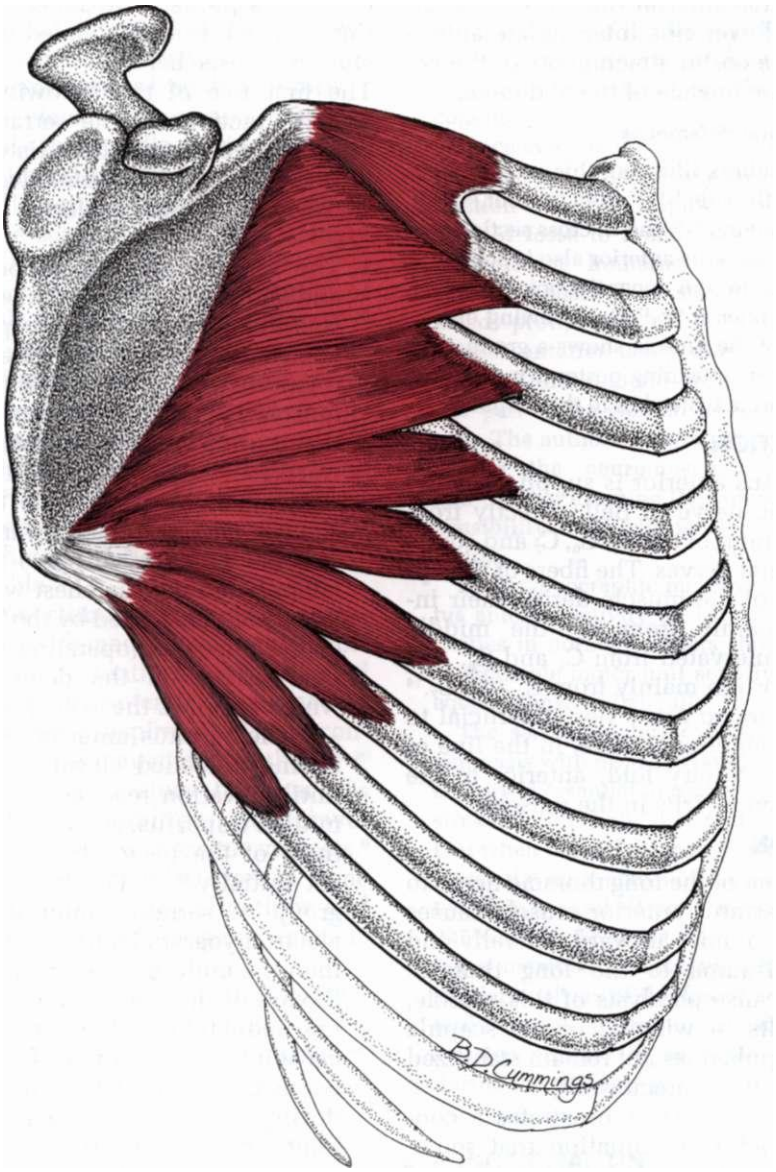


Figure 46.2. Attachments of the right serratus anterior muscle (red). The clavicle has been removed and the scapula rotated backward. The fibers of the muscle are divided into three groups and are identified by their fiber direction and the rib to which each digitation or segment attaches (see Section 2 of text).

(Fig. 46.2). These lowest serrations can develop central TrPs near the midpoint of each group of muscle fibers near the midaxillary line. In a study of 818 cadavers,[34] the most inferior anterior attachment in the third group of fibers included the 6th to 12th ribs. The most common attachment was the 8th rib in males (42.5%) and the 9th rib in females (43.3%). Bilateral symmetry was the rule (70%) for both sexes.

The serratus anterior digitations that attach to the lower ribs interdigitate anteriorly with the costal attachments of the external oblique muscle of the abdomen.

Supplemental References

Other authors illustrate this muscle as seen from the side,[1,19,47] from in front,[15,16] **21.45 from** behind,[2,37] and in cross section.[3, 12.20, 36] **The serratus** anterior also is shown in relation to the long thoracic nerve, which supplies it.[4,5, 17, 38] A drawing of a variation of the muscle shows a group of interior fibers attaching posteriorly to superficial fascia instead of to the scapula.[22]

3. INNERVATION

The serratus anterior is supplied by the long thoracic nerve of Bell, directly from the anterior rami of the C_5, C_6, C_7 and sometimes C_8 spinal nerves. The fibers of the upper portion of the muscle derive their innervation mainly from C_5; the middle portion is innervated from C_5 and C_6, and the lower portion mainly from C_6 and C_7.[14] The long thoracic nerve lies superficial to the serratus anterior muscle, in the line of the anterior axillary fold, anterior to the usual location of TrPs in the muscle.

4. FUNCTION

Stimulation of the long thoracic nerve to the entire serratus anterior muscle causes the scapula to move upward, laterally and forward.[18] Trauma to the long thoracic nerve may cause paralysis of this muscle, which results in winging of the scapula (i.e., the scapula does not remain stabilized snug against the thoracic wall).

The serratus anterior illustrates a confusingly paradoxical situation that sometimes arises with regard to the effect of TrPs and their functional implications. Because of the increased tension caused by the taut bands, one would not expect winging of the scapula to be a symptom of TrPs in the serratus anterior. However, TrPs can have potent reflex effects that are largely uninvestigated and poorly understood. *Clinically, winging can sometimes be relieved by inactivating serratus anterior TrPs.* Weakness in this case could reflect a combination of reflex facilitation of antagonist muscles and inhibition of the serratus anterior muscle. Janda identifies this muscle as one prone to weakness and inhibition,[25] which is substantiated by two EMG studies[23,44] (see below).

The first five of the following eight reported functions of the serratus anterior muscle have been substantiated by electromyography, the last three were not substantiated in normal subjects:

1. The serratus anterior supports flexion and abduction of the arm (because of its stabilizing effect on the scapula and its contribution to upward rotation). Contraction of the most caudal fibers of the muscle laterally rotates the scapula so that it turns the glenoid fossa to face upward.[9, 14, 26, 30, 40] These fibers, when stimulated, initially rotate the inferior angle of the scapula forward.[18] As the serratus contracts to move the scapula laterally around the chest wall, the displacement is resisted by the lower fibers of the trapezius (operating to maintain the position of the deltoid tubercle, which becomes the axis of rotation).[27,28]

   The serratus anterior is not active during unloaded elevation of the arm until elevation reaches about 30°. The middle trapezius, rhomboid, and upper third of the pectoralis major muscles act initially.[18,40] The lower, triangular group of serratus anterior fibers are electromyographically more active than the middle trapezius fibers during flexion of the arm, and *vice versa* during abduction of the arm.[9] Partly because of the migration of the instantaneous center of rotation of the scapula[7] during elevation of the glenohumeral joint, the lowest, triangular group of serratus anterior fibers (with the stabilization provided by the lower trapezius fibers) continue to have an effective lever arm for rotation. This mechanical

advantage becomes increasingly evident when the elevation of the clavicle ends at about 140° of arm elevation and the remaining motion depends more on increased acromioclavicular joint motion.[7]

2. By abducting the scapula, the serratus anterior protracts the shoulder girdle, as when the individual exerts effort to push an object forward.[14,26,40] This also is described as an oblique lateral motion.[46] Thus, this muscle helps to stabilize the scapula against the posterior thorax during forward-pushing efforts.
3. It helps to elevate the scapula. Stimulation of only the *middle* portion (two flat sheets of fibers) elevates the acromion.[18] The middle portion contributes to elevation of the scapula and is increasingly activated as the arm is elevated.[24]
4. This muscle holds the medial border of the scapula firmly against the thorax.[26,30]
5. With the upper limb fixed against a surface, it displaces the thorax posteriorly during a pushup[30] from the floor, or during a push back from the wall.
6. The lowest fibers are said to depress the scapula,[26,30,46] although neither direct stimulation[18] nor electromyography[9] support this contention. This function is questionable.
7. The original stimulation studies and observations were made in severely abnormal muscular situations and indicated that the serratus anterior functioned to support forceful inspiration.[18] This conclusion was perpetuated by others.[8,30] However, an inspiratory function has been refuted by multiple electromyographic studies in *normal* subjects.[9] Clinically, it assists inhalation in some demanding or abnormal situations and is recognized as an "accessory" muscle of inspiration.[17a]
8. Motor unit activity of the serratus anterior is *not* needed to support the shoulder girdle against gravity,[9] as first reported.[24]

Electromyographic monitoring of the serratus anterior during simulated automobile driving showed activity in almost all cases when the top of the steering wheel was rotated contralaterally.[28] Monitoring of the serratus anterior during freestyle swimming revealed that, during most of the pull-through phase of the swimming stroke, in subjects with painful shoulders[44] the EMG activity was less than half of that in subjects with pain-free shoulders.

Fine-wire electrode recordings of EMG activity of the serratus anterior muscle while the subject was pitching a baseball[23] revealed that the activity in subjects with chronic anterior instability of the shoulder was markedly reduced compared to the muscle's activity in subjects with pain-free shoulders. The authors[23] concluded that the neuromuscular imbalance evidenced by the diminished activity of the muscle may decrease protraction of the scapula, causing the glenoid fossa to remain behind the forward-flexing humerus during the late cocking phase. They stated that diminished protraction of the scapula increases anterior laxity due to increased stress of the humeral head on the anterior part of the glenoid labrum and capsule. The authors[23] never explained what caused the neuromuscular imbalance and did not examine the subjects for the possibility that TrPs were a significant factor.

Electromyographic monitoring of serratus anterior activity during 13 sports activities in normal subjects showed slight to moderate motor unit activity of nearly equal intensity bilaterally.[11]

The serratus anterior deserves special emphasis with regard to tennis players. Its activity is essential to each of three tennis strokes.[43] Similarly, it is critically important when normal subjects pitch fast balls. It was the most active of the five muscles tested in 4 baseball pitchers and reached 225% of the EMG activity recorded during a maximal manual muscle test![27] A similar analysis of the free-style and butterfly strokes in swimming[35] showed the serratus anterior to be active primarily during the recovery phase of a stroke.

5. FUNCTIONAL UNIT

Synergistic muscles include the pectoralis minor and upper fibers of the pectoralis major, which also act to protract the

shoulder girdle. The serratus anterior is synergistic with the trapezius in upward rotation of the glenoid fossa. The more vertical fibers of the levator scapulae assist in elevating the scapula as a whole.

Antagonists for abduction are the more horizontal fibers of the latissimus dorsi, the rhomboidei, and the middle trapezius muscles. Glenoid rotation upward is countered by the more vertical fibers of the latissimus dorsi, the levator scapulae muscle, and the pectoral muscles.

6. SYMPTOMS

Chest pain from serratus anterior TrPs may be present at rest in severe cases. When the TrPs are less hyperirritable, pain may be precipitated by deep breathing (i.e., a "stitch in the side") while running. Similar pain also may arise from TrPs in the external abdominal oblique muscle, which interdigitates with the lowest group of serratus anterior fibers, or if the "stitch" is a little lower, it may result from diaphragm TrPs. The runner may press against, or squeeze, the painful area for relief in order to keep going; taking a few slow *full* breaths also may help. Patients have difficulty finding a comfortable position at night and often are unable to lie on the affected muscle. See Section 1 for the referred pain distribution.

Patients with this serratus anterior myofascial syndrome may report that they are "short of breath," or that they "can't take a deep breath, it hurts." They frequently are unable to finish an ordinary sentence without stopping to breathe[49] and patients find this especially bothersome when talking on the telephone. Although these patients are likely to receive a cardiopulmonary workup for dyspnea, at least part of the cause is reduced tidal volume due to restriction of chest expansion by pain or by increased tension of the TrP-afflicted serratus anterior.

Serratus anterior TrPs can contribute to the pain associated with myocardial infarction. The pain has been relieved by inactivating pectoral muscle and serratus anterior TrPs on the left side.[42]

Pain is rarely aggravated by the usual tests for range of motion at the shoulder, but may result from a strong effort to protract the shoulder girdle. Scapulohumeral rhythm may be disrupted by TrPs in the serratus anterior.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Serratus anterior TrPs may be activated by muscle strain during excessively fast or prolonged running, push-ups, lifting heavy weights overhead, or severe coughing due to respiratory disease. Serratus anterior TrPs appear to be particularly vulnerable to torsional stresses-for example, when an automobile driver makes an abrupt forceful turn of a steering wheel without power steering to avoid (or attempt to avoid) an accident or when the thorax rotates vigorously while the upper limb is in a fixed position. High levels of anxiety appear to increase the likelihood of serratus anterior TrPs.[49]

Patients with emphysema do not seem to be especially prone to develop these TrPs, which may be accounted for by the usually over-expanded barrel chest in these patients. This condition would tend to stretch the serratus anterior muscle and would prevent leaving it in a shortened position for any length of time.

8. PATIENT EXAMINATION

Round-shouldered posture and prominence of the superior border and spine of the scapula on the affected side can result from abduction and rotation of the scapula by the tense serratus anterior fibers. Viewed from the back, the scapula stands out. From the front, the patient has a unilateral round-shouldered posture similar to that seen when the pectoralis major muscle develops unilateral active TrPs, but the pectoralis major muscle is usually nearly equally affected on both sides of the body. Some patients may show winging of the scapula due to TrP inhibition of the serratus anterior and facilitation of its antagonists.

The examiner should observe the patient's thoracic movement during respiration. Active TrPs in the serratus anterior muscle inhibit expansion of the lower chest. On inspiration, the patient can expand the upper thoracic cage, but measurement of chest expansion around the lower margin of the rib cage is likely to show marked restriction. After inactivation of

TrPs in this muscle, there is a smaller minimum and a larger maximum lower chest circumference. The resultant marked increase in volume of tidal air is associated with immediate relief of respiratory pain and dyspnea. Also, in the patients who experience a feeling of "air hunger" associated with rapid shallow respirations, the respiratory cycles usually revert to normal depth when all active serratus anterior TrPs have been inactivated.[49]

Before treatment for the serratus anterior TrPs, the patient is likely to overuse the accessory muscles of respiration in the neck, and also to make poor use of the diaphragm. The diaphragmatic dysfunction and the reduced lower chest expansion appear to represent reflex inhibitory influences on respiration since the serratus anterior is normally an accessory respiratory muscle for increased demand rather than a primary muscle of respiration.

The serratus anterior can be tested directly for restricted range of motion by placing the patient in the same position used for spray and stretch *(see* Fig.46.4A). As the patient's elbow is moved posteriorly and lowered toward the table, the position of the scapula is monitored by palpation. Scapular range of adduction may be limited by TrPs and the patient is likely to experience pain at the end of available movement, in contrast to the greater and pain-free range on the contralateral, uninvolved side.

The examiner should stand behind the patient and observe scapulohumeral rhythm while the patient performs arm flexion and abduction. Although the *range* of arm elevation may be within normal limits, scapulohumeral rhythm and muscle balance can be disrupted by serratus anterior TrPs.

Kendall, *et al.*[30] illustrate and describe ways of testing this muscle for weakness. However, muscle weakness is not as reliable an indicator of TrPs as increased muscle tension, shortening, and painful limitation of full stretch range of motion. With sufficiently active TrPs in the muscle, maximum voluntary effort may evoke pain, especially in the shortened position.

9. TRIGGER POINT EXAMINATION (Fig. 46.3)

The TrPs in the serratus anterior muscle are usually located in the subcutaneous portion of the muscle in the midaxillary line at approximately the level of the nipple, over the fifth or sixth ribs,[50] but occasionally they are located higher or lower, as was illustrated by Webber.[52] For examination, the recumbent patient lies (semisupine) turned half-way toward the opposite side with the ipsilateral arm partly extended (Fig. 46.3). When the operator ex-

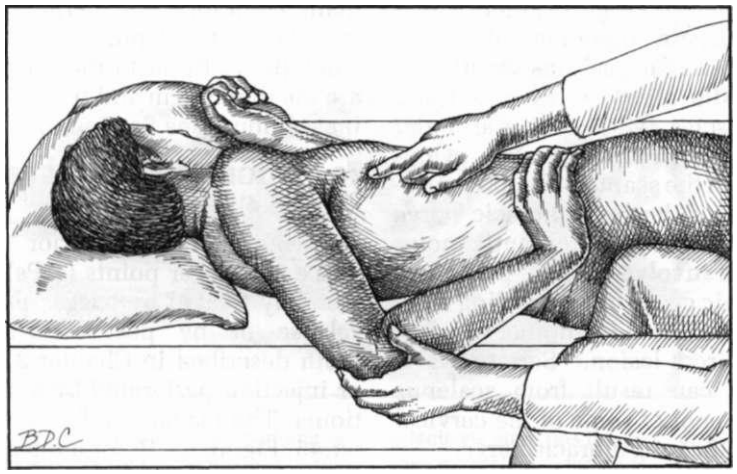


Figure 46.3. Palpation of a trigger point in the right serratus anterior muscle at the level of the sixth rib just anterior to the midaxillary line.

tends the arm backward to adduct the scapula for palpation to locate the TrPs, the midaxillary line projected down the chest appears to be aligned with the anterior axillary fold. Flat palpation against the ribs in this area reveals a tender nodule in a palpable band within the muscle, just under the skin. Pressure on this nodule reproduces the patient's pain complaint. Also, snapping palpation at this point of exquisite tenderness can induce a local twitch response in the palpable taut band.

10. ENTRAPMENT

No reports were found or cases observed of nerve entrapment by the serratus anterior muscle. However, two of the three cervical roots that form the long thoracic nerve pass through the scalenus medius muscle[6,14] and are potentially vulnerable to entrapment by TrP activity in that scalene muscle. Thus, the nerve supply to the serratus anterior muscle may suffer entrapment due to TrPs in the scalenus medius.

11. DIFFERENTIAL DIAGNOSIS

Diagnoses to be distinguished from serratus anterior TrPs include costochondritis, intercostal nerve entrapment, C_7-C_8 root lesions, and herpes zoster.

The chest pain part of the serratus anterior pain pattern must be distinguished from a broken rib and intercostal muscle TrPs. In one patient, the stress fracture of a rib was attributed to serratus anterior tension.[33] The back pain component of the serratus pain pattern requires consideration of TrPs in the middle trapezius, rhomboid, and paraspinal muscles. Midthoracic articular dysfunctions can produce similar symptoms. Winging of the scapula due to a neuropathic process of the long thoracic nerve may not be painful if predominantly motor nerve fibers are involved. Thus, painful or not, a neurogenic cause for the finding must be carefully considered.[13] Another cause of winging is a C_7 root lesion.[32] Serratus anterior weakness can result from scalenus medius entrapment of some of the cervical roots that form the long thoracic nerve.

In the presence of active serratus anterior TrPs, one can sometimes see what looks like elevation or inhalation lesions of ribs 2 through ribs 8 or 9. However, this must be carefully assessed. Abnormal tension of the serratus anterior alone can make it appear as if there is an articular dysfunction when in fact, the apparent articular dysfunction is simply the result of the increased muscle tension caused by the myofascial TrPs. In that case, inactivation of the myofascial TrPs alleviates whatever apparent articular dysfunction is present.

Patients with active TrPs in the serratus anterior muscle often have involvement of only this muscle. They may show no clinical involvement of other muscles in its myotatic (functional) unit. On the other hand, the serratus anterior can be part of a multiple-TrP, predominantly unilateral interscapular pain problem that involves TrPs in the ipsilateral upper and midthoracic paraspinal muscles, including the rhomboids, the middle trapezius, and possibly the serratus posterior superior. Much like the key role that the subscapularis plays in a TrP-caused frozen shoulder syndrome, this back pain syndrome will not clear up until the serratus anterior TrP component has been identified and inactivated.

The other muscles that may become overloaded due to shortening and reduced function of the serratus anterior include the latissimus dorsi, and surprisingly, neck muscles of inspiration, namely, the scalene muscles and the sternocleidomastoid muscle (accessory for inspiration). These associated muscles may develop TrPs that remain latent for a long period of time. Other muscles that can produce a "stitch in the side" (in addition to the serratus anterior) are the diaphragm and the external abdominal oblique *[see* Section 6).

12. TRIGGER POINT RELEASE (Fig. 46.4)

If the serratus anterior is primarily weak, its trigger points (TrPs) are most effectively treated by **trigger point pressure release** or by postisometric relaxation (both described in Chapter 3, Section 12), or injection performed by a skilled practitioner. The reader is referred also to Chapter 45, Figure 45.10 for a release procedure for upper intercostal muscle tension which, with a variation in hand position, can be effective for releasing TrP tension in

the upper and middle portions of the serratus anterior muscle.

For **spray and stretch,** the patient lies on the uninvolved side with the back toward the operator and the uppermost arm drawn backward (Fig. 46.4A) so that the weight of the patient's arm can help to initiate passive stretch of the serratus anterior. Before and during release, the clinician sprays the vapocoolant anteroposteriorly over the muscle (icing strokes can be used instead of spray, as described in Chapter 3). The patient assumes the position shown in Figure 46.4B and the clinician monitors the progress of release and assists in taking up the slack with the other hand as the patient's arm weight assists the scapular adduction. The patient's pelvis is blocked from rotation by the clinician's hip. During this stretch, the patient takes a deep breath and momentarily holds it to enlarge the lower rib cage. This further stretches the muscle, while the vapocoolant spray is applied in slow parallel sweeps as in Figure 46.4A, from the TrP area backward along the line of the muscle fibers, then over the

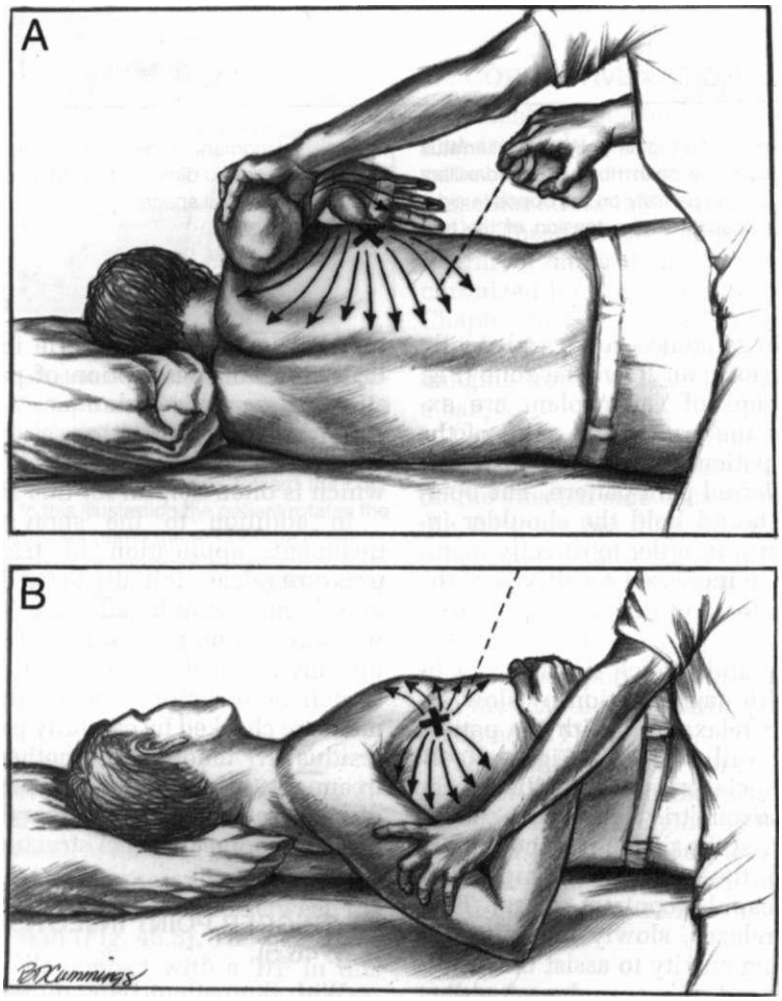


Figure 46.4. Stretch position and spray patterns *(arrows)* for a trigger point (**X**) of the right serratus anterior muscle in the midaxillary line. **A,** initial side-lying position. **B,** full adduction of the right scapula, which effectively stretches the serratus anterior if the operator's hip stabilizes the patient's pelvis to prevent backward body rotation. The operator's hand should hold the patient's right shoulder to guide the scapula back.

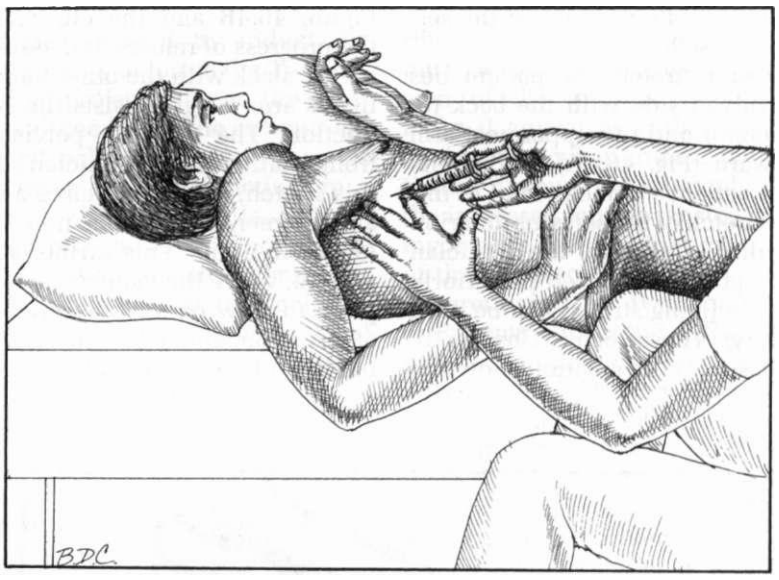


Figure 46.5. Injection of a trigger point in the serratus anterior muscle over the sixth rib in the midaxillary line. The patient is lying partially on the opposite side, Adduction of the scapula and extension of the arm moves the landmark of the anterior axillary fold backward. The needle is directed toward an underlying rib, avoiding intercostal spaces.

posterior pain reference zone, and finally over the anterior pain reference zone (Fig. 46.4B). Sweeps of vapocoolant are extended down the arm to the palm of the hand in the patient who experiences this part of the referred pain pattern. The operator's hand should hold the shoulder instead of the arm in order to directly monitor and have an increased sensitivity of the progressive release of the serratus anterior muscle.

This spray and stretch method can be combined with augmentation by slow exhalation with relaxation. With the patient in the position illustrated in Figure 46.4B, with the clinician's hand stabilizing the scapula, postisometric relaxation can be performed. First the patient reaches anteriorly (toward the ceiling), attempting to abduct the scapula (contract phase). Then the patient relaxes, slowly breathing out while allowing gravity to assist the arm to drop down and the scapula to adduct, lengthening the serratus anterior. Lewit described and illustrated the use of postisometric relaxation for releasing tension (due to TrPs) in this muscle.[31] He included illustration and description of patient self-stretch using this technique. Addition of *gentle* voluntary effort to assist the stretch adds the effect of **reciprocal inhibition,** which is often helpful for this muscle.

In addition to the spray-and-stretch treatment, application of **trigger point pressure release** initially to the TrPs in this muscle may be quite effective. This finger-pressure technique also is useful to "clean up" any residual TrPs following spray and stretch or injection. The results of treatment are checked by carefully palpating for residual TrP tenderness. Another beneficial treatment technique for this region is myofascial release of the pectoral, thoracic, and lumbodorsal fascial structures.

13. TRIGGER POINT INJECTION (Fig. 46.5)

With the patient lying on the contralateral side, as for spray and stretch, a serratus anterior trigger point (TrP) is located by flat palpation and pinned against a rib between

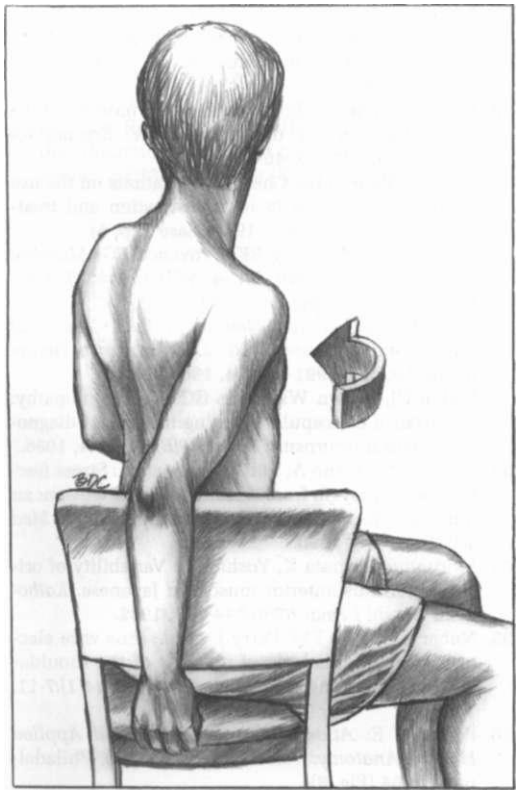


Figure 46.6. Self-stretch of the serratus anterior, patient seated. The patient stabilizes the scapula of the involved side by placing the ipsilateral arm behind the chair back. After taking in a deep breath, the patient exhales slowly and turns the thorax toward the contralateral side. In this illustration the patient rotates the thorax toward the left (turns the front of the chest toward the left) in order to stretch the right serratus anterior. Some patients learn how to facilitate the muscle relaxation and muscle lengthening by including postisometric relaxation.

the fingers of one hand. The needle is directed toward the rib, at a shallow angle *nearly* tangential with the chest wall until the needle tip encounters the TrP. The TrP lies in the thin layer of muscle between the rib and the skin (Fig. 46.5). The pain reaction on needle contact with a TrP in this muscle is often less intense than the response from TrPs in many other muscles.

One should be scrupulously careful to replace immediately any needle that has developed a burr on its tip due to contact with bone.

Since the long thoracic nerve supplies exclusively the serratus anterior muscle, some degree of anesthesia of this motor nerve is to be expected when injecting an anesthetic. However, the patient is not likely to notice only temporary weakness of part of the serratus anterior muscle in the absence of any change in skin sensation.

In our experience, no patient has reported symptoms indicating that a nerve block had resulted from the injection. Rachlin[39] illustrated and described a similar injection technique.

14. CORRECTIVE ACTIONS

Patients must avoid or modify activities that are likely to reactivate TrPs in the serratus anterior muscle, particularly the muscular stress that activated the TrPs initially. These patients should learn to clear the throat rather than to cough, to use synchronized (not paradoxical) breathing *[see* Chapter 20, Section 14), to avoid push-ups and heavy overhead lifting, and to avoid hanging from, or chinning themselves on, a bar.

Patients with very irritable TrPs in the serratus anterior often are unable to sleep on the affected side because of pressure on the TrPs, nor are they able to sleep on the other side if the arm of the affected side falls forward onto the bed and places the muscle in a cramped, shortened position. The latter problem is remedied by use of a pillow to support the arm, and to keep it and the scapula from falling forward, as illustrated in Figure 22.6.

The seated patient can do the Serratus Anterior Self-stretch Exercise as described and illustrated in Figure 46.6. The patient also can do the In-doorway Stretch Exercise in the lower and middle hand-positions *[see* Fig. 42.9).

Case *Reports*

The management of a patient with serratus anterior TrPs, including injection of procaine, is presented by Dr. Travell.[49]

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991, p. 83 (Fig. 2.7).
2. *Ibid.* p. 234 (Fig. 4.48).
3. *Ibid.* p. 371 (Fig- 6.19A).
4. *Ibid.* p. 378 (Fig- 6.28).
5. *Ibid.* pp. 375, 376 (Figs. 6.25, 6.26).
6. *Ibid.* p. 555 (Fig. 8.4).
7. Bagg SD, Forrest WJ: A biomechanical analysis of scapular rotation during arm abduction in the scapular plane. *Am J Phys Med Rehabil 67(6):238-*245, 1988.
8. Bardeen CR: The musculature. Section 5. In: *Morris's Human Anatomy.* Ed. 6. Edited by Jackson CM. Blakiston's Son & Co., Philadelphia, 1921 (p. 394).
9. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 263, 267, 426, Fig. 12.1).
10. Bonica JJ, Sola AE: Chest pain caused by other disorders. Chapter 58. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, Philadelphia, 1990, pp. 1114-1145 (see p. 1133).
11. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sports Skill.* Charles C Thomas, Springfield, Ill., 1967.
12. Carter BL, Morehead J, Wolpert SM, ef *al.: Cross-Sectional Anatomy.* Appleton-Century- Crofts, New York, 1977 (Sects 20-29).
13. Chandler FA: Isolated paralysis of the serratus anterior muscle. *Surg Clin North Am* 25:21-27, 1945.
14. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 521, 1207, 1209).
15. *Ibid.* (Fig. 6-45).
16. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 12, 233).
17. *Ibid.* (Figs. 19, 20).
17a. De Troyer A: Mechanics of the chest wall muscles. Chapter 6. In: *Neural Control of the Respiratory Muscles.* Edited by Miller AD, Bianchi AL, Bishop BP. CRC Press, New York, 1997:59-73 (p. 68).
18. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 24-36, 45).
19. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (Fig. 52).
20. *Ibid.* (Fig. 68).
21. *Ibid.* (Fig. 76).
22. *Ibid.* (Fig. 77).
23. Glousman R, Jobe F, Tibone J, *et al.:* Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg 70A(2):220-226,* 1988.
24. Inman VT, Saunders JB, Abbott LC: Observations on the function of the shoulder joint. *J Bone Joint Surg* 26.1-30, 1944 (p. 26).
25. Janda V: Evaluation of muscular imbalance. Chapter 6. In: *Rehabilitation of the Spine: A Practitioner's Guide.* Edited by Liebenson C. Williams & Wilkins, Baltimore, 1996 (pp. 97-112).
26. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Back.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 83, 84).
27. Jobe FW, Moynes DR, Tibone JE, *et al.:* An EMG analysis of the shoulder in pitching: a second report. *Am J Sports Med 12(3) :2\8-220,* 1984.
27a. Johnson G, Bogduk N, Nowitzke A, et al .'Anatomy and actions of the trapezius muscle. *Clin Biomech* 9:44-50, 1994.
28. Jonsson S, Jonsson B: Function of the muscles of the upper limb in car driving, Part IV. *Ergonomics* 28:643-649, 1975 (p. 464).
29. Kelly M: Pain in the Chest: Observations on the use of local anaesthesia in its investigation and treatment. *Med J Aust 1:4-7,* 1944 (Case 2, p. 5).
30. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (pp. 288, 289).
31. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 24, 198-200).
32. Makin GJ, Brown WF, Ebers GC: *C,* radiculopathy: importance of scapular winging in clinical diagnosis. *J Neurol Neurosurg Psych* 49(6):640-644, 1986.
33. Mintz AC, Albano A, Reisdorff EJ, *et al:* Stress fracture of the first rib from serratus anterior tension: an unusual mechanism of injury. *Ann Emerg Med 19(4):411-414,* 1990.
34. Morimoto I, Hirata K, Yoshida S: Variability of origin of serratus anterior muscle in Japanese. *Kaibogaku ZasshiJ Anat 67(6):744-748,* 1992.
35. Nuber GW, Jobe FW, Perry J, *et al.:* Fine wire electromyography analysis of muscles of the shoulder during swimming. *Am J Sports Med 14(l):7-\\,* 1986.
36. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy.* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 8).
37. *Ibid.* (Fig. 28).
38. *Ibid.* (Fig. 39).
39. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (p. 212).
40. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Ed. 6. Lea & Febiger, Philadelphia, 1978 (pp. 153, 154).
41. Rinzler SH: *Cardiac Pain.* Charles C Thomas, Springfield, Ill., 1951 (pp. 79, 80, 82).
42. Rinzler SH, Travell J: Therapy directed at the somatic component of cardiac pain. *Am Heart J* 35:248-268, 1948 (pp. 255-257, Case 1).
43. Ryu RK, McCormick J, Jobe FW, *et al.:* An electromyographic analysis of shoulder function in tennis players. *Am J Sports Med 16(5):481-485,* 1988.
44. Scovazzo ML, Browne A, Pink M, *et al:* The painful shoulder during freestyle swimming: an electromyographic cinematographic analysis of twelve muscles. *Am J Sports Med 19(B):577-582,* 1991.
45. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 283).
46. Steindler A: *Kinesiology of the Human Body.* Charles C Thomas, Springfield, Ill., 1955 (pp. 468, 469).
47. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (p. 277).
48. Travell J: Referred pain from skeletal muscle: the pectoralis major syndrome of breast pain and soreness and the sternomastoid syndrome of headache and

dizziness. *NY State J Med* 55:331-339,1955 (p. 333).
49. Travell J, Bigelow NH: Role of somatic trigger areas in the patterns of hysteria. *Psychosom Med* 9:353-363, 1947 (pp. 354, 355).
50. Travell J, Rinzler SH: Pain syndromes of the chest muscles: Resemblence to effort angina and myocardial infarction, and relief by local block. *Can Med Assoc J* 59:333-338, 1948 (Case 1, p. 256).
51. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 23:425-434,1952 (p. 429, Fig. 3).
52. Webber TD: Diagnosis and modification of headache and shoulder-arm-hand syndrome. *J Am Osteopath Assoc* 72:697-710, 1973 (p. 10, Fig. 31).
53. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little, Brown & Company, Boston, 1988 (p. 212, Fig. 12-3).

CHAPTER 47
Serratus Posterior Superior and Inferior Muscles

Section A
Serratus Posterior Superior

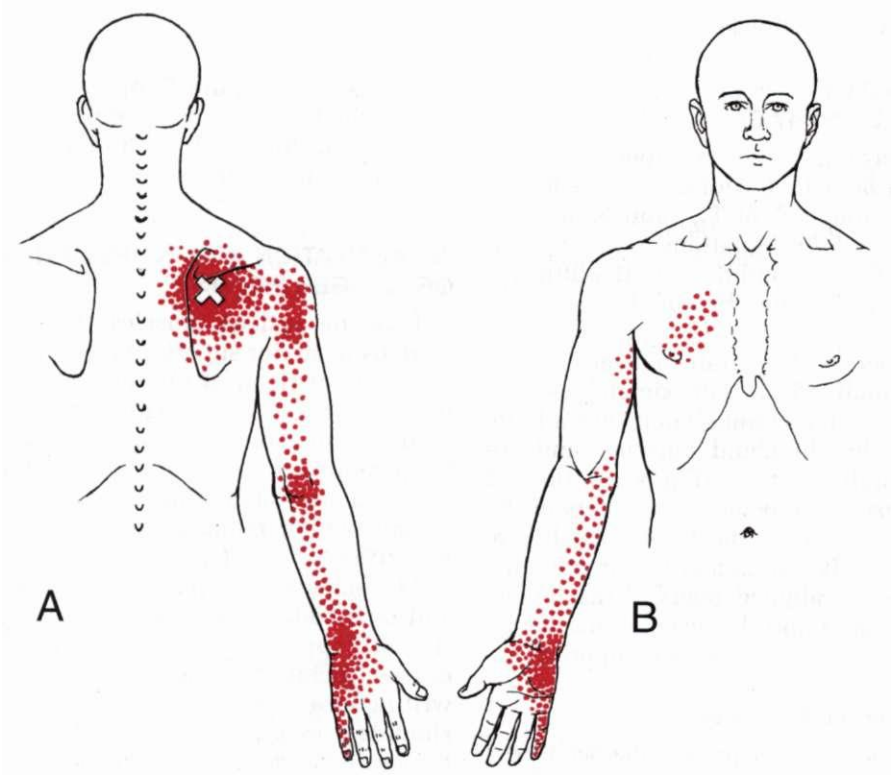
HIGHLIGHTS: Referred pain from trigger points (TrPs) in the serratus posterior superior is a frequent source of deep scapular pain. **REFERRED PAIN** from this muscle is strongly felt deep under the upper portion of the scapula, often with extension to the back of the shoulder, the upper triceps area, the elbow, ulnar side of the forearm and hand, and to the entire little finger. **ANATOMY:** The attachments of the serratus posterior superior are to the dorsal midline fascia from C_6 through T_2, above, and to the second through fifth ribs, below and laterally. The **FUNCTION** established for this muscle is to assist inspiration. The **SYMPTOM** of pain may be increased by reaching out forward with the hands or by sidelying on the ipsilateral side. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** can occur from overloading caused by posture and activities or by vigorous respiratory effort, as by coughing or paradoxical breathing. **TRIGGER POINT EXAMINATION** requires strong abduction of the scapula to uncover the TrPs and make the sensitive attachment TrPs accessible to palpation against the ribs. **TRIGGER POINT RELEASE** can be performed by prespray and release of the TrPs or by trigger point pressure release. Sometimes injection of serratus posterior superior TrPs is necessary because of their relatively inaccessible location for manual release techniques. For **TRIGGER POINT INJECTION** the needle is directed into the TrP, which is pinned down against a rib, taking care not to penetrate between ribs. **CORRECTIVE ACTIONS** include learning abdominal breathing and the home application of trigger point pressure release.

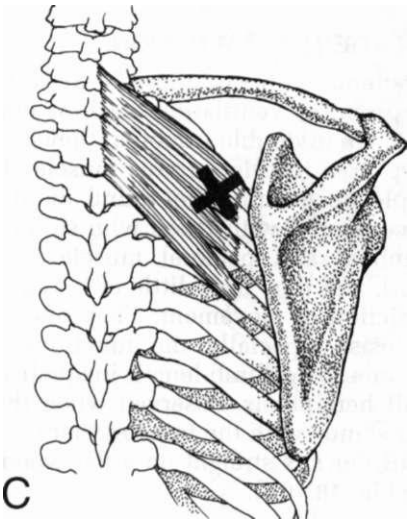
1A. REFERRED PAIN
(Fig. 47.1)

The most annoying trigger points (TrPs) in the serratus posterior superior muscle are the attachment TrPs (ATrPs), one of which is illustrated in Figure 47.1C. The problem occurs when the bony scapula squeezes the sensitive region of enthesopathy against the underlying rib to which the muscle fibers attach. Among 76 painful shoulders in 58 patients, this muscle was a cause of pain in 98%, and the single source of pain in 10%.[25]

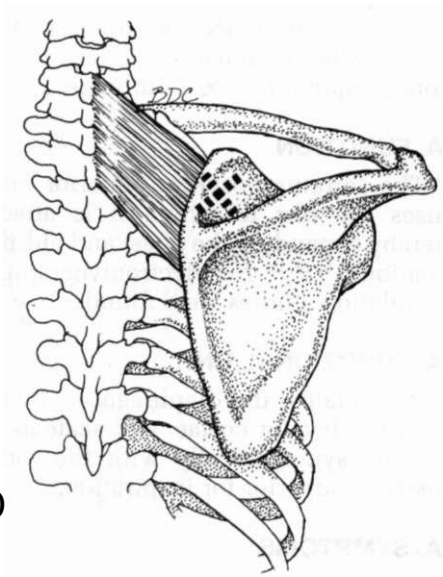
The essential pain reference of this muscle is a *deep* ache under the upper portion of the scapula (Fig. 47.1A). When asked to point to the painful area, patients usually reach back with the opposite arm, but are unable to touch the sore area because the shoulder blade covers it. This pain is perceived as deeper than the similar upper back pain that arises from TrP_5 in the middle trapezius. Pain is also usually felt intensely over the posterior border of the deltoid and the long head of the triceps brachii muscles.[24,26] It often covers the entire triceps region with an accent on the olecranon process of the elbow and occasionally includes the ulnar side of the forearm, hand, and all of the little finger. Anteriorly, the pectoral region may occasionally be painful (Fig. 47.**1B)**

Not only do these TrPs cause referred pain, but they also often refer what the patient interprets as numbness into the C_8-T_1 distribution of the hand.[15]





Trigger point
palpable



Trigger point
not palpable

Figure 47.1. Referred pain pattern of a trigger point (X) in the right serratus posterior superior muscle. Essential pain is *solid red,* spillover pain is *stippled red.* **A,** back view of pain pattern. **B,** front view of pain pattern. **C,** scapula abducted, making the attachment trigger point (X) accessible to palpation and injection. **D,** scapula in the normal rest position, and the attachment trigger point *(dashed* X) is inaccessible.

2A. ANATOMY (Figs. 47.2 and 47.3)

The serratus posterior superior muscle attaches ***above*** to the dorsal midline fascia, from C_6 through T_2 or T_3,[1a] and ***below*** and ***laterally*** by four digitations, to the cranial borders of the second through the fifth ribs (Fig. 47.2). The number of digitations is variable.[5]

The fibers of the serratus posterior superior are inclined at approximately 45° to the horizontal, lie immediately beneath the fibers of the rhomboid muscles, and are nearly parallel to them (Fig. 47.3). Both of these muscles lie beneath the fibers of the trapezius muscle, most of which are aligned nearly horizontally. Paraspinally, the vertically aligned fibers of the longissimus thoracis and iliocostalis muscles lie deep to the serratus posterior superior.

Supplemental References

Anatomy atlases present the serratus posterior superior as seen from behind,[1, 6, 9, 11, 18, 22, 23] from the side,[7] and in cross section.[3]

3A. INNERVATION

The serratus posterior superior muscle is innervated by the anterior primary divisions of spinal nerves T_1 through T_4.[5]

4A. FUNCTION

The serratus posterior superior muscle raises the ribs to which it is attached, thereby expanding the chest and aiding inhalation.[2, 5, 14, 20] No electromyographic or stimulation studies were found.

5A. FUNCTIONAL UNIT

Presumably, the diaphragm and the intercostal, levator costae, and scalene muscles act synergistically with the serratus posterior superior for inspiration.

6A. SYMPTOMS

The patient complains of a steady deep ache at rest, as described in Section 1A. Little or no change in the intensity of pain occurs with unloaded movements. However, pain may be increased by lifting objects with outstretched hands, or by other activities or a posture (lying on the same side) which cause the scapula to press against attachment TrPs in the serratus posterior superior muscle.

7A. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

Like the scalene muscles, TrPs in the serratus posterior superior muscle are activated by overload of the thoracic respiratory effort because of coughing, as in pneumonia, asthma or chronic emphysema, and by paradoxical breathing (use of the diaphragm and abdominal muscles out of phase), which reduces tidal volume (see Fig. 20.15A).

Movements and postures that stretch and overload the serratus posterior superior also appear to activate its TrPs. Those causes include sitting for long periods writing at a high desk or table, when the shoulders are elevated and rotated forward to permit the arms to reach the high surface; repeatedly reaching to the rear of a high work surface, as by laboratory technicians, and protrusion of the thorax against the scapula by scoliosis.

8A. PATIENT EXAMINATION

Patients with intrathoracic disease that compromises ventilation, such as emphysema, are in double trouble if they also develop TrPs in this serratus muscle. These people are generally *not* round-shouldered (as compared with those who suffer from rhomboid and pectoral muscle involvement), and they have little or no apparent restriction of movement. They often have scoliosis, especially the functional type due to a lower limb-length inequality and small hemipelvis, observed when the patient stands with the feet together (see Fig. 48.9B), or sits straight on a flat wood seat (see Fig. 48.10B).

9A. TRIGGER POINT EXAMINATION (Fig. 47.4)

The patient sits and leans forward slightly, with the arm hanging forward and down on the side to be examined (Fig. 47.4), or with the homolateral hand placed

in the opposite axilla, to fully abduct the scapula.[25] The scapula **must** be abducted and pulled laterally to uncover the serratus TrPs beneath the scapula (Figs. 47.1C and 47.4). The serratus posterior superior is palpated through the trapezius and rhomboid muscles (Fig. 47.3), as also illustrated by Michele *et al.*[17] Snapping palpation may elicit local twitch responses of TrPs in the overlying trapezius fibers, which can be identified because of the nearly horizontal orientation of those superficial fibers. However, local twitch responses in the deeper, obliquely oriented rhomboid and serratus fibers are not so readily perceived but may be palpable.

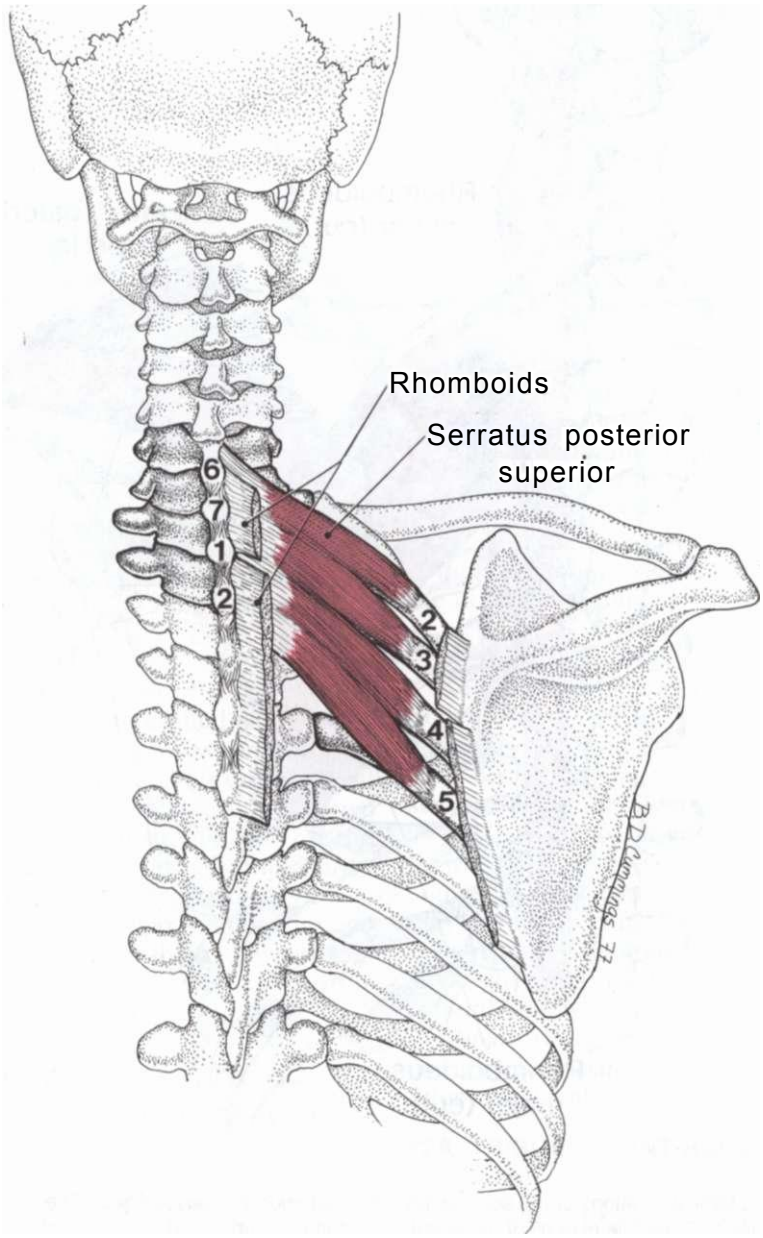


Figure 47.2. Attachments of the serratus posterior superior muscle (red) to numbered vertebrae and ribs.

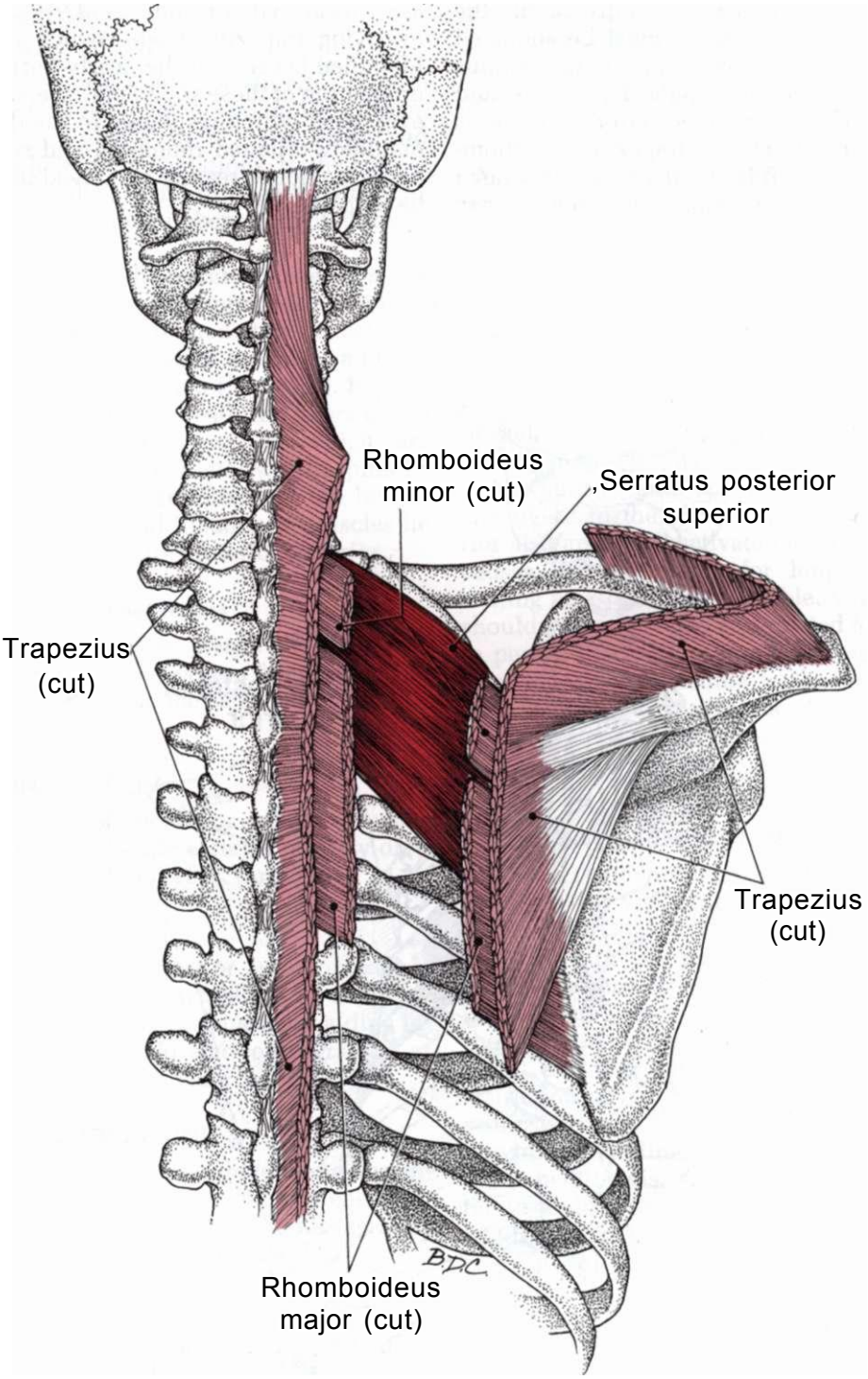


Figure 47.3. Anatomical relations of the serratus posterior superior muscle (*dark red*). The cut trapezius and rhomboid muscles *(light red)* lie over all of the serratus posterior superior, and the iliocostalis and longissimus thoracis muscles (not shown) lie beneath part of this muscle.

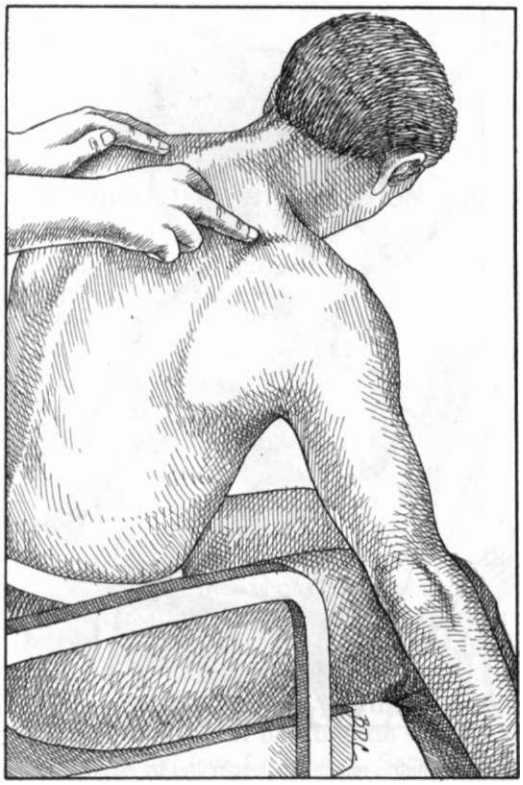


Figure 47.4. Digital examination of the right serratus posterior superior muscle. The scapula **must** be abducted to uncover the exquisitely tender attachment trigger point area (see Fig. 47.1 **C** and **D).**

A serratus TrP is identified as a spot of exquisite deep tenderness when palpated against an underlying rib. It is unlikely that a taut band will be palpable through two muscles. When pressure on either a central TrP or attachment TrP induces the characteristic serratus referred pain pattern that patients recognize as their pain, it convincingly demonstrates to them the relationship between this myofascial TrP and the pain they are suffering.'

10A. ENTRAPMENT

No nerve entrapment has been attributed to this muscle.

11A. DIFFERENTIAL DIAGNOSIS

Differential diagnoses for this muscle include thoracic outlet syndrome, C_7-C_8 radiculopathy, olecranon bursitis, and ulnar neuropathy. The referred pain pattern of this muscle mimics the distribution of pain caused by eighth cervical root compression[21] and this diagnosis must be considered. This confusion is further aggravated by the referred numbness into the C_8-T_1 distribution of the hand[15] so that physicians commonly make the diagnosis of C_8-T_1 radiculopathy when the symptoms are caused by TrPs in this muscle. The serratus myofascial syndrome causes no primary neurological deficit. A radiculopathy *per se* causes no TrP tenderness, palpable bands, or referred pain evoked by pressure applied to the muscle.

Fourie[12] described a scapulocostal syndrome associated with fibrositis (old terminology that included myofascial TrPs). The pain and tenderness was caused by enthesopathy of the lateral attachments of the serratus posterior superior digitations to the ribs.

Articular dysfunction associated with this muscle usually occurs at the T_1 level. There is usually exquisite tenderness directly over the spinous process of this segment. On inspection, this configuration of articular dysfunctions presents as a regional extension of the upper thoracic spine with inability to flex forward across the involved segments.

Related Trigger Points

Key TrPs in the scalene muscles can induce satellite TrPs in the serratus posterior superior" and occasionally the relationship occurs in the reverse direction; the serratus posterior superior can be the key.

The TrPs in the serratus posterior superior lie within the pain reference zone of the synergistic scalene muscles. The scalene TrPs may mimic, in part, the pain pattern of the serratus posterior superior. The neck should always be examined for scalene TrPs if a TrP is found in the serratus posterior superior.

The overlying rhomboid and nearby iliocostalis, longissimus thoracis, and multifidus muscles also may have associated TrPs.

12A. TRIGGER POINT RELEASE (Fig. 47.5)

In addition to the manual spray and release technique described here, other techniques including trigger point (TrP) pressure release of the central TrPs (CTrPs) and

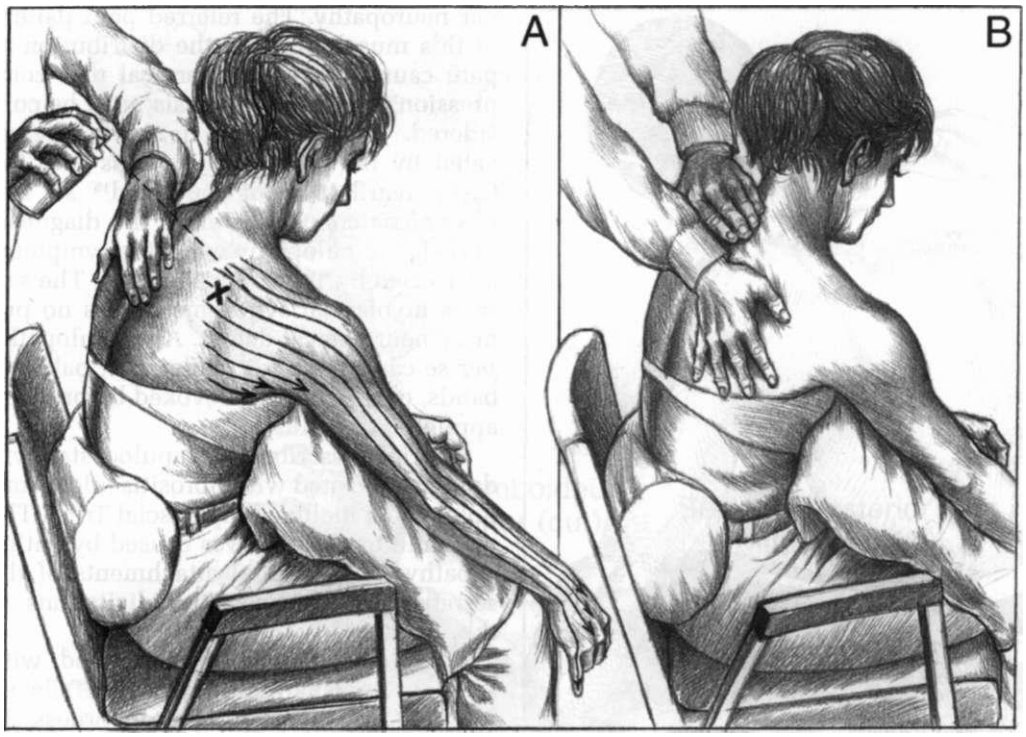


Figure 47.5. Application of vapocoolant *(arrows)* and manual release for a trigger point region **(X)** in the serratus posterior superior muscle. **A,** patient seated comfortably and relaxed with the upper thoracic spine flexed, and with the arm supported in a forward position to swing the scapula laterally. The operator applies sweeps of vapocoolant (or ice) in the pattern indicated, preparatory to manual release. **B,** manual release of trigger point tension is accomplished with postisometric relaxation, beginning by having the patient look up to the left and breathe in. Then, as the patient augments relaxation by looking down and **slowly** breathing out, the operator takes up slack in the muscle. One hand (left in this case) stabilizes the spinous processes; the other hand applies light steady pressure downward and laterally directly on the rib cage medial to the scapula, helping to release the serratus posterior superior muscle.

limited iontophoretic application of steroids (as described in Chapter 3, Section 12 for attachment TrPs) are also effective for release of TrPs in the serratus posterior superior muscle. The primary therapeutic approach to *attachment* TrPs is to inactivate the central TrPs that are causing them. The CTrPs may be difficult to locate accurately in this muscle because they lie under two other muscles or the scapula and may be situated over soft intercostal muscle instead of a firm rib.

The spray and release technique is performed as described and illustrated in Figure 47.5. An initial prespray (Fig. 47.5A) of vapocoolant (or stroking with ice) is applied in slow parallel sweeps laterally and downward over the course of the muscle fibers and then outward over the shoulder and down the arm. Lines of spray should cover the referred pain pattern, which includes the 5th digit (Figs. 47.1A and 47.5). The prespray is followed immediately by manual release (Fig. 47.5B).

When the patient is in the spray and release position, trigger point pressure release is easily applied to any central TrPs that lie directly over a rib. This finger pressure therapy is often helpful and it is most effective if the muscle is on moderate (nonpainful) stretch while pressure is applied.

The position of placing the arm across the chest and the hand under the opposite axilla, while useful for examination of the

muscle, should not be used for treatment. This extreme position of the arm tends to elevate, rather than to lower, the rib cage and makes it difficult for the patient to achieve full relaxation of chest musculature.

13A. TRIGGER POINT INJECTION (Fig. 47.6)

Even if spray and stretch and trigger point pressure release are not effective, injection of the trigger points (TrPs) usually succeeds, but this carries a significant hazard of pneumothorax if performed without precautions and adequate skill.

With the patient lying on the opposite side and the scapula fully abducted (Fig. 47.6), a TrP is precisely located and fixed with the fingers against an underlying rib. The needle is directed nearly tangent to the skin and at all times is pointed toward a rib, not toward an intercostal space, as the operator or the patient might sneeze or unexpectedly startle and jump. This technique is also illustrated by Rachlin.[19] In this location, the Hong method of holding the syringe is recommended (see Chapter 3, Section 13). Here, the possibility of causing a pneumothorax must always be kept in mind.

Following TrP injection, the patient flexes the upper thorax forward during inhalation and takes three *slow* full-in, full-out breaths to move this muscle through its full range of motion. Spray and stretch are repeated, as above, and moist heat applied over the muscle.

14A. CORRECTIVE ACTIONS

It is most important that the patient uses coordinated chest and abdominal breathing *[see* Fig. 20.15C and **D)** and not paradoxical breathing, to minimize overload of the upper-chest accessory muscles of inspiration.

The patient should maintain normal lumbar lordosis, both standing and sitting. When seated, this is facilitated by placing an appropriately sized lumbar pillow in the small of the back, then relaxing and leaning against the back of the chair so that the pillow maintains both the normal lumbar and thoracic curves without muscle strain (*see* Fig. 41.4E).

While supine, the patient may find it possible to apply trigger point pressure release by lying on a tennis ball placed under the interscapular region *[see* Chapter 18 text relating to Fig. 18.4 and *see* Chapter 22, Section 14), if, for this muscle, the scapula is abducted sufficiently. As an alternate part of the home program, a companion may be taught to apply trigger point pressure release to this TrP.

Figure 47.6. Injection of a trigger point in the serratus posterior superior muscle. The scapula must be abducted to reach the tender attachment trigger points in this muscle. The needle is directed nearly tangent to the chest wall and toward a rib to *avoid* penetrating an intercostal space and causing a pneumothorax.

Section B
Serratus Posterior Inferior

HIGHLIGHTS: **REFERRED PAIN** from the serratus posterior inferior muscle is relatively local, near the trigger point (TrP) and usually is identified as an annoying ache that remains after the pain from associated paraspinal TrPs has been relieved. The serratus posterior inferior pain extends over and around the muscle. **ANATOMY:** The attachments of this muscle anchor, above and laterally, to the lowest four ribs. Below and medially, it attaches by an aponeurosis to the spinous processes of the last two thoracic and the first two lumbar vertebrae. **FUNCTION** of this muscle is to depress the lower ribs, and probably to rotate the lower thorax when acting on one side and to extend it with bilateral activation. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** usually results from an acute back strain, which may also activate TrPs in the nearby major back muscles at the same time. **TRIGGER POINT EXAMINATION** is made by flat palpation across the direction of the muscle fibers. Central TrPs are distinguished from attachment TrPs. **DIFFERENTIAL DIAGNOSES** include renal diseases, lower thoracic radiculopathy, and articular dysfunction. **TRIGGER POINT RELEASE** employs a manual release technique that can include prespray. Trigger point pressure release is often helpful. **TRIGGER POINT INJECTION** of this muscle requires that the needle be directed toward a rib, not between ribs. **CORRECTIVE ACTIONS** include relief of chronic stresses on the muscle by correcting a small hemipelvis or leg-length discrepancy, by adding a lumbar support to the straight backrest of a chair, by sleeping on a nonsagging mattress, and by the normalization of paradoxical breathing.

1B. REFERRED PAIN
(Fig. 47.7)

An active trigger point (TrP) in the serratus posterior inferior muscle produces aching discomfort over and around the muscle (Fig. 47.7). The pain extends across the back and over the lower ribs. Patients are likely to identify this annoying ache as muscular in origin. Occasionally, the pain is perceived as extending through the chest to the front.

2B. ANATOMY
(Fig 47.8)

The serratus posterior inferior muscle attaches ***medially*** to the thin aponeurosis from the spinous processes of the last two thoracic and the first two lumbar vertebrae. ***Laterally*** its four digitations attach to the lowest four ribs just medial to their angles[5] (Fig. 47.8). The digitations to one or more ribs, especially to the ninth and twelfth ribs, are sometimes missing. Occasionally the entire muscle is absent.[7]

Supplemental References

Other authors have illustrated the muscle clearly as seen from behind,[1,6,9,16,18,22] from the side,[8,22] and in cross section.[4] A variation of the muscle is viewed from behind.[10]

3B. INNERVATION

The serratus posterior inferior is supplied by branches of the anterior primary divisions of thoracic spinal nerves 9 through 12.[5] It is not supplied by the posterior divisions, as are the paraspinal muscles.

4B. FUNCTION

This muscle attaches to the lower ribs and has been reported as an exhalation[20] muscle or as a muscle that stabilizes the lower ribs against the upward pull of the diaphragm.[5,14] However, an electromyographic study found no respiratory activity attributable to the muscle.[2] Unilateral contraction should contribute effectively to trunk rotation, and bilateral contraction to extension of the lower thorax.

5B. FUNCTIONAL UNIT

The serratus posterior inferior muscle appears to act synergistically with the iliocostalis and longissimus thoracis muscles of the same side, unilaterally for rotation

and bilaterally for extension of the spine. As an accessory muscle of exhalation, it would likely act synergistically with the quadratus lumborum muscle.

6B. SYMPTOMS

After symptoms due to active TrPs in associated major muscles of the back have been eliminated, the patient may be left with a nagging ache in the lower thoracic region. The ache is annoying, but not a severely threatening pain. Patients may report that squirming and stretching provide some relief.

Maximal deep inhalation and coughing usually do not evoke pain from the serratus posterior inferior, as they may from active TrPs in the serratus anterior, quadratus lumborum, and deep abdominal wall muscles.

7B. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

This is one of the many back muscles that are susceptible to strain during the combined movement of lifting, turning and reaching. Active TrPs in the serratus posterior inferior develop due to overload strain at the same time as TrPs in associated muscles. Standing on a ladder with the back hyperextended to reach up and work overhead has activated TrPs in this muscle, and paradoxical breathing and unequal leg lengths may perpetuate them.

8B. PATIENT EXAMINATION

Patients may have slight restriction of thoracolumbar flexion and of spinal extension due to pain, and may be limited in rotating the torso away from the painful side.

9B. TRIGGER POINT EXAMINATION

A nodule in a taut band in this inferior serratus muscle may be difficult to palpate through or distinguish from the overlying latissimus dorsi muscle (for this anatomical relationship *see* Fig. 4.25 in Volume 2). However the midfiber spot tenderness of central TrPs is usually identifiable. The exquisite tenderness of attachment TrPs at the

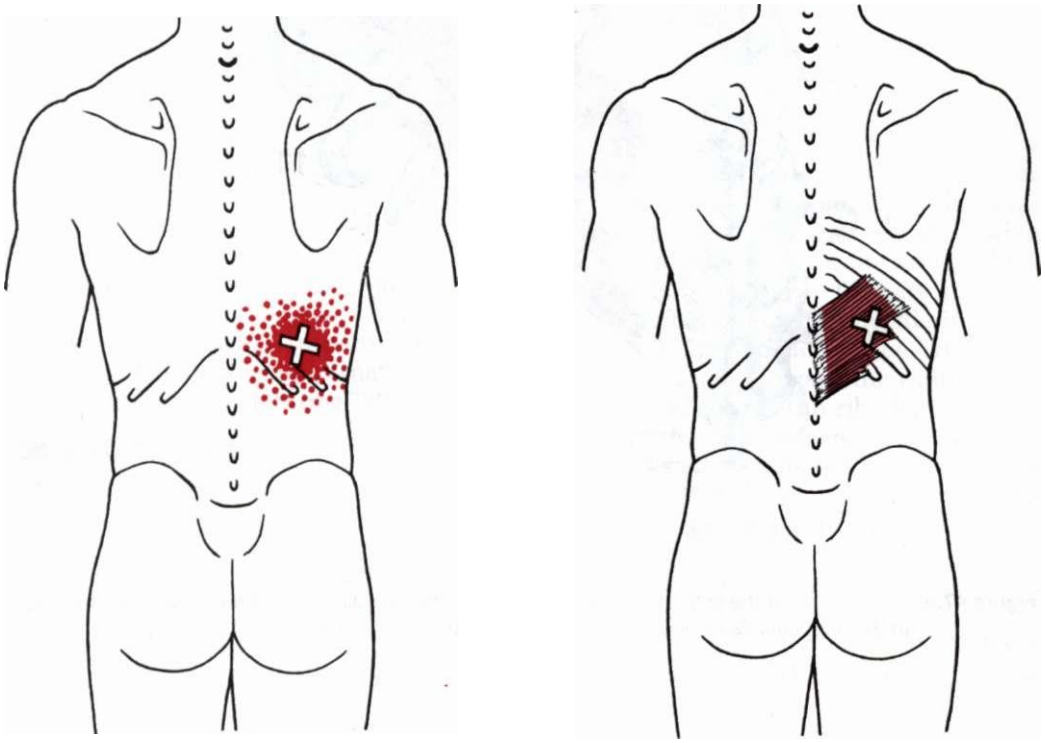


Figure 47.7. Referred pain pattern (essential zone is *solid dark red*, spillover zone is *stippled dark red)* of an active trigger point (**X**) in the right serratus posterior inferior muscle *(light red)*.

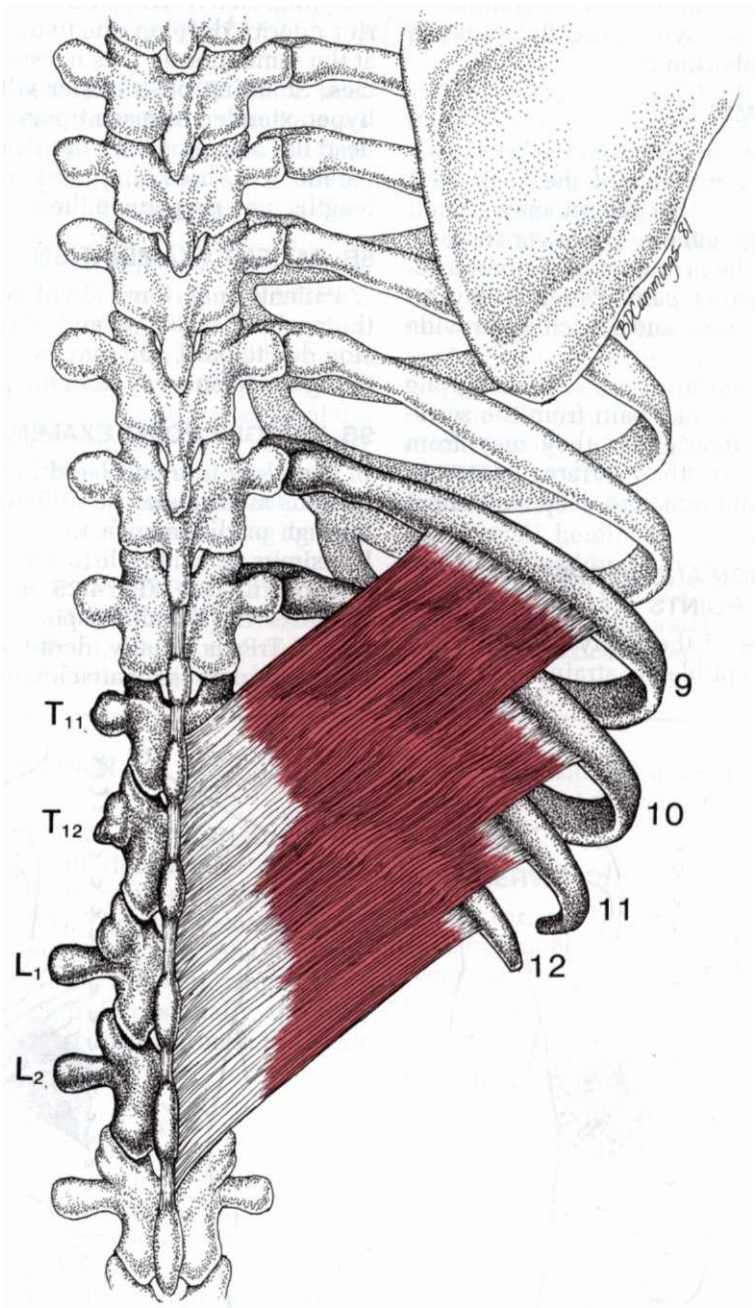


Figure 47.8. Attachments of the serratus posterior inferior muscle laterally to the lowest four ribs and medially to the aponeurosis extending from the spinous processes of the T_{11} to L_2 vertebrae.

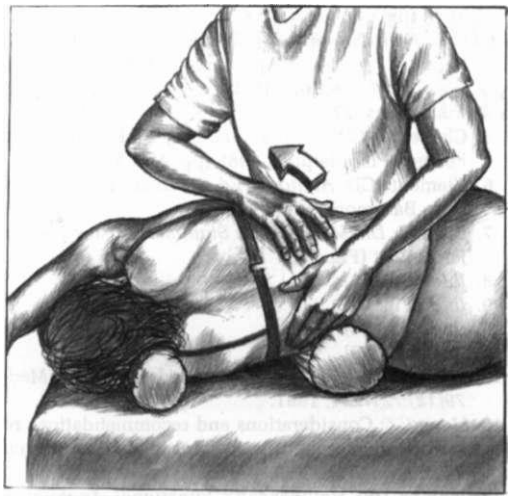


Figure 47.9. Manual release of right serratus posterior inferior, patient lying on left side with right arm elevated. Release of trigger point tension begins by having the patient look up to the right and breathe in. Then, as the patient looks back down to the left, **slowly** breathes out, and reaches toward the floor with the right arm, the operator takes up slack in the muscle. One hand of the operator (left in this case) stabilizes the lower thoracic and upper lumbar spinous processes; the other hand applies light steady pressure upward and laterally, directly on the right lower rib cage, taking up slack in the muscle as it relaxes. During this stretch phase, the operator can place one finger directly on the trigger point and use light pressure (as in pressure release) to facilitate release. Repeat the inhalation-exhalation and the release procedure if needed for complete release of the trigger point tension.

lateral end of the muscle close to the muscle's rib attachments is usually more readily located. Local twitch responses are difficult to elicit and detect by palpation in this muscle but may be felt during TrP injection.

10B. ENTRAPMENT

No entrapment of a peripheral nerve is attributed to this muscle.

11B. DIFFERENTIAL DIAGNOSIS

Differential diagnoses of the symptoms caused by TrPs in this muscle include renal diseases (caliectasis, pyelonephritis, or ureteral reflux), and a lower thoracic radiculopathy. The most common articular dysfunction associated with serratus posterior inferior TrPs is a simple neutral dysfunction extending from T_{10} to L_2. Occasionally, one finds a concurrent depression or "exhalation" dysfunction of the lower four ribs.

Related Trigger Points

This patch of discomfort is likely to be noticed only after successful treatment of myofascial symptoms arising from TrPs in associated muscles. In this case, the associated muscles are the adjacent iliocostalis and longissimus thoracis.

12B. TRIGGER POINT RELEASE (Fig. 47.9)

A manual release technique with respiratory augmentation as described and illustrated in Figure 47.9 is recommended. Its effectiveness can often be augmented by preliminary application of serial sweeps of vapocoolant spray in the direction of the muscle fibers, covering the entire muscle and pain zone. The patient's ipsilateral arm is placed overhead to pull the rib cage upward and the torso rotated toward the opposite side to take up slack in the muscle.

This muscle also responds well to trigger point (TrP) pressure release described in Chapter 3, Section 12.

13B. TRIGGER POINT INJECTION (Fig. 47.10)

Injection of trigger points (TrPs) in this muscle is recommended only for those who are experienced and skillful in doing TrP injections. For injections of the serratus posterior inferior muscle, the patient lies on the side opposite the muscle to be injected and the active TrPs are precisely located by palpation. The needle is angled (Fig. 47.10) for injection of the TrP so that its point is aimed toward the ninth, tenth, eleventh, and/or twelfth rib, depending on which digitations are involved. This technique is also illustrated by Rachlin.[19] Penetration between the ribs must be avoided. Injection of the TrPs in this muscle characteristically elicits palpable local twitch responses, and affords prompt relief of the nagging discomfort.

After injection, the muscle is stretched and sprayed, as described above, and moist heat applied.

14B. CORRECTIVE ACTIONS

Many of the corrective actions to be considered are covered in other chapters.

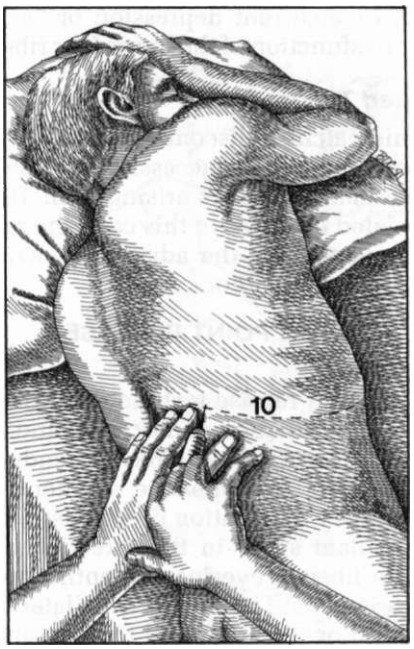


Figure 47.10. Injection of a central TrP in a digitation of the serratus posterior inferior muscle that attaches to the ninth rib. The needle is directed toward the tenth rib, not between ribs.

These include use of lifts to correct the compensatory scoliosis caused by a small hemipelvis when sitting, or by a lower limb-length discrepancy when standing (see Chapters 4 and 48 and a more detailed discussion in Chapter 4 of Volume 2), normalization of paradoxical breathing (see Figs. 20.15 and 20.16), sitting in chairs that fit and having adequate lumbar support (see Figs. 41.4E and 41.5B and C), standing with a normal lordotic lumbar curve (see Fig. 41.4C); and sleeping on a firm mattress that does NOT sag.

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991, p. 234 (Fig. 4.48).

1a. Bardeen CR: The musculature, Sect. 5. In *Morris's Human Anatomy,* edited by C. M. Jackson, Ed. 6. Blakiston's Son & Co., Philadelphia, 1921 (p. 490).

2. Campbell EJ: Accessory muscles. Chapter 9. In: *The Respiratory Muscles.* Ed. 2. Edited by Campbell EJ, Agostoni E, Davis JN. W.B. Saunders, Philadelphia, 1970 (pp. 181-195).

3. Carter BL, Morehead J, Wolpert SM, *et al: Cross-Sectional Anatomy.* Appleton-Century- Crofts, New York, 1977 (Sections 19-21).

4. *Ibid.* (Sects. 27-29).

5. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 478, 479).

6. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 524).

7. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (Fig. 50).

8. *Ibid.* (Fig. 52).

9. *Ibid.* (Fig- 53).

10. *Ibid.* (Fig. 54).

11. *Ibid.* (Fig. 55).

12. Fourie LJ: The scapulocostal syndrome. S *Afr MedJ 79(12):72\-724,* 1991.

13. Hong CZ: Considerations and recommendations regarding myofascial trigger point injection. *J Musculoske Pain* 2(1):29-59, 1994.

14. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Rack.* Ed. 6. W. B. Saunders, Philadelphia, 1991 (pp. 198, 200).

15. Lynn P: Personal communication, 1993.

16. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 96).

17. Michele AA, Davies JJ, Krueger FJ, *et al.:* Scapulocostal syndrome (fatigue-postural paradox). *NY State J Med* 50.1353-1356, 1950 (Fig. 2).

18. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 29).

19. Rachlin ES: Injection of specific trigger points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (pp. 208, 209).

20. Rasch PJ, Burke RK: *Kinesiology and Applied Anatomy.* Lea & Febiger, Philadelphia, 1978 (p. 256).

21. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981 (Table 2).

22. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. Hirzel, Leipzig, 1922 (p. 307).

23. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (pp. 267, 269).

24. Travell J: Basis for the multiple uses of local block of somatic trigger areas (procaine infiltration and ethyl chloride spray). Miss Valley Med *J* 71:12-21,1949 (p. 18, Fig. 4).

25. Travell J, Rinzler S, Herman M: Pain and disability of the shoulder and arm: treatment by intramuscular infiltration with procaine hydrochloride. *JAMA* 220:417-422, 1942 (p. 418, Fig. 2).

26. Travell J, Rinzler SH: Pain syndromes of the chest muscles: Resemblance to effort angina and myocardial infarction, and relief by local block. *Can Med Assoc* /59:333-338, 1948 (p. 336, Fig. 5).

CHAPTER 48
Thoracolumbar Paraspinal Muscles

HIGHLIGHTS: The paraspinal musculature consists of a superficial group of long-fibered longitudinal muscles, and a deep group of short diagonal muscles. In the superficial (erector spinae) group, the longissimus thoracis, iliocostalis thoracis, and iliocostalis lumborum are most likely to develop active trigger points (TrPs). Patients are likely to call this pain lumbago. The deep paraspinal group includes, at successively deeper levels, the semispinalis, multifidus and rotatores. **REFERRED PAIN** from TrPs in the iliocostalis thoracis is projected medially toward the spine and may spill over anteriorly in the abdomen and up toward the back of the shoulder. The lumbar iliocostalis TrPs refer pain to the mid-buttock. Active TrPs in the longissimus thoracis muscle at the low thoracic and high lumbar levels also refer pain downward to the sacroiliac region and the buttock. Pain from the multifidus and rotatores muscles centers on the spinous processes at the segmental level of the TrP or, in the lumbar region, it may be referred a few segments caudal to the TrP. **FUNCTIONS** of the paraspinal muscles are primarily to extend the spine, and to contribute to rotation to some extent, particularly for stabilization. The superficial fibers are extensors. The successively deeper, shorter and more diagonal fibers supply an increasing rotational component for fine adjustments. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the paraspinal muscles is caused by either sudden overload, as when lifting objects with the back twisted and flexed, or by sustained overload in the stooped posture, or when these back muscles are maintained in a fully shortened (hyperlordotic) position. **PATIENT EXAMINATION** reveals restricted range of back motion especially in flexion or rotation. Tightness of the more superficial group of muscles can be felt best when the patient is positioned between side-lying and prone. **TRIGGER POINT EXAMINATION:** Identification of the deeper paraspinal TrPs is aided by eliciting focal deep tenderness and noting the resulting referred pain pattern. **ENTRAPMENT** of the posterior primary rami of both thoracic and lumbar spinal nerves may be due to TrPs and their tense bands in the paraspinal muscles. **TRIGGER POINT RELEASE** of the long-fibered erector spinae muscles is accomplished by flexing the spine of the seated patient, while a jet stream of vapocoolant is applied in downward parallel sweeps. Successively deeper muscle layers require progressively more spinal rotation as the patient's chest turns further toward the affected side. **TRIGGER POINT INJECTION** of the deep paraspinal TrPs may require needle penetration to the depth of the laminae of the vertebrae, followed with full stretch by thoracic rotation. **CORRECTIVE ACTIONS** include relief of postural strain, compensation for body asymmetries, modification of the patient's daily activities to reduce stress on the back muscles, self-administered trigger point pressure release of TrPs by use of a tennis ball, and graduated stretch and strengthening exercises.

1. REFERRED PAIN (Figs. 48.1 and 48.2)

Trigger points (TrPs) are one of the most common causes of enigmatic back pain (see Chapter 41 Section B). Among 283 patients referred to a chronic pain treatment program who fit the diagnosis of chronic intractable benign lumbar pain, 96% had tender/trigger points.[108]

The referred pain patterns illustrated for these back muscles at specific segmental levels are common examples, but TrPs may develop at any segmental level. Determining the depth and muscular length for a deeper-layer TrP is sometimes difficult but often important for selecting appropriate treatment.

Pain patterns similar to those observed in adults were reported from TrPs in the longissimus and multifidus muscles of children.[11]

Superficial Paraspinal (Erector Spinae) Muscles (Fig. 48.1)

In the middle and lower back, the two muscles of this group that are most likely to develop TrPs are the longissimus thoracis and the iliocostalis thoracis. The iliocostalis thoracis refers pain both cephalad and caudad, while the iliocostalis lumborum and the longissimus thoracis refer pain mainly caudad.[134]

The pattern of referred pain from TrPs in the iliocostalis thoracic at the midthoracic level (Fig. 48.1 A) is upward toward the shoulder and laterally to the chest wall which, on the left side, is easily mistaken for cardiac angina,[51,99] or as pleurisy on either side.[70] At the low thoracic level (Fig. 48.1B), iliocostalis thoracic TrPs may refer pain upward across the scapula, around to the abdomen, and downward over the lumbar area.[15,134,138] This pain referred to the abdomen from a back muscle may be mistaken for visceral pain.[58,99,137] These low iliocostalis thoracis TrPs may be in the iliocostalis lumborum since these two iliocostalis muscles overlap in this region.

From iliocostalis lumborum TrPs at the upper lumbar level (Fig. 48.1C), pain is referred strongly downward, concentrating on the midbuttock,[15,132,134,137] and is a frequent source of unilateral posterior hip pain. "Fibrositis" of the iliocostalis muscles (frequently, the term fibrositis was used to identify TrPs) is one common cause for the pain described as "lumbago."[82,105] The patient usually draws an up-and-down pattern to represent the pain referred from iliocostalis TrPs, but a crosswise pattern in the same region of the back to demonstrate the pain referred from TrPs in the lower rectus abdominis muscle.

> A quadriparetic patient had pain and tenderness in the right lower quadrant, right flank, and right subcostal area with a right subcostal TrP in the iliocostalis lumborum muscle. Pressure on the TrP reproduced the patient's pain. The abnormal iliocostalis muscle tension and the patient's pain were relieved by spray and stretch of that muscle.[112] This pain pattern was more like what one would expect from a low iliocostalis thoracis TrP than from a high iliocostalis lumborum TrP. The spinal cord may not make that clear a distinction this close to the transition zone, and this patient did not have a normal spinal cord.

Myofascial TrPs at the low thoracic level in the longissimus thoracis muscle (Fig. 48.ID, *right side)* refer pain strongly low in the buttock.[15,134,138] This remote source of buttock pain is easily overlooked. Longissimus TrPs toward the caudal end of the muscle fibers in the upper lumbar area usually refer pain several segments caudally, but still within the lumbar region[15,134,138] (Fig. 48.1D, left side). This is another muscular source of "lumbago."

Lange,[74] in 1931, identified myogelosis (completely compatible with myofascial TrPs) of the erector spinae muscles at the lumbar level as a frequent cause of "lumbago" and sacral pain. Gutstein[57] reported numerous patients with referred pain from myalgic spots or muscular rheumatism in the erector spinae muscles.

Kellgren[68] mapped experimentally induced referred pain patterns of the erector spinae muscles by injecting hypertonic salt solution into normal muscles. He reported that the superficial erector spinae muscles at the midlumbar level referred pain to the upper part of the buttock. In a similar study, hypertonic saline injection of the structures along the edge of the interspinous ligament at the L_1 level[69] referred pain characteristic of renal colic to the loin, inguinal, and scro-

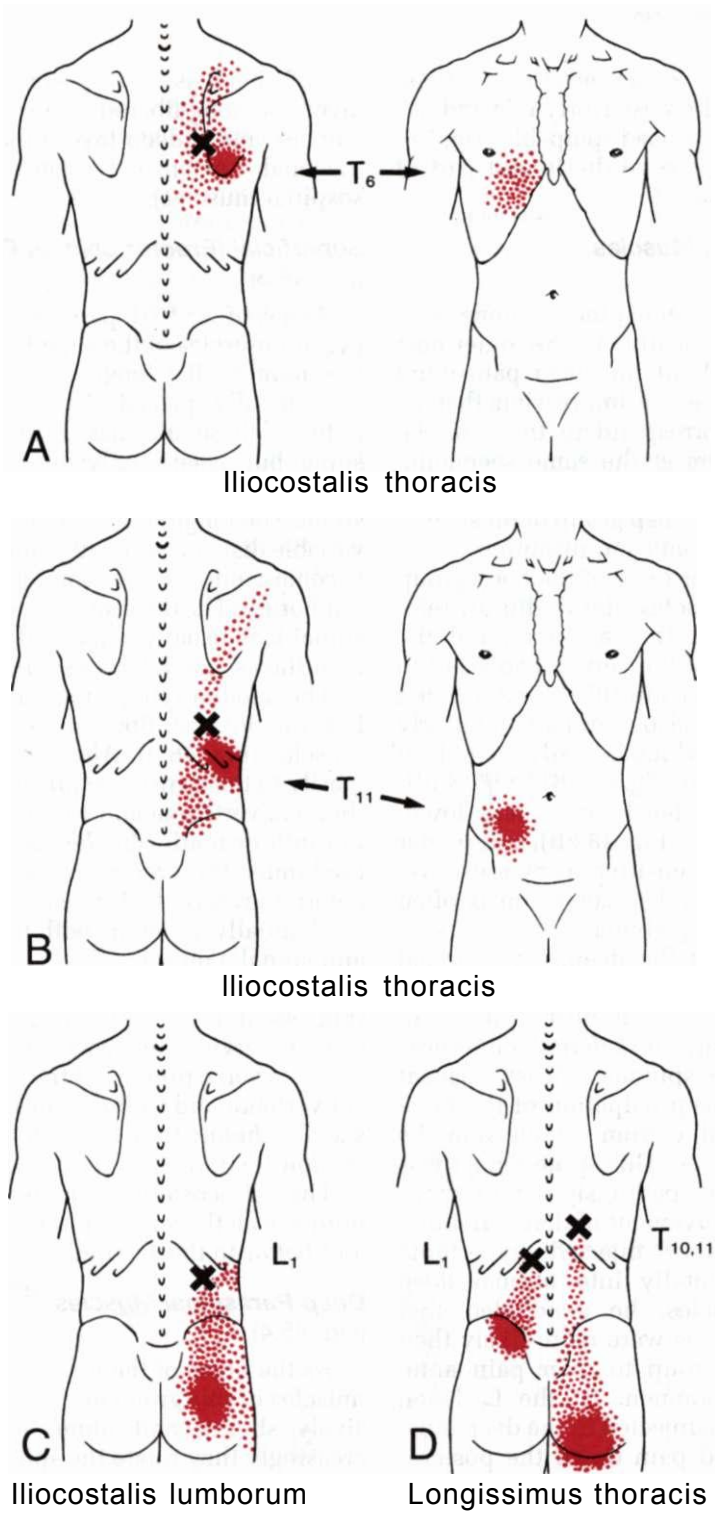


Figure 48.1. Examples of referred pain patterns (essential reference zones are ***solid red,*** spillover areas are ***stippled red)*** with their corresponding trigger points (**Xs**), at several levels in the erector spinae (superficial paraspinal) muscles. **A,** the mid-level of the right iliocostalis thoracis. **B,** the caudal portion of the right iliocostalis thoracis. C, the upper end of the right iliocostalis lumborum. **D,** the lower thoracic *(right)* and upper lumbar (***left)*** longissimus thoracis. Longissimus fibers often reach the upper lumbar region.

tal areas, causing retraction of the testicle. At the T_9 level, the posteriorly injected hypertonic saline caused palpable rigidity and deep tenderness of the lowest part of the abdominal wall.[76]

Deep Paraspinal Muscles (Fig. 48.2)

Although the semispinalis thoracis is classified anatomically as the outermost (most superficial) of the deep paraspinal muscles, we have the impression that its pain patterns correspond to those of the longissimus fibers at the same segmental level. The severe aching "bone" pain from TrPs in any of this deep group of muscles is persistent, worrisome and disabling.

The next deeper layer of the deep group of paraspinal muscles, the multifidi, refer pain primarily to the region around the spinous process of the vertebra adjacent to the TrP (Fig. 48.2A). Multifidus TrPs located from to L_5 may also refer pain anteriorly to the abdomen, which is easily misjudged as visceral in origin (Fig. 48.2B).[134,138] Multifidus TrPs at the S_1 level project pain downward to the coccyx (Fig. 48.2B), and render the coccyx hypersensitive to pressure (referred tenderness). The condition is often identified as coccygodynia.

Involvement of the deepest paraspinal muscles, the rotatores, throughout the length of the thoracolumbar spine produces midline pain and referred tenderness to tapping on the spinous process adjacent to a TrP. Only deep palpation of the muscles can determine from which side the midline pain arises. This spine tenderness is used as an osteopathic sign of articular-dysfunction involvement of that vertebra.

When Kellgren[68] injected hypertonic saline experimentally into normal deep paraspinal muscles, he concluded that these deep muscles were more likely than the superficial group to refer pain anteriorly to the abdomen. At the L_5 level, hypertonic saline injected in the deep muscles also referred pain down the posterolateral aspect of the thigh and leg.

2. ANATOMY (Figs. 48.3 and 48.4)

The bewildering complexity of the paraspinal muscles is simplified by thinking of them as two layers, a superficial layer of long-fibered extensors (erector spinae), and a deep layer of shorter, more diagonal extensor rotators (transversospinal muscles).

Superficial (Erector Spinae) Group (Fig. 48.3)

As a source of TrP pain, the two most important muscles of the superficial group are the more medial longissimus thoracis and the laterally placed iliocostalis thoracis. Both of these muscles span the thoracic spine, but usually only the iliocostalis extends to the sacrum beyond the lumbar spine. The longissimus thoracis continues a variable distance across the lumbar region to become a more or less complete longissimus lumborum. The third superficial muscle, the spinalis, is usually small and has not been identified separately as a source of TrP pain.

The medial-lying longissimus thoracis has the longest fibers of the paraspinal muscles (Fig. 48.3). ***Above*** it attaches primarily to the transverse processes of all the thoracic vertebrae and to the adjacent first to ninth or tenth ribs; ***below*** it attaches to the lumbar transverse processes, and to the anterior layer of the lumbocostal aponeurosis. Caudally, it blends with the iliocostalis and spinalis muscles.[27]

The more lateral iliocostalis thoracis (Fig. 48.3) is a continuation of the iliocostalis cervicis. Its fibers connect ***above*** to the transverse process of the seventh cervical vertebra and to the angles of the *upper* six ribs; ***below*** they attach to the angles of the *lower* six ribs.[27]

The iliocostalis lumborum extends ***above*** from the angles of the lowest six ribs and *below* to the sacrum.

Deep Paraspinal Muscles (Fig. 48.4)

As the fibers of the progressively deeper muscles of this group also become progressively shorter and more horizontal, increasingly they rotate the spine rather than primarily extending it.[27] Among the deep group of paraspinal muscles, the semispinalis thoracis extends caudally as far at T_{10}, overlying the multifidi (Fig. 48.4). The multifidi and rotatores continue beyond the lumbosacral junction where they fill

the multifidus triangle of the sacrum[81] and are covered by the tendinous extensions of the more superficial longissimus and iliocostalis muscles.

The deeper multifidi and rotatores muscles attach ***medially*** and ***above*** near the base of a vertebral spinous process. ***Laterally*** and ***below*** they attach to a transverse process (Fig. 48.4), spaced as follows: the semispinalis thoracis fibers cross at least five vertebrae and extend caudally to the tenth thoracic vertebra (Fig. 48.4). Multifidus fibers cross 2 to 4 segments throughout the thoracic and lumbar spine, and sometimes extend to S_1. The short rotatores attach to adjacent vertebrae. The long rotatores span one segment throughout the spine,[27] but ordinarily do not include sacral segments. The fibers of the lumbar multifidus are divided by distinct cleavage planes into five segmental bands. Each band arises from a lumbar spinous process and is innervated unisegmentally.[86]

Supplemental References

Other authors have clearly illustrated the longissimus thoracis, and the iliocostalis thoracis and lumborum as seen from behind,[1,28,88,89,101,117,127,128] from the side,[131] and in cross section.[24,66,67] The semispinalis thoracis has been presented as seen from behind,[1,29,42,44,101,118,128] and in cross section.[25] The multifidus has been illustrated from behind,[29,30,42,101,102,119,128,129] from the side,[43] and in cross section.[2,66,67] The rotatores have been shown from behind[3,44,89,101,102,120] and from the oblique rear view.[130]

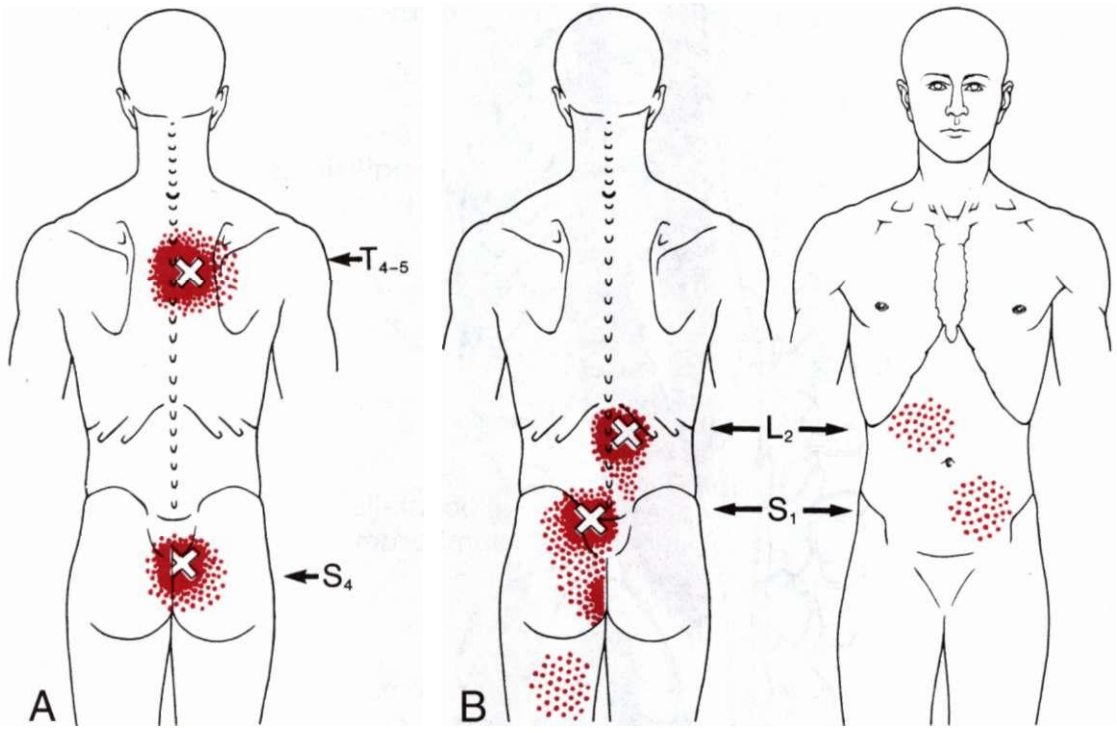


Figure 48.2. Referred pain patterns (red), and their corresponding trigger points (**Xs**) in the deep paraspinal muscles. Pain referred by the rotatores is felt essentially in the midline. **A,** examples of local patterns characteristic of trigger points at the midthoracic level and in multifidi at the low sacral level. **B,** local and projected pain patterns of trigger points in these muscles at the intermediate L_2 and S_1 levels.

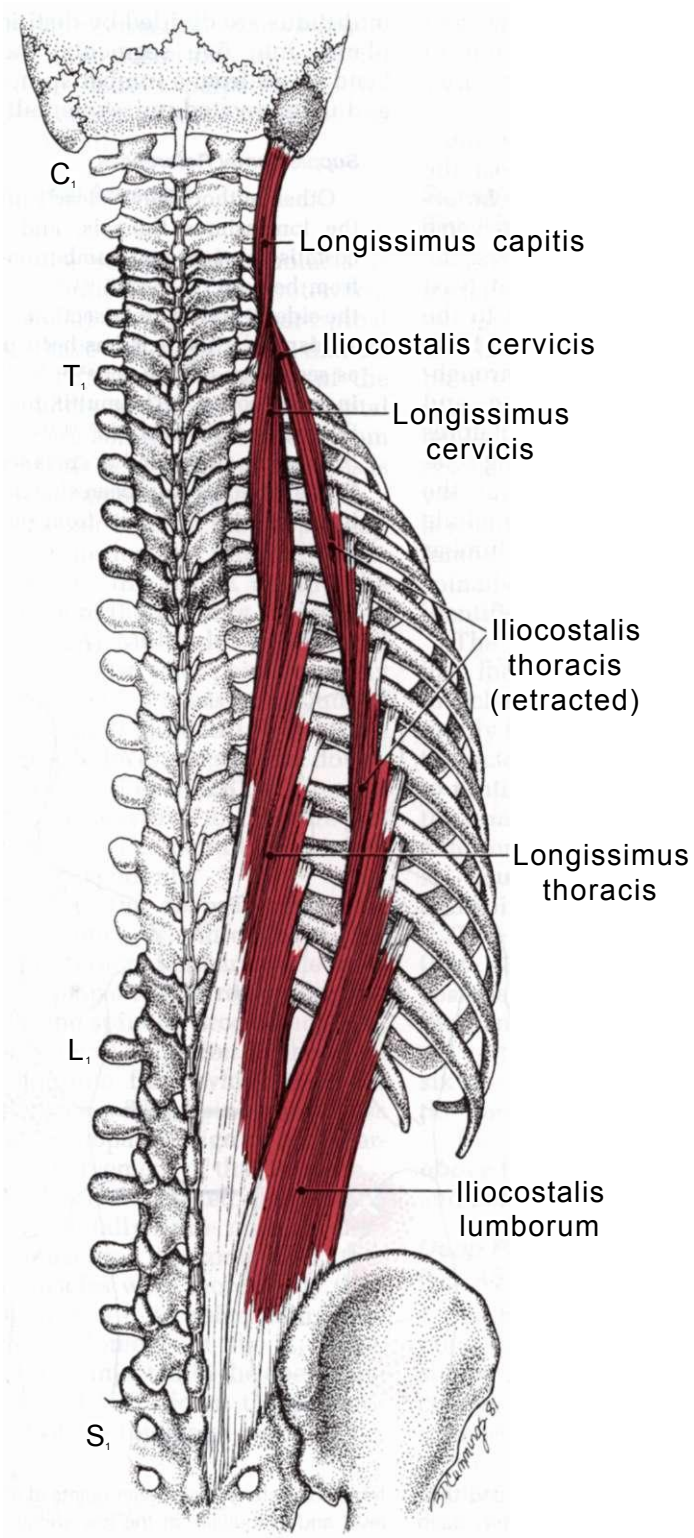


Figure 48.3. Attachments of the two most important of the superficial (erector spinae) group of paraspinal muscles *(red): medially* the longissimus thoracis, and *laterally* the iliocostalis thoracis and iliocostalis lumborum.

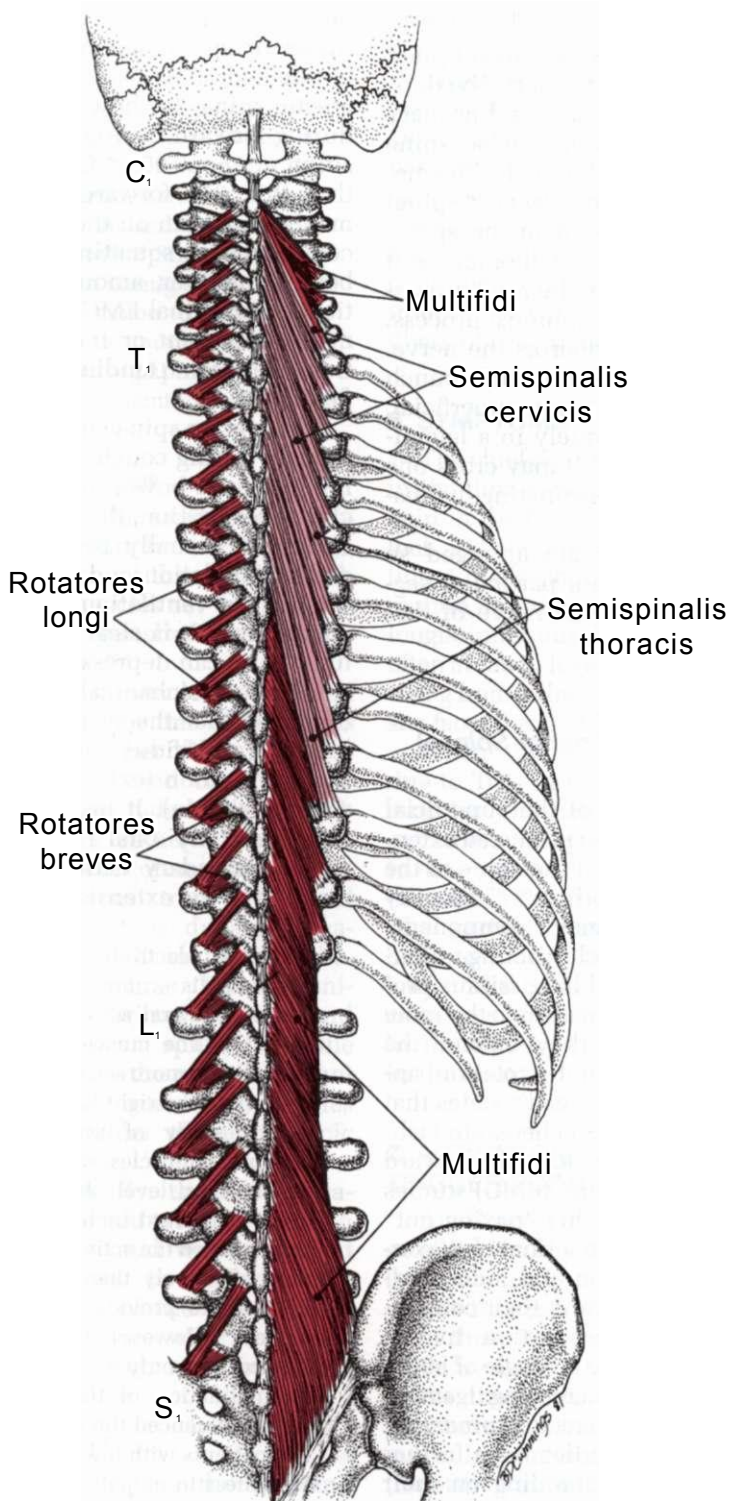


Figure 48.4. Attachments of the deep group of paraspinal muscles. ***Right,*** The more superficial of this group are the semispinalis thoracis at the thoracic level (*light red)*, which overlies the multifidi, and the multifidi at the thoracic, lumbar and sacral levels *(dark red)*. ***Left,*** The rotatores form the deepest layer at both the thoracic and lumbar levels. The rotatores occur above the sacral level. Only the multifidi extend across sacral segments.

3. INNERVATION

All the paraspinal muscles are supplied by branches of the dorsal primary divisions of the spinal nerves.[27] Each dorsal primary division in the thoracic and lumbar spine has a medial and a lateral branch. The medial branch innervates the *deepest* spinal muscles at the level of exit of the spinal nerve, so that in the lower thoracic and lumbar regions, the nerve, the rotator muscle, and the tip of the spinous process, which has the same number as the nerve, are at the same level.[14] The lateral branch innervates the longer, more superficial, muscles by running obliquely in a lateral-caudal-dorsal direction.[65] It may cross one or two segments before terminating in muscle fibers.[52,69]

The lumbar multifidi are arranged so that the fibers that move a particular segment are innervated by the nerve of that segment.[14]

4. FUNCTION

Superficial Paraspinal (Erector Spinae) Muscles

Electrical stimulation of the superficial lumbar paraspinal muscles produced extension and lateral bending of the spine to the same side.[40] Some authorities[64,67] identify three functions for both major components of the erector spinae muscles: acting unilaterally, the iliocostalis and longissimus produce lateral flexion and rotation to the same side, acting bilaterally, they extend the spine. Their contribution to rotation appears to be minor. Hollinshead[64] states that these muscles function in a checkrein fashion to resist gravity in the stooping-forward position. Electromyographic (EMG) studies support a major role for this "paying out" action (lengthening contractions for control) during spinal flexion and side-bending.[10] When bending forward, their contraction is increased in proportion to the amount of flexion down to an angle of about 45°.[7] Beyond that angle, increasing ligamentous tension unloads the erector spinae.

Electromyographic studies have further shown that, in persons standing on their feet, the erector spinae can achieve complete relaxation: when standing erect, when bending forward with the spine fully flexed,[10,75] and when side bending if all traces of spinal flexion or extension are eliminated.[100] An earlier author found the erector spinae to show a maximum activity during forward flexion in the standing position at about 90°.[96] Calculations showed that this 90° forward-bent position put maximum strain on the lumbosacral joints compared to squatting with the knees bent.[94] However, among 87 back pain patients, paraspinal EMG activity was abnormally persistent or increased in all positions between standing and full forward flexion.[114]

The erector spinae muscles contract vigorously during coughing and when straining to have a bowel movement.[10] As muscles of respiration, the bilateral iliocostalis lumborum usually become active at the end of inhalation and also during exhalation, if the ventilation rate is close to its maximum.[23] It is clear that the iliocostalis lumborum can depress the lower ribs.

Detailed mechanical measurements[85] established that the principal action of the lumbar multifidus muscle is posterior sagittal rotation (extension without posterior translation). It had no translatory action. The only axial rotation effect was a minor secondary action which must be coupled to the extension movement.

> Surface electrodes over the lumbar sacrospinalis muscles consistently showed bilateral activity during 13 sport activities. The muscles on the left side were clearly more active than those on the right when the right hand was used.[19]
>
> In a study of seated subjects, the paraspinal muscles were more active at the thoracic level than at the lumbar level.[6] Backward inclination of the chair back reduced the activity of these muscles more effectively than did contouring of the chair to provide lumbar or thoracic support.[56] However, a radiographic study showed that only a lumbar support, not the inclination of the backrest, significantly influenced the lumbar lordosis.[4]
>
> In patients with low back pain and with tenderness to palpation of the paraspinal muscles, the superficial layer tended to show less than a normal amount of EMG activity until the test movement became

painful. Then, these muscles showed increased motor unit activity, or "splinting."[103] During 6 min of standing, the root mean square amplitude of electrical activity of the L_4 and L_5 paraspinal muscles, recorded from surface electrodes, increased in seven patients with recent onset of low back pain, and decreased in four pain-free controls.[63] Since the cause of the low back pain is enigmatic, the relevance of this observation to TrPs is conjectural, but trapezius muscles with TrPs were more electromyographically active with movement than contralateral muscles without TrPs.[38]

Deep Paraspinal Muscles

Acting bilaterally, the semispinalis thoracis, the thoracic and lumbar multifidi, and the rotatores extend the vertebral column. When these muscles act unilaterally, they can rotate the vertebrae to the contralateral side.[10,67,100,103]

The deep group of muscles is believed to function primarily for **fine adjustments** between vertebrae, rather than for gross spinal movements.[39] Based on EMG evidence, Basmajian[9] concluded that the multifidi are stabilizers rather than prime movers of the vertebral column as a whole. Specifically, the deepest transversospinal (rotatores) muscles act as dynamic ligaments that adjust small movements between individual vertebrae. The abdominal muscles are the primary flexors and rotators of the lumbar spine, and the quadratus lumborum is the most important for side-bending. The intercostal muscles are the primary rotators of the thoracic spine (see Chapter 45).

Electromyographically, the deep paraspinal muscles were activated by rotation to the opposite side, and were activated in complex patterns by flexion, extension and rotation of the spine.[10] Responses recorded by fine wire electrodes were illustrated for each of these movements.[91]

5. FUNCTIONAL UNIT

Spinal extension by the thoracic and lumbar paraspinal muscles is assisted by the serratus posterior inferior and the quadratus lumborum muscles, and is opposed by the rectus abdominis and abdominal oblique muscles.

Rotation of the lumbar spine is provided primarily by the oblique abdominal muscles, and is assisted most by the deepest paraspinal muscles. Rotation of the thoracic spine is provided primarily by the intercostal muscles assisted by the multifidi. Rotation also may be assisted in the thoracolumbar region by the serratus posterior inferior and one group of diagonal deep fibers of the quadratus lumborum.

6. SYMPTOMS

The chief complaint caused by active TrPs in the thoracolumbar paraspinal muscles is pain in the back and sometimes in the buttock and abdomen as was described in Section 1. This pain markedly restricts spinal motion and the patient's activity. When the longissimus muscles are involved bilaterally, often at the L_3 level, the patient has difficulty rising from a chair and climbing stairs if he or she faces forward in the usual manner.

When the complaint of "lumbago" is due to TrPs in the deep lumbar paraspinal muscles, the pain usually is a unilateral, extremely disagreeable, steady ache deep in the spine. It becomes bilateral as the muscles on both sides become involved. The patient may point to a one-sided bulging of the long muscles of the low back. The patient finds little relief by changing position, and is often convinced by the way it feels that the pain originates in the bony spine, not in the muscles.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

An iliocostalis thoracis TrP may be a satellite of a key TrP in the latissimus dorsi (which must be treated effectively to clear up the iliocostalis).

In these back muscles, TrPs may be activated by sudden overload (a specific activity or traumatic event that is clearly remembered), or by sustained or repeated muscular contraction over a period of time (repetitive microtrauma).

A quick awkward movement that combines bending and twisting of the back, especially when the muscles are fatigued or

chilled, is likely to activate TrPs in the iliocostalis, even though no additional loading (lifting) is involved; this may be caused by disproportionate loading of one group of muscle fibers as the result of poor coordination.

Trigger points in these muscles can be activated or perpetuated by any mechanical factor that disturbs axial symmetry, such as a lower limb-length inequality *(see* Chapter 4, Section B and Chapter 4 of Volume 2), a disturbance of pelvic symmetry due to structural asymmetries, or due to a wallet carried in a back pocket.[53] The relevance of structural asymmetries as perpetuating factors is illustrated by the study of Strong and Thomas.[122] Paraspinal muscles should increase activity on the side of the longer leg, and on the side of the concavity in the presence of spinal curvature.

Lange[74] and Lindstedt[79,80] both thought that flat feet caused muscular strain which activated myogelosis or muscular rheumatic symptoms (described in terms indicative of TrPs) in many back, hip, and thigh muscles and produced pain patterns commonly identified as "sciatica" or "lumbago." The authors of this manual find that equinus valgus and structural disproportions, such as leg and pelvic asymmetry, can overload specific muscles to perpetuate, and sometimes initiate, the pain-producing TrPs. Almost any factor that contributes to a significant gait deviation can activate TrPs in the iliocostalis.

The whiplash type of accident that causes sudden acceleration or deceleration is likely to rapidly stretch protectively stiffened spinal muscles which, in turn, is likely to activate TrPs in them.

Prolonged immobility, as when sitting for hours in an aircraft or automobile with the seat belt fastened, may activate TrPs in the paraspinal muscles. Substantiating this, an EMG study of the thoracic and lumbar erector spinae showed that typists who remained immobile in their optimally relaxed position (initial electrical silence) developed muscular activity in about 1/2 hr or sooner; repositioning temporarily quieted this motor unit activity at rest.[83] It is noteworthy that immobility built up muscle tension in *everyone* tested, in some much sooner than in others.

8. PATIENT EXAMINATION

Superficial Paraspinal (Erector Spinae) Muscles

When standing, the patient with involvement of the superficial (erector spinae) muscles may be unable to flex the torso more than a few degrees. Palpation of specific paraspinal muscles is less effective with the patient standing because of postural muscle tension and protective splinting by normal muscles. The examiner must obtain relaxation of the patient's back muscles so that abnormally taut muscle fibers are distinguishable. When the seated patient leans forward, dangles the arm between the legs, and relaxes, an involved lumbar longissimus on one side is evident and feels like a hard rope. For greatest sensitivity to palpation, the patient lies on one side and brings the knees toward the chest just far enough to take up the slack in the long erector spinae. Janda[62] identified the erector spinae as being prone to facilitation and tightness which is in accordance with EMG studies of referred motor activity from lower body TrPs *(see* Chapter 2, Section B).

After the erector spinae on the painful side have been passively stretched during vapocooling and the muscles on that side have relaxed, mirror-image pain and muscular tension may appear, so that the opposite lumbar longissimus now stands out and feels tense. The two sides frequently function together as a unit and are likely to develop TrPs together.

Deep Paraspinal Muscles

Active TrPs in the deep paraspinal muscles cause guarded movements and restrict side bending, rotation, and hyperextension of the trunk. Deep lumbar paraspinal TrPs are likely to occur in patients with either an excessive or absent lumbar lordosis; deep thoracic paraspinal TrPs tend to occur in patients with marked thoracic kyphosis.

Active TrPs in the deep group of paraspinal muscles impair movement between two vertebrae during flexion or side bending of the spine. During flexion, a hollow or a flat area develops in the smooth

curve formed by the spinous processes. The flattening usually spans one to three vertebrae. Involvement of a multifidus or a rotator muscle on either side produces midline tenderness over the adjacent spinous process. This tenderness is easily located by tapping each spinous process in succession; it disappears after inactivation of the responsible TrPs, which may be located on either or both sides of the spine.

General

Local areas of reduced skin resistance to direct current were identified as characteristic of the musculoskeletal and myofascial symptoms of backache with limitation of spinal motion.[71,72]

The skin overlying involved lumbar paraspinal muscles often exhibits superficial tenderness and resistance to skin rolling (panniculosis),[8] or trophedema,[56] which disappears after therapeutic skin rolling and inactivation of the underlying myofascial TrPs. For patients who have a marked degree of panniculosis over low thoracic and lumbar paraspinal muscles with myofascial TrPs, the identification and treatment of both may be critical to their prompt recovery.[84]

9. TRIGGER POINT EXAMINATION (Fig. 48.5)

Superficial Paraspinal (Erector Spinae) Muscles

The patient lies on the uninvolved side in a comfortable, relaxed position with a pillow under the side of the abdomen for semiprone support. The full prone position often strains the patient's neck and tends to over-slacken the paraspinal muscles for examination. The back muscles must have an intermediate degree of stretch, so that the taut bands containing the TrPs can be distinguished from the adjacent normal, slackened muscle fibers. The degree of stretch is regulated by bringing the patient's knees toward the chest (Fig. 48.5). Flat palpation of the muscles then elicits spot tenderness (of a palpable nodule in a taut band in superficial muscles) and often elicits patient-recognized referred pain.

Deep Paraspinal Muscles

With the patient recumbent as above, or seated and leaning forward to flex the spine slightly, a flattened region or slight hollow that extends over one to three vertebrae indicates the probable TrP source of trouble. The examiner taps or presses on the tips of successive spinous processes to elicit tenderness. When a spinous process in the flat area is hypersensitive, the deep musculature on each side of it is palpated by firm pressure in the groove between the process and the longissimus muscle. Deep finger pressure is directed along the side of the spinous process to exert pressure on the rotatores against the underlying laminae to locate a spot of maximum tenderness. If two or three spinous processes are tender, one expects to find adjacent TrPs on at least one side at each level of tenderness.

10. ENTRAPMENT

The dorsal primary divisions (rami) of the spinal nerves supply skin sensation to the back. Since these dorsal rami pass through the paraspinal muscles to reach the skin, it is not surprising that many patients with active TrPs in these muscles, in addition to pain, complain of nerve-entrapment symptoms. In the presence of entrapment, symptoms include hyperesthesia, dysesthesia or hypoesthesia of the skin of the back. The medial branches of these rami supply afferent fibers to the skin for most of the thoracic segments above T_8, where they pass through the semispinalis thoracis and longissimus thoracis muscles. The lateral branches supply most of the skin below T_8, including the lumbar region, and are likely to be entrapped by the more lateral iliocostalis muscle.[55,61]

Symptoms in the high lumbar region were usually due to compression of the low thoracic dorsal rami by bands of tense fibers in the iliocostalis lumborum muscle.[106] Richter[106] reported permanent relief in 144 patients by surgically excising the entrapped nerve or releasing herniated fat. Subsequently, Richter[107] reported 500 patients with these symptoms of nerve entrapment in whom focal TrP tenderness was found. Nearly half of the patients were successfully treated solely by injection of the tender area with a local anesthetic.

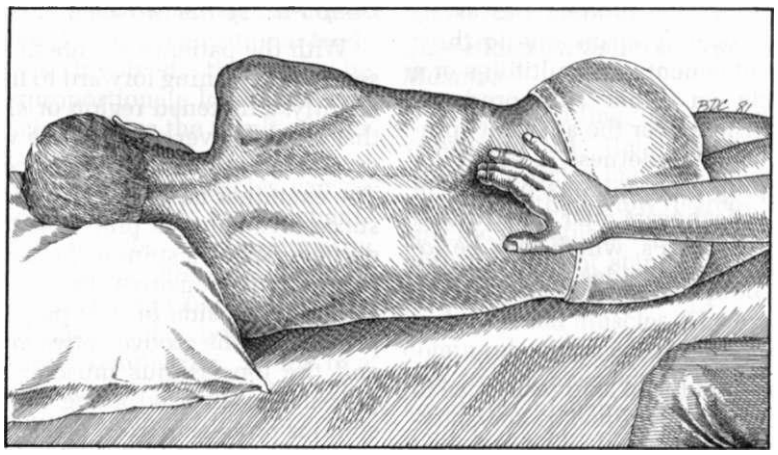


Figure 48.5. Examination of the right erector spinae muscles for trigger points. The back muscles are relaxed by placing the patient on the side, and the slack in the muscles is taken up by bringing the knees toward the chest.

11. DIFFERENTIAL DIAGNOSIS

Some of the most important differential diagnoses of symptoms caused by TrPs in thoracolumbar paraspinal muscles include articular dysfunctions, fibromyalgia, radiculopathy, visceral disease, thoracolumbar osteoarthritis, and fat lobules. Articular dysfunction and surface tears of intervertebral discs were covered in Chapter 41, Section B. Chapter 16, Section 11 includes a thorough discussion of arthritic disorders, some of which can affect this region as well.

In addition one may need to consider strain of spinal ligaments,[46] renal diseases, and gallstones in the case of right-side unilateral involvement. The rupture of an intervertebral disc, ligamentous strain, and paraspinal muscular overload that activates myofascial TrPs are all likely to be caused by similar strains. These strains are especially likely when lifting with the back twisted and flexed, instead of erect and straight.[93]

Some additional causes of back pain that may need to be considered are metastatic tumors from the breast, ovaries or prostate; retrocecal appendicitis; dissecting aortic aneurysm or saddle thrombus; biliary, renal or ureteral stones; torsion of the kidney; pelvic inflammatory disease or endometriosis; ankylosing spondylitis; Paget's disease; leukemia with enlarged retroperitoneal nodes; Hodgkin's disease; tumors of the kidney; prostatitis and seminal vesiculitis;[113] sacroiliitis; and, very rarely, primary psychogenic backache.

Referred pain from lumbar zygapophysial (facet) joints overlaps pain referred from multifidi muscles (see Volume 2, Chapter 3, Section 2).

Articular Dysfunction

Segmental dysfunction associated with TrPs in the thoracolumbar paraspinal musculature may occur anywhere in this region. The number of segments involved depends on the muscles involved. For example, TrPs in the rotatores can induce a concurrent single level dysfunction. Trigger points in the multifidi are more likely to induce articular dysfunction involving two or three adjacent segmental levels. Semispinalis TrPs at any level will usually be associated with four to six segmental levels of dysfunction. The apex segment is often exquisitely tender to palpation. The most superficial and longest muscles are the iliocostalis and the longissimus. Their TrPs are associated with group dysfunctions. If the patient compensates proximally to level the shoulders, he or she can present with a double curve (S curve) that is easily misinterpreted as a primary scoliosis.

Trigger points in the iliocostalis lumborum are also associated closely with pelvic obliquity secondary to tension applied to the muscle's insertional aponeurosis onto the sacral base. Therefore, this can also present as a sacroiliac dysfunction, which is demonstrated by a positive seated-flexion test. Be aware that the side of the positive seated-flexion test is not the side of the sacroiliac dysfunction. Detailed description of the seated-flexion test is found in Greenman.[54]

Schneider[111] emphasized that the symptoms caused by multifidus TrPs mimic those of lumbar facet or sacroiliac syndromes and that an L_4-L_5 lateral disc herniation produces tightness of the left L_4-L_5 multifidus muscle, causing a segmental motion block. Referred pain characteristic of lumbar facet joints is illustrated in Volume 2, Chapter 3, Figure 3.2.

Manual release techniques for these articular dysfunctions have been reviewed.[50,54]

Fibromyalgia

Any patient with chronic low back pain and additional widespread pain should be examined for fibromyalgia. The diagnostic criteria are found in Chapter 2, Section B. Patients with fibromyalgia frequently also have myofascial TrPs and each diagnosis requires its own therapeutic approach.

Radiculopathy

Radiculopathy may be caused by pressure from a ruptured disc, by encroachment within the spinal foramen as from osteoarthritis, or by a tumor.

Lumbar radiculopathy usually causes pain that radiates into the lower extremity; paraspinal TrPs alone do not. However, when active TrPs in the back muscles induce satellite TrPs in the gluteal muscles, the latter TrPs often refer myofascial pain down the lateral or posterior aspect of the thigh or leg, sometimes extending to the foot.[116,132-134] Radiculopathy is characterized by neurological deficits including decreased tendon reflexes, impaired cutaneous sensation, and motor weakness with atrophy. Myofascial TrPs *per se* do not cause such neurological deficits unless the TrP tautness of the muscle fibers entraps a peripheral nerve. The number of these specific muscle-nerve entrapment syndromes is limited, and the degree of nerve damage is rarely more than neuropraxia.

The muscles supplied by a compressed nerve root[55] or any cause of mild entrapment neuropathy[26] are likely to develop TrPs. The pain caused by a myofascial TrP may be identified by the muscle-specific referred pain pattern, by reproduction of pain that the patient recognizes as familiar in response to pressure on the TrP, by the physical findings of spot tenderness of a nodule in a palpable band, and in superficial muscles by a local twitch response of the band. When radiculopathy activates TrPs, they may persist long after the nerve root compression has been relieved; these TrPs produce symptoms of stiffness and pain similar in distribution to the radicular pain, and may explain the complication known as the postlumbar-laminectomy pain syndrome,[109] or failed-back syndrome.

Osteoarthritis

The presence of osteoarthritis does not by itself identify the cause of the patient's pain. The radiographic signs of degenerative joint disease correlate poorly with the occurrence of pain.[121] In the absence of intervertebral disc degeneration, about one-third of a group of industrial cases with low back pain were labeled simply "low back strain." On the other hand, over one-third of 50 asymptomatic control subjects had radiographic evidence of minor degenerative changes; one had slight narrowing of the lumbosacral disc space.[55] Thus, it cannot be assumed that these degenerative changes cause back pain. In another study, only 40% of 936 symptom-free Air Force Academy or Air Cadet applicants between 17 and 27 years of age were free of congenital variations and other abnormalities of the spine.[32] Many patients with these spinal abnormalities are completely relieved of their pain when the responsible TrPs are inactivated.

Fat Lobules

Another, less common source of low back pain is herniation of fat lobules through subcutaneous fascia.[31, 34] Orr, ef *al.*[97] reported similar paraspinal fibrolipomatous nodules at the T_{12} through L_2 levels that referred pain to the back, abdomen, groin and testicle; the pain was temporarily relieved by local injection of 2% procaine solution and permanently relieved by surgical excision.

Dittrich[37] identified fibrosis of the subcutaneous lumbosacral fascia, presumably in response to tears caused by muscular strain, as a cause of low back pain. In 109 patients, these lesions with some attachment TrP characteristics referred pain from either the midsacral, midlumbar, or the low cervical areas, as judged by the relief afforded for days, weeks, or months, by the injection of procaine.[36] Local surgical intervention relieved 14 of 19 patients.[35] It is very likely that these findings frequently related to the enthesopathy of attachment TrPs.

Pain localized at the posterior portion of one iliac crest (iliolumbar syndrome) was frequently relieved by injections of a local anesthetic that penetrated sometimes the iliolumbar ligament, sometimes the quadratus lumborum muscle, and sometimes both.[60] Some of these cases may have had fascial TrPs in the ligaments.

Fat lobules and herniations of fat through the subcutaneous fascia in the lumbosacral area were identified as the source of referred backache[31] and were considered the cause of coccygodynia when they were located at the midsacral level, lateral to the midline.[34]

Swezey[123] observed that lumbar subcutaneous nodules occur in 25% of white adults, are rarely a cause of back pain, and seldom should require biopsy.

Related Trigger Points

The muscles that can cause or contribute to low back pain because of TrPs were presented in a 1983 review[116] and are summarized in Chapter 41, Section B. The back pain that is referred from TrPs in the abdominal wall musculature is presented in more detail in Chapter 49.

The deep paraspinal group is more likely to show isolated muscle involvement, whereas the more superficial paraspinal muscles are likely to accumulate associated TrPs in functionally related muscles, especially the contralateral superficial muscles.

When TrPs are active in the longissimus and iliocostalis muscles, the latissimus dorsi and quadratus lumborum also are often involved, either secondarily, or by the same initiating event that activated the paraspinal group. Frequently, the iliocostalis has a TrP that is a satellite induced by a key TrP in the latissimus dorsi muscle. In this case, the latissimus dorsi TrP must be treated. The serratus posterior inferior, and sometimes the serratus posterior superior, also may develop associated TrPs.

Not uncommonly, articular dysfunction of the thoracolumbar junction will be associated with active TrPs in the adjacent erector spinae, psoas muscle, and the quadratus lumborum muscle. Remarkably, if one treats the dysfunction of the thoracolumbar junction, or TrPs in one of the three muscles, the treatment often relieves TrPs in another one of the muscles.[77]

12. TRIGGER POINT RELEASE (Figs. 48.6 and 48.7)

In addition to the spray-and-stretch technique described here, other techniques including augmented postisometric relaxation (PIR) and contract relax as described in Chapter 3, Section 12 are also effective for releasing trigger points (TrPs) in many of these paraspinal muscles. Trigger-point pressure release is most helpful for inactivating TrPs in the most superficial layers of the erector spinae. A TrP in the iliocostalis thoracis that is refractory to treatment may be a satellite TrP induced by a key TrP in the latissimus dorsi muscle. Then Latissimus dorsi then must be released (the TrP inactivated) for full recovery.

Superficial Paraspinal (Erector Spinae) Muscles (Fig. 48.6)

Either of two seated stretch positions can be used. The less strenuous seated po-

sition (Fig. 48.6A) stretches chiefly the long thoracic paraspinal muscles. The more strenuous long-sitting position (Fig. 48.6B), in addition to strongly stretching the thoracic paraspinal muscles, also stretches the lumbosacral, gluteal and hamstring muscles.

To apply the less strenuous technique, the patient sits in a chair with the feet placed comfortably on the floor and the legs apart. The patient leans forward, lets the head hang forward, and lets the arms drop between the knees (Fig. 48.6A). After a few initial sweeps of spray, the operator gradually increases pressure on the upper back to guide the patient's movement as the vapocoolant spray is directed over the paraspinal muscles *bilaterally* in long downward parallel sweeps. At the same time, in order to hyperflex the thoracic spine, the patient is told to take a deep breath, to exhale fully, and to curl or "Hump the back!" The wrong instruction, "Arch your back!" usually causes the patient to extend, rather than to flex the spine. Vapocooling is followed promptly by application of moist heat to rewarm the skin, in the recumbent position, and then followed by active range of motion.

To obtain greater stretch of the low paraspinal muscles, the patient assumes the long-sitting position on a flat surface with the hips flexed and the knees straight (Fig. 48.6B). The paraspinal and gluteal muscles are then sprayed in parallel down sweeps, as in A for the seated position, but the sweeps continue over the buttocks (Fig. 48.6B). This position places a strong stretch on the gluteus maximus and hamstring muscles, which, if tight, should first be released by stretch and spray during straight-leg raising to permit the full range of flexion at the hips. This technique is presented in detail in Volume 2, Chapters 7 and 16.

An effective and comfortable way to lengthen the lumbar paraspinal muscles

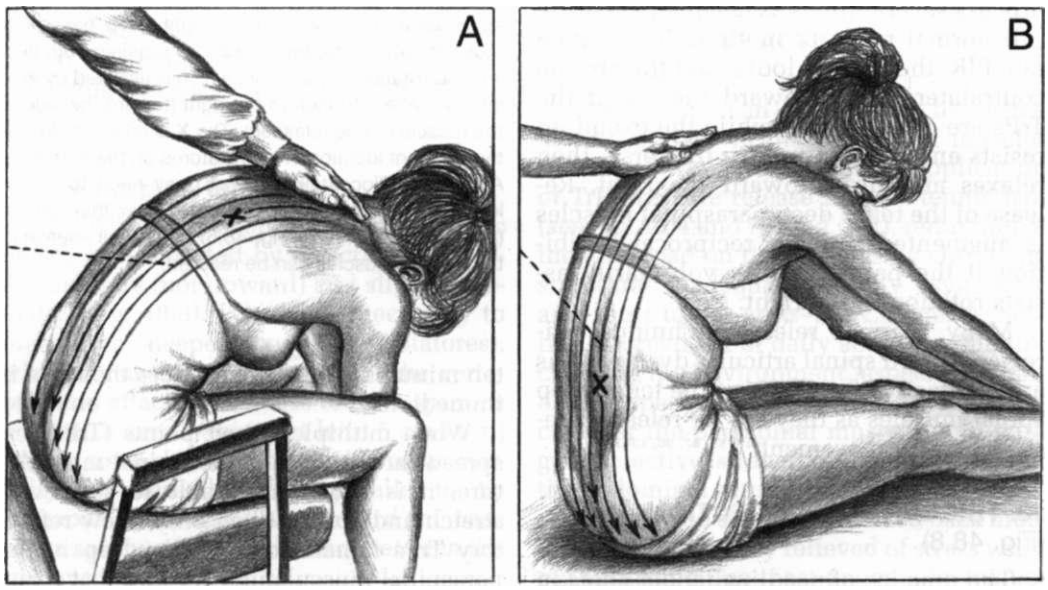


Figure 48.6. Stretch positions and spray pattern *(arrows)* for release of erector spinae muscles bilaterally. Typical locations of trigger points in the longissimus thoracis and iliocostalis lumborum muscles are indicated by the "**Xs**." **A,** stretch of chiefly the thoracic segments when the patient is seated with the knees bent. **B,** spray and stretch of the low thoracic and lumbar paraspinal muscles, plus the gluteal and hamstring muscles, with the patient in the long-sitting position with the knees straight. The posterior spinal musculature is stretched bilaterally, and must be vapocooled ***bilaterally***. The practitioner guides the patient's movement into flexion, but does not force it. (For a more isolated stretch of the lumbar area, see Fig. 48.14.)

without excessive flexion at higher levels is illustrated in Chapter 49 (see Fig 47.7B). In the supine position, with the hips and knees flexed and held by the hands, the patient can augment the low back stretch using PIR. The patient presses the buttocks downward against the bed (contracting the lumbar extensors) and then relaxes, pulling the thighs up toward the chest. A similar method is illustrated later in this chapter (see Fig. 48.14B).

Lewit[78] fully describes and illustrates the use of PIR for release of tension in the erector spinae muscles that is caused by TrPs.

Deep Paraspinal Muscles (Fig. 48.7)

To stretch the multifidus and rotatores muscles, the seated patient's spine is flexed and simultaneously rotated, turning the chest toward the side of the involved muscle. After initial sweeps of spray (Fig. 48.7), the operator takes up the slack that develops and repeats the process several times as needed to achieve full normal range of motion. To incorporate PIR, the patient looks first toward the contralateral side (toward the left if the TrPs are on the right) while the examiner resists any attempt to turn the torso, then relaxes and turns toward the right. Release of the tense deep paraspinal muscles is augmented through reciprocal inhibition if the patient gently voluntarily assists rotation to the right.

Many manual release techniques directed toward spinal articular dysfunctions are as effective for releasing the tense deep spinal muscles as they are for releasing restricted joint movement.[16,33,87,190,135]

Figure 48.7. The position of stretch and diagonal spray pattern *(arrows)* for the right deep paraspinal muscles. Stretching these muscles requires both flexion and rotation of the torso, which is assisted by asking the patient to look to the right (toward the side of the muscles to be released). The **X** is an example of a trigger point location in the rotatores on the right side. A tight left iliocostalis thoracis may need to be released by adding sweeps of spray over that muscle (as on the left side of Fig. 48.6) before full release of the deeper muscles can be realized.

13. TRIGGER POINT INJECTION (Fig. 48.8)

The practice of needling tender spots in lumbar muscles for the treatment of low back pain is not new. In 1912 Osier[98] wrote, "For lumbago, acupuncture is, in acute cases, the most efficient treatment. Needles of from three to four inches in length (ordinary bonnet-needles, sterilized, will do) are thrust into the lumbar muscles at the seat of pain, and withdrawn after five or ten minutes. In many instances the relief is immediate ..."

When multiple trigger points (TrPs) are spread throughout the paraspinal musculature, it is usually desirable to start with stretch and spray. When only a few refractory TrPs remain, or they lie deep in the paraspinal musculature, we find that injection is best. Injection of TrPs in the paraspinal musculature has previously been reported extensively.[12,13,18,20,45,48,49,59,73,81,110,137] Sometimes injection of TrPs in abdominal muscles is required to relieve back pain.[17,116]

The TrP injection is followed at once by a repetition of stretch and spray, and then by moist heat and active range of motion.

Superficial Paraspinal (Erector Spinae) Muscles

The longissimus and iliocostalis TrPs are clearly palpable and readily located for injection in all but very obese patients. When injecting the iliocostalis thoracis muscle, the needle must be directed tangent to, and not between, the ribs, to avoid pneumothorax.

When injecting TrPs in the superficial group of muscles at the mid- to low-thoracic level, needle penetration of TrPs located more medially in the longissimus thoracis muscle refers pain caudally. The patient sometimes expresses surprise when injection of another TrP located 1-2 cm (about 3/4 in) more laterally in the iliocostalis thoracis muscle refers pain upward toward the shoulder, instead of downward.

Rachlin[104] illustrates the injection of a TrP in the longissimus thoracis and one in the iliocostalis lumborum.

Distinguishing central from attachment TrPs in the paraspinal muscles can be difficult even in the more superficial muscles. The presence of a tender nodule with a taut band extending in either direction is highly suggestive of a central TrP. Sometimes, the tenderness can be identified as occurring at the attachment of the tense muscle fibers.

Deep Paraspinal Muscles

The TrPs in the deep paraspinal thoracic muscles are injected by directing the needle caudally (not upward) and slightly medially (Fig. 48.8). When it is necessary to inject the deepest muscles (rotatores), which lie against the laminae of the vertebrae and attach at the base of each spinous process, a needle that is at least 5 cm (2 in) long is used. It is directed somewhat caudally and medially, nearly parallel to the long axis of the spine and toward the base of the spinous process, but *not between* the spinous processes.

This angle of the needle, while reaching the tender spots in the deepest paraspinal muscles, eliminates the possibility of introducing the needle between the ribs into the pleural cavity, or between the vertebrae into the epidural space. The caudal slant of the needle is indicated because of the shingle-like overlap of the laminae. Penetration to a depth greater than the laminae is unnecessary and undesirable.

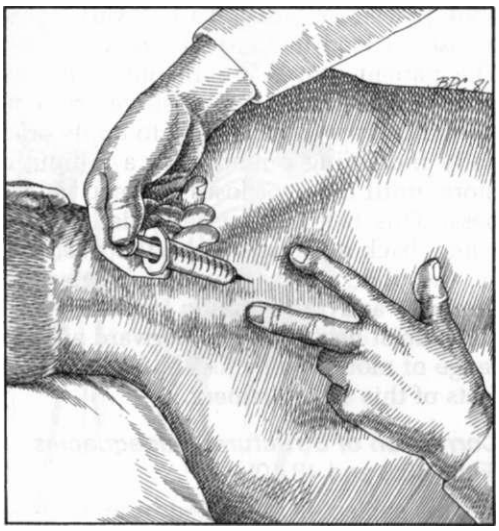


Figure 48.8. Injection of the right multifidus and rotatores muscles at the upper thoracic level. The needle is aimed slightly caudad to avoid penetrating between the vertebral laminae.

14. CORRECTIVE ACTIONS (Figs. 48.9-48.14)

The patient can reduce the hyperirritability of TrPs in the paraspinal muscles in several ways including self-application of TrP pressure release using a tennis ball *[see* Fig. 18.4 and related text), reduction of the total load on the muscles by correcting structural inadequacies (body asymmetry and short upper arms-see Chapter 4, Section A), revision of daily activities, modification of the environment, especially chair design, performing passive stretch exercises for the paraspinal muscles, and with graded active strengthening exercises for the abdominal muscles.

Acutely and severely involved back muscles may be partially relieved of stress without incapacitating most patients by temporary application of a corset or brace for low back support, as described by Cailliet.[21]

Trigger Point Pressure Release

The patient can apply this release therapy to TrPs in the superficial back muscles by lying supine on a tennis ball, either on the floor, or on a bed with a large, thin

book placed under the ball. A variation is to use an iced tennis ball (*see* Fig. 18.4). The patient moves around until the ball presses directly on the sensitive TrP; controlled body weight is used to apply gradually increasing pressure for a minute or more, until the spot loses its deep tenderness. This technique is especially useful where back muscles overlie the ribs, such as the iliocostalis thoracis, longissimus thoracis, and the serratus posterior muscles. Moist heat applied afterward and full range of motion enhance the beneficial effects of this self-treatment.

Correction of Structural Inadequacies (Figs. 48.9 and 48.10)

This subject is presented in more detail in Chapter 4. The following summarizes the essential facts.

A functional scoliosis develops in order to compensate for lateral tilting of the pelvis that is caused by a short leg when standing, or by a small hemipelvis when sitting. Such body asymmetry imposes persistent muscle strain that perpetuates TrPs in the paraspinal and associated musculature, and must be corrected.

Limb Length Inequality. Chapter 4 of Volume 2 presents lower limb-length inequality and an analysis of its effects on spinal curvatures in considerably more detail. Nichols[93] recognized that a lower limb length inequality can make a critical contribution to musculoskeletal pain. Ordinarily, a difference of as much as 1.3 cm (1/2 in) in leg length alone does not activate TrPs and cause pain, but it is a powerful perpetuating factor that can convert an acute TrP problem into a chronic one. The persistent TrP activity causes chronic referred pain. In addition, focal reduction of skin resistance over the tender spots in the muscles demonstrates one of their autonomic effects.[72] The functional scoliosis (Fig. 48.9B and *see* Volume 2, Chapter 4) due to the short leg and tilted pelvis requires continuous compensatory muscular activity in the upright position, which overloads the paraspinal muscles.

The scoliotic spine also tilts the shoulder-girdle axis. Usually, the shoulder sags on the side of the longer leg (Fig. 48.9B); but if the disparity in leg length is about 1.3 cm (1/2 in) or more, the shoulder is likely to be lower on the shorter side. The patient often stands on the shorter leg with the longer leg in front or to the side (Fig. 48.9A).

To ensure lasting relief from the myofascial pain, it is important to correct a leg length discrepancy of as little as 0.3 cm (1/8 in) in a short person. The correction must be worn *whenever* these patients are on their feet, including the use of bedroom slippers. The patient should avoid walking, or jogging, on slanted ground or a slanted beach.

To make a functional determination of the leg length difference, the patient stands with the feet together, or at most 7.6 cm (3 in) apart, and is observed from behind. To identify a short leg, the patient is examined (1) for asymmetry of the body silhouette between the ribs and the pelvis, (2) for lateral tilt of the lumbar spine as it leaves the sacrum, (3) for the ensuing lateral scoliosis, (4) for a tilted shoulder-girdle axis (the symmetry of the scapular bulges are more reliable than the line of the shoulders, which is easily modified by trapezius muscle involvement, (5) for a low posterior superior iliac spine, by palpation or by eye for one low dimple, and (6) by palpation for a low iliac crest on one side.

A sufficient lift (pages of a pad or magazine) is placed under the heel of the side on which the pelvis is low in order to level the pelvis and straighten the spine. This usually corrects the other signs of asymmetry. The necessity for the correction is convincingly demonstrated to the patient by calling attention to the asymmetry seen in a full-length mirror, especially when the heel lift is briefly transferred under the heel of the longer leg, doubling the existing difference. The patient feels uncomfortable with the correction on the wrong side, and the resulting marked aggravation of the indicators of asymmetry confirms the shortness of the other leg. A difference of 0.5 cm (3/16 in) is often a significant source of muscle strain that requires correction.

The difference in leg length is corrected temporarily by inserting the correct thickness of firm felt inside the heel of the shoe, or permanently by building up the outside thickness of the shoe heel under the short side if the shoe has a low heel (Fig. 48.9C),

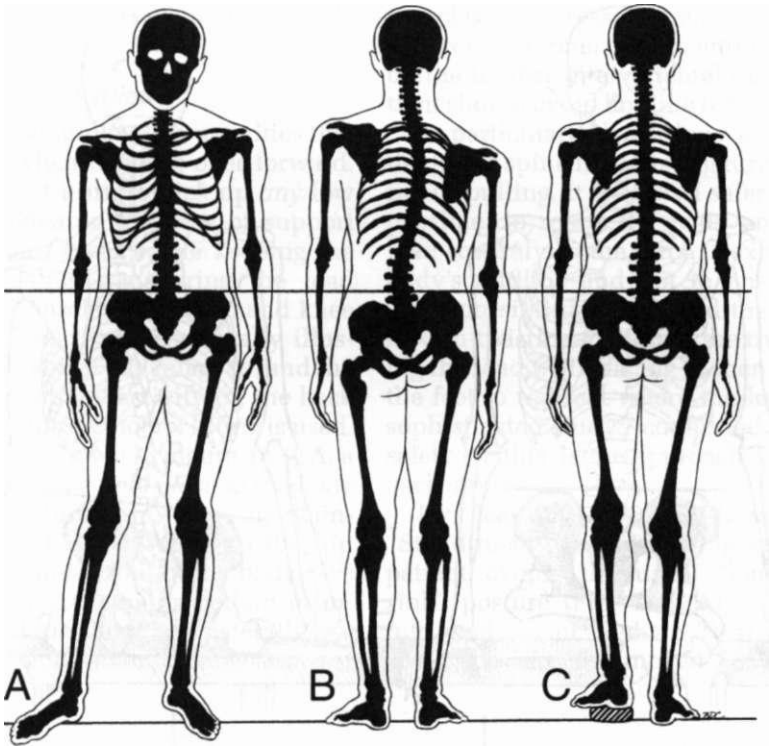


Figure 48.9. Example of skeletal asymmetry due to a relatively short left leg. **A,** to compensate, the patient stands on the shorter left leg, placing the longer right leg forward and slightly to the side. This levels the pelvis. **B,** tilted pelvis, functional scoliosis, and tilted shoulder-girdle axis when the patient stands with the feet nearly together. **C,** the discrepancy in leg length is corrected by adding the precise lift under the shorter (left) leg. This levels the pelvis with the feet nearly together.

or by cutting down the shoe heel on the long side, if it has a high heel. With large corrections of about 1.3 cm (1/2 in) or more, it is wise to divide the difference by removing half of the correction from the heel on the long side and by adding half to the other heel.

Correcting the leg-length discrepancy alone may be sufficient to relieve the pain of muscular origin.[113] By removing the perpetuating factors, the active TrPs causing the pain may spontaneously revert to latent TrPs in a few days or weeks.

Asymmetrical Pelvis. Usually, the vertical dimension of the pelvis is smaller on the side of the shorter leg. This tilts the pelvis when sitting, just as the limb-length inequality tilts it when standing, and with the same musculoskeletal effects (Fig. 48.10B and *see* Volume 2, Chapter 4).

While the patient sits on a flat level wood seat, pelvic tilt is estimated and corrected by placing enough pages or sheets of paper under the ischial tuberosity on the shorter side to level the pelvis exactly (Fig. 48.10**C**). A hard surface requires less correction than a well padded seat, since the softness of the seat allows the body to tilt to the short side. This shifts more weight to that side, and increases the pelvic tilt (Fig. 48.10D). The patient's muscles become very discriminating as to the size of the "butt" lift needed, in relation to each chair seat. Some seats are domed and others are scooped, as in the bucket seat.

A pelvic tilt also may be produced unwittingly by sitting on a wallet in the back pocket, causing "back-pocket sciatica,"[57] or by sitting regularly in a tilted office chair, or on a piano bench that has two rubber feet missing at one end or is placed on a slanted stage.

The patient often tries to compensate for a small hemipelvis by crossing one knee

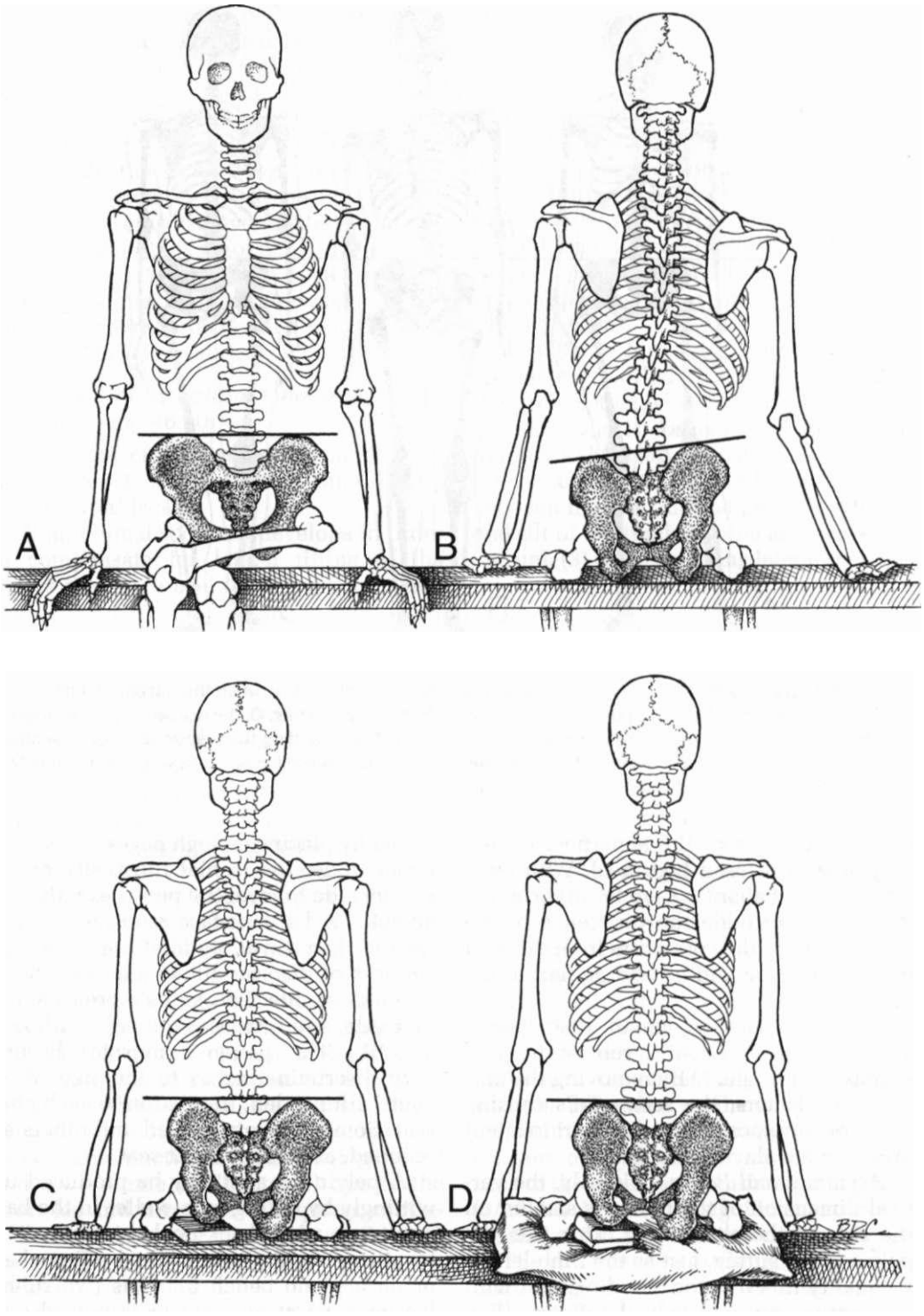


Figure 48.10. Effects of skeletal asymmetry due to a smaller left hemipelvis are demonstrated by sitting on a flat level wood bench. **A,** crossing the leg on the shorter side over the other knee helps to level the pelvis. **B,** the tilted pelvis causes compensatory scoliosis, which tilts the shoulder-girdle axis. **C,** a small ischial lift levels the pelvis on a hard surface. **D,** on a soft cushioned surface, a thicker ischial lift is required to provide the same correction as that obtained on a hard surface.

over the other to cantilever up the low side (Fig. 48.10A).

Modification of Activities

The patient should modify activities that induce stress when bending over forward. The patient must learn to pick up *any* low object by broadening the base of support and bending the knees while keeping the back upright, thus transferring the load from the back muscles to the hip and knee extensors. Tichauer[124,126] graphically illustrated the mechanical advantage to, and the reduction of electrical activity in, the back muscles when this method of lifting is used. During lifting, as shown in Figure 48.11 A, a heavy object must be *held close to the body* with the pelvis "tucked in," thus maintaining the center of gravity close to the hip joints, rather than in front of the body.[22,125] Increasing the base of support both in an anteroposterior dimension and laterally reduces strain. Contracting the abdominal muscles to increase intra-abdominal pressure relieves some of the compressive forces on the lumbar intervertebral discs. The patient should avoid holding his or her breath.

A particularly hazardous movement to the lower spine is a twisting turn while lifting or pulling. It is MUCH safer, whenever possible, to rotate the body and face the load squarely so the force is exerted in the body's midline and not to one side. The other possibility is to avoid the combined flexion-twisting motion of the trunk by lifting the load while facing it, then pivot with the feet to redirect where the load goes. A sophisticated study[47] confirmed the greater safety of this latter approach in terms of back strain.

By learning the "Sit-to-stand" and "Stand-to-sit" Technique (Fig. 48.12B), the patient avoids the usual "bent-over-the-sink" posture (Fig. 48.12A) when getting into, and out of, a chair. To rise from the chair, the hips are moved forward to the

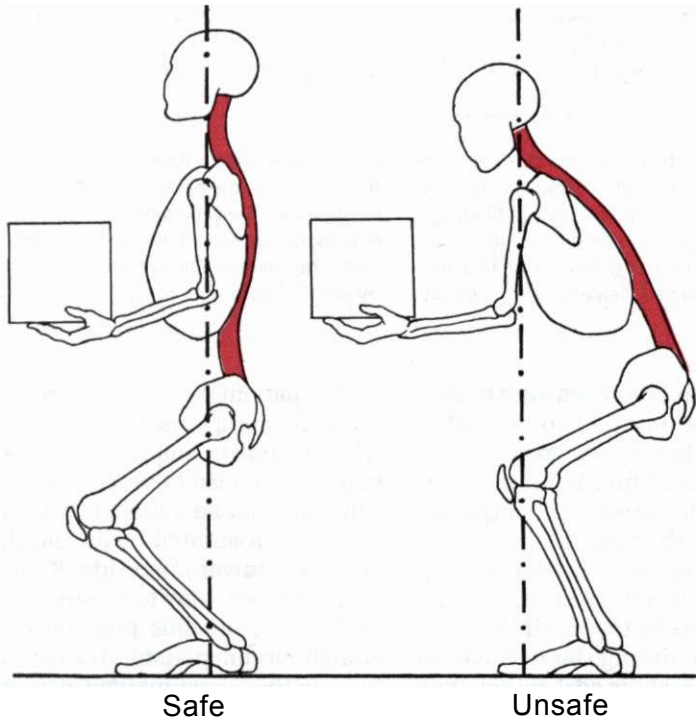


Figure 48.11. Safe and unsafe ways of lifting. **Left side,** safe position, keeping the object close to the body and lifting with the hip and knee extensors. The center of gravity falls through the pelvis. **Right side,** unsafe way with the object held out in front of the body. Here, the trunk leans forward, which forces the paraspinal muscles to lift like a crane, overloading them and increasing compressive forces on the lumbar intervertebral discs.

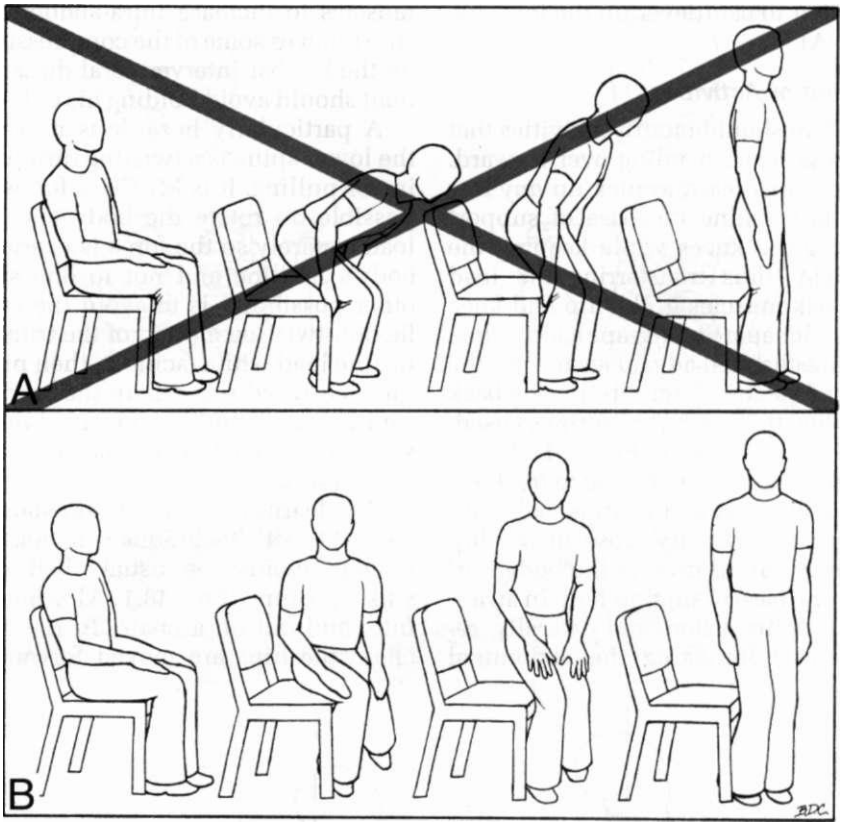


Figure 48.12. The Sit-to-stand and Stand-to-sit Technique is a method for minimizing strain on the low back muscles and the intervertebral fibrocartilaginous discs while getting up from or sitting down in a chair. **A,** poor, usual method of rising from a chair which places the back in a strained "leaning-over" posture. **B,** the better Sit-to-stand Technique (reading from left to right) keeps the spine erect throughout, from sitting to standing. This movement loads the hip and knee extensors instead of the thoracolumbar back muscles. The reverse Stand-to-sit technique is illustrated by reading from right to left.

front of the chair seat before starting to rise; the body and hips are turned somewhat to the side, and one foot is placed beneath the front edge of the chair; finally, the torso is held erect while the knees and hips are straightened, lifting the body.

The process is reversed in Stand-to-sit by turning to the side and placing one foot under the front edge of the chair, keeping the torso erect, and aiming the buttocks at the *front* edge of the chair seat rather than at the *rear* of the seat. The person then slides backward on the seat to meet the backrest. This procedure again maintains the back in an erect position and transfers the load from the paraspinal muscles to the hip and thigh muscles.

The patient with myofascial back pain has difficulty going upstairs or climbing a ladder while facing the steps or rungs because of the tendency to lean forward and flex the spine. The pain can be avoided by turning the entire body at an angle with the steps, to face about 45° toward one side. It may be slower climbing, and the feet must travel around each other, but this position automatically straightens the patient's posture, lightens the load on the paraspinal muscles, and may enable the patient to go up and down steps without pain. A tension-reducing way to ascend stairs is illustrated in Figure 41.6B.

Correction of faulty body posture when standing and sitting, as described in Chapter 41, Section C, reduces muscle strain

and, therefore, the likelihood of recurrence of myofascial pain.

Modification of Environment

The paraspinal musculature can be relieved of much unnecessary stress by modifying the seating to fit the person and the task, and by changes of position (see Fig. 41.5B and C). The backrest of a chair should provide enough lumbar support to maintain the normal lumbar lordotic curve when the muscles relax. The chair, not the muscles, should do the work of maintaining correct posture. Simply reclining the backrest does not affect lumbar lordosis.[4,5] If the seat has a straight back with no forward curvature at waist level (a fault of many chairs), support for the normal lumbar lordosis should be supplied by a pad, such as a small pillow or a roll of folded bath towel (see Figs. 16.4D and 41.4E). It is placed at belt level against the back of the chair, or auto seat, and adjusted up or down for comfort and upright posture. Seated posture which completely eliminates lumbar lordosis[136] may be helpful for brief periods as a postural variation but can, by itself, cause muscle strain if maintained for a prolonged time, as when driving a car. To relieve tension during prolonged sitting, the paraspinal muscles should be stretched regularly by changing position.

In an extensive study to determine what chair design causes minimum muscular stress, as measured electromyographically when typing, Lundervold[83] found that the chair should have a backrest with a backward slope, a seat which is slightly hollowed out, no casters, and firm upholstery. Seat height should be low enough so that the feet rest flat on the floor without compression of the thigh by the front edge of the seat. A footrest may be used to avoid underthigh compression. The lower edge of the backrest is positioned to support that part of the lumbar spine which flexes the most when bending forward. The upper edge of the backrest should reach high enough to cover and support at least the inferior angles of the scapulae.

The under surface of a keyboard support should fit just above the operator's knees, keeping the keyboard close to lap-level. Short armrests can be helpful, if they are the correct height for that person's body structure and work set-up.

A bed that is too soft and sags in the middle like a hammock aggravates tension in the back muscles. This is remedied by placing a plywood bed board, nearly as large as the mattress, between the mattress and the bed spring. Alternatively, several separate boards 1.3 cm ($^1/_2$ in) thick and 15-20 cm (6-8 inches) wide, cut three-quarters of the length of the mattress, may be placed lengthwise. The separate boards are more readily installed under the mattress, and also may be transported on a trip. If boards or slats are placed crosswise underneath the mattress, a sufficient number must be used to provide a smooth correction for a hammock-like longitudinal sag of the bed.

When sleeping on the side rather than supine in a firm flat bed, the patient with myofascial back pain is usually more comfortable with a pillow placed under the uppermost knee. This prevents the rotary torsion of the lumbar spine that occurs when the knee drops forward onto the bed.

Exercises

The In-bathtub Stretch Exercise (Fig. 48.13) should be performed in comfortably warm water (provided there is no medical contraindication to the increased cardiovascular load caused by the heat). The patient actively leans forward with the knees straight, and assists dorsal relaxation by letting the head hang forward. The patient then walks the fingers down the shins until a pull is felt on the stretched paraspinal muscles, and then a little further to slight discomfort. After holding this degree of stretch for several seconds, tautness usually slackens. The patient leans back, relaxes, and breathes deeply with abdominal respiration, then leans forward to take another step of the fingers to "take up the slack." This re-establishes the previous degree of tension on the slightly longer paraspinal muscles. This slow, step-wise passive stretch helps to recapture the lost range of motion of the long back muscles. At the same time, the hamstring muscles are passively stretched as the pelvis rotates. The patient must be warned that if iliopsoas

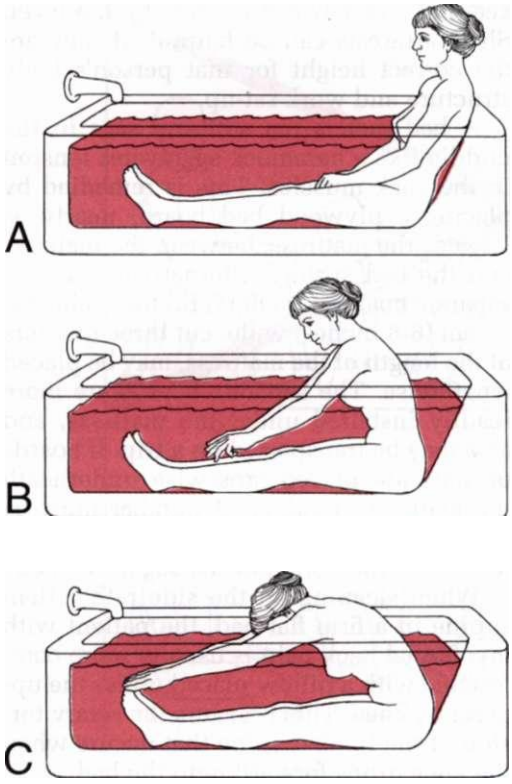


Figure 48.13. The In-bathtub Stretch Exercise. **A,** relaxed position in the bathtub with warm water covering the lower limbs and the lumbosacral area. **B,** partial stretch. **A** comfortably tolerable forward stretch effort is maintained until the erector spinae and/or hamstring tightness releases enough to allow another progression of the fingertips forward on the shins, ankles or feet. **C,** maximum stretch by reaching forward, while keeping the neck and back completely limp and relaxed. This long-sitting position puts a full stretch on both the hamstring and paraspinal muscles (tightness of either group of muscles limits this reach).

TrPs are also present, a forward flexion effort in this seated position contracts the iliopsoas muscles in a shortened position and can activate any latent TrPs in them. These TrPs should be inactivated *(see* Volume 2, Chapter 5) first, and the patient shown how to stretch them in case the bathtub stretch initiates a reactive cramp due to TrPs in the iliopsoas muscles. If lacking a bathtub, a similar "dry-land" stretch performed daily can be very helpful.

Patients should be reminded that whenever a muscular problem arises that compromises their ability to get out of a bathtub, they should try to roll over on their hands and knees and then crawl over the side of the tub.

The Pelvic Tilt Exercise *(see* Fig. 49.12 in the next chapter) stretches the paraspinal muscles while strengthening the muscles of the abdomen. Further strengthening of the abdominals is achieved by using the Sit-back, Abdominal-curl, and Sit-up Exercises *(see* Fig. 49.13 A-C). The sit-back phase should be performed *slowly,* not rapidly. Strong abdominal muscles can provide 30% to 50% additional weight-carrying support to the thoracolumbar spine.[45, 92]

The Low-back Stretching Exercise in the supine position begins by drawing one knee to the chest with the hands clasped around the thigh behind the knee. This stretches the hip and low back extensors (Fig. 48.14A). Next, that lower limb is returned to the straight-leg starting position, and the other thigh is flexed to the chest and returned. Finally, both legs are pulled to the chest (Fig.

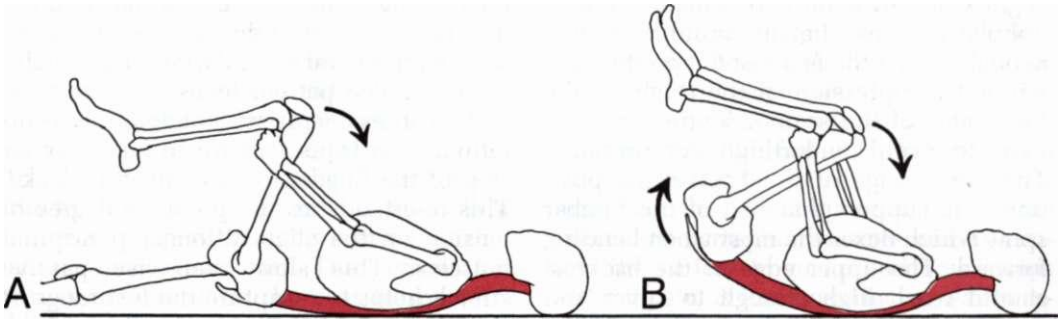


Figure 48.14. Low-back Stretching Exercise. **A,** first phase: flexion of ***one*** thigh at a time by rhythmically and gently bringing the knee toward the corresponding armpit. (Toward the opposite armpit provides a gluteus maximus stretch.) **B,** second phase: flexion of ***both*** thighs together, brought tight onto the chest. The thighs, rather than the knees, are grasped to avoid forced knee flexion. (See also Fig. 49.7B.)

48.14B). Postisometric relaxation can be incorporated as described in Section 12.

SUPPLEMENTAL REFERENCES, CASE REPORTS

Travell has reported the total management of patients with myofascial TrPs of the paraspinal muscles.[132,135]

REFERENCES

1. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 235, Fig. 4.49).
2. *Ibid.* (p. 237, Fig. 4.53).
3. *Ibid.* (p. 238, Fig. 4.54).
4. Andersson BG, Murphy RW, Ortengren R, *et al.:* The influence of backrest inclination and lumbar support on lumbar lordosis. *Spine* 4:52-58, 1979.
5. Andersson BJ, Jonsson B, Ortengren R: Myoelectric activity in individual lumbar erector spinae muscles in sitting. A study with surface and wire electrodes. *Scand f Rehabil Med 3(Suppl):91-108,* 1974.
6. Andersson BJ, Ortengren R: Myoelectric back muscle activity during sitting. *Scand J Rehabil Med 3(Suppl):73-90,* 1974.
7. Andersson GB, Ortengren R, Herberts P: Quantitative electromyographic studies of back muscle activity related to posture and loading. *Orthop Clin North Am* 8:85-96, 1977.
8. Baker DM: Changes in the corium and subcutaneous tissues as a cause of rheumatic pain. *Ann Rheum Dis* 24:385-391, 1955.
9. Basmajian JV: Electromyography—its significance to the manipulator. Chapter 3. In: *Back Pain: An International Review.* Edited by Paterson JK, Burn L. Kluwer Academic Publishers, Boston, 1990 (pp. 21-26).
10. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 261, 355-358, 360).
11. Bates T, Grunwaldt E: Myofascial pain in childhood. *JPediatr* 55:198-209, 1958.
12. Berges PU: Myofascial pain syndromes. *Postgrad Med* 53:161-168, 1973.
13. Blank VK: Bort bei Lumbalgien, Ischialgien, vertebragenen Syndromen und Muskelharten Verspanmmgszustanden. *Hippokrates* 38:528-530, 1967.
14. Bogduk N, Twomey LT: *Clinical Anatomy of the Lumbar Spine.* Churchill Livingstone, New York, 1987 (p. 99).
15. Bonica JJ, Sola AE: Other painful disorders of the low back. Chapter 72. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, et *al.* Lea & Febiger, Philadelphia, 1990, pp. 1490-1498.
16. Bourdillon JF: *Spinal Manipulation.* Ed. 2. Appleton-Century-Crofts, New York, 1973.
17. Bourne IH: Treatment of painful conditions of the abdominal wall with local injections. *The Practitioner* 224:921-925, 1980.
18. Bourne IH: Treatment of chronic back pain. *The Practitioner* 228:333-338, 1984.
19. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sport Skills, an Electromyographic Study.* Charles C Thomas, Springfield, Ill., 1967.
20. Brown BR: Diagnosis and therapy of common myofascial syndromes. *JAMA* 239:646- 648, 1978.
21. Cailliet R: *Soft Tissue Pain and Disability,* F.A. Davis, Philadelphia, 1977.
22. Cailliet R: *Low Back Pain Syndrome.* Ed. 3. F.A. Davis, Philadelphia, 1981 (pp. 109- 115, Figs. 76, 77, 98).
23. Campbell EJ: Accessory muscles. Chapter 9. In: *The Respiratory Muscles,* edited by Campbell EJ, Agostoni E, Davis JN. Ed. 2. W.B. Saunders, Philadelphia, 1970 (p. 188).
24. Carter BL, Morehead J, Wolpert SM, *et al: Cross-Sectional Anatomy.* Appleton- Century- Crofts, New York, 1977 (Sects. 20-35).
25. *Ibid.* (Sects. 23-26, 28, 29).
26. Chu J: Dry needling (intramuscular stimulation) in myofascial pain related to lumbosacral radiculopathy. *Eur J Phys Med Rehabil* 5(4):106-121, 1995.
27. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 466- 471, 1198, Fig. 12-32).
28. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Figs. 524, 525).
29. *Ibid.* (Fig. 526).
30. *Ibid.* (Fig. 529).
31. Copeman WS, Ackerman WL: Edema or herniations of fat lobules as a cause of lumbar and gluteal "fibrositis." *Arch Intern Med* 79:22-35, 1947.
32. Crow NE, Brodgon BG: The "normal" lumbosacral spine. *Radiology 72:97,* 1959.
33. Cyriax J: *Textbook of Orthopaedic Medicine.* Ed. 8, Vol. 2 *Treatment by Manipulation, Massage and Injection.* Williams & Wilkins, Baltimore, 1971.
34. Dittrich RJ: Coccygodynia as referred pain. *J Rone Joint Surg* 33A.-715-718, 1951.
35. Dittrich RJ: Low back pain—referred pain from deep somatic structure of the back. *J Lancet 73:63-*68, 1953.
36. Dittrich RJ: Somatic pain and autonomic concomitants. *Am J Surg* 87:66-73, 1954.
37. Dittrich RJ: Soft tissue lesions as cause of low back pain: anatomic study. *Am J Surg* 92:80-85, 1956.
38. Donaldson CC, Skubick DL, Clasby RG, *et al:* The evaluation of trigger-point activity using dynamic EMG techniques. *Am J Pain Manage* 4:118-122, 1994.
39. Donisch EW, Basmajian JV: Electromyography of deep back muscles in man. *Am J Anat* 233:25-36, 1972.
40. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 505, 506).
41. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (Fig. 56).
42. *Ibid.* (Fig. 59).
43. *Ibid.* (Fig. 62).
44. Ferner H, Staubesand J: *Sobotta Atlas of Human Anatomy.* Ed. 10, Vol. 2, *Thorax, Abdomen, Pelvis, Lower Extremities, Skin.* Urban & Schwarzenberg, Baltimore, 1983 (p. 237).
45. Finneson BE: *Low Back Pain.* J.B. Lippincott, Philadelphia, 1973 (pp. 31-33, 99, 100).
46. Fischer AA: New developments in diagnosis of myofascial pain and fibromyalgia. *Phys Med Rehabil Clin North Am* 8(1):1-21, 1997.
47. Gagnon M, Plamondon A, Gravel D: Pivoting with the load. An alternative for protecting the back in

asymmetrical lifting. *Spine 18(11):1515-1524* 1993.
48. Garvey TA, Marks MR, Wiesel SW: A prospective, randomized double-blind evaluation of trigger-point injection therapy for low-back pain. *Spine* 24(9):962-964, 1989.
49. Gerwin RD: Myofascial aspects of low back pain. *Neurosurg Clin North Am* 2(4):761-784, 1991.
50. Gitelman R: A chiropractic approach to biomechanical disorders of the lumbar spine and pelvis. Chapter 14. In: *Modern Developments in the Principles and Practice of Chiropractic.* Edited by Haldeman S. Appleton-Century-Crofts, New York, 1980, pp. 297-330 (see p. 307).
51. Good MG: The role of skeletal muscles in the pathogenesis of diseases. *Acta Med Scand* 238:285-292, 1950 (p. 286).
52. Gough JG, Koepke GH: Electromyographic determination of motor root levels in erector spinae muscles. *Arch Phys Med Rehabil* 47:9-11, 1966.
53. Gould N: Back-pocket sciatica. *N Engl J Med* 290:633, 1974.
54. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996 (pp. 26, 316).
55. Gunn CC, Milbrandt WE: Tenderness at motor points. *J Bone Joint Surg* 58A:815- 825, 1976.
56. Gunn CC, Milbrandt WE: Early and subtle signs in low-back sprain. *Spine* 3:267-281, 1978.
57. Gutstein M: Diagnosis and treatment of muscular rheumatism. *Br J Phys Med* 2:302- 321, 1938.
58. Harman JB, Young RH: Muscle lesions simulating visceral disease. *Lancet* 2:1111- 1113, 1940.
59. Hench PK: Nonarticular rheumatism. In: *Rheumatic Diseases: Diagnosis and Management.* Edited by Katz WA. J.B. Lippincott, Philadelphia, 1977 (p. 624).
60. Hirschberg GG, Froetscher L, Naeim F: Iliolumbar syndrome as a common cause of low back pain: Diagnosis and prognosis. *Arch Phys Med Rehabil* 60:415-419, 1979.
61. Hollinshead WH: *Anatomy for Surgeons.* Ed. 3, Vol. 3, *The Head and Neck.* Harper & Row, Hagerstown, 1982 (p. 79, Fig. 2-2).
62. Janda V: Evaluation of muscular imbalance. Chapter 6. In: *Rehabilitation of the Spine: A Practitioner's Guide.* Edited by Liebenson C. Williams & Wilkins, Baltimore, 1996 (pp. 97-112).
63. Jayasinghe WJ, Harding RH, Anderson JA, *et al.:* An electromyographic investigation of postural fatigue in low back pain—a preliminary study. *Electromyo Clin Neurophysiol* 28:191-198, 1978.
64. Jenkins DB: *Hollinshead's Functional Anatomy of the Limbs and Rack.* Ed. 6. W.B. Saunders, Philadelphia, 1991 (pp. 203, 204).
65. Jonsson B: Morphology, innervation, and electromyographic study of the erector spinae. *Arch Phys Med Rehabil* 50:638-641, 1969.
66. Jonsson B: Topography of the lumbar part of the erector spinae muscle. *Z Anat Entwickl Gesch* 230:177-191, 1970.
67. Jonsson B: Electromyography of the erector spinae muscle. In: *Medicine and Sport,* Vol. 8: *Biomechanics III.* Edited by Jokl E. Karger, Basel, 1973.
68. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3.175-190, 1938 (pp. 180, 181, 185, 186; Figs. 3, 5, 9).
69. Kellgren JH: The anatomical source of back pain. *Rheumatol Rehabil* 26:3-12, 1977 (p. 7, Fig. 3; and p. 9, Fig. 4).
70. Kelly M: Pain in the chest: observations on the use of local anaesthesia in its investigation and treatment. *Med J Aust* 2:4-7, 1944 (pp. 5, 6, Case 4).
71. Korr IM, Wright HM, Chace JA: Cutaneous patterns of sympathetic activity in clinical abnormalities of the musculoskeletal system. *Acta Neurovegetativa* 25:489-606, 1964.
72. Korr IM, Wright HM, Thomas PE: Effects of experimental myofascial insults on cutaneous patterns of sympathetic activity in man. *Acta Neurovegetativa* 23:329-355, 1962.
73. Kraus H: *Clinical Treatment of Back and Neck Pain.* McGraw-Hill, New York, 1970 (pp. 83, 98, 105, 106).
74. Lange M: *Die Muskelhaerten (Myogelosenj; Ihre Entstehung und Heilung.* J.F. Lehmanns, Munchen, 1931 (pp. 30, 91, 137, 138, 152, 158).
75. Letts RM, Quanbury AO: Paraspinal muscle activity. *Phys Sportsmed* 6(9):80-90, 1978.
76. Lewis T, Kellgren JH: Observations relating to referred pain, viscero-motor reflexes and other associated phenomena. *Clin Sci 1*:47-71, 1939.
77. Lewit K: Muscular pattern in thoraco-lumbar lesions. *Manual Med* 2.105-107, 1986.
78. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (pp. 205-207).
79. Lindstedt F: Zur Kenntnis der Aetiologie und Pathogenese der Lumbago and ahnlicher Riickenschmerzen. *Acta Med Scand* 55:248-280, 1921.
80. Lindstedt F: Uber die Nature der muskelrheumatischen (myalgischen) Schmerzsymptome. *Acta Med Scand 30(Suppl):1-180,* 1929.
81. Livingston WK: *Pain Mechanisms, A Physiologic Interpretation of Causalgia and Its Related States.* Macmillan, New York, 1943, reprinted by Plenum Press, New York, 1976 (pp. 134, 135).
82. Llewellyn LJ, Jones AB: *Fibrositis.* Rebman, New York, 1915 (Fig. 39).
83. Lundervold A: Electromyographic investigations during sedentary work, especially typing. *Br J Phys Med* 24:32-36, 1951.
84. Lynn P: Personal communication, 1995.
85. Macintosh JE, Bogduk N: The biomechanics of the lumbar multifidus. *Clinical Biomechanics* 2:205-213, 1986.
86. Macintosh JE, Valencia F, Bogduk N, *et al.:* The morphology of the human lumbar multifidus. *Clinical Biomechanics* 2:196-204, 1986.
87. Maigne R: *Diagnosis and Treatment of Pain of Vertebral Origin: A Manual Medicine Approach.* Williams & Wilkins, Baltimore, 1996 (pp. 480-502).
88. McMinn RM, Hutchings RT, Pegington J, ef *al: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, St. Louis, 1993 (p. 96).
89. *Ibid.* (p. 95).
90. Mennell JM: *Back Pain.* Little, Brown & Company, Boston, 1960.
91. Morris JM, Banner G, Lucas DB: An electromyographic study of the intrinsic muscles of the back in man. *J Anat (Lond)* 96:509-520, 1962.
92. Morris JM, Lucas DB, Bresler B: Role of the trunk in stability of the spine. *J Bone Joint Surg 43A:327-351*, 1961.

93. Nachemson A, Lindh M: Measurement of abdominal and back muscle strength with and without low back pain. *Scartd J Rehabil Med* 2:60-65,1969.
94. Nemeth G: On hip and lumbar biomechanics. A study of joint load and muscular activity. *Scand J Rehabil Med 10(Suppl):1-35,* 1984.
95. Nichols PJ: Short-leg syndrome. *Br Med J* 2:1863-1865, 1960.
96. Okada M: An electromyographic estimation of the relative muscular load in different human postures. *J Human Ergol 1*:75-93, 1972.
97. Orr LM, Mathers F, Butt T: Somatic pain due to fibrolipomatous nodules, simulating ureterorenal disease: a preliminary report. *J Urol* 59:1061-1069, 1948.
98. Osier W: *The Principles and Practice of Medicine.* D. Appleton and Co., New York, 1912 (p. 1131).
99. Patton II, Williamson JA: Fibrositis as a factor in the differential diagnosis of visceral pain. *Can Med Assoc J* 58:162-166, 1948 (Cases 2 and 3).
100. Pauly JE: An electromyographic analysis of certain movements and exercises, I—some deep muscles of the back. *Anat Rec* 355:223-234, 1966.
101. Pernkopf E: *Atlas of Topographical and Applied Human Anatomy,* Vol. 2. W.B. Saunders, Philadelphia, 1964 (Fig. 30).
102. *Ibid.* (p. 35).
103. Price JP, Clare MN, Ewerhardt FH: Studies in low backache with persistent muscle spasm. *Arch Phys Rehabil Med* 29:703-709, 1948.
104. Rachlin ES: Injection of Specific Trigger Points. Chapter 10. In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (see p. 203).
105. Reynolds MD: Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil* 62:111-114, 1981.
106. Richter HR: Fettgewebe "Hernien." In: *Der Weichteilrheumatismus,* Vol. 1. Fortbildungskunde Rheumatol. Karger, Basel, 1971 (pp. 49-59).
107. Richter HR: Einklemmungsneuropathien der Rami Dorsales als Ursache von akuten und chronischen Rueckenschmerzen. *Ther Umsch* 34:435-438, 1977.
108. Rosomoff HL, Fishbain D, Goldberg M, *et al.:* Myofascial findings in patients with "chronic intractable benign pain" of the back and neck. *Pain Manage* 3(2):114-118, 1990.
109. Rubin D: An approach to the management of myofascial trigger point syndromes. *Arch Phys Med Rehabil* 62:107-110, 1981 (p. 110).
110. Samberg HH: The trigger point syndromes. *GP* 35:115-117, 1967.
111. Schneider MJ: The traction methods of Cox and Leander: the neglected role of the multifidus muscle in low back pain. *Chiropract Techn 3(3):*109-115,1991.
112. Schwartz RG, Gall NG, Grant AE: Abdominal pain in quadriparesis: myofascial syndrome as unsuspected cause. *Arch Phys Med Rehabil* 65:44-46, 1984.
113. Sicuranza BJ, Richards J, Tisdall L: The short leg syndrome in obstetrics & gynecology. *Am J Obstet Gynecol 1*07:217-219, 1970.
114. Sihvonen T, Partanen J, Hanninen O, *et al.:* Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. *Arch Phys Med Rehabil* 72:1080-1087, 1991.
115. Simmons EE: Referred low back pain. *J Omaha Mid-West Clin Soc 1*:3-6, 1954.
116. Simons DG, Travell JG: Myofascial origins of low back pain. Parts 1,2,3. *Postgrad Med* 73:66-108, 1983.
117. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (p. 309).
118. *Ibid.* (p. 311).
119. *Ibid.* (p. 312).
120. *Ibid.* (p. 313).
121. Stimson BB: The low back problem. *Psychosom Med* 9:210-212, 1947.
122. Strong R, Thomas PE: Patterns of muscle activity in the leg, hip, and torso associated with anomalous fifth lumbar conditions. *J Am Osteopath Assoc* 67:1039-1041, 1968.
123. Swezey RL: Non-fibrositic lumbar subcutaneous nodules: prevalence and clinical significance. *Br J Rheumatol* 30(5):376-378, 1991.
124. Tichauer ER: Ergonomics: the state of the art. *Am Ind Hyg Assoc J* 28:105-116, 1967.
125. Tichauer ER: Industrial engineering in the rehabilitation of the handicapped. *J Ind Eng* 29:96-104, 1968.
126. Tichauer ER: A pilot study of the biomechanics of lifting in simulated industrial work situations. *J Safety Res* 3:98-115, 1971.
127. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (pp. 268, 269).
128. *Ibid.* (p. 270).
129. *Ibid.* (p. 271).
130. *Ibid.* (p. 272).
131. *Ibid.* (p. 343).
132. Travell J: Basis for the multiple uses of local block of somatic trigger areas (procaine infiltration and ethyl chloride spray). Miss *Valley Med J* 71:13-22, 1949 (pp. 19, 20; Case 4).
133. Travell J: Symposium on mechanism and management of pain syndromes. *Proc Rudolf Virchow Med Soc* 16:128-136, (p. 135) 1957.
134. Travell J, Rinzler SH: The myofascial genesis of pain. *Postgrad Med* 11:425-434, 1952.
135. Travell J, Travell W: Therapy of low back pain by manipulation and of referred pain in the lower extremity by procaine infiltration. *Arch Phys Med Rehabil* 27:537-547, 1946 (pp. 544, 545; Case 3).
136. Williams PC: *Low Back and Neck Pain, Causes and Conservative Treatment.* Charles C Thomas, Springfield, Ill., 1974 (Fig. 19, Panel 3).
137. Young D: The effects of novocaine injections on simulated visceral pain. *Ann Intern Med* 29:749-756, 1943.
138. Zohn DA: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Ed. 2. Little, Brown & Company, Boston, 1988 (p. 212, Fig. 12-3).

CHAPTER 49
Abdominal Muscles

HIGHLIGHTS: Myofascial trigger point (TrP) phenomena of the abdominal musculature show strong reciprocal somatovisceral and viscerosomatic interactions and commonly produce pseudo-visceral pain that can be diagnostically very misleading. **REFERRED PAIN** from myofascial TrPs in the abdominal musculature is likely to appear in the same quadrant and, occasionally, in any other quadrant of the abdomen, as well as in the back. In addition to pain, these TrPs are capable of initiating somatovisceral responses, including projectile vomiting, anorexia and nausea, intestinal colic, diarrhea, urinary bladder and sphincter spasm, and dysmenorrhea. When such visceral symptoms occur with abdominal pain and tenderness, the combination can closely mimic acute visceral disease, especially appendicitis and cholelithiasis. **ANATOMY** of the three lateral abdominal wall muscles, the internal and external obliques, and the transversus abdominis, produce a diagonal crisscross and radial fiber arrangement like the plies in a tire. The fibers of the two medial muscles, the rectus abdominis and its pubic appendage, the pyramidalis muscle, are aligned vertically. **FUNCTIONS** of the abdominal musculature are chiefly to increase intra-abdominal pressure, and to flex and rotate the vertebral column. **ACTIVATION AND PERPETUATION OF TRIGGER POINTS** in the abdominal wall musculature secondary to visceral disease represents a viscerosomatic response. Examples of visceral diseases that can initiate and perpetuate TrPs include peptic ulcer, intestinal parasites, dysentery, ulcerative colitis, diverticulosis, diverticulitis, and cholelithiasis. Once activated, TrPs may then be perpetuated by emotional stress, occupational strain, paradoxical respiration, faulty posture, and over-enthusiasm for misdirected "fitness" exercises. **DIFFERENTIAL DIAGNOSIS,** in addition to the above perpetuating factors, includes consideration of articular dysfunctions, fibromyalgia, and a misdiagnosis of appendicitis. **TRIGGER POINT RELEASE** of the involved abdominal muscles by spray and stretch calls for extension of one hip, protrusion of the abdomen, and a downsweep spray pattern. **TRIGGER POINT INJECTION** begins with a pincer grasp, when possible, and injection proceeds with careful attention to the location and depth of needle penetration. **CORRECTIVE ACTIONS** include self-administration of TrP pressure release, learning how to breathe with coordinated abdominal (diaphragmatic) respiration, and learning to do the Pelvic-tilt and the Sit-back Exercises. Laughter is good medicine.

REFERRED PAIN
(Figs. 49.1 and 49.2)

Abdominal trigger points (TrPs) may cause as much distress from induced visceral dysfunction or from pain-limited activity as from referred pain. Symptoms referred from these myofascial TrPs commonly confuse the diagnostic process by mimicking visceral pathology. Pain patterns of TrPs in the abdominal muscles, especially the obliques, are less consistent from patient to patient than are the patterns for most other muscles. Abdominal pain referred from TrPs has little respect for the midline; abdominal TrPs on one side frequently cause bilateral pain. Gutstein[53] observed that the patient is likely to describe the distress caused by abdominal TrPs as "burning," "fullness" "bloating," "swelling," or "gas," although objective evidence for the symptoms is frequently missing. The pain patterns that are presented here were repeatedly observed by the authors and by others. Each of the abdominal muscle will be considered separately.

Abdominal Obliques (Fig. 49.1)

The abdominal oblique TrPs have multiple referred pain patterns that may reach up into the chest, may travel straight or diagonally across the abdomen, and may extend downward. Whether this variability represents different characteristics of the successively deeper layers of muscle, or less consistency in the patterns of pain referred from TrPs in this musculature, is not clear. One must palpate the abdomen carefully and thoroughly to identify all of the TrPs potentially responsible for abdominal symptoms.

Active TrPs in the upper portion of the abdominal external oblique muscle, which overlies the rib cage anteriorly, are likely to produce "heartburn" (Fig. 49.1A) and other symptoms commonly associated with hiatal hernia.[94] These "costal" and "subcostal" TrPs in abdominal muscles also may produce deep epigastric pain that occasionally extends to other parts of the abdomen.[95] Bonica, *et al.*[14] reported similar visceral symptoms and illustrated comparable pain patterns from TrPs in the external oblique muscle.

Active TrPs located in the musculature of the lower lateral abdominal wall, possibly in any one of the three layers of muscle, refer pain into the groin and testicle, and may project fingers of pain to other parts of the abdomen (Fig. 49.1C). The experimental injection of hypertonic saline into the external obliques near the anterior superior iliac spine induced referred pain over the lower portion of that quadrant of the abdomen, along the inguinal ligament and into the testicle.[69] A left external abdominal oblique TrP in a 10-year-old child referred severe pain from the left upper quadrant to the left inguinal region.[3]

Active TrPs along the upper rim of the pubis and the lateral half of the inguinal ligament may lie in the lower internal oblique muscle, and possibly in the lower rectus abdominis. These TrPs can cause increased irritability and spasm of the detrusor and urinary sphincter muscles, producing urinary frequency, retention of urine and groin pain;[47,92,117] they have been associated with enuresis in older children. When needled, such TrPs often refer pain to the region of the urinary bladder. Bonica, *et al.*[14] observed similar symptoms and illustrated comparable pain patterns from TrPs in the lower lateral abdominal wall musculature.

Melnick[94,95] identified TrPs in the muscles of the lower abdomen as sources of chronic diarrhea (Fig. 49.1D). In our experience, when TrPs that produce this symptom are identified and injected in a fold of the abdominal wall between the fingers, they seem to be in the superficial layer of the lateral abdominal wall musculature.

Transversus Abdominis

Active TrPs in the more craniad portion of the transversus abdominis refer pain as a band across the upper abdomen between the anterior costal margins. Sometimes the distressing pain concentrates on the region of the xiphoid process. Transversus TrPs in fibers attaching to the lower costal cartilage are likely to cause marked enthesitis along the inferior costal margin. This can be very distressing when coughing.

"Belch Button"

The "belch button" is a TrP that is uncommon, but may be of critical importance to the patient who has one. It has not been consistently localized to a specific muscle. It is a dorsal TrP that may lie in the posterior fringe of a lateral abdominal wall muscle, such as the external oblique, or it may be a fascial TrP in the lumbodorsal fascia. The patient is likely to complain of a "stomach problem" with much belching of gas. In our experience, this TrP is found on the left or right side usually at, or just below, the angle of the twelfth rib. When one locates it by palpation, a rib is beneath the finger (Fig. 49.1B), and the patient belches as pressure is applied to the TrP. When sufficiently active, this TrP causes spontaneous belching and in severe cases, projectile vomiting, which can be deeply embarrassing and a serious postoperative complication. Alvarez[7] reported that some patients belched every time the physician touched a trigger area in the back. Gutstein[53] reported that 7 patients responded with belching following injection of fibrositic spots (interpreted by us as TrPs) in the abdominal musculature, and that a few patients belched in response to pressure applied to tender abdominal spots.

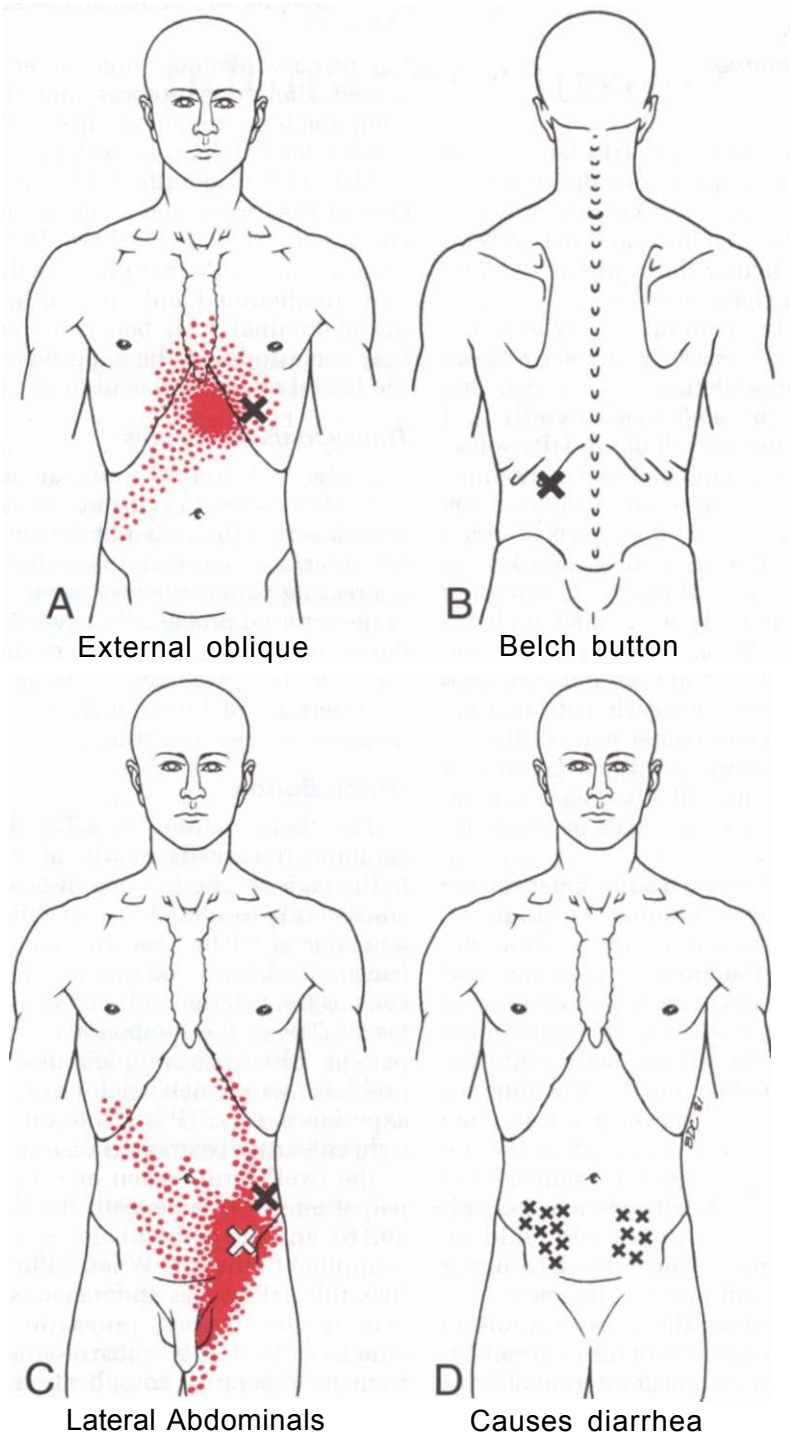


Figure 49.1. Referred pain patterns (red) and visceral symptoms of trigger points (**Xs**) in the oblique (and possibly transverse) abdominal muscles. **A,** "heartburn" from an attachment trigger point of the external oblique overlying the anterior chest wall. **B,** projectile vomiting and belching from the "belch button," which is usually located in the most posterior abdominal wall musculature or in connective tissue and may be on either side. **C,** groin and/or testicular pain, as well as chiefly lower quadrant abdominal pain, referred from central trigger points in the lower lateral abdominal wall musculature of either side. **D,** diarrhea from various trigger-point sites in lower abdominal quadrant muscles (after Melnick[93]).

Rectus Abdominis (Fig. 49.2)

The symptoms caused by TrPs in this muscle are varied, but largely dependent on the location of the TrPs. Symptoms will be considered in three groups, those due to TrPs in the upper portion of the muscle (above the umbilical region), those caused by periumbilical TrPs, and those from TrPs in the lower rectus abdominis.

Upper Rectus Abdominis. An active TrP high in the rectus abdominis muscle on either side can refer pain to the mid-back bilaterally, which is described by the patient as running horizontally *across* the back on both sides at the thoracolumbar level (Fig. 49.2A).[109] Gutstein[83] also noted that treatment that relieved tender spots in abdominal wall muscles relieved pain in the back. Unilateral backache at this level, however, more frequently originates in TrPs of the latissimus dorsi muscle.

In addition to back pain, TrPs high in the rectus abdominis can also refer pain to the region of the xiphoid process similar to the pain referred to that location by TrPs in the upper transversus abdominis muscle.

Several authors have described symptoms of abdominal fullness, "heartburn," indigestion and sometimes nausea and vomiting due to paraxiphoid TrPs located in the upper rectus abdominis.[47,92,94,95] In our experience, nausea and epigastric distress occur more often when these uppermost rectus abdominis TrPs are on the left, rather than on the right side. These TrPs also may refer pain across the upper abdomen between the costal margins.

Injection of hypertonic saline into the rectus abdominis at about 2.5 cm (1 in) above the umbilicus caused brief referred pain throughout the same quadrant of the abdomen and on the same side in the back.[69] A TrP in the upper rectus abdominis, when located on the left side, also may produce precordial pain.[48,73,92] When it has been established that the chest pain is myofascial and not cardiac in origin, it is usually due to TrPs in the pectoralis or a sternalis muscle; a rectus abdominis source of the pain is easily overlooked.

TrPs in the upper rectus abdominis and focal tender points characteristic of TrPs were observed to refer pain to the same abdominal quadrant,[46,74] and to simulate the symptoms of cholecystitis, gynecological disease[92] and peptic ulcer.[92,95]

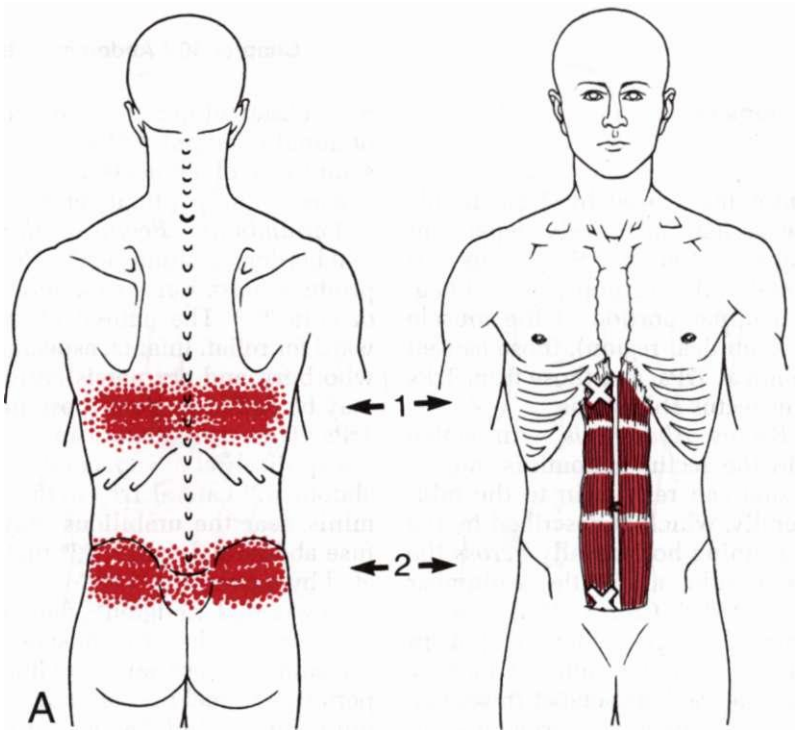
Periumbilical Rectus Abdominis. Lateral border, periumbilical TrPs are likely to produce sensations of abdominal cramping or colic.[94,95] The patient often bends forward for relief. Infants, especially neonates who burp and cry persistently with colic, may be suffering from these periumbilical TrPs. Their symptoms can be relieved by the application of vapocoolant spray to the abdomen.[11] Lateral TrPs in the rectus abdominis near the umbilicus may evoke diffuse abdominal pain[47,74,76] that is accentuated by movement.[72,124]

Lewis and Kellgren[82] demonstrated experimentally that this muscle can generate the pain of intestinal colic. Injection of hypertonic saline into normal rectus abdominis muscle induced a familiar colic-like pain, which was much stronger anteriorly than toward the back and extended diffusely over *several segments* in front.[69]

Lower Rectus Abdominis. Inactivation of TrPs in the lower rectus abdominis, about half-way between the umbilicus and the symphysis pubis (or in the overlying skin), may relieve dysmenorrhea[117,118] (Fig. 49.2C). See Section 6 for a relevant experiment by Theobald.

In the lowest part of the rectus abdominis, TrPs may refer pain bilaterally to the sacroiliac and low back regions.[109] The patient portrays this pain with a crosswise motion of the hand (Fig. 49.2A), rather than the up-and-down pain pattern characteristic of the iliocostalis thoracis and other more superficial paraspinal muscles.

Several authors have noted that a TrP in the lateral border of the right rectus abdominis in the region of McBurney's point, which is halfway between the anterior superior iliac spine and the umbilicus (Fig. 49.2B), is likely to produce symptoms closely simulating those of acute appendicitis.[47,48,88,92] This pain pattern was reported as often occurring when the patient was tired, worried or premenstrual.[47] In one case, the myalgic spot for this "pseudo-appendicitis" pain was reported to be located in the rectus abdominis just above the level of the umbilicus.[48]



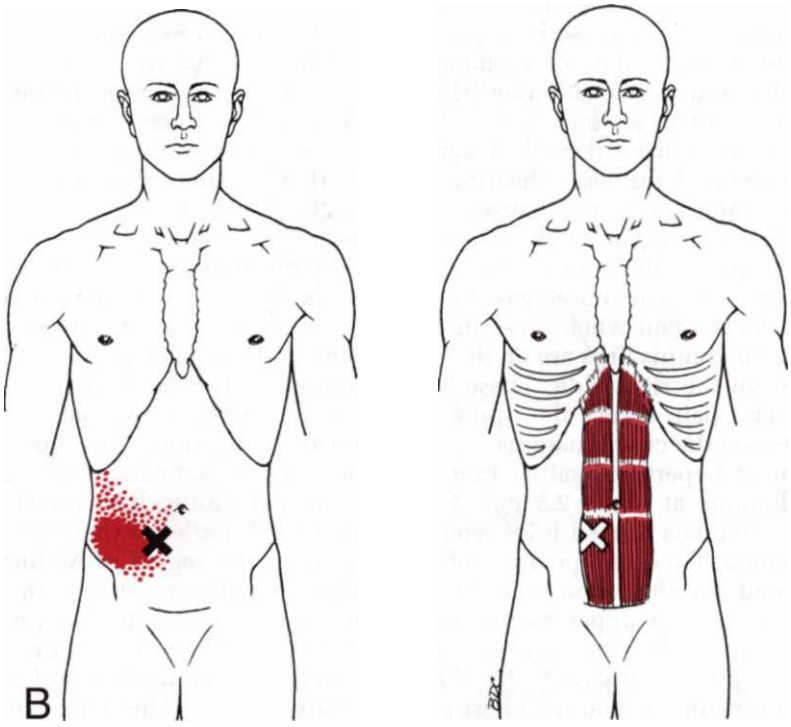
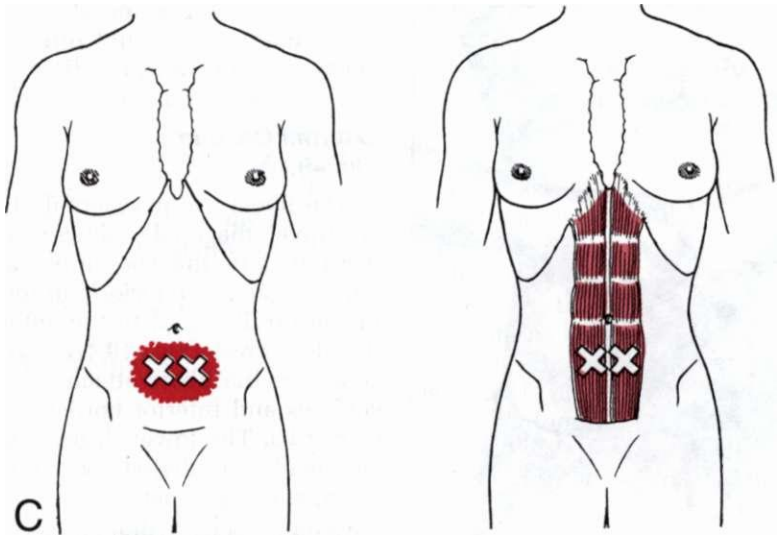


Figure 49.2. Referred pain patterns *(red)* and visceral symptoms of trigger points (**Xs**) in the rectus abdominis muscle. **A,** bilateral pain across the back, precordial pain, and/or a feeling of abdominal fullness, nausea and vomiting can be caused by a trigger point *(1)* in the right (pictured) or left upper rectus abdominis. A similar pattern of bilateral low back pain is referred from what is often an attachment trigger point (2) in the caudal end of the rectus muscle on either side. **B,** lower right quadrant pain and tenderness may occur in the region of McBurney's point due to a nearby trigger point in the lateral border of the rectus abdominis.



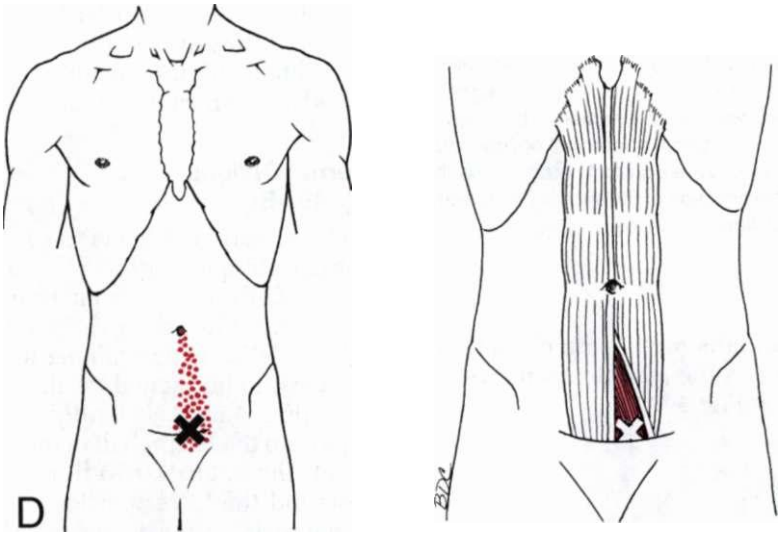


Figure 49.2—*continued.* **C,** dysmenorrhea may be greatly intensified by trigger points in the lower rectus abdominis. **D,** referred pain pattern of the pyramidalis muscle.

Other authors also have observed that TrPs in the region of McBurney's point may refer pain to the same lower quadrant,[46, 88] throughout the abdomen,[93] and to the right upper quadrant.[74] These TrPs also may refer sharp pain to the iliac fossa, the iliacus muscle, and to the penis.[47] The pain may simulate renal colic.[92] An active TrP in the right lower rectus abdominis may cause diarrhea[47, 95] and symptoms mimicking diverticulosis or gynecological disease.[92] A TrP just above the pubis may cause spasm of the detrusor and urinary sphincter muscles.

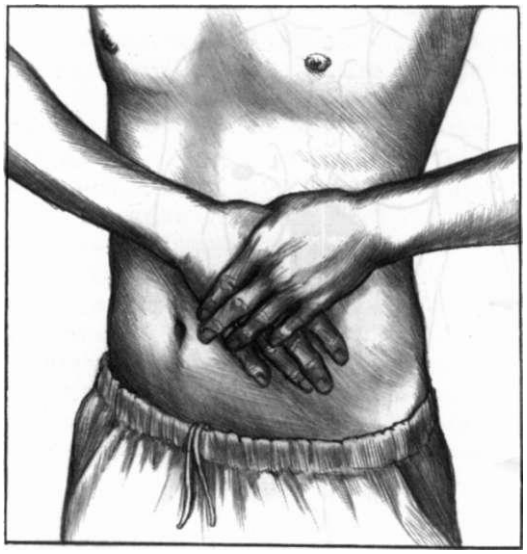


Figure **49.3.** Technique for remembering the fiber direction of the oblique abdominal muscles. By placing one hand (right hand in this illustration) over the lower abdomen on the opposite side, the fingers indicate the direction of fibers of the internal abdominal oblique muscle (left in this illustration). By then placing the other hand on top of the first hand, the fingers of the top hand now indicate the direction of the fibers of the more *superficial* external abdominal oblique muscle. This procedure provides a tactile memory aid that also applies to the direction of the intercostal muscles. (See Fig. 49.4 for fiber directions.)

Pyramidalis

The pyramidalis refers pain close to the midline between the symphysis pubis and the umbilicus (Fig. 49.2D).

2. ANATOMY
(Figs. 49.3-49.6)

The external and internal abdominal oblique muscles, like the external and internal intercostals have a diagonal criss-cross arrangement and the two groups of muscles have corresponding orientation. At times, it is important to know which layer runs in which direction and Figure 49.3 illustrates a mnemonic for the fiber directions. The fingers of the (right) hand on the left side of the abdomen against the skin represent the fiber direction of the left internal abdominal oblique (and intercostals), while the (left) hand on top represents the fiber direction of the left external abdominal oblique (and intercostals). The transversus fibers run radially around the abdomen as their name implies.

External Oblique
(Fig. 49.4A)

The fibers of the external oblique muscle travel diagonally downward and forward to join the abdominal aponeurosis which attaches *anteriorly* to the linea alba in the midline and to the anterior half of the iliac crest (Fig. 49.4A). *Laterally* and *cephalad* the fibers attach to the external surfaces and inferior borders of the lower eight ribs. The lower three of these rib attachments interdigitate with the latissimus dorsi, and the upper five, with the serratus anterior muscle. Although these three muscles appear in anatomy books to be quite separate, in dissection the external oblique may seem to form with the other two an unbroken sheet of muscle. The fasciculi from the lowest two ribs lie nearly vertically, and thus are parallel and adjacent to those fibers of the quadratus lumborum that also connect the iliac crest and the twelfth rib.[19]

Internal Oblique
(Fig. 49.4B)

The direction of fibers in the fan-shaped internal oblique abdominal muscle in the upright body ranges from nearly vertical, *posteriorly,* through a diagonally upward and medial direction among its intermediate fibers, to horizontal for the most caudal fibers (Fig. 49.4B). *Laterally* all fibers converge onto the lateral half of the inguinal ligament, the anterior two-thirds of the iliac crest, and the lower portion of the lumbar aponeurosis. *Above* the nearly vertical fibers attach to the cartilages of the last three or four ribs. *Above* and *medially* diagonal fibers attach to the linea alba through the anterior and posterior rectus sheath. *Medially* the horizontal fibers from the inguinal ligament attach to the arch of the pubis through the conjoined tendon, which this muscle forms with the transversus abdominis.

Transversus Abdominis
(Fig. 49.5)

These fibers run nearly horizontally across the abdomen and attach *anteriorly*

to the midline linea alba *via* the rectus sheath (Fig. 49.5), which surrounds the rectus abdominis muscle above the arcuate line, and attach to the pubis through the conjoined tendon. Below that line, the sheath occurs only anterior to the rectus. *Laterally* the transversus muscle attaches to the lateral one-third of the inguinal ligament, to the anterior three-quarters of the crest of the ilium, to the thoracolumbar fascia, and to the inner surface of the cartilages of the last six ribs, where it interdigitates with fibers of the diaphragm.[21]

Rectus Abdominis (Fig. 49.6)

The rectus abdominis attaches *below* along the crest of the pubic bone (Fig. 49.6). The fibers of the paired muscles interlace across the symphysis. *Above* the muscle attaches to the cartilages of the fifth, sixth, and seventh ribs.

The fibers of the rectus abdominis are usually interrupted by three or four, more or less complete, transverse tendinous inscriptions. Of the three most constant inscriptions one is found near the tip of the xiphoid process, one close to the level of the umbilicus, and one midway between them. Sometimes, there are also one or two partial inscriptions below the umbilicus.[22] In 115 cadavers, the total number of inscriptions per muscle ranged from one to four.[98]

The abdominal section of the pectoralis major muscle *(see* Fig. 42.5) may overlap fibers of the upper rectus abdominis, and thus may account for the occasional reference of pain to the anterior chest from TrPs in this region.

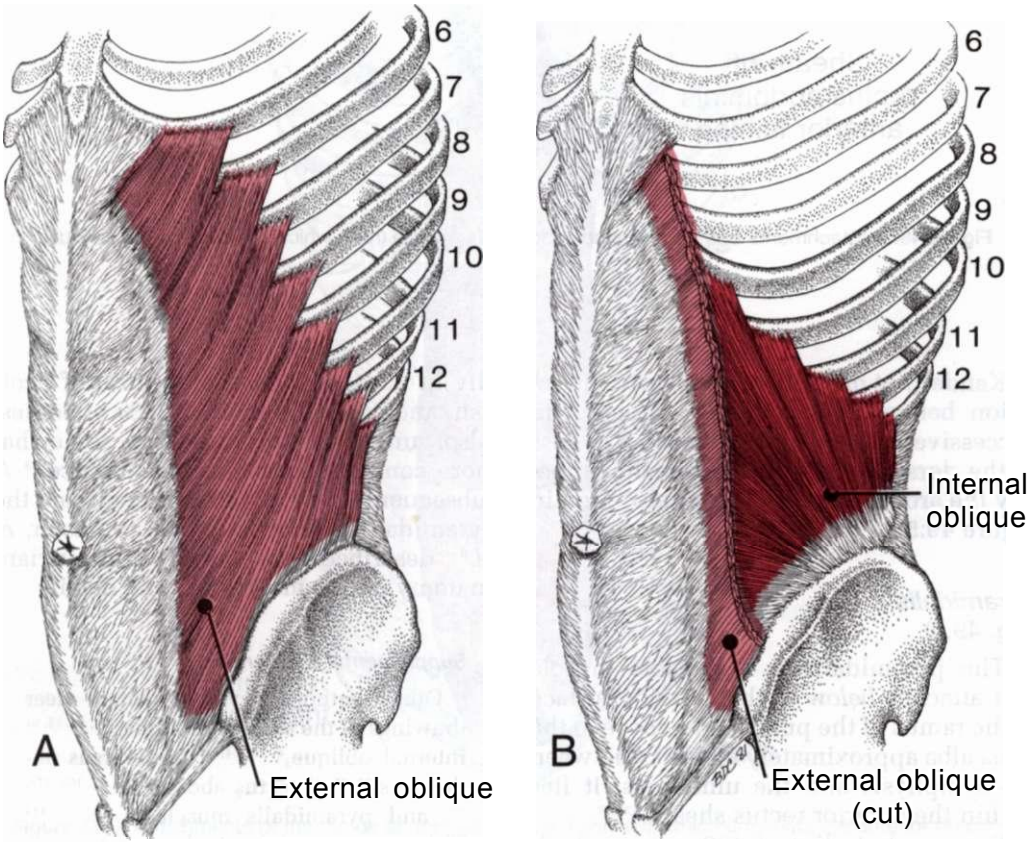


Figure 49.4. Attachments of two lateral abdominal wall muscles. A, external oblique *(light red).* **B,** internal oblique *(dark red);* the external oblique *(light red)* is cut.

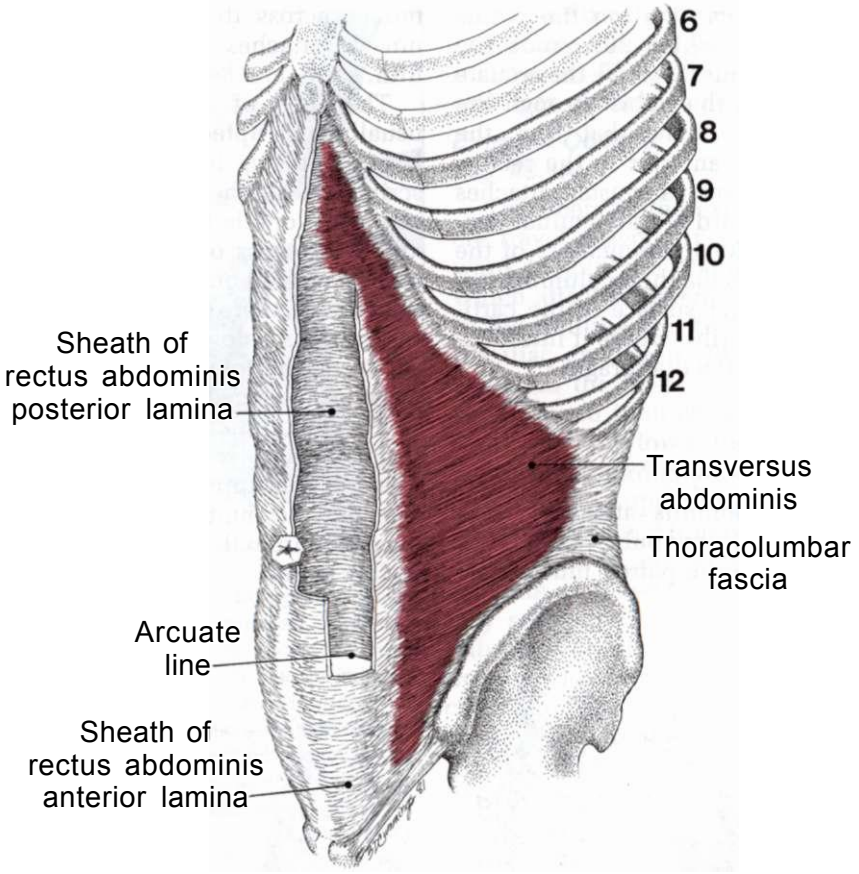


Figure 49.5. Attachments of the transversus abdominis muscle *(red)*, which lies deep to the obliques.

Kendall, *et al.*[78] illustrate clearly the relation between the surface anatomy and successive cross-sectional views. The loss of the dorsal half of the rectus sheath below the arcuate line is clear as also seen in Figure 49.5.

Pyramidalis
(Fig. 49.6)

The pyramidalis is a variable muscle that attaches *below* to the anterior surface of the ramus of the pubis,[8] and *above* to the linea alba approximately mid-way between the symphysis and the umbilicus. It lies within the anterior rectus sheath.[22,33]

In several studies of 100 or more bodies,[33] the pyramidalis was absent bilaterally in 3.3% of Japanese, in 25% of Scottish, and generally in 15%-20% of bodies. Also, unilateral absence was somewhat more common than bilateral absence.[33] A subsequent study of 430 sides reported the pyramidalis absent in 17.7%.[12] Anson, *et al.*[8] described the usual and variant anatomy of this muscle in great detail.

Supplemental References

Other authors have presented clear drawings of the external oblique,[2,5,19,23,91] internal oblique,[4,20,24,91] transversus abdominis,[21,25,91] rectus abdominis,[25,102,114,122] and pyramidalis muscles.[24,33,102,114] The anterior abdominal muscles are shown in cross section.[3,26,102]

3. INNERVATION

The three lateral abdominal wall muscles, the external and internal obliques and the transversus abdominis, are innervated by branches of the eighth through the twelfth intercostal nerves. The internal oblique and transversus abdominis muscles are also supplied by branches of the iliohypogastric and ilioinguinal nerves which stem from the first lumbar nerve. Segmental innervation is from T_8-T_{12}. The transversus is supplied, in addition, by the seventh intercostal nerve.[19-21]

The rectus abdominis is innervated by the seventh through the twelfth intercostal nerves derived from the corresponding spinal nerves; usually different segmental nerves innervate fibers between different tendinous inscriptions, especially in the upper half of the muscle.[22]

The pyramidalis is supplied by a branch of the twelfth thoracic nerve.[22]

4. FUNCTION

In advance of activation of the prime movers in response to lower limb movements in the standing position,[59] all of the abdominal wall muscles and some spinal muscles, including the transversus abdominis (which always started first), internal oblique, external oblique, rectus abdominis, and the lumbar multifidus are activated in a feed-forward manner. This response was independent of the movement

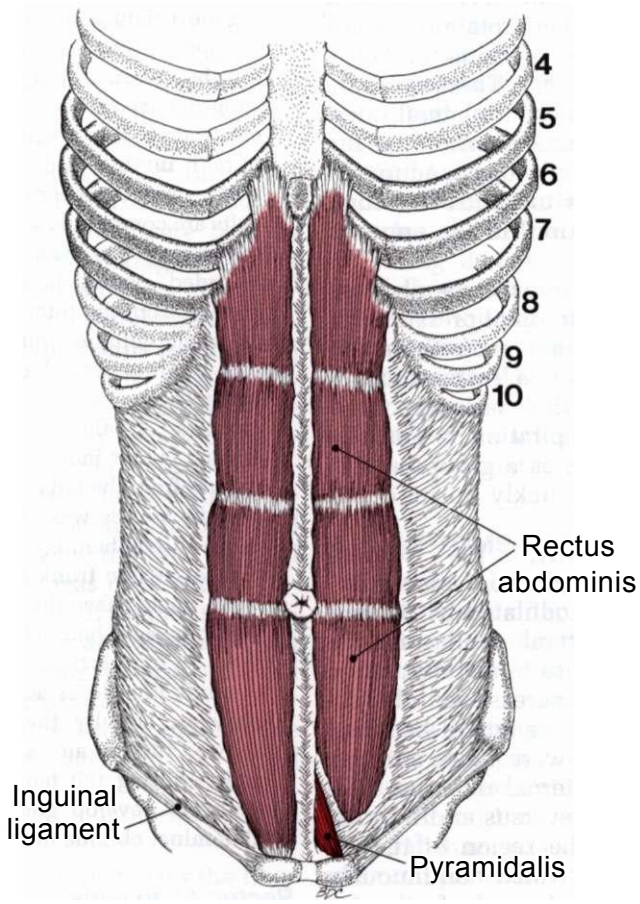


Figure **49.6.** Attachments of the rectus abdominis muscle *(light red)*, which connects the anterior rib cage to the pubic bone close to the symphysis, and attachments of the variable pyramidalis muscle *(dark red)*, which lies just above the symphysis pubis within the anterior rectus sheath.

and therefore was not a response to reactive forces, but was linked to control of stability of the spine against perturbations.[59]

Special functions of the abdominal muscles, particularly as related to respiratory activities, are covered in Chapter 45, Section 5.

Lateral Abdominal Wall Muscles

The internal and external abdominal oblique muscles function (1) bilaterally, to increase intra-abdominal pressure (e.g., for micturition, defecation, emesis, parturition and forced exhalation), (2) bilaterally, to flex the vertebral column, (3) unilaterally, to bend the vertebral column toward the same side, and (4) unilaterally, to assist vertebral column rotation. The external oblique muscle rotates the vertebral column toward the contralateral side; most of the internal oblique fibers rotate it toward the contracting muscle, bringing the opposite shoulder forward.[19,20] Therefore, one external oblique and its contralateral internal oblique affect trunk rotation in the same direction. They also can function by eccentric contractions to control rotation (provide a braking action) in the opposite direction.

Contraction of the transversus abdominis increases intra-abdominal pressure.[21]

Stimulation experiments on these lateral abdominal wall muscles produced a powerful exhalation that seriously compromised normal respiration.[29] The abdominal wall muscles as a group help to *complete* exhalation quickly during rapid breathing.[10]

Electromyographically,[10] the external and internal obliques showed some activity during walking, modulated by the gait cycle. All three lateral wall muscles showed marked increase in activity with a sudden or sustained increase in intra-abdominal pressure. During lateral bending, the internal obliques were more strongly activated than the external obliques. The fibers of both the transversus and internal oblique muscles in the region of the inguinal canal were activated continuously during standing. They showed a further increase in motor unit discharges during activities that would increase intra-abdominal pressure. Selective activation of the internal oblique and transversus abdominus muscles doubles when a curl-back (see Fig. 49.13A) is performed with the feet unsupported as compared to when the feet are supported, which emphasizes recruitment of other muscles such as the iliopsoas.[97]

Activity of the abdominal muscles helps to pump venous blood out of the abdomen. Relaxation of the abdominal wall during inhalation increases blood flow into the abdominal veins from the lower extremities. As the abdominal wall muscles contract for exhalation, the blood is forced upward toward the heart if the valves of the lower extremity veins are competent.

> Surface electrodes placed obliquely over each upper quadrant consistently disclosed more "external oblique" activity on the left than on the right side during most right-handed throwing activities in sports.[15] Since the left internal oblique, not the external oblique, brings the right shoulder forward, and since this type of electrode in this position should respond to both internal and external oblique activity, one interpretation is that these results are compatible with kinesiology and the findings of other authors, provided the recorded potentials are interpreted as coming from the internal, rather than the external, oblique. Surface electrodes in this location would not distinguish between the motor unit activity of these two muscles. Another interpretation of the finding is that indeed the recorded activity was from the left external oblique, and that the muscle was functioning in an *eccentric* (lengthening) contraction to prevent excessive trunk rotation that otherwise might have been produced by a vigorous right-handed throw. In none of these 13 sport activities was the rectus abdominis muscle as active as the oblique muscles. Usually the rectus abdominis showed little, if any, activity; it was most active during the tennis serve.[15] Discus throwers develop greatly hypertrophied abdominal oblique musculature.

Rectus Abdominis

The rectus abdominis muscle serves as the prime mover for spinal flexion, especially of the lower thoracic and lumbar spine, and it tenses the anterior abdominal

wall to increase intra-abdominal pressure.[26] Experimental stimulation of all portions of the rectus abdominis produced strong forward flexion of the trunk.[29]

Electromyographically, the rectus abdominis is active when a weight is carried on the back, but not when the weight is carried anterior to the thighs. The muscle responds consistently and clearly to the gait cycle during walking, but is not generally activated by efforts that increase intra-abdominal pressure, except by vigorous maneuvers, such as coughing.[10] This muscle is consistently active as the feet leave the ground when jumping, and inconsistently active during landing from the jump.[68]

Electromyographically, the rectus abdominis was essentially inactive during 14 static upright postures.[101] Sit-ups generated much more electrical activity in the rectus abdominis than did let-backs (sit-backs).[10, 36, 44, 127] **THE MUSCULAR** activity was greatest during the initial phase of the sit-up between 15° and 45°,[36] or between scapular lift and hip lift from the floor.[44] Little difference was seen in this muscle's electrical activity whether the knees were bent to 65°, or were straight.[36] Flexing the knees and anchoring the feet during a sit-up increased the activity of the abdominal muscles as compared to the rectus femoris, the fibers of which were said to become too short to pull effectively.[44] Electromyographic recording of four levels of difficulty of abdominal muscle testing (elevating progressively more of the weight of the lower extremities in the supine position) showed that at all levels, the lower half of the rectus abdominis was most active, followed by the upper half of that muscle, followed by the abdominal obliques. With maximum load, the relative proportion of oblique activity increased.[43]

The abdominal muscles are more active during uphill walking than on level ground.

Pyramidalis

The pyramidalis muscle tenses the linea alba.[22]

5. FUNCTIONAL UNIT

To increase intra-abdominal pressure for nonrespiratory reasons, the four muscles of the belly wall are synergistic with the quadratus lumborum and with the diaphragm.

For gross spinal rotation and flexion, anatomically, the external abdominal oblique appears synergistic with the external intercostals, lower serratus anterior (with which the external oblique interdigitates), and also the vertical costal fibers of the latissimus dorsi with which the lower part of the external oblique interdigitates and forms a continuous line of pull.[19] For lumbar spinal rotation, the external oblique muscle on one side is synergistic with the deepest (most diagonal) paraspinal muscles on the same side, and with the contralateral serratus posterior inferior and internal abdominal oblique muscles. For thoracolumbar spinal rotation, the internal oblique is synergistic with the internal intercostals.

For sidebending, the lateral, most vertical, fibers of the external and internal oblique muscles are synergistic with the vertical fibers of the quadratus lumborum, and with the most lateral of the paraspinal muscles, the iliocostalis.

During flexion/extension of the vertebral column, the rectus abdominis is antagonistic to the paraspinal group, especially the longissimus thoracis muscle, and synergistic with the psoas muscle if the lumbosacral spine is flexed. Functions of the abdominal muscles as related to respiration are covered in Chapter 45, Sections 4 and 5.

6. SYMPTOMS

Abdominal symptoms are commonly enigmatic and often a source of diagnostic confusion. Understanding the reciprocal somatovisceral and viscerosomatic effects of TrPs helps to unravel some of this uncertainty. Myofascial TrPs in an abdominal muscle may produce referred abdominal pain and visceral disorders (somatovisceral effects) that, together, closely mimic visceral disease. Conversely, visceral disease can profoundly influence somatic sensory perception and can activate TrPs in somatic structures that may perpetuate pain and other symptoms long after the patient has recovered from the initiating visceral disease.

Table 49.1 *Frequency of Serious Complaints among 156 Patients with Abdominal Trigger Points.*[a]

Symptoms	Number of Patients	Prevalence*
		%
Pain	40	71
Pressure and bloating	14	25
Heartburn	6	11
Vomiting	6	11
Diarrhea	2	4

[a] Adapted from Melnick J: Treatment of trigger mechanisms in gastrointestinal disease. NY State J Med 54:1324-1330, 1954.
*Percentage and numbers total more than 100% because some patients had more than one symptom.

Symptoms Due to Myofascial Trigger Points in Abdominal Muscles

Melnick[93] reported the relative frequency of serious symptoms arising from trigger areas in the abdominal musculature among 56 patients (Table 49.1).

Long[88] distinguished the "anterior abdominal wall syndrome" from visceral disease. The syndrome was attributed to TrPs in the musculature of the abdominal wall. Its distinguishing feature was nearly continuous pain that might relate to movement, but not to the ingestion of food or to evacuation. On careful inquiry, some of his patients localized the pain to the abdominal wall.

Good[46] observed that abdominal pain referred from TrPs in the lateral border of the rectus abdominis muscle near mid-abdomen was typically aggravated by bending over when lifting (an activity which shortens and often causes contraction of the rectus abdominis). In the experience of the authors of this manual, prolonged vigorous activity that requires forceful abdominal breathing also may increase the pain referred from abdominal wall TrPs.

Kelly[72] noted that patients with myalgic lesions (described like TrPs) of the abdominal wall musculature were likely to complain of abdominal discomfort or distress, rather than of pain *per se.* In the authors' experience, active TrPs of the abdominal muscles, especially in the rectus abdominis, may cause a lax, distended abdomen with excessive flatus. Contraction of the abdominal muscles is inhibited by the TrPs so that the patient cannot "pull the stomach in." This apparent distension is readily distinguished from that due to ascites by physical examination.

Right upper quadrant pain due to TrPs in either the oblique abdominal muscles or in the lateral border of the rectus abdominis of the same quadrant is easily confused with the pain of gallbladder disease. Pain simulating appendicitis was projected from "fibrositic nodules" (described like palpable bands and TrPs) in the region covered by the costal portion of the external oblique,[131] and from TrPs in the lateral border of the rectus abdominis in the right lower quadrant.[48,64,92]

Weiss and Davis[128] demonstrated that local anesthetic injection of a visceral pain reference zone can relieve the pain just as does infiltrating the area of pain referred from a TrP in a muscle.[58,70] Relief of pain in this way does not guarantee that the pain site is the site of origin.

7. ACTIVATION AND PERPETUATION OF TRIGGER POINTS

A posture or activity that activates a TrP, if not corrected or if continued, can also perpetuate it. In addition, many structural and systemic factors (*see* Chapter 4) will perpetuate a TrP that has been activated by an acute or chronic overload.

Abdominal TrPs are likely to develop in a muscle that is subject to acute or chronic overload, or in muscles that lie within the zone of pain referred from a viscus. In general, these TrPs may develop in response to visceral disease, direct trauma, and to mechanical, toxic or emotional stress.

Visceral Disease

As indicated in Section 11, visceral diseases in general,[53] and specifically peptic ulcer,[93,95] have been identified as often responsible for abdominal myofascial TrPs. Abdominal TrPs are especially likely to develop during an infestation with such intestinal parasites as *Entamoeba histolytica,* and beef or fish tapeworm. Such an infestation can be a potent perpetuator of myo-

fascial TrPs, and also may activate TrPs, in the abdominal musculature.

Trauma

Acute trauma[95] and chronic occupational strain[53] are important activating factors. In the authors' experience, TrPs are likely to occur close to an abdominal scar, as after an appendectomy or hysterectomy; the initiating stresses during surgery may be the combination of excessive stretch on the muscles by retractors and associated ischemia. Connective tissue TrPs within the scar tissue itself also are seen.[51] The skin and muscles around an incision have been infiltrated effectively with procaine at the time of suturing the wound to prevent the development of active TrPs following surgery and to reduce postoperative incisional discomfort.

Rectus abdominis TrPs may be initiated in conjunction with an abdominal operation and perpetuated by paradoxical breathing that develops as the result of postoperative abdominal soreness. The TrPs also discourage abdominal muscle activity, which contributes to paradoxical breathing (see Chapter 20, Section 14).

Stress

Several commonly encountered stress factors may activate abdominal TrPs: total body fatigue,[95] over-exercise (too many sit-ups or, for activation of rectus abdominis TrPs, too much heavy resistance "curl-type" exercises to develop the biceps and pectorals), emotional tension,[53,95] cold exposure, viral infections, straining at stool due to constipation, and poor posture[53] (such as sitting and leaning forward for hours on a bed or at a desk with the abdominal muscles shortened and tense, with the back not supported). On the other hand, forward-head posture or slumped posture (*see* Chapter 4, Section C) can sometimes be a result of TrP tension and shortening in the upper rectus abdominis. Structural inadequacies, such as a short leg or small hemipelvis, may add unnecessary overload. These stresses are additive.

The external oblique is vulnerable to a sustained twisted position (sitting at a desk, turned sideways because of lighting). This muscle also is vulnerable in sports activities that require a vigorous twisting body motion (throwing the discus).

The region of attachment of the internal oblique to the costal margin in the region of the eleventh rib seems to be vulnerable to developing enthesitis in response to overload. An example is continued coughing. Each cough can become excruciatingly painful. Latent TrPs in that muscle would increase the likelihood of this development.

8. PATIENT EXAMINATION

The examiner should observe the patient's posture in sitting, standing, walking, and reaching (see Chapter 41, Section C for postural considerations).

After establishing the event(s) associated with the onset of the pain complaint, the clinician should make a detailed diagram representing the pain described by the patient. The drawing should be in the style of the pain patterns in this volume using a copy of an appropriate body form such as those found in Chapter 3, Section 1, Figures 3.2-3.4.

Several authors have noted the value of *increasing* the abdominal muscle tension during examination to help distinguish the pain that is due to muscular TrPs from that due to underlying visceral disease. To conduct the Abdominal Tension Test according to Long,[88] the sensitive area is compressed with sufficient pressure to cause steady pain. When the supine patient then raises the legs high enough to bring *both* heels a few inches above the examining surface, the tensed abdominal muscles lift the palpating finger away from the viscera, while the digital pressure on the muscle itself is increased. If the pain increases, that indicates that it originates in the abdominal wall; if the pain decreases, it more likely originates inside the abdomen. To achieve increased abdominal tension, Llewellyn and Jones[87] recommended that the patient hold a partial sit-up. The similar Carnett technique (the supine patient crossed the arms and sat half-way forward) reliably distinguished abdominal wall tenderness from visceral tenderness.[119] Wilson,[131] like Long,[88] asked the supine patient to lift both heels off the bed, while de-Valera and Raftery[134] had the patient elevate both the feet and the head. Hunter[64]

and Kelsey[77] merely requested the patient to tense the abdominal muscles. By having patients raise only the head and shoulders free of the table, the test can be performed by those unable to do a sit-up and they can confirm the test for themselves, assuring themselves of no abdominal origin of the pain.[54]

The examiner should observe the displacement of the patient's umbilicus during various movement activities while the patient is supine (activities such as laughing, coughing, raising one leg up from the bed, or pressing his hand against resistance by the examiner). If there is abdominal muscle imbalance, the umbilicus will deviate away from a weaker (or inhibited) muscle and toward a stronger (or more hyperactive) muscle. Simply observing the umbilicus while the patient rests quietly may reveal a deviation toward a muscle with TrP shortening or away from an abdominal muscle that is inhibited by TrPs.

Abdominal Obliques

To ensure contraction of the lateral wall abdominal muscles when performing the Abdominal Tension Test, the supine patient must elevate the heels, or elevate the head and shoulders high enough to lift both scapulae off any support. When the patient elevates only the head, usually only the rectus abdominis muscles contract, and not the obliques.

Rectus Abdominis

When the patient with active TrPs in the rectus abdominis muscle stands, the abdomen is likely to sag and become pendulous. Clinically, TrPs in this muscle inhibit its supportive function. Janda[66] classified this muscle as prone to inhibition and weakness, and others agree.[50,84] The tense palpable band associated with an active TrP would extend through and would shorten only the segment of muscle (between inscriptions) in which it lies. However, the TrP activity apparently inhibits contraction of adjacent segments to reduce tension on the involved fibers, thereby causing lengthening, rather than shortening, of the muscle as a whole. The rectus abdominis has no parallel muscle, except its contralateral mate, that could contract and unload it to provide protective splinting.

If asked to take a deep breath, these patients are likely to exhibit paradoxical breathing (see Chapter 20). Although during quiet respiration, exhalation is essentially performed by the elasticity of the lungs and requires little muscular assistance, the threat of pain due to stretching of the involved rectus abdominis apparently subconsciously inhibits the normal diaphragmatic contraction on inspiration. This may be a rectus abdominus-diaphragmatic reflex inhibition. When the patient inhales deeply with the diaphragm, thus protruding the abdomen, referred pain due to rectus abdominis TrPs may be exacerbated.

The bilateral, transverse, midback pain referred from TrPs high in the rectus abdominis muscle is usually aggravated by taking a deep breath, especially when the back is arched in marked lumbar lordosis, which further stretches the rectus abdominis. Back pain from paraspinal TrPs is not usually influenced by respiration. Herniation through the abdominal musculature is detected in some cases only when the patient is standing rather than recumbent.

9. TRIGGER POINT EXAMINATION

Gerwin, *et al.*[41] established that the most reliable criteria for making the diagnosis of myofascial TrPs were the detection of a taut band, the presence of spot tenderness, the presence of referred pain, and reproduction of the patient's symptomatic pain. For several muscles, agreement on the presence of a local twitch response was low. Of the five muscles tested it was lowest for the upper trapezius and infraspinatus muscles. Examination of the more superficial external oblique and rectus abdominis muscles should be comparable in difficulty to those two test muscles. Local twitch responses are not a reliable diagnostic test for these muscles for most examiners. The new understanding of the nature of TrPs (see Chapter 2) makes it clear that a fundamental palpable characteristic of a TrP is a tender palpable nodule in the middle of a taut band. The deeper internal oblique and transversus abdominal muscles are not reliably accessible for these palpable diagnostic findings.

When the abdomen is examined for myofascial TrPs, the supine patient should take a deep breath using diaphragmatic (abdominal) breathing and hold the breath to passively stretch these muscles (it helps to relax them) and to increase their sensitivity to palpation. To optimize palpation of lateral abdominal TrPs, the patient lies on the contralateral side and holds a similar deep breath. Gutstein[53] warned that a few TrPs are more easily found when the *relaxed* abdomen is palpated, and repeated palpation may be required before TrP tenderness is definitely established.

External Oblique

Attachment TrPs of the external oblique muscle are found along the lower border of the rib cage[53] and along the line where this muscle attaches to the iliac crest.[53,80] The authors of this manual frequently found central TrPs in superficial palpable bands that extend between the tip of the twelfth rib and the crest of the ilium (Fig. 49.1C).

In addition to examining the abdomen of the supine patient by flat palpation, the patient's hips may be flexed to slacken the abdominal muscles, so that the abdominal wall in the flank area (external, internal obliques and transversus muscles) can be grasped between the fingers and thumb, as shown later in Figure 49.9A. When the most tender part of a palpable band is briskly rolled within the pincer grasp, the band usually responds with a vigorous local twitch response. Some thin patients with lax abdominal musculature may be examined most effectively with their thighs extended at the hip.

Internal Oblique

"Fibrositic nodules" (a description that was compatible with attachment TrPs) in this muscle were located along the inferior margins of the tips of the six lower ribs, and also close to the pubic bone.[87] In our experience, to find them, the examiner must press down against the *upper edge* of the pubic arch, not on the flat anterior surface of the pubis. These TrPs feel like small buttons, or short bands at the region of attachments of the internal oblique fibers.

Rectus Abdominis

Active TrPs in this muscle are commonly found in the angle between the costal arch and the xiphoid process,[53] or between the xiphoid process and the umbilicus. In addition, they may be found in the middle or lower portions of the rectus abdominis, especially along its lateral border and at its attachment to the pubic bone.

10. ENTRAPMENT

An anterior branch of a spinal nerve may become entrapped in the rectus abdominis muscle or sheath, is frequently referred to as the rectus abdominis syndrome, and produces lower abdominal and pelvic pain that can simulate gynecological disease in female patients. This syndrome was diagnosed by a test injection of procaine to block the nerve; if the test injection afforded relief, the entrapped nerve was cauterized by injection of 0.5 ml of 6% aqueous phenol solution.[134] Others injected 5% and 7% phenol into the lateral border of the rectus sheath.[28,92] Some of these "entrapments" may have been unrecognized TrPs receiving unusually vigorous therapy.

Two surgeons[55] treated 24 patients for nerve entrapment in the rectus abdominis muscle by treating a site identified as a TrP. Eleven patients were cured by Bupivacaine injection of the site with or without steroid, 10 by a nerve-destructive procedure, and 2 had persistent symptoms. A neurologist[107] attributed the rectus abdominis syndrome to spinal nerve entrapment of enigmatic etiology. A gynecologist[45] observed temporary relief in 30 patients of what he called the Ibrahim Syndrome by injecting the tender spot in the lateral rectus abdominis with Xylocaine. Subsequent surgical loosening of adhesions and dilation where the spinal nerve exits from the lateral part of the rectus abdominis muscle produced lasting relief in 80% of patients.

Three internists[79] reported 14 cases of definite entrapment of the ilioinguinal nerve where it pierces in a step-like or zig-zag fashion the transversus abdominis and internal oblique muscles at a point 3

cm medial to and slightly below the anterior superior iliac spine. They identified this point as a typical TrP that referred pain to the iliac fossa groin and/or to the back.

When the entrapment is due to tension from TrP activity in fibers of the rectus abdominis, inactivation of the TrPs by injecting them with 0.5% procaine solution provides a simple way to relieve the symptoms.

One report[81] described neurolysis of an ilioinguinal nerve entrapped by fibers of the internal oblique muscle that permitted the teacher to return to work without symptoms.

Several of these descriptions sound as if the patients were experiencing nerve compromise due to loss of mesoneural mobility. This loss of mobility is a restriction of the normal sliding movement of the nerve in the tissue through which it passes as described by Butler and Jones.[16]

11. DIFFERENTIAL DIAGNOSIS

It has been recognized since the 1920s that persistent abdominal pain is as likely to originate in abdominal-wall muscles or be referred from chest-wall muscles as it is to originate in abdominal viscera.[18] Trigger points in the diaphragm can also cause chest pain.[65] The differential diagnosis of diseases that produce symptoms which are commonly caused by or may mimic the pain caused by abdominal muscle TrPs includes articular dysfunctions, fibromyalgia, appendicitis,[48,53] peptic ulcer,[53,95] gallstone colic,[53] colitis,[53] painful rib syndrome,[31] intractable dysmenorrhea,[126] enigmatic pelvic pain syndrome caused by abdominal wall TrPs,[112] chronic pelvic pain,[86] and urinary tract disease.[62]

The referred pain patterns of a number of abdominal diseases are mimicked by TrPs in abdominal wall muscles.[113] Additional differential diagnostic considerations can include hiatal hernia (gastroesophageal reflux), gastric carcinoma, chronic cholecystitis or ureteral colic, inguinal hernia, hepatitis, pancreatitis, gynecologic pathology (such as ovarian cysts), diverticulosis, umbilical hernia, thoracic radiculopathy, upper lumbar radiculopathy, costochondritis, ascariasis,[6] epilepsy,[108] and rectus abdominis hematoma.[52]

Abdominal pain in an upper quadrant may be attributed to Tietze's syndrome of the costal cartilages,[121] reported also as affecting the xiphisternal joint,[67] or to abnormal mobility of the lower intercostal joints, which has been variously referred to as the "slipping rib syndrome,"[56] or the "rib-tip syndrome."[90] This has been diagnosed by the "hooking maneuver," in which the fingers are hooked under the costal margin to pull the ribs forward, demonstrating their abnormal mobility and reproducing the pain.[90] Temporary, sometimes permanent, relief from this symptom was obtained by the local injection of an anesthetic agent.[90] Some patients experienced surgical removal of the hypermobile rib segment and reported permanent relief.[56] There is a strong likelihood that many of these patients were suffering from enthesitis of muscular attachments to the chondral cartilages. The chondral intercostal muscles, pectoralis major, and transverse abdominal muscles are likely candidates for central TrPs that could be causing the enthesitis.

Abdominal pain, particularly in the lower quadrant of the abdomen, may be referred from TrPs in the paravertebral muscles *(see* Chapter 48).[53,93,95,133] Gastrointestinal pain and cramping has been reported from TrPs specifically in the erector spinae bilaterally.[37] Conversely, TrPs in the lower rectus abdominis muscle can cause pain in the thoracolumbar region[109] and similar pain in that region can also be caused by an avulsion injury of the lumbar multifidus and rotator muscles,[61] or from apophysial joints.[89] Also, nausea and belching may result from TrP activity in the paraspinal muscles at the upper thoracic level.[7,27] Three examples of abdominal pain were attributed to remote TrPs in the skin itself.[110] Lower abdominal pain, tenderness and muscle spasm may be referred from TrPs located in the vaginal wall about 2.5-3.8 cm (1 to 1 1/2-in) inside the introitus, in a region that is normally insensitive to digital pressure.[94]

Urinary frequency, urinary urgency and "kidney" pain may be referred from TrPs in the skin of the lower abdomen, as well as from TrPs in lower abdominal muscles. In-

jection of a TrP in an old appendectomy scar in the right lower quadrant has relieved frequency and urgency, and increased the bladder capacity from 240 ml to 420 ml. Similar symptoms from a TrP in the skin close to McBurney's point were relieved for at least 8 months by its injection with a local anesthetic.[62]

A TrP high in the adductor muscles of the thigh may refer pain upward into the groin and to the lower lateral abdominal wall.[123]

> Feinstein, *et al.*[35] injected hypertonic saline into paraspinal musculotendinous tissues, 1.3-2.5 cm (1/2 to 1 in) from the midline at each segmental level. The abdominal pain patterns referred from paraspinal muscles at the T_7 to T_{12} levels were similar, but without the precise degree of segmental correspondence that was suggested earlier by Melnick.[94] Clinically, these authors found only an approximate anterior segmental correspondence.
>
> Lewis and Kellgren,[72] and later Kellgren,[70] described pain referred to the abdomen from interspinous ligaments when they were injected with hypertonic saline. Hockaday and Whitty[58] subsequently found that pain was referred from these ligaments only to dorsal areas. The more extensive pain patterns observed by Kellgren[71] may have been due to his injection of paraspinal (non-midline) structures, which Hockaday and Whitty scrupulously avoided.

An unusual source of continuous severe lower abdominal pain is hematoma of the rectus abdominis muscle;[32, 104, 105, 111, 115] Murray[100] reported three such cases in 55,900 pregnancies, and all three had been coughing heavily when the pain began.

Articular Dysfunctions

Articular dysfunctions associated with abdominal TrPs include pubic and innominate dysfunctions, and depressed lesions of the lower half of the rib cage on the side of involvement. Movement restriction of the thoracolumbar junction that responds to mobilization is sometimes associated with a shortened rectus abdominis muscle with palpable TrPs that respond to postisometric relaxation. Similar involvement of the psoas and quardatus lumborum muscles are even more commonly associated with this articular dysfunction.[83]

Fibromyalgia

Whenever a patient who complains of abdominal pain and also has widespread pain complaints that have been present for at least 3 months, they should be examined for fibromyalgia *(see* Chapter 2, Section B). Fibromyalgia and TrPs are different diseases that cause pain for different reasons and respond to different treatment approaches.[40] More than half of fibromyalgia patients also have TrPs.

Appendicitis

Active TrPs in the lateral border of the rectus abdominis (Fig. 49.2B) may induce recurrent pain in the area of McBurney's point,[64] or pain in the iliac fossa.[48] These TrPs simulate the symptoms of appendicitis,[53, 116] with marked local tenderness and rigidity. Surgeons who are unaware of the common myofascial sources of lower right quadrant pain are understandably frustrated by the poor correlation between the patient's symptoms and the pathological state of the excised appendix.[49] Nearly 40% of the appendices removed in one large series were normal.[129] One would suspect that many of the 22.4% of these operated patients who obtained only partial relief, and most of the 8.2% who had no relief from their "appendicular" pain by surgery,[129] had active TrPs that contributed to their symptoms. A more recent study found normal appendices in 12.4% of "appendicitis" patients.[132]

When the abdominal pain suggestive of appendicitis is due to TrPs in the rectus abdominis, that muscle shows a palpable nodule and ropiness, which differ from the more generalized, board-like rigidity of all layers of the abdominal musculature found in acute appendicitis. Tenderness relief by the Abdominal Tension Test *(see* Section 8) and positive laboratory findings indicative of infection favor appendicitis. Rovsing's sign (pain from pressure on the left side of the abdomen due to colonic gas being pushed to the right),[120] and rebound tenderness are usually present only in visceral disease.

Abnormal sensitivity of the iliopsoas or obturator internus muscles to passive stretch due to an inflamed retrocaecal appendix[120] must be distinguished from a similarly reduced range of motion due to active TrPs in these two pelvic muscles. In the latter case, it is specifically the muscles that are tender to palpation.

The leucocyte count and erythrocyte sedimentation rate are normal in the uncomplicated myofascial pain syndromes, but elevated in acute appendicitis and other acute inflammatory visceral disease.

Urinary Tract Symptoms

Myofascial TrPs also can induce pain in the urinary bladder,[62] with associated sphincter spasm and residual urine. Some patients have received urethral dilation and urethrotomy without relief. The referred TrP sensations have been diagnosed as cystitis.[77] Urinary tract symptoms indicating prostatitis can be and often are caused by intrapelvic TrPs.

Somatovisceral Effects

Myofascial TrPs can induce visceral disturbances and dysfunctions. Also, modification of the sensory input to the central nervous system in somatic areas of pain referred from visceral nociceptive input can modify the perception of pain.

Good[47] reported that a myalgic condition of the abdominal musculature (description compatible with TrPs) often caused functional disturbance of an abdominal viscus.[47] Abdominal TrPs may induce diarrhea, vomiting, food intolerance,[53] colic and dysmenorrhea in adults or excessive burping in an infant. Diarrhea may be a concomitant of TrP activity in the rectus abdominis, but is more likely to depend on TrPs in the lower-quadrant oblique muscles (Fig. 49.1D).

Weiss and Davis[128] demonstrated another somatovisceral relationship by modulating the somatic limb of primarily visceral pain. They relieved the pain referred to the abdominal wall from a diseased viscus by infiltrating the pain reference zone subcutaneously (not intramuscularly) with a local anesthetic. This effectively anesthetized the skin of the painful area, and probably the underlying superficial layers of muscle. Three patients were tested who suffered from acute gallbladder disease; one felt pain over the epigastrium, the other two felt pain in the right upper quadrant. Subcutaneous infiltration of from 12-30 ml of 2% procaine solution into the area of referred pain provided relief lasting from 30 min to several hours. In one case, following the infiltration, pain appeared in an adjacent area, and this pain also was relieved by local anesthetic infiltration. One patient with acute, and another with chronic, appendicitis had pain and tenderness in the right lower quadrant. Subcutaneous infiltration of the painful zone with 8 and 15 ml of 2% procaine, respectively, provided complete temporary pain relief in both patients. Similar temporary results were reported for pain due to nephrolithiasis, salpingitis and carcinoma of the esophagus.

Theobald[118] electrically stimulated the endometrium to simulate dysmenorrhea by producing abdominal wall pain centrally over the rectus abdominis muscles midway between the umbilicus and the pubis. The visceral-referred uterine pain was eliminated somatically by procaine infiltration of the painful skin and subcutaneous tissues in the reference zone, suggesting a convergence-facilitation mechanism of referred pain.[106] However, referred abdominal pain produced by sufficiently strong electrical stimulation of the uterus was not blocked by local anesthetic infiltration of the abdominal reference zone, suggesting a central activation mechanism. Clinically, complete relief was usually, but not always, obtained when dysmenorrhea was treated by procaine infiltration of this painful area over the rectus abdominis muscles.[118]

Lewis and Kellgren[82] established experimentally that the clinical symptom of intestinal colic can be referred from normal rectus abdominis muscle by injecting 0.3 ml of 6% sodium chloride solution just *below,* and 2.5 cm (1 in) *outside,* the navel. This irritant solution produced continuous pain for 3-5 min; the pain was referred deeply in the front of the body, and was indistinguishable from the pain of colic.

Viscerosomatic Effects

A reciprocal influence of visceral structures on somatic regions including muscles can be equally important. The reflex spasm (rigidity) of the abdominal muscles in response to the inflammation of acute appendicitis is well known.[96]

Pain, which previously had responded to medical therapy for a duodenal ulcer, became unresponsive and persisted until TrPs in the abdominal musculature were found and inactivated.[93] The ulcer apparently had activated these abdominal TrPs before it was healed by medical treatment. Then the TrPs continued to refer pain that was similar to that previously caused by the ulcer.

In normal subjects, stimulation of the splenic flexure of the small intestine by acute distention induced pain referred to the upper abdomen.[30] In patients with an irritable colon, this stimulus projected pain also to the precordium, left shoulder, neck and arm.[30] The upper and lower gastrointestinal tract of 21 patients with "functional" abdominal pain with no organic cause was systematically explored using an inflatable balloon.[99] The authors found trigger areas in the esophagus, small intestine, and colon that produced the patients' symptoms. The trigger areas could refer pain anywhere in the abdomen. Both ileal and jejunal hypermobility have been found coincident with abdominal pain in these patients. This raises the question of how commonly do TrPs of the intestinal mucosa cause serious gastrointestinal and somatic symptoms. This is an essentially unexplored possibility.

Specific viscero-rectus abdominis and viscero-pannicular reflexes were reported in the cat. Pinching the pancreas, or the mesentery, or a loop of the duodenum consistently produced a marked contraction of the rectus abdominis muscle.[82] Dilatation of the gallbladder by a balloon caused contraction of the subcutaneous panniculus carnosus muscle over the lateral and dorsal thorax of the cat.[9]

Giamberardino, et al.[42] studied responses to ureteral stone implants in rats for as long as 10 days. They observed a direct linear correlation between severity of visceral pain episodes and hyperalgesia of the ipsilateral external abdominal oblique muscle. The amount of referred lumbar muscle hyperalgesia appears to be a direct function of the amount of colic pain experienced.

Trinca[125] demonstrated a viscerosomatic reflex when stimulation of the gastric mucosa by drinking a cup of hot tea caused reddening of epigastric skin that had been previously irritated by a rubefacient.

Related Trigger Points

Although one first thinks of TrPs in the abdominal musculature to explain nonvisceral abdominal pain, there are other TrP sites to be considered. Epigastric pain suggestive of a duodenal ulcer may arise from "fibrositic nodules" (TrPs) in the region of the serratus anterior muscle, and has been effectively treated by digital pressure on the nodules.[131]

The TrPs in the lower lateral abdominal wall are often associated with active TrPs high in the adductor muscles of the thigh, which may refer pain upward inside the abdomen.

Gutstein[53] emphasized, and we agree, that it is important to look for additional tender points above and below the inguinal ligament on the same side as the pain and, if found, for corresponding points on the opposite side.

12. TRIGGER POINT RELEASE (Figs. 49.7 and 49.8)

In addition to the spray-and-stretch technique described here, other techniques including postisometric relaxation[85] and contract relax as described in Chapter 3, Section 12 are also effective for release of *central* trigger points (TrPs) in all of the abdominal muscles. Trigger point pressure release is usually only applicable to the superficial external oblique and rectus abdominis muscles. The primary therapeutic approach to *attachment* TrPs is to inactivate the central TrPs that are causing them.

Active TrPs in the abdominal muscles of infants and young children are particularly responsive to stretch and spray. In adults, before injecting the abdominal TrPs, one should first look for and inactivate any TrPs

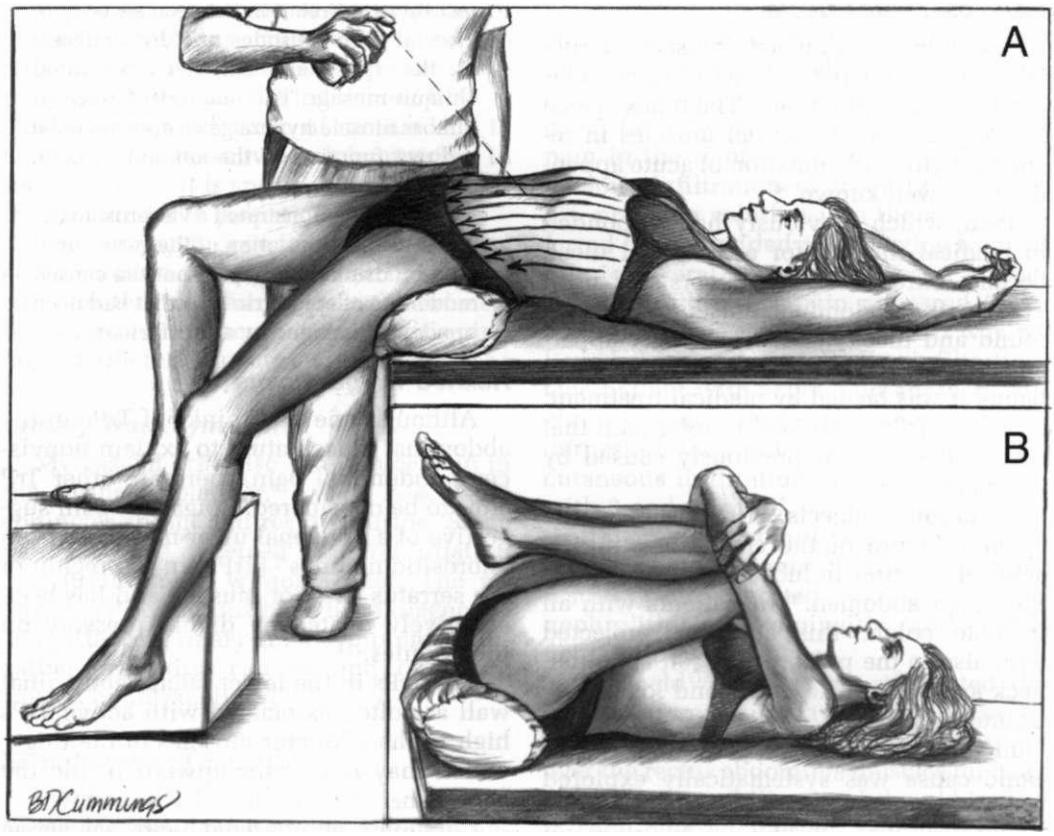


Figure 49.7. Stretch position and spray pattern *(arrows)* for trigger points in abdominal muscles on the *left* side of the body with follow-up full range of motion. **A,** The patient lies supine with the hip joint at the edge of the treatment table, and with the lower limbs extending over the end of the table. The hips are padded with a pillow. The arms are raised and one lower limb is supported on a stool or chair seat. The lower limb on the treatment side at first is supported by the stool or by the therapist in order to allow gradual stretch. After the operator initially applies vapocoolant with sweeps in the caudal direction, the patient allows the lower limb on the treatment side (left) to hang free and then takes a very deep breath, allowing the downward-moving diaphragm to strongly protrude the relaxed abdominal musculature. This is a critical step to stretch the abdominal muscles effectively. As the patient completes the inhalation and begins to slowly exhale, sweeps of spray are applied in a caudal direction and extend to the attachment of the iliopsoas muscle, since that muscle (which often has trigger points) also is stretched by this procedure. *The procedure should be repeated for the contralateral abdominal muscles.* **B,** bilateral knee-to-chest position that unloads stress that might have been placed on the lumbosacral spine. The patient assumes this position after release of the muscles on *both* sides of the abdomen. In this position, the abdominal muscles are fully shortened when the patient gently and fully exhales. To restore full functional range of motion, the patient should gently alternate between the fully stretched and the fully shortened position three times, one leg at a time.

in the back muscles that refer pain to the abdomen, since TrPs in the abdomen may be satellites of the dorsal TrPs. Satellite TrPs from the reverse direction can also occur.

To stretch and spray the rectus abdominis muscle (Fig. 49.7), the patient lies supine on a plinth or firm support with the legs extending over the end, with the arms positioned upward over the head, and with one foot supported on a stool so that initially the thighs are not extended at the hips. The procedure is illustrated and described in Figure 49.7. In the authors' clinical experience, an up-pattern of vapocooling, which was recommended by others,[93] is not as effective as the down-pattern (Fig. 49.7). Both right and left rectus abdominis muscles should always be treated, since

they function as a team and usually are both involved. Range of motion through flexion and extension can be carried out as illustrated in Figure 48.14

The patient should be taught how to self-stretch the rectus abdominis as illustrated and described in Figure 49.8.

To stretch the more lateral external oblique muscle, the patient lies on the contralateral side and the uppermost shoulder is lowered backward toward the table. This action rotates the thoracolumbar spine, as when stretching the serratus anterior muscle *(see* Fig. 46.4B). To release the underlying internal oblique muscle, the patient rotates the uppermost hip rather than the shoulder backward toward the table, turning the thorax in the opposite direction. In each position, the spray pattern follows the line of the muscle fibers in a caudal direction.

Starting with the lower limbs in the position illustrated in Figure 49.7 ***A***, the patient can perform full active range of motion of the oblique muscles by moving the thigh from the fully extended position to a fully flexed position with the knee moving toward the opposite axilla and then returning to the diagonally extended position. The contralateral muscles are taken through range of motion by repeating the procedure with the other lower limb. Then, moist heat is applied promptly over the treated muscles.

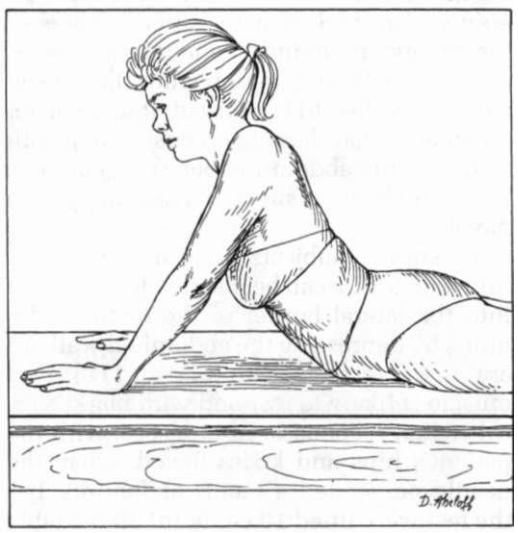


Figure 49.8. Self-stretch of the abdominal muscles. In the prone position, the patient does a press-up, weight-bearing on the upper limbs to arch the back, while being careful to keep the pelvis firmly against the base of support. Deep inhalation using diaphragmatic breathing protrudes the abdomen, which further lengthens and releases tight abdominal muscles. See text for a more detailed explanation.

Dysmenorrhea may be relieved by directing parallel sweeps of vapocoolant spray downward over the painful region of the abdomen[38] for 15 or 20 sec.[34] The authors of this volume are careful to avoid frosting the skin, by continuously moving the stream of spray in parallel lines. The patients may be taught to apply the Gebauer Spray and Stretch Vapocoolant themselves, if repeated applications are necessary. Ethyl chloride is not recommended for use by patients.

The application of effective TrP pressure release to individual TrPs in the abdominal muscles requires that the muscle be placed on sufficient tension. Pressure release is most successful for the TrPs close to the arch of the pubic bones, and less successful in patients with excess adipose tissue.

13. TRIGGER POINT INJECTION (Figs. 49.9-49.11)

Melnick[95] reported that, in a series of 36 patients whose epigastric pain had become refractory to ulcer treatment, 32 responded successfully to myofascial trigger point (TrP) inactivation and were returned to a normal diet without symptoms or need for medication. He injected their abdominal TrPs once or twice weekly until no further muscular hypersensitivity was present. Other authors[63] who appear to be unaware of TrPs identify rectus abdominis spot tenderness as rectus abdominis muscle syndrome when injection of the spot with lidocaine relieved patients' pain. Ling and Slocumb[86] demonstrated the importance of identifying and injecting abdominal wall TrPs for relief of chronic pelvic pain.

Most TrPs in abdominal muscles can be reached with a 3.8-cm (1 1/2in) needle unless the patient is obese. Better control is obtained by inserting it at a shallow angle than by inserting it nearly perpendicular to the skin. The shallower angle makes it easier to align the shaft of the needle with the muscle fibers and to feel the changes in

consistency of fat, fascia, and muscle as the needle penetrates successive layers. One should be careful to avoid penetrating the peritoneal cavity with the needle.

Active full range of motion as described in the previous section, together with repetition of vapocooling, should be performed slowly after the TrP injection and then followed by moist heat.

Lateral Abdominal Muscles (Figs.49.9 and 49.10)

Injection of TrPs in the part of the external oblique muscle overlying the ribs employs a technique similar to the injection of the serratus anterior or serratus posterior muscles, with precautions to avoid penetrating an intercostal space and the pleura.

Injection of the lateral wall obliques is preferably done when possible by pinching the abdominal wall between the fingers and thumb so that no abdominal contents remain within the pincer grasp (Fig. 49.9A and C). The TrP is located by rolling the musculature between the digits to identify a tender nodule in a palpable band. The needle is then directed precisely into the TrP, which is fixed within the operator's grasp.

Suprapubic attachment TrPs are felt as little buttons with bands extending into the muscle where the musculature attaches to the upper border of the pubic bone (Fig. 49.9B). These are injected from above, directing the needle toward the pubis. These attachment TrPs may be responsive also to TrP pressure release.

Injection of the transversus abdominis attachment TrPs along the costal margin (Fig. 49.10) requires special care. The muscle attaches to the underside of the costal margin where the fibers interdigitate with the diaphragm, beyond which lies the pleura. The exact position of the needle tip can be established by gently contacting the costal cartilage and walking the needle down from there.

Rectus Abdominis (Fig. 49.11)

Several authors have noted the effectiveness of injecting TrPs in the rectus abdominis muscle for relief of abdominal pain.[74,88,117] These may be central or attachment TrPs depending on their relation to the tendinous inscriptions. Gutstein[83] warned of postinjection soreness and stiffness for 6-12 hr following injection of the upper rectus abdominis. Kelly[75] estimated that only one-third of these injections relieved the patient's pain as compared with Melnick's 91% success rate;[93] the patients were selected very differently. Hunter[64] emphasized to his patients the importance of their emancipation from the fear of pain. Among 21 cases, he reported that 12 (57%) were fully relieved of pain and 5 (24%) were partly relieved of pain. Dry needling leads to more postinjection soreness than injection of an anesthetic.[60] We find that close attention to perpetuating factors is essential to a high success rate.

Injection of upper rectus abdominis TrPs in the space between the costal margin and the xiphoid process (Fig. 49.11A) again requires careful technique with attention to the depth of needle penetration to avoid entering the abdominal cavity, as also described and illustrated by Rachlin.[103] Experience gained by injecting TrPs in other muscles teaches one to recognize the difference in the feel of the tissue as the needle penetrates skin, subcutaneous fat, epimysium, and then the muscle fibers of the rectus abdominis. Penetration beyond the second layer of epimysium (the posterior rectus sheath) is avoided; it must be remembered that there is no posterior sheath to the rectus abdominis below the arcuate line, which lies a short distance below the navel.

In supine subjects who are relatively thin, the needle can be inserted horizontally into the lateral border of the rectus abdominis by depressing the abdominal wall lateral to the rectus sheath (Fig. 49.11C). This muscle is likely to respond with marked local twitch responses. In one case, with the patient's hips and knees flexed, when the needle penetrated a rectus abdominis TrP, the feet were lifted 10 cm (4 in) off the table by the vigor of the local twitch response.

Injection of the fibers close to the pubic attachment of the rectus abdominis is accomplished by directing the needle toward the pubic bone (Fig. 49.11B).

Injection of TrPs in the pyramidalis muscle is accomplished by directing the

needle cephalad close to the midline, away from the pubis, rather than toward the bone.

14. CORRECTIVE ACTIONS (Figs. 49.12 and 49.13)

Visceral Disease and Other Causal Factors

Myofascial TrP activity may persist long after the initiating acute visceral disease has resolved. However, if the initiating visceral lesion persists (e.g., peptic ulcer, neoplasm, or intestinal parasites), treatment directed only to the TrPs provides merely transient or partial relief. Causative factors must be resolved for lasting relief.[95,117]

Likewise, perpetuating stresses on the muscles must be reduced or eliminated to obtain prolonged relief. Included are emotional stress, viral infections, and mechan-

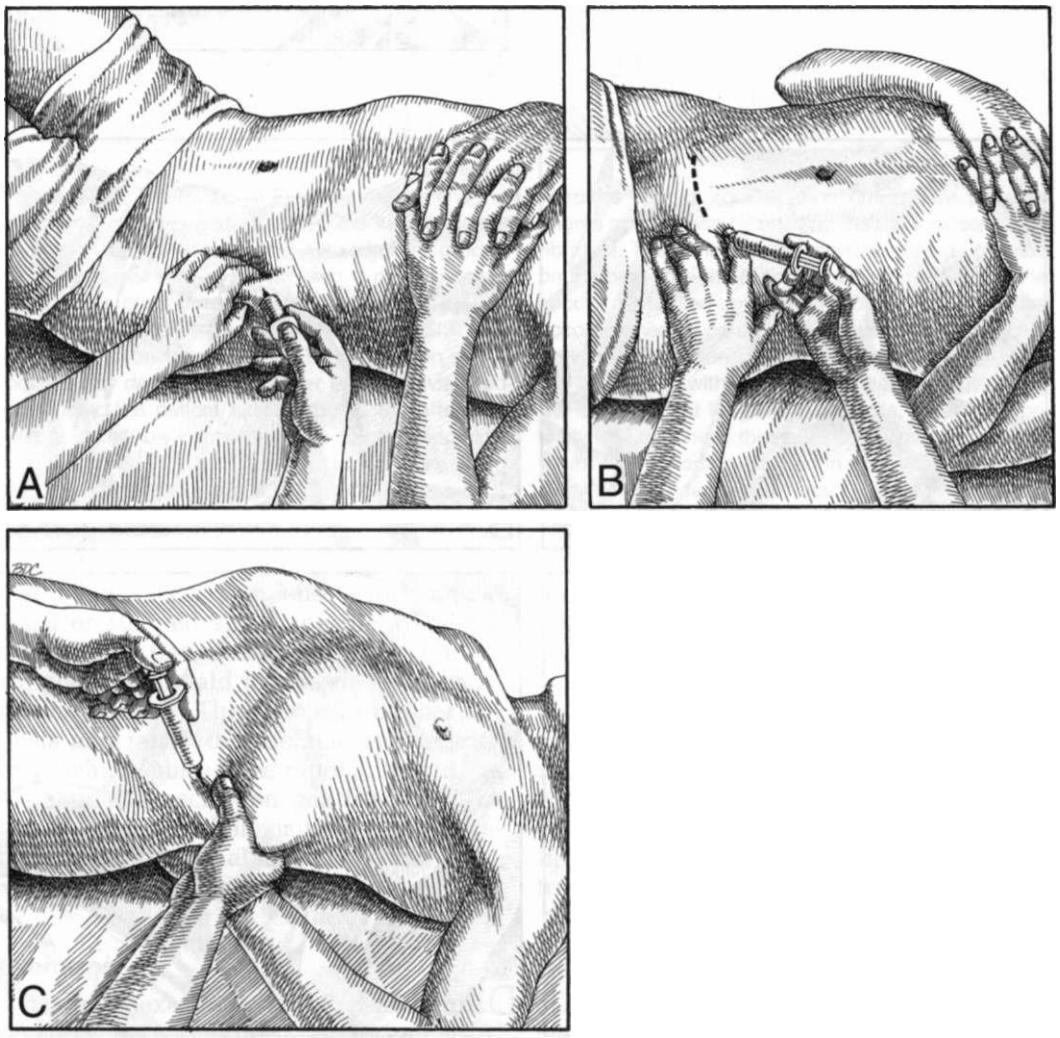


Figure 49.9. Injection of the external abdominal oblique muscle. **A,** pinching the abdominal wall permits grasping the muscle and its trigger points between the digits without any abdominal contents. **B,** suprapubic attachment trigger points are injected against the upper border of the pubic arch. *Dashed line* marks upper border of the pubic bones. **C,** alternate manner of grasping the abdominal wall to avoid injecting abdominal contents while injecting myofascial trigger points in the oblique or transverse abdominal muscles.

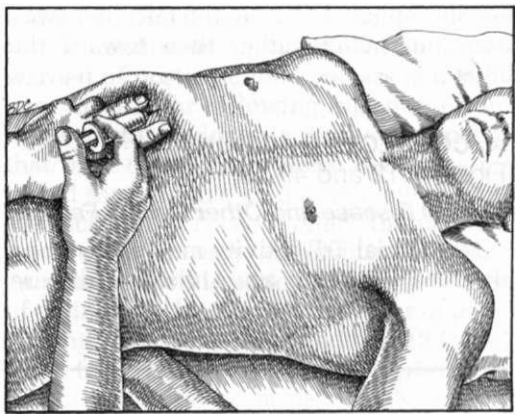


Figure 49.10. Injection of the right transversus abdominis muscle for attachment trigger points along the costal margin. The needle is directed at the caudal border of the rib, not deep to it.

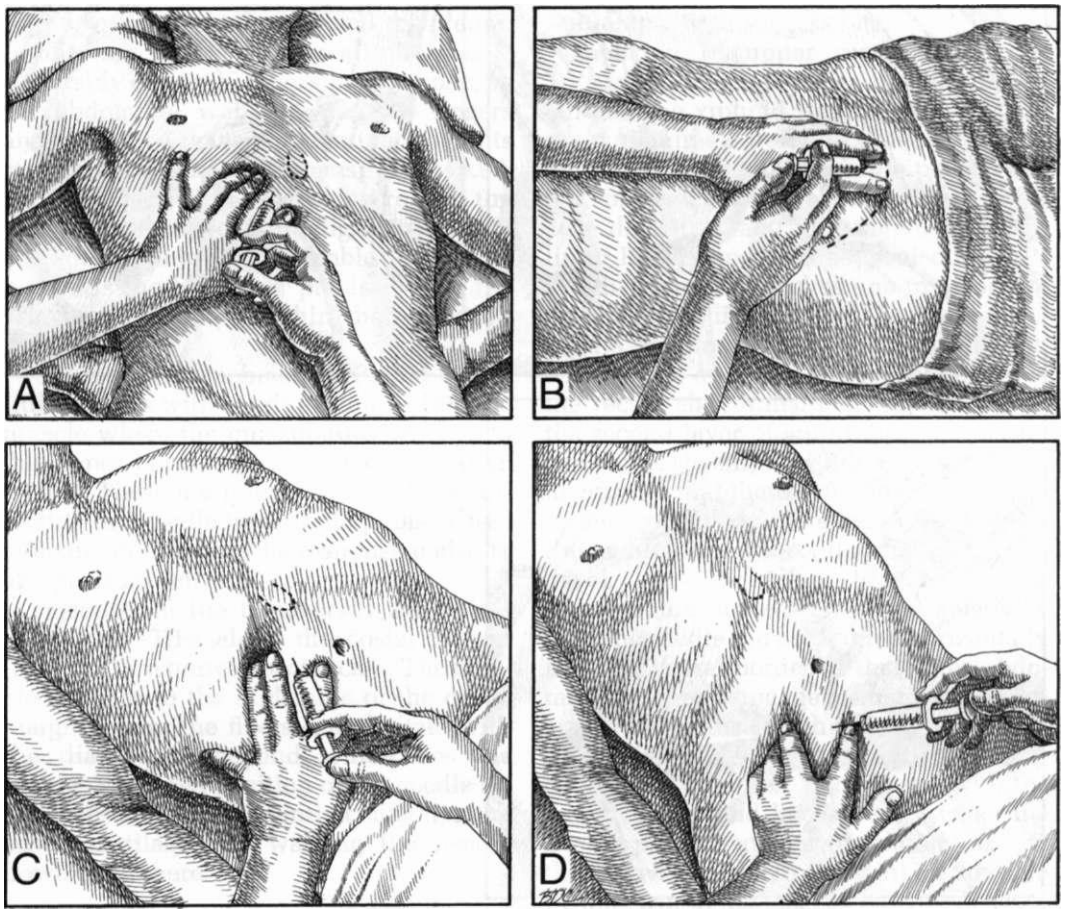


Figure 49.11. Injection of trigger points in the right rectus abdominis muscle. The *dotted line* outlines the xiphoid process in **Parts A, C** and **D**, and in **B** *the dotted line* outlines the upper border of the inguinal ligament and pubis. A, in the para-xiphoid space, with close attention to the depth of needle penetration. **B**, in the suprapubic region. The pyramidalis muscle also lies in this region, but the needle is directed cephalad to inject that muscle. **C**, along the lateral border of the muscle, just above the umbilicus. **D**, in the lower rectus abdominis adjacent to McBurney's point.

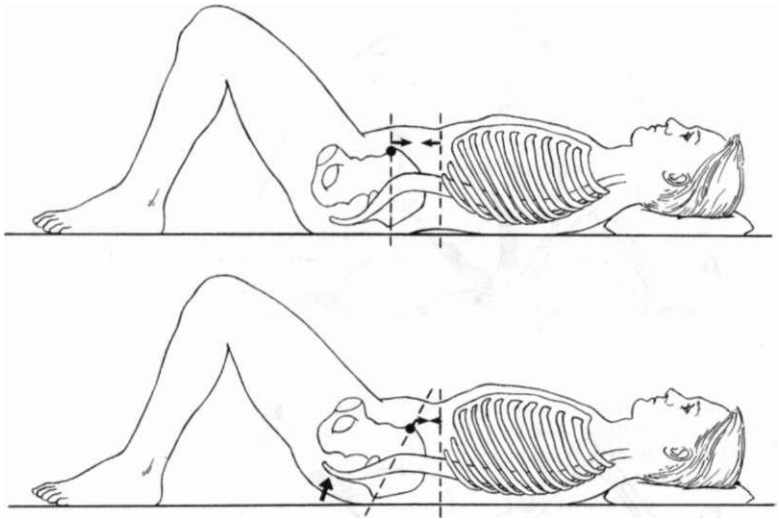


Figure 49.12. The Pelvic-tilt Exercise strengthens the abdominal muscles and stretches the lumbar spinal muscles. **Upper Panel,** normal relaxed starting position. Hands (not shown) can be used to monitor pelvic position by placing each hand so that a finger is touching the anterior superior iliac spine (ASIS) and the thumb is touching the lower rib cage (open space indicated by *clashed lines).* **Lower Panel,** pelvis tilted posteriorly: the patient tilts the pelvis to flatten the lumbar spine by contracting the lower abdominal muscles, pulling the anterior pelvis up while breathing out. This contraction brings the symphysis pubis toward the xiphoid process, approximating the patient's fingers and thumbs by bringing the ASIS closer to the rib cage. The lumbar spine remains firmly supported on the bed while the distal tip of the buttocks and the coccyx are rocked upward, as shown. (This should be accomplished by contracting the muscles in the lower abdomen, *NOT* the gluteal muscles [buttocks], and *not* by pushing with the feet.) The patient should hold the low back flat for several seconds, breathing normally with the chest, then relax and allow the pelvis to return to the starting position in the upper panel. Repeat the exercise several times.

ical distortions to compensate for an awkward or stooped sitting posture. The patient should use a small pillow for lumbar support and should lean against the backrest of the chair. This increases lumbar lordosis and raises the thoracic cage anteriorly, which places the more longitudinal abdominal muscles on gentle stretch. A very tight elastic belt or girdle may compress the abdominal muscles, interfering with their circulation.

Exercises

Helpful exercises for the abdominal musculature include abdominal (diaphragmatic) breathing, the Pelvic-tilt and the Sit-back/Sit-up Exercises, and laughter.

Abdominal (Diaphragmatic) Breathing. The most effective active stretch exercise for these muscles is abdominal breathing[39] (*see* Chapter 20). Abdominal breathing, especially with the patient *prone,* stretches the lateral abdominal wall muscles.

Pelvic Tilt. The Pelvic-tilt Exercise is a gentle and effective strengthening movement for the lower rectus abdominis. It is illustrated as a flexion exercise, by Williams,[130] and as "pelvic tilting" by Cailliet.[17] The exercise is performed as illustrated and described in Figure 49.12.

Sit-back/Abdominal-curl/Sit-up. The Sit-back/ Abdominal-curl/Sit-up Exercise is the smooth combination of three exercises (Fig. 49.13). This combination exercise should always *begin with the* Sit- back Exercise (Fig. 49.13A), which is presented by Cailliet[17] as a progressive "uncurl." It results in a *lengthening,* not shortening contraction of the abdominal musculature. The lengthening contraction of the Sit-back places relatively less load on the involved

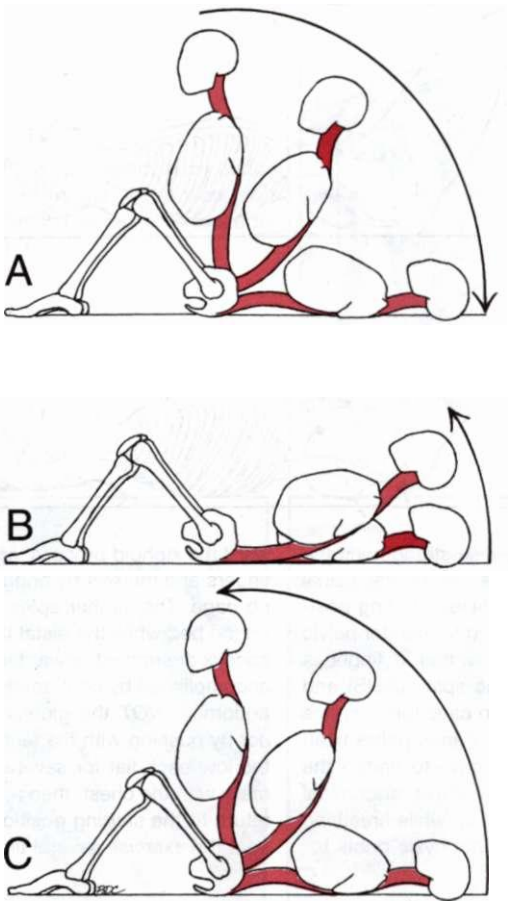


Figure 49.13. A, The Sit-back Exercise is a progressive uncurling that starts in the sitting position and ends supine. The initial sitting position is attained with the help of the arms (not shown). Knees and hips should be bent and the feet fixed. From this initial sitting position, the patient leans back slightly. After a few degrees of uncurling, the patient returns to the starting position. Progressive uncurling, with assisted return to the starting position, is repeated until uncurling reaches the full supine position. **B,** When a full Sit-back has been achieved, the Abdominal-curl Exercise involves rolling up with the patient supine. Progressively, the head is raised free of support, then the shoulders, and finally the scapulae, while the lumbar spine remains firmly supported. **C,** the Sit-up Exercise requires rolling up through an abdominal curl to the full sitting position. The strength required to do this exercise increases as the hands (not shown) are held, first at the level of the hips, next at the abdomen, then at the chest and, finally, at the back of the head. The Sit-up should not be done unless it is pain-free.

abdominal muscles because of the greater strength and efficiency of a lengthening, as compared with a shortening contraction.[57] First, the patient pushes himself or herself up into the sit-up position with the arms and then does a slow Sit-back (Fig. 49.13A). The curl-down movement of the Sit-back should be made smoothly and slowly, without jerks.

The pause between each cycle of the exercise is as important as the movement, and should be equally long. During the pause, the muscle has time to recharge with blood and to wash out waste products. A full inspiration/expiration at the end of each Sit-back helps to re-establish complete relaxation of the muscles and to pace the exercise.

The patient starts by doing the exercise on alternate days or, if the abdominal musculature is still sore, skips two days; then the number of Sit-backs is gradually increased to a goal of 10 per daily session.

Only when the Sit-back goal is reached does the patient proceed to the Abdominal-curl (Fig. 49.13B), which is a partial Sit-up, described by Williams[130] as a flexion exercise. This is done as a "peel-up" with the spine flexed, so that each successive vertebra leaves the floor in turn.

When the Abdominal-curl Exercise can be done comfortably 10 times, the patient may start the Sit-up Exercise (Fig. 49.13C), as illustrated by Williams[130] and by Cailliet[17] as an abdominal flexion exercise.

Laughter. Laughter is a vigorous isometric exercise for all of the abdominal muscles and is "pleasant medicine."

Other Actions

The patient should learn how to apply trigger point pressure release to individual TrPs. While lying in a tub of warm bath water, the patient locates a tender spot, protrudes the abdomen and then applies steady and increasing pressure directly on the sore spot until it is no longer sensitive to the sustained pressure. Subsequently, that spot should still be nontender, but others that remain can be similarly inactivated, TrP by TrP. This self-treatment is especially valuable, between menstrual periods, to minimize dysmenorrhea.

Skin-rolling for panniculosis over the affected abdominal muscles also may be effectively performed by the patient, while relaxing in a warm bath. Patients with paradoxical breathing (asynchrony of the chest *versus* the diaphragm and abdominal muscles) must learn proper respiratory mechanics (see Figs. 20.15 and 20.16).

REFERENCES

1. Aftimos S: Myofascial pain in children. *N Z Med J 102(874)440-441,* 1989.
2. Agur AM: *Grant's Atlas of Anatomy.* Ed. 9. Williams & Wilkins, Baltimore, 1991 (p. 81, Fig. 2.5).
3. *Ibid.* (p. 80, Fig. 2.4).
4. *Ibid.* (p. 82, Fig. 2.6; p. 85, Fig. 2.10).
5. *Ibid.* (p. 88, Fig. 2.15).
6. Aiken DW, Dickman FN: Surgery in obstruction of small intestine due to ascariasis. *JAMA 164(12):* 1317-1323, 1957.
7. Alvarez WC: *An Introduction to Gastro-enterology,* Ed. 3. Paul B. Hoeber, New York, 1940 (p. 144).
8. Anson BJ, Beaton LE, McVay CB: The pyramidalis muscle. *Anatomical Record* 72:405- 411, 1938.
9. Ashkenaz DM, Spiegel EA: The viscero-pannicular reflex. *Am J Physiol* 2 22:573-576, 1935.
10. Basmajian JV, DeLuca CJ: *Muscles Alive.* Ed. 5. Williams & Wilkins, Baltimore, 1985 (pp. 262, 385, 386, 391-397).
11. Bates T, Grunwaldt E: Myofascial pain in childhood. *J Pediatr* 53:198-209, 1958.
12. Beaton LE, Anson BJ: The pyramidalis muscle: its occurrence and size in American white and negroes. *Am J Phys Anthropol* 25:261-269, 1939.
13. Bloomfield AL: Mechanism of pain with peptic ulcer. *Am J Med* 27.165-167, 1954.
14. Bonica JJ, Johansen K, Loeser JD: Abdominal pain caused by other diseases. Chapter 64. In: *The Management of Pain.* Ed. 2. Edited by Bonica JJ, Loeser JD, Chapman CR, *et al.* Lea & Febiger, 1990, pp. 1254-1282, 1990.
15. Broer MR, Houtz SJ: *Patterns of Muscular Activity in Selected Sport Skills.* Charles C Thomas, Springfield, Ill., 1967.
16. Butler DS, Jones MA: *Mobilisation of the Nervous System.* Churchill Livingstone, New York, 1991 (p. 19).
17. Cailliet R: *Low Rack Pain Syndrome.* Ed. 3. F.A. Davis, Philadelphia, 1981 (pp. 115-121; Figs. 81, 85, 86).
18. Carnett JB: Intercostal neuralgia as a cause of abdominal pain and tenderness. *Surg Gynecol Obstet* 42:625-632, 1926.
19. Clemente CD: *Gray's Anatomy.* Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 484,485, Fig. 6-26).
20. *Ibid.* (pp. 488-489, Fig. 6-29).
21. *Ibid.* (pp. 490-491, Fig. 6-31).
22. *Ibid.* (pp. 491-493).
23. Clemente CD: *Anatomy.* Ed. 3. Urban & Schwarzenberg, Baltimore, 1987 (Fig. 12).
24. *Ibid.* (Figs. 235, 237).
25. *Ibid.* (Figs. 238, 242).
26. *Ibid.* (Fig. 241).
27. DonTigny RL: Inhibition of nausea and headaches. *Phys Ther* 54:864-865, 1974.
28. Doouss TW, Boas RA: The abdominal cutaneous nerve entrapment syndrome. *NZ Med J* 82:473-475, 1975.
29. Duchenne GB: *Physiology of Motion,* translated by E.B. Kaplan. J.B. Lippincott, Philadelphia, 1949 (pp. 488-490).
30. Dworken HJ, Biel FJ, Machella TE: Subdiaphragmatic reference of pain from the colon. *Gastroenterology* 22:222-228, 1952.
31. Dyer NH: Painful rib syndrome. *Gut 35(3):420,* 1994. [Letter]
32. Egan TF: Zenker's degeneration of rectus abdominis complicated by spontaneous rupture. *J Ir Med Assoc* 42.127-128, 1957.
33. Eisler P: *Die Muskeln des Stammes.* Gustav Fischer, Jena, 1912 (pp. 571-575, Fig. 93).
34. Ellis M: The relief of pain by cooling of the skin. *Rr Med J* 2:250-252, 1961.
35. Feinstein B, Langton JN, Jameson RM, *et al:* Experiments on pain referred from deep somatic tissues. *J Bone Joint Surg* 36/1:981-997, 1954.

36. Flint MM: An electromyographic comparison of the function of the iliacus and the rectus abdominis muscles. *J Am Phys Ther Assoc* 45:248-253, 1965.
37. Gardner DA: The use of ethyl chloride spray to relieve somatic pain. *J Am Osteopath Assoc* 49:525-528, 1950.
38. Gardner DA: Dysmenorrhea: a case report. *J Osteopath* 58:19-22, 1951.
39. Gelb H: *Killing Pain Without Prescription.* Harper & Row, 1980 (pp. 138, 139).
40. Gerwin RD: Myofascial aspects of low back pain. *Neurosurg Clin North Am 2(4):761-784,* 1991.
41. Gerwin RD, Shannon S, Hong CZ, *et al:* Interrater reliability in myofascial trigger point examination. *Pain* 69:65-73, 1997.
42. Giamberardino MA, Valente R, de Bigontina P, *et al.:* Artificial ureteral calculosis in rats: behavioural characterization of visceral pain episodes and their relationship with referred lumbar muscle hyperalgesia. *Pain* 61:459-469, 1995.
43. Gilleard WL, Brown JM: An electromyographic validation of an abdominal muscle test. *Arch Phys Med Rehabil* 75.1002-1007, 1994.
44. Godfrey KE, Kindig LE, Windell J: Electromyographic study of duration of muscle activity in sit-up variation. *Arch Phys Med Rehabil* 58.132-135, 1977.
45. Goecke C: Die Reizung der vorderen Bauchdeckennerven-Ibrahim-Syndrom. *Zent bl Gynakol* 224:555-556, 1992.
46. Good MG: What is "fibrositis?" *Rheumatism 5:117-*123, 1949 (pp. 120, 121; Fig. 8).
47. Good MG: The role of skeletal muscles in the pathogenesis of diseases. *Acta Med Scand* 238:285-292, 1950.
48. Good MG: Pseudo-appendicitis. *Acta Med Scand* 238:348-353, 1950.
49. Gorrell RL: Appendicitis: failure to correlate clinical and pathologic diagnoses. *Minn Med 34:137-*138, 151, 1951.
50. Greenman PE: *Principles of Manual Medicine.* Ed. 2. Williams & Wilkins, Baltimore, 1996.
51. Gross D: *Therapeutische Lokalanasthesie.* Hippokrates, Stuttgart, 1972.
52. Guivarch M, Boche O, Roullet-Audy JC, *et al:* [33 cases of hematoma of the rectus abdominis muscle in a surgical department]. *Chirurgie* 226(8-9);602-608, 1990.
53. Gutstein RR: The role of abdominal fibrositis in functional indigestion. *Miss Val Med J* 66:114-24, 1944.
54. Hall MW, Sowden DS, Gravestock N: Abdominal wall tenderness test [Letter]. *Lancet* 337:1606,1991.
55. Hall PN, Lee AP: Rectus nerve entrapment causing abdominal pain. *Br J Surg 75(9):917,* 1988.
56. Heinz GJ III, Zavala DC: Slipping rib syndrome. *JAMA* 237:794-795, 1977.
57. Hill AV: The mechanics of voluntary muscle. *Lancet* 2:947-951, 1951.
58. Hockaday JM, Whitty CW: Patterns of referred pain in the normal subject. *Brain* 90:481- 496, 1967.
59. Hodges PW, Richardson CA: Contraction of the abdominal muscles associated with movement of the lower limb. *Phys Ther* 77(2):132-141, 1997.
60. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Behabil* 73:256-263, 1994.
61. Howarth D, Southee A, Cardew P, et *al:* SPECT in avulsion injury of the multifidus and rotator muscles of the lumbar region. *Clin Nucl Med 19(7):571-*574, 1994.
62. Hoyt HS: Segmental nerve lesions as a cause of the trigonitis syndrome. *Stanford Med Bull* 2 2:61-64, 1953.
63. Hughes GS Jr, Treadwell EL, Miller J: Syndrome of the rectus abdominis muscle mimicking the acute abdomen. *Ann Emerg Med* 14(7);694-695, 1985.
64. Hunter C: Myalgia of the abdominal wall. *Can Med Assoc J*28.157-161, 1933.
65. Ingber RS: Atypical chest pain due to myofascial dysfunction of the diaphragm muscle: a case report [Abstract]. *Arch Phys Med Rehabil* 69:729, 1988.
66. Janda V; Evaluation of muscular imbalance. Chapter 6. In: *Rehabilitation of the Spine: A Practitioner's Guide.* Edited by Liebenson C. Williams & Wilkins, Baltimore, 1996 (pp. 97-112).
67. Jelenko C III: Tietze's disease predates "chest wall syndrome." *JAMA* 242:2556, 1979.
68. Kamon E: Electromyographic kinesiology of jumping. *Arch Phys Med Rehabil* 52:152- 157, 1971.
69. Kellgren JH: Observations on referred pain arising from muscle. *Clin Sci* 3:175-190, 1938 (pp. 180, 181, 185).
70. Kellgren JH: On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. *Clin Sci* 4:35-46, 1939.
71. Kellgren JH: The anatomical source of back pain. *Rheumatol Rehabil* 26:3-12, (Plates facing p. 16) 1977.
72. Kelly M: Lumbago and abdominal pain. *Med J Aust 1*:311-317, 1942.
73. Kelly M: Pain in the Chest: Observations on the use of local anaesthesia in its investigation and treatment. *Med J Aust* 2:4-7, 1944 (p. 6, Case V, Fig. 3).
74. Kelly M: The nature of fibrositis. II. A study of the causation of the myalgic lesion (rheumatic, traumatic, infective). *Ann Rheum Dis* 5:69-77, 1946.
75. Kelly M: Some rules for the employment of local analgesia in the treatment of somatic pain. *Med J Aust* 2:235-239, 1947.
76. Kelly M: The relief of facial pain by procaine (novocaine) injections. *J Am Geriatr Soc 11*:586-596,1963.
77. Kelsey MP: Diagnosis of upper abdominal pain. *Tex State J Med* 47:82-86, 1951.
78. Kendall FP, McCreary EK, Provance PG: *Muscles: Testing and Function.* Ed. 4. Williams & Wilkins, Baltimore, 1993 (p. 147).
79. Knockaert DC, D'Heygere FG, Bobbaers HJ: Ilioinguinal nerve entrapment: a little-known cause of iliac fossa pain. *Postgrad Med* 65:632-635, 1989.
80. Lange M: *Die Muskelhdrten (Myogelosen).* J.F. Lehmanns, Munchen, 1931.
81. Lausten GS, Riegels-Nielsen P: Entrapment of the ilioinguinal nerve. *Acta Orthop Belg* 51(6):988-991, 1985.
82. Lewis T, Kellgren JH: Observations relating to referred pain, visceromotor reflexes and other associated phenomena. *Clin Sci* 4:47-71, 1939 (pp. 50, 51, 58, 61).

83. Lewit K: Muscular pattern in thoracolumbar lesions. *Manual Med* 2:105-107, 1986.
84. Lewit K: *Manipulative Therapy in Rehabilitation of the Locomotor System.* Ed. 2. Butterworth Heinemann, Oxford, 1991 (p. 24).
85. *Ibid.* (pp. 209, 218).
86. Ling FW, Slocumb JC: Use of trigger point injections in chronic pelvic pain. *Obstet Gyn Clin North Am* 20(4):809-815, 1993.
87. Llewellyn LJ, Jones AB: *Fibrositis.* Rebman, New York, 1915 (pp. 266-268, Fig. 47).
88. Long C II: Myofascial pain syndromes, part Ill—some syndromes of the trunk and thigh. *Henry Ford Hosp Med Bull* 4:102-106, 1956 (pp. 103, 104).
89. Maigne R: Low back pain of thoracolumbar origin. *Arch Phys Med Rehabil* 62:389-395, 1980.
90. McBeath AA, Keene JS: The rib-tip syndrome. *J Bone Joint Surg* 57A.-795-797, 1975.
91. McMinn RM, Hutchings RT, Pegington J, *et al.: Color Atlas of Human Anatomy.* Ed. 3. Mosby-Year Book, Missouri, 1993 (p. 201).
92. Mehta M, Ranger I: Persistent abdominal pain: treatment by nerve block. *Anaesthesia* 26.-330-333, 1971.
93. Melnick J: Treatment of trigger mechanisms in gastrointestinal disease. *NY State J Med* 54:1324-1330, 1954.
94. Melnick J: Symposium on mechanism and management of pain syndromes. *Proc Rudolf Virchow Med Soc City NY* 26.135-142, 160, 1957.
95. Melnick J: Trigger areas and refractory pain in duodenal ulcer. *NY State J Med* 57:1073- 1076, 1957.
96. Mendeloff Al, Seligman AM: Acute appendicitis. Chapter 287. In: *Harrison's Principles of Internal Medicine.* Ed. 7. Edited by Wintrobe MW, Thorn GW, Adams RD, *et al.* McGraw-Hill, New York, 1974 (p. 1486).
97. Miller MI, Medeiros JM: Recruitment of internal oblique and transversus abdominis muscles during the eccentric phase of the curl-up exercise. *Phys Ther* 67(8):1213-1217, 1987.
98. Milloy FJ, Anson BJ: The rectus abdominis muscle and the epigastric arteries. *Surg Gynecol Obstet* 220:293-302, 1960.
99. Moriarty JK, Dawson AM: Functional abdominal pain further evidence that whole gut is affected. *Br Med J* 284:1670-1672, 1982.
100. Murray J: Rectus abdominis haematoma in pregnancy. *Aust NZ J Obstet Gynaecol* 25:173- 176, 1975.
101. Okada M: An electromyographic estimation of the relative muscular load in different human postures. *J Human Ergol* 1:75-93, 1972.
102. Pernkopf E: Arias *of Topographical and Applied Human Anatomy,* Vol. 2, W.B. Saunders, Philadelphia, 1964 (Figs. 177, 181, 186-188).
103. Rachlin ES: Injection of specific trigger points. Chapter 10 In: *Myofascial Pain and Fibromyalgia.* Edited by Rachlin ES. Mosby, St. Louis, 1994, pp. 197-360 (seep. 214).
104. Reid JD, Kommareddi S, LanderaniM, *et al:* Chronic expanding hematomas. *JAMA* 244:2441-2442,1980.
105. Rogatz P, Rubin IL: Hematoma of the rectus abdominis muscle. *NY State J Med* 54:675- 679, 1954.
106. Ruch TC, Patton HD: *Physiology and Biophysics.* Ed. 19. W.B. Saunders, 1965 (pp. 357- 359).
107. Rutgers MJ: The rectus abdominis syndrome: a case report. *J Neurol* 233:180-181, 1986.
108. Sheehy BN, Little SC, Stone JJ: Abdominal epilepsy. *J Pediatr* 56:355-363, 1960.
109. Simons DG, Travell JG: Myofascial origins of low back pain. Parts 1,2,3. *Postgrad Med* 73:66-108, 1983.
110. Sinclair DC: The remote reference of pain aroused in the skin. *Brain* 72:364-372, 1949.
111. Slipyan A, Batongbacal VI: Massive right rectus muscle hematoma simulating signs and symptoms of coarctation of the aorta. *NY State J Med* 58:3851-3852, 1958.
112. Slocumb JC: Neurological factors in chronic pelvic pain: trigger points and the abdominal pelvic pain syndrome. *Am J Obstet Gynecol* 149:536-543, 1984.
113. Smith LA: The pattern of pain in the diagnosis of upper abdominal disorders. *JAMA* 156:1566-1573, 1954.
114. Spalteholz W: *Handatlas der Anatomie des Menschen.* Ed. 11, Vol. 2. S. Hirzel, Leipzig, 1922 (pp. 291, 294).
115. Staff D, Heudebert G, Young MJ: Hematoma of the rectus abdominis manifested as severe pain in the right lower quadrant. *South Med J 84(10):1275-*1276, 1991.
116. Telling WH: The clinical importance of fibrositis in general practice. *Br Med* /1.-689-692, 1935.
117. Theobald GW: The relief and prevention of referred pain. *J Obstet Gynaecol Br Commonw* 56:447-460, 1949 (pp. 451, 452; Case 3; Fig. 3).
118. Theobald GW: The role of the cerebral cortex in the perception of pain. *Lancet* 2:41-47, 94-97,1949 (p. 41, Fig. 3).
119. Thomson WH, Dawes RF, Carter SS: Abdominal wall tenderness: a useful sign in chronic abdominal pain. *Br J Surg* 78:223-225, 1991.
120. Thorek P: The acute abdomen. *Can Med Assoc J* 62:550-556, 1950.
121. Tietze A: Ueber eine eigenartige Haufung von Fallen mit Dystrophie der Rippen Knorpel. *Berl Klin Wochenschr* 58:829-831, 1921.
122. Toldt C: *An Atlas of Human Anatomy,* translated by M.E. Paul. Ed. 2, Vol. 1. Macmillan, New York, 1919 (pp. 274, 276).
123. Travell J: The adductor longus syndrome: A cause of groin pain. Its treatment by local block of trigger areas (procaine infiltration and ethyl chloride spray). *Bull NY Acad Med* 26:284-285, 1950.
124. Travell JG: A trigger point for hiccup. *J Am Osteopath Assoc* 77:308-312, 1977.
125. Trinca F: New diagnostic method: manipulation of the hypersensitive visceral reflex as a clue to more exact diagnosis. *Med J Aust* 2:493-495, 1940.
126. Tung AS, Tenicela R, Giovanitti J: Rectus abdominis nerve entrapment syndrome. *JAMA 240(8):73S-739,* 1978.
127. Walters CE, Partridge MJ: Electromyographic study of the differential action of the abdominal muscles during exercise. *Am J Phys Med* 36:259-268, 1957.
128. Weiss S, Davis D: The significance of the afferent impulses from the skin in the mechanism of visceral pain. Skin infiltration as a useful therapeutic measure. *Am J Med Sci* 276:517-536, 1928.

129. Willauer GJ, O'Neill IF: Late postoperative follow-up studies on patients with recurrent appendicitis. *Am J Med Sci* 205:334-342, 1943.
130. Williams PC: *Low Back and Neck Pain, Causes and Conservative Treatment.* Charles C Thomas, Spring-field, Ill., 1974 (Panels 1A, 1B, and 2, Fig. 19).
131. Wilson TS: Manipulative treatment of subacute and chronic fibrositis. *Br Med J* 2:298-302, 1936.
132. Wittman A, Bigler FC: Preoperative diagnosis. *J Kans Med Soc* 78:411-414, 1977.
133. Young D: The effects of novocaine injections on simulated visceral pain. *Ann Intern Med* 29:749-756, 1943.
134. deValera E, Raftery H: Lower abdominal and pelvic pain in women. In: *Advances in Pain Research and Therapy.* Vol 1. Edited by Bonica JJ, Albe-Fessard D. Raven Press, New York, 1976 (pp. 933-937).

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The page numbers of the definitive presentation on a topic are set in bold face type. A page number that refers to an illustration or table is *italicized.*