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Myofascial Pain and Dysfunction

The Trigger Point Manual

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need for rest to permit recuperation from injury.[281] When neither interpretation applies, but pain persists, the patient is confronted with chronic pain, as often happens with neglected myofascial TPs. The suffering associated with pain is enormously influenced at the cortical level by the meaning ascribed to it. The powerful effect of meaning on the perception of pain is eloquently demonstrated by religious rites that involve serious tissue damage without anesthesia, but also without evidence of pain or suffering.[166] Acute pain that diminishes in the course of the natural healing process is generally manageable psychologically. However, recurrent or persistent pain due to an unrecognized or untreatable cause threatens future function and well-being, which often leads to frustration, depression and progressive disability.[248]

When patients mistakenly believe that they must "live with" TP pain because they think it is due to arthritis or a pinched nerve that is inoperable, they restrict activity in order to avoid pain. Such patients must learn that the pain comes from muscles, not from nerve damage, and not from permanent arthritic changes in the bones. Most important, they must know it is responsive to treatment. This gives the pain a new *meaning*. When these patients realize the twin facts that their pain is myofascial and is treatable, their lives take on new meaning and they are started on the road to recovery of function.

Referred Pain. Referred pain from any source, TP or visceral, is prohibitively difficult to study in animals and, at this time, the exact mechanisms responsible for it are far from resolved. Hypotheses to account for it include peripheral branching of axons, convergence-projection, convergence-facilitation, reflex constriction of the vasa nervorum, and autonomic nociceptive feedback.

The **periphral branching of axons**[234] would permit the response to a stimulus in one branch to be interpreted as coming from the other branch of the axon. This would require individual sensory neurons to have both visceral and somatic branches. To account thus for the pain referred from myofascial TPs, one branch would have to extend to the TP zone of a muscle and the other branch to the referred pain zone for that muscle. Extensive branching of many sensory nerves in this manner has not been reported.

The **convergence-projection** mechanism[211] is the one generally cited and can occur when cutaneous afferents and visceral (or skeletal muscle) afferents converge on the same spinal neuron. Such convergence has been demonstrated on spinal neurons of the spinothalamic tract in primates.[175] In this model, it is assumed that the sensory cortex has become accustomed to interpreting nociceptor responses of such neurons as coming from the cutaneous structures. The cortex thus misinterprets a strong visceral (or muscular) input as arising from the corresponding skin site. It is argued that, if this mechanism is exclusively responsible for referred visceral pain, anesthetizing the painful skin region should have no effect on the perception of the pain.[284]

The **convergence-facilitation** theory[211] proposes that the normal background activity of sensory afferents in the reference zone is facilitated sufficiently by abnormal visceral afferent activity to be registered as pain. Good[85] reviewed Mackenzie's introduction of this mechanism in 1921 as an "irritable focus" in the spinal cord. Should this be the mechanism responsible for referred pain, it is argued that local anesthesia of the somatic reference zone eliminates the pain for the duration of the block. In fact, local anesthetic block of the region of referred pain often provides relief that long outlasts the duration of the anesthesia, indicating that this mechanism plus another factor, such as suppression of a reverberating feedback in the central nervous system, is present.[166, 288] Considering the strong preponderance of inhibitory synapses in the dorsal horn,[131] this effect of convergence is as likely to be caused by disinhibition as by facilitation.

Roberts[206] proposed that a visceral disturbance might cause **reflex constriction of the vasa nervorum**. If these vessels nourished sensory nerves supplying the reference zone, their ischemia might cause pain to be felt in that area.

Theoretically, **autonomic nociceptive feedback** could occur if autonomic nerves reflexly released nociceptive substances in the referred pain zone.[197, 297] This mechanism would be self-sustaining if the resul-

1. REFERRED PAIN

The patient's pattern of referred pain is usually the key to the diagnosis of a myofascial pain syndrome. This section explains how to draw and interpret distribution of the patient's pain.

Surprisingly, the patient is rarely aware of trigger points (TPs) in the muscle that are causing myofascial pain; pain evoked by lying on an infraspinatus TP at night is perceived in the shoulder, not at the guilty TP in the muscle overlying the scapula. The myofascial TP pain patterns presented throughout this manual were described by patients as situated deep (subcutaneous and muscular) and intensely aching in character, unless stated otherwise in our description.

The patterns of pain referred from TPs 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 disease of a viscus 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 this distribution is mapped at every visit.

We know of no general rule for guessing the referred pain pattern of a muscle. Pain is more likely to be projected distally than proximally in the limbs, and it is often referred to a joint moved by that muscle. However, there are many exceptions. Some muscles, like the deltoid, refer pain only locally; others, like the scaleni, refer pain extensively. The pain pattern of each muscle must be learned individually.

For individual muscles, the *solid red area* in each drawing of referred pain depicts the essential pain zone, which is present in nearly every patient when the identified TP is active. Spillover pain zones, which may or may not be present, appear as *red stippling*. A black (or white) × identifies the usual location of the TP, or TPs, in that muscle; this is intended only as a general guide. The actual location for the individual patient must be determined by physical examination, as described in Section 9 of this chapter.

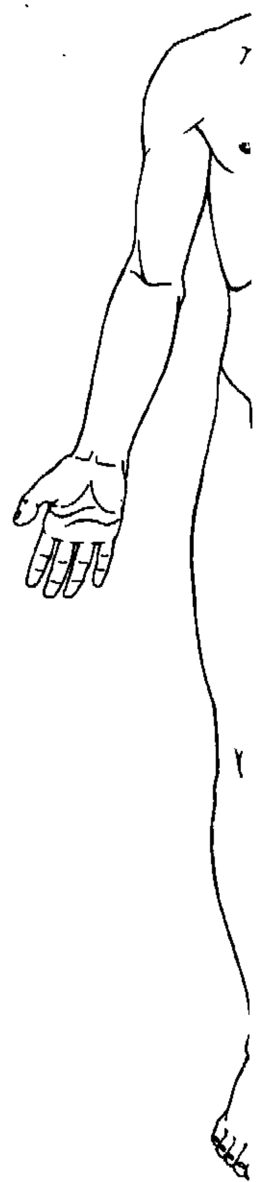
Drawing the Pain Pattern

A precise pictorial representation of the patient's pain is critical for an accurate diagnosis of myofascial pain; verbal descriptions are often imprecise and misleading; a blank body form is used routinely to record the patient's pain. Figures 3.1, 3.2, and 3.3 are forms useful for this purpose.

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. The *patient* delineates the pain on his or her body using one finger, while the *practitioner* draws it on the blank form. The patient may 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 recent pains and the dates of their first appearance are noted for future reference. Other authors also strongly endorse the use of pain drawings.[12,61,85] *Precise* delineation of the patient's pain areas is required to match them with the known pain patterns of individual muscles.

To represent 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, are used for numbness and tingling. A black (or white) × locates the TP area. After treatment, black diagonal lines record the areas that were stretched and sprayed. A circled × locates a TP injected with procaine. Marginal notes tell the date of onset and associated event, unusual depth of the pain (if superficial or deep in the bones and joints), and any unusual quality. 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 direction 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



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1. REFERRED PAIN
(Fig. 16.1)

A different referred pain pattern is observed for each of the three trigger point (TP) areas (Fig. 16.1*A*). The common TP_1 location lies above the base of the neck at the C_4, C_5 level. The TPs in this area refer pain and tenderness upward to the suboccipital region and sometimes down the neck to the upper vertebral border of the scapula (Fig. 16.1*B*) in adults[16, 41, 43] and in children.[3] These TPs may lie as deep as the semispinalis cervicis and multifidus muscles.

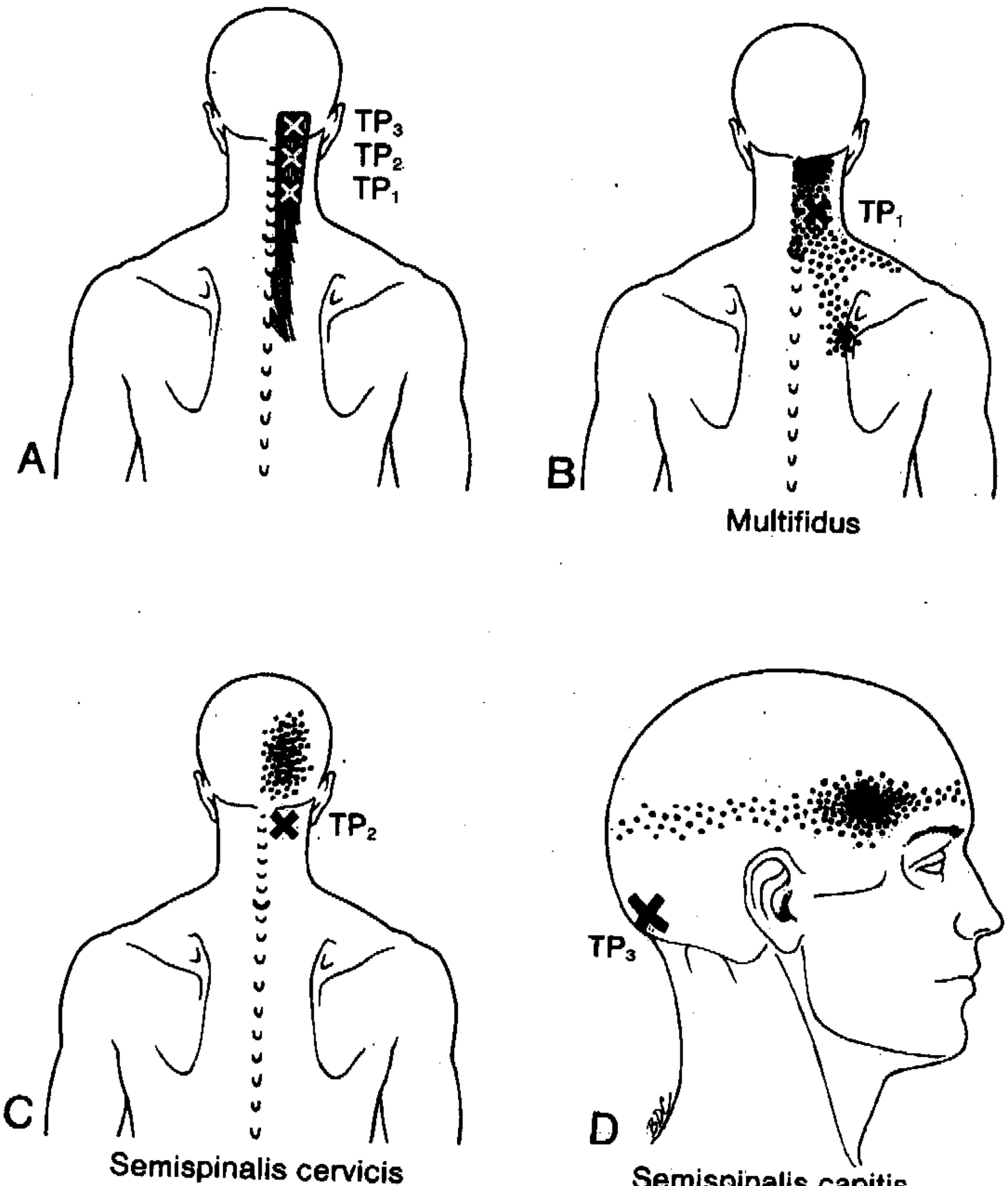

Figure 16.1. Referred pain patterns (*red*) and their trigger points (×s) in the medial posterior cervical muscles. *A*, three major trigger point locations. *B*, TP_1 lies deep at the C_4 or C_5 level in the multifidi or rotatores; it is the posterior cervical trigger point most commonly found and often leads to entrapment of the greater occipital nerve. *C*, TP_2 in the third-layer semispinalis cervicis. *D*, the uppermost TP_3 in the semispinalis capitis.

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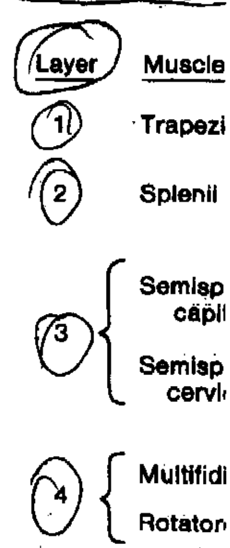

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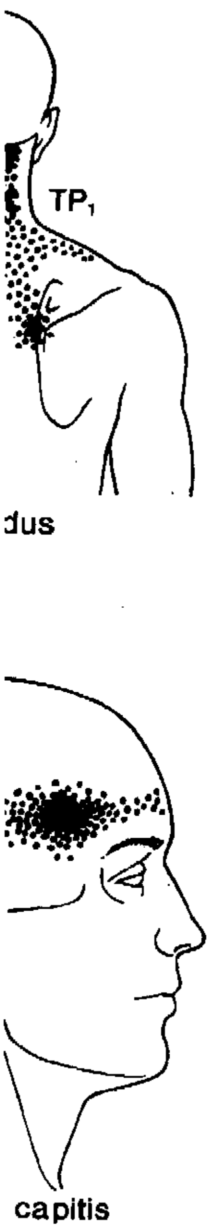

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Activity of TP_2, located 2-4 cm (1-2 in) below the occiput, refers pain over the occiput and toward the vertex (Fig. 16.1C).

TP_3 lies just below the occipital ridge at the insertion of the semispinalis capitis. Its referred pain travels forward like a band that half encircles the head and reaches its maximum intensity in the temple and forehead over the eye (Fig. 16.1D). This TP does not refer pain to the neck. Infrequently, TPs in the posterior cervical muscles below the occiput may refer pain to the hands and feet bilaterally, or to the body below the shoulder on the same side.[40]

The referred pain patterns from all three locations were reproduced by the injection of hypertonic salt solution into these posterior cervical muscles.[6, 42]

2. ANATOMICAL ATTACHMENTS (Figs. 16.2 and 16.3)

The four layers[13] of the posterior cervical muscles alternate direction, suggesting the plies of a tire (Fig. 16.2).

The most superficial, the upper trapezius fibers, converge above, tending to form a "∧", or roof-top shape. The next deeper, the splenius fibers, converge below to form a "V" shape. The semispinalis capitis fibers of the third layer lie nearly vertical, parallel with the spine. All of the remaining, deepest fibers return to the "∧" shape. These include the more deeply placed semispinalis cervicis of the third layer and the multifidi and rotatores fibers, which constitute the fourth layer. The angle becomes progressively flatter (more obtuse) as successively deeper fibers span fewer segments.

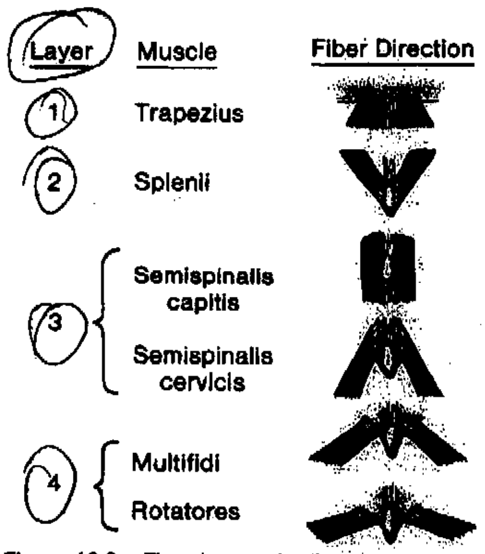

Figure 16.2. The changes in direction of successively deeper fibers in the four layers of the posterior cervical muscles.

Knowledge of this fiber arrangement is helpful in order to stretch and spray these muscles effectively.

Semispinalis Capitis and Semispinalis Cervicis

The semispinalis capitis muscle overlies the semispinalis cervicis. Both attach *below* to the transverse processes of the thoracic vertebrae, T_1 to T_6, and sometimes T_7 (Fig. 16.3). The semispinalis capitis also attaches below to the transverse processes of cervical vertebrae C_4 to C_6 and often has a tendinous inscription that runs across the muscle opposite the C_6 vertebra, most marked where the fibers come from the thoracic vertebrae. ***Above*** the semispinalis capitis attaches to the occiput between the superior and inferior nuchal lines, while the cervicis (illustrated in Fig. 48.4, Chapter 48) attaches to spinous processes of the C_2 to C_5 vertebrae.[13] The fibers of the semispinalis cervicis cross four or five vertebrae.[1] The longissimus capitis has common thoracic attachments below with the semispinalis capitis (T_1 to T_6), but connects, above, to the skull just lateral to the semispinalis capitis, so that both muscles have similar actions; the longissimus capitis is treated in this manual as part of the semispinalis capitis.

Multifidi and Rotatores

The cervical multifidi attach ***above*** to the spinous processes of vertebrae C_2 to C_5. They attach ***below*** to the articular processes of the last four cervical vertebrae, C_4 to C_7. Its fibers cross two to four vertebrae (Fig. 16.3).

The cervical rotatores have comparable attachments, but are shorter and connect adjacent or alternate vertebrae (Fig. 16.3). Since they lie immediately beneath the multifidi and have essentially the same functions, the rotatores are considered as part of the multifidi in this manual.

Supplemental References

Other authors have illustrated the semispinalis capitis as seen from be-

being careful to include skin overlying all of the muscle. No therapeutic harm is done with some additional coverage. Full coverage also may release latent TPs in other parts of the same muscle or in adjacent muscles, where they can be limiting the range of motion.

Many patients are startled by the cold spray if they are not warned that it is coming. The impact of the spray on the operator's hand, and then on the patient's hand, should be demonstrated before starting treatment. When vapocooling the face, the eye on that side should be covered. It is startling, but not damaging, if Fluori-Methane spray accidentally hits the conjunctiva, or the eardrum.

The patient should tell the operator if he or she wants an area of skin covered that was missed by the spray. Vapocooling such an overlooked region usually further releases muscle tension. It is remarkable how precisely the skin that needs to be sprayed overlies the muscle fibers on maximum stretch. A path that is a few centimeters to one side fails to afford the release of tension that an accurately centered jet stream provides. It also is remarkable how the muscle tension sometimes melts away as the stream of spray is extended over the most distant portion of the referred pain pattern.

The direction of spraying was initially determined by subjective testing on patients by the senior author, who noted the direction of spraying that the patients preferred and that gave the maximum relief of tension and pain.

When the spray is applied for the first time over very irritable TPs, the skin may be hypersensitive to the cold, even unbearably so. However, after several passes of the spray, this extreme sensitivity usually abates. The initial distress can be alleviated by using a bottle saved for its fine-bore nozzle, by holding the bottle closer to the skin so that the spray has cooled very little before impact, and by wafting the jet stream across the skin more rapidly.

No cases are known of skin irritation or allergic reaction to Fluori-Methane spray. A white trace left behind on the skin is sebum dissolved from its pores. When patients use the spray often, the sebum can be replaced with cold cream or lanolin.

Patients generally learn quickly to self-spray their masticatory and calf muscles. However, it requires unusually skillful selective relaxation to effectively stretch and spray by oneself the shoulder-girdle, arm and neck muscles. Self treatment is useful during the transition period, while the perpetuating factors are still being identified and resolved, and for patients who seem unavoidably prone to reactivation of TPs and need to inactivate their TPs promptly themselves.

Substitute Stimuli. A variety of distracting sensory stimuli can be applied to the skin over the muscle while it is being stretched, but generally, none of these substitutes are quite as effective as vapocoolant spray. Nearly as effective is hot running water, especially if the person sits and relaxes under a hot shower, thus avoiding antigravity tension (see Fig. 16.10). Ice molded in a paper drinking cup, or an ice cube wrapped in plastic, can be stroked over dry skin, simulating the parallel sweeps of spray.[27, 164] The prolonged skin wetness caused by bare ice produces generalized cooling instead of a spot of rapid temperature change. Skin rolling, as used to diagnose and treat panniculosis, helps to relieve underlying muscle tension. One can use the neurologist's pinwheel, designed for testing pinprick sensation, by running it over the same paths that one would run the spray, and call it the "stretch-and-prickle" technique.

Other Uses. Ethyl chloride spray was initially used for **joint sprains**;[93] Flouri-Methane is equally effective. The sooner the vapocoolant is applied after the sprain, the more fully it relieves pain and disability. Vigorous stretching is avoided in the presence of torn tissues, but the full range of motion at the joint should be reestablished within a reasonable period.

The vapocoolant spray is effective for relieving the pain of **thermal burns**, and it also reduces their secondary hyperalgesia and erythema, as demonstrated in experimental studies.[127, 138] Burns that were sprayed until pain-free did not blister, compared with untreated control burns that did. The spray is applied to the painful area as soon as possible after the burn until it stops hurting, which usually takes several seconds. The spray is reapplied

Lewis and Kellgren[38] 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.

Other sources of chest pain to be considered include intercostal neuritis or radiculopathy; irritation of the bronchi, pleura or esophagus; hiatal hernia with reflux; distension of the stomach by gas; mediastinal emphysema[53]; gasseous distension of the splenic flexure of the colon[10]; and lung cancer.

Some of the less common non-cardiac skeletal syndromes that cause pain and tenderness in the chest include the chest wall syndrome.[13] Tietze's syndrome,[32, 58] costochondritis, the hypersensitive xiphoid, the precordial catch syndrome,[5] the slipping rib syndrome[27] and the rib-tip syndrome.[43] Each patient should be carefully examined to determine if the symptoms are partially or entirely due to myofascial referred pain and tenderness, especially from TPs 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. Relief by injection is characteristic of TPs.

The patient who presents with a painful or tender breast, often with hypersensitivity of the nipple to light contact, may harbor responsible TPs in the lateral margin of the pectoralis major muscle[63, 69] (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.

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 TP in the right pectoralis major muscle between the fifth and sixth ribs at the specific site[64] (Fig. 42.2B). Although this TP is tender to palpation, it is not a source of spontaneous pain. Inactivation of the TP promptly restores normal sinus rhythm when an ectopic supraventricular rhythm is present, and often eliminates 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.[16] Another 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.[71] Even the application of only vapocoolant spray to the area of chest pain referred from a myocardial infarct relieved the pain at once.[44, 64] 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 TPs in the chest wall muscles.[52]

Another example of somatic modulation of visceral cardiac pain was observed using the intravenous ergonovine test, which induces 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 min 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.[64] 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 just *before* the ergonovine injection to the areas that were known to become painful in response to the myocardial ischemia caused by this test and by

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Weiss and Davis[90] eloquently 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 TP in a muscle.[39, 45] Relief of pain in this way does not identify the source of the pain.

Symptoms Due to Trigger Points NOT in Abdominal Muscles

Although one first thinks of TPs in the abdominal musculature to explain nonvisceral abdominal pain, there are numerous other TP sites to be considered. Epigastric pain suggestive of a duodenal ulcer may arise from "fibrositic nodules" (TPs) in the region of the serratus anterior muscle, and has been effectively treated by digital pressure on the nodules.[93]

Occasionally, abdominal pain in an upper quadrant may be due to Tietze's syndrome of the costal cartilages,[84] reported also as affecting the xiphisternal joint,[42] or to abnormal mobility of the lower intercostal joints, which has been variously referred to as the "slipping rib syndrome,"[37] or the "rib-tip syndrome."[57] This may be 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.[57] Temporary, sometimes permanent, relief was obtained from this symptom by the local injection of an anesthetic agent.[57] Some patients required surgical removal of the hypermobile rib segment to obtain permanent relief.[37]

Abdominal pain, particularly in the lower quadrant of the abdomen, may be referred from TPs in the paravertebral muscles,[36, 60, 62, 95] (Chapter 48). Gastrointestinal pain and cramping has been reported from TPs specifically in the erector spinae bilaterally.[19] Also, nausea and belching may result from TP activity in the paraspinal muscles at the upper thoracic level.[2, 10] Three examples of abdominal pain were attributed to remote TPs in the skin itself.[72] Lower abdominal pain, tenderness and muscle spasm may be referred from TPs located in the vaginal wall about 2.5–3.8 cm (1 to 1½ in) inside the introitus, in a region that is normally insensitive to digital pressure.[61]

Urinary frequency, urinary urgency and "kidney" pain may be referred from TPs in the skin of the lower abdomen, as well as from TPs in lower abdominal muscles. Injection of a TP 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 TP in the skin close to McBurney's point were relieved for at least 8 months by its injection with a local anesthetic.[40]

A TP high in the adductor muscles of the thigh may refer pain upward into the groin and to the lower lateral abdominal wall.[86]

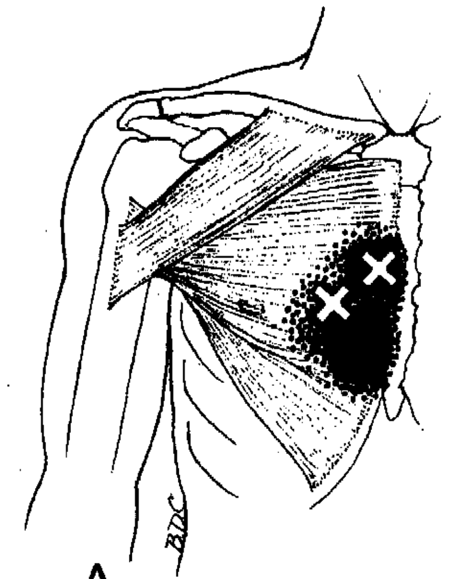
Feinstein, et al.[17] injected hypertonic saline into paraspinal musculotendinous tissues, 1.3–2.5 cm (½ to 1 in) from the midline at each segmental level. The abdominal pain patterns referred from paraspinal muscles at the T_7 to T_{12} levels were similar, but without the precise degree of segmental correspondence suggested by Melnick.[61] Clinically, these authors found only an approximate anterior segmental correspondence.

Lewis and Kellgren,[54] and later Kellgren,[46] described pain referred to the abdomen from interspinous ligaments when they were injected with hypertonic saline; Hockaday and Whitty[39] subsequently found that pain was referred from these ligaments only to dorsal areas. The more extensive pain patterns observed by Kellgren[46] may have been due to his injection of paraspinal (non-midline) structures, which Hockaday and Whitty scrupulously avoided.

Differential Diagnosis

Non-abdominal diseases that may cause abdominal symptoms include coronary artery insufficiency,[52] which may present as acute "indigestion";[53] pneumonia of the lower lobe with pleurisy; herpes zoster; a ruptured upper lumbar vertebral disc with nerve root compression; diabetic acidosis; and abdominal migraine.

The abdominal distress[52] and referred pain patterns[74] caused by a number of abdominal diseases is mimicked by the



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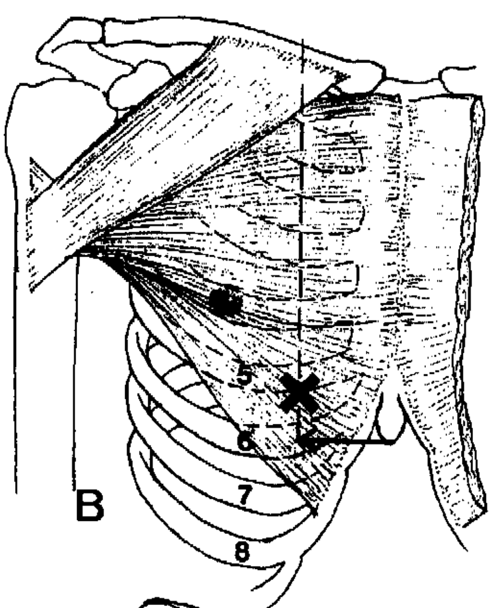

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Figure 42.2. Right pectoralis major muscle trigger-point phenomena. *A*, overlapping referred pain patterns (*red*) of two parasternal trigger points (xs) located in the medial sternal section of the muscle. *B*, location of the "cardiac arrhythmia" 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*).

leaves of a fan. Hollinshead[28] clearly described this relationship between the clavicular and sternocostal sections. A few other authors recognized the overlap of these sections.[2,23,28,40,49,56] Many illustrations of the muscle show a variable degree of this overlap,[2,22,28,40,45,48,56,59] while others do not.[22,23,54]

Eisler[12] 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[28] also described this folding process and illustrated it diagrammatically.[28] Some illustrations of the muscle also portray this feature,[12,22,23,28,40,45,54,56,59] but others do not.[2,22,48] Drawings of the muscle with and without this fold sometimes appear in the same volume.

Frustrated by these inconsistencies, Ashley[1] dissected 60 adults and 8 fetuses to establish the facts. He reported clear schematic drawings 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[1] 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[1] found (Fig. 42.5) that the tendinous pectoralis major attachment ***laterally*** to the humerus has two layers, each of which is made up of laminae. The ventral layer at the humerus, as previously described by Eisler,[12] 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 of this superficial layer.

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 pectoralis major to attach on the humerus as most, if not all, of the dorsal patients with this pressure callus also would have a short first metatarsal bone. Even though the bone disproportion was not primarily responsible for the callus, it would tend to perpetuate it.

To illustrate the usefulness of this Morton correction technique, a 2-year-old child with a 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.

The Dudley J. Morton foot is unrelated to the metatarsalgia of Morton's neuroma, described by Thomas G. Morton[3] as due to pressure on an interdigital neuroma of the plantar nerve, usually between the third and fourth metatarsal heads.

Surprisingly, TPs in the lower extremity muscles can interact with tense muscles of the head and neck to restrict movement of the latter. Release of tension in the lower extremity muscles, by inactivation of their TPs, such as those perpetuated by a short first, long second, metatarsal relationship, may at once increase a TP-restricted interincisal opening of the jaws by 20 or 30%.

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 TPs in the shoulder-girdle musculature. Short upper arms are characteristic of the body structure of the American Indian, but are not limited to this race. If the shoulder-elbow segment of the upper extremity 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 (Fig. 6.10C). For most adults, the average armrest height from the compressed seat bottom is 22 cm (8½ in), and ranges from 18–25 cm (7–10 in).[83]

This disparity places undue stress on the shoulder-girdle elevators, thus perpetuating TPs in the upper trapezius and the levator scapulae muscles. One must be able to recognize shortness of the upper arms in patients with persistent TPs in these muscles and to make the seating corrections discussed and illustrated in Section 14 of Chapter 6.

Postural Stresses

Here we consider postural stresses due to misfitting furniture, poor posture, abuse of muscles, and immobility.

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[291] 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."[291] Body proportions that are the basis for the design of comfortable chairs have been meticulously detailed.[83] The value of an adequate lumbar support is illustrated in Fig. 42.9E of Chapter 42; auto seats are among the worst offenders in this respect.

Poor Posture. This is another frequent source of chronic muscular strain that perpetuates myofascial TPs. Common examples of poor posture that contribute to continued TP activity are unphysiologic positioning at a desk or work surface (see Fig. 16.4 *C* and *D*) and head tilt resulting from poorly adjusted reading glasses (see Fig. 16.4 *A* and *B*), 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.[286] Correction of a kyphotic, round-shouldered posture when standing (see Fig. 42.9*A*, *B* and *C*, in Chapter 42) and when sitting (see Fig. 42.9 *D* and *E*) relieves the upper back and more caudal back muscles, as well as easing chronic shortening of the pectoral

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Section 14 of Chapte

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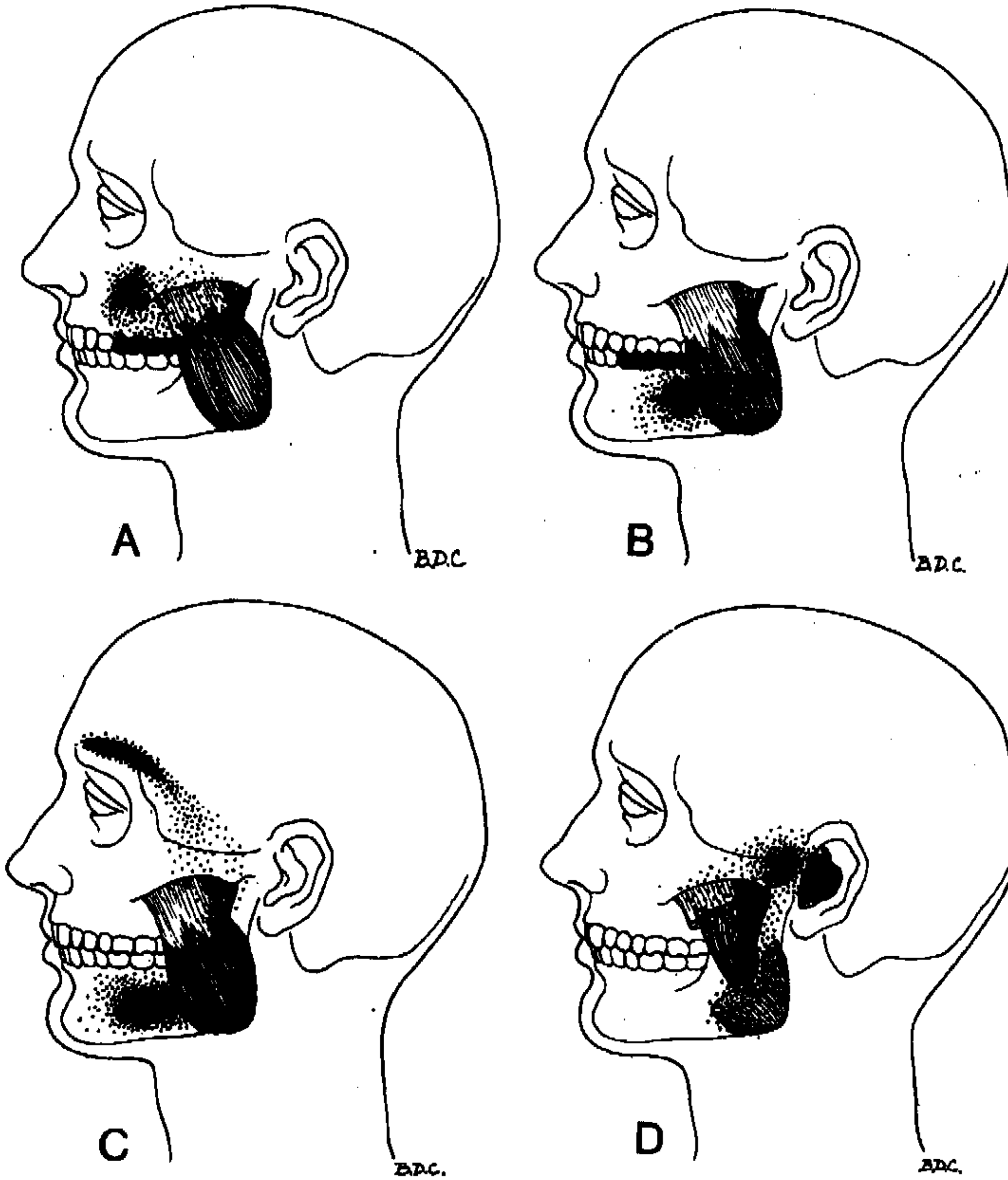

Figure 8.1. The ×s locate trigger points in various parts of the masseter muscle. *Solid red* shows essential referred pain zones, and the *stippled areas* are spillover pain zones. *A*, superficial layer, upper portion. *B*, superficial layer, mid-belly. *C*, superficial layer, lower portion. *D*, deep layer, upper part—just below the temporomandibular joint.

located just *below* the mid-belly of the muscle, they refer pain to the lower molar teeth and mandible (Fig. 8.1B).[80, 86] From TPs 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).[31, 80-82] A masseter TP at the gonial angle may refer pain preauricularly in the region of the TMJ.[89] Prolonged pain responses to a thermal stimulus to a tooth may indicate a pulpitis, whereas sensitivity to percussion and pressure can result from inflammation of

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Deep Layer

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ate maxillary teeth
[6,44,49,55] In the pos-

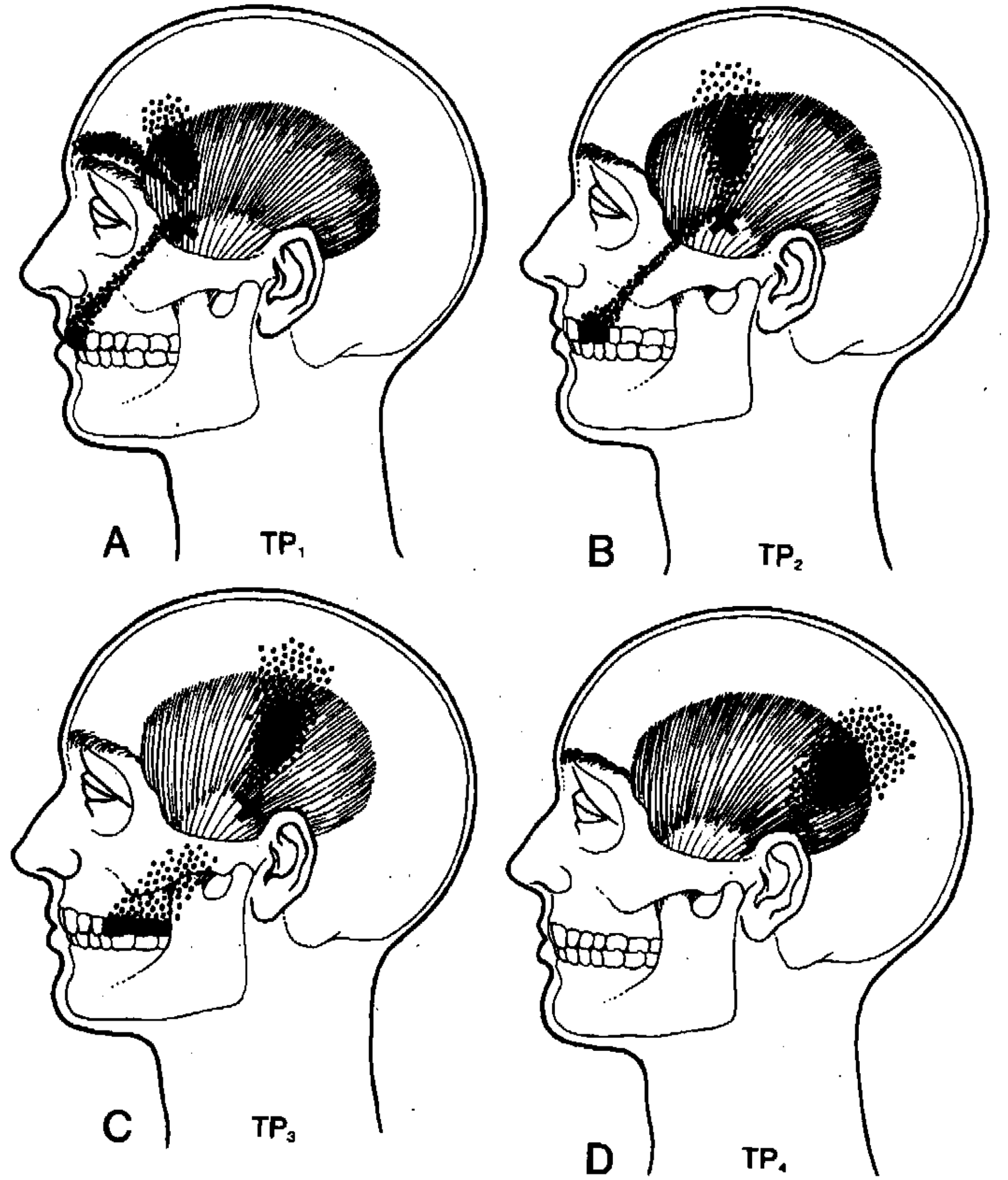

Figure 9.1 Referred pain patterns from trigger points (Xs) in the left temporalis muscle (essential zone *solid red*, spillover zone *stippled*). *A*, anterior "spokes" of pain arising from the anterior fibers (trigger point one region). *B* and *C*, middle "spokes" (trigger point two and trigger point three regions). *D*, posterior supra-auricular "spoke" (trigger point four region).

terior section, active TPs in the TP_4 region refer pain backward and upward (Fig. 9.1*D*).[44] Fibers of the temporalis deep in the TP_3 region, like the deepest masseter fibers, may refer pain to the maxilla and the TMJ.[6,44]

Deep tenderness may be found in each of these pain reference zones even when the corresponding TPs 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.[44]

Wolff[53] studied the vascular response and neuromuscular changes in 10 non-

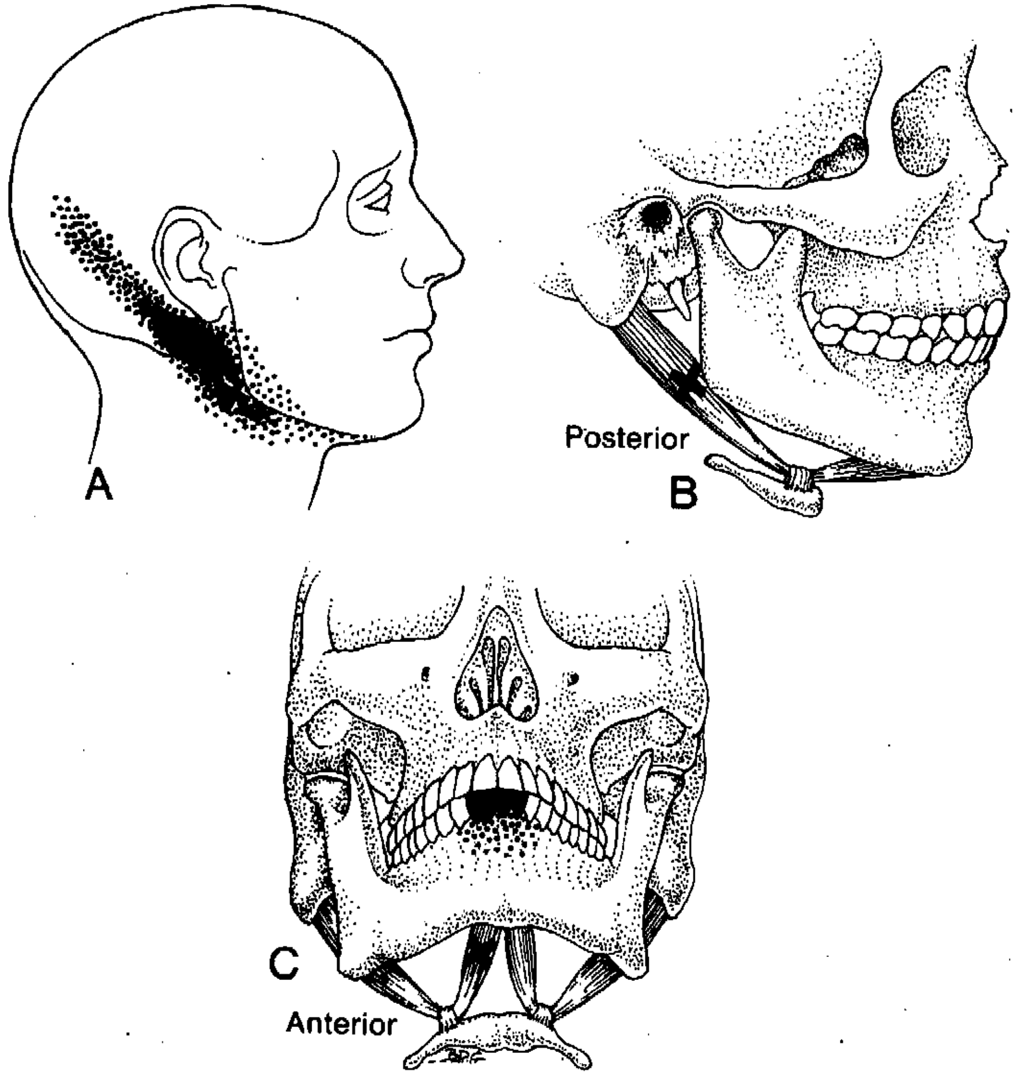

Figure 12.1. Referred pain pattern (essential portion, *solid red*; spillover portion, *stippled red*) of trigger points (×s) in the right digastric muscle. *A* and *B*, posterior belly, side view. *C*, anterior belly, front view.

united end-to-end by a common tendon that attaches to the hyoid bone through a fibrous loop or sling. ***Behind and above*** the posterior belly attaches to the mastoid notch deep to the attachments of the longissimus capitis, splenius capitis and sternocleidomastoid muscles on the mastoid process. In ***front and above*** the anterior belly attaches to the inferior border of the mandible, close to its symphysis.

The common tendon perforates the stylohyoid muscle, which lies near the front half of the posterior belly of the digastric. The common tendon of the digastric connects to the hyoid bone either directly by bands, or by sliding through a fibrous loop.[1]

Supplemental References

Anatomy textbooks illustrate both bellies of the digastric muscle in level side view[12, 20, 22] and as seen from below in side view,[1, 5, 10, 16, 21] from inside the mouth,[6] and from the front.[15] The relationship between the muscle and underlying neurovascular structures is clearly illustrated in

Figure '
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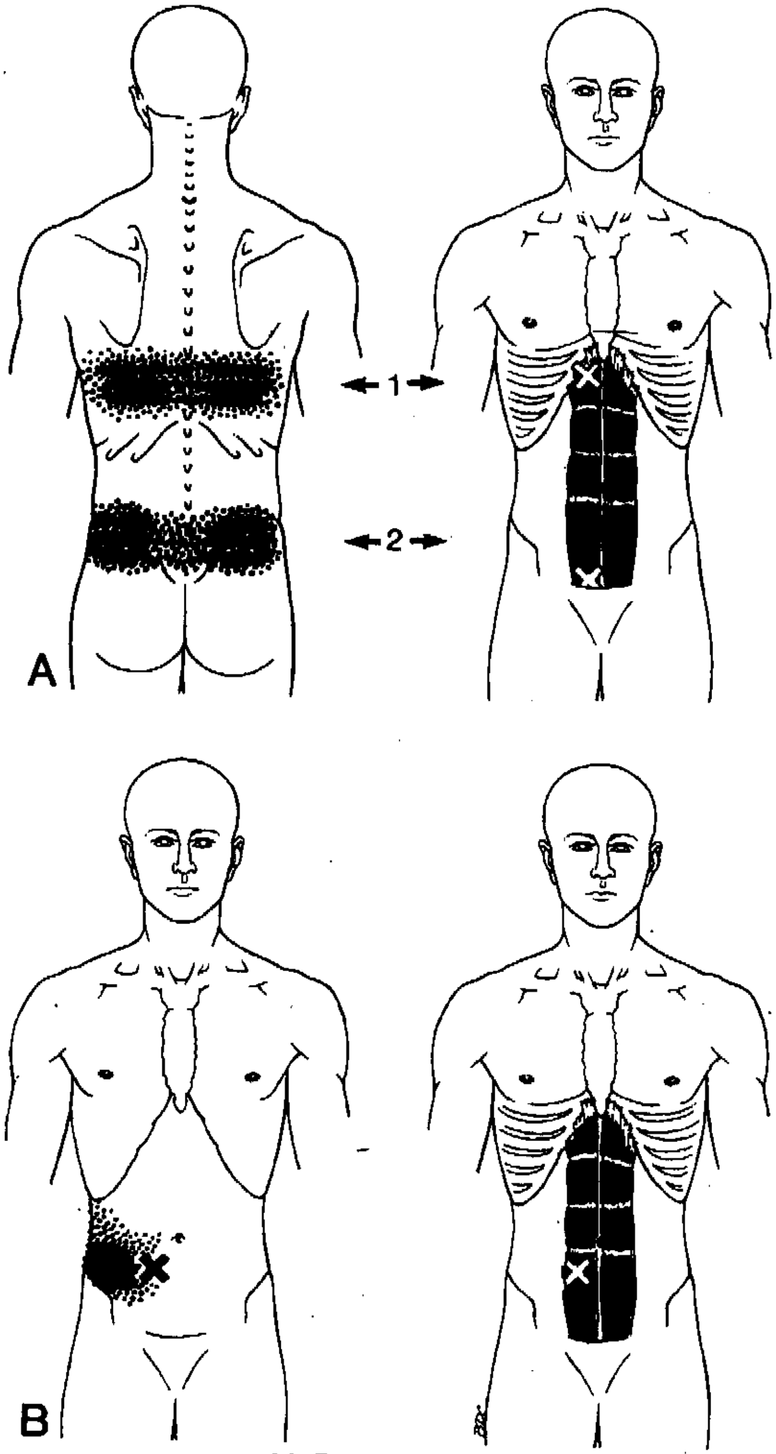


Figure 49.2 *A* and *B*.

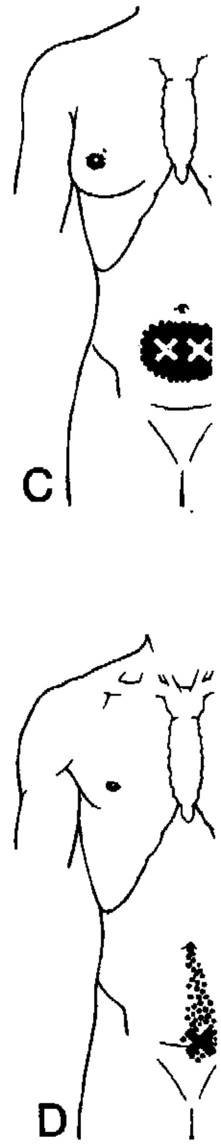


Figure 49.2A–D. R
trigger points (xs) ir
red). *A*, bilateral pa
abdominal fullness, n
the left (or right) upp
pain is referred from
either side. *B*, lower r
of McBurney's point
rectus abdominis. *C*,
in the lower rectus. *L*

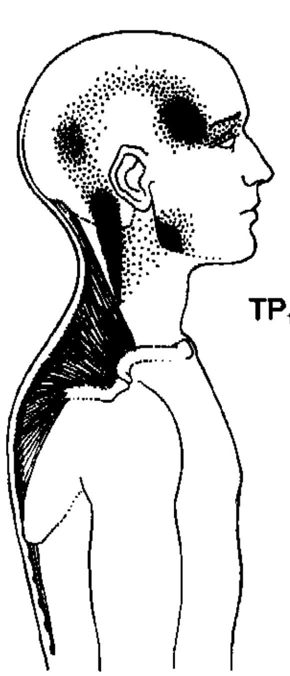
mus dorsi and the upper f
serratus anterior muscle. Al
three muscles appear in anat

ɔwer portions of the trapezius; two re located in each portion. A seventh ɛfers a non-painful autonomic re- e. The TPs are numbered in their ximate order of prevalence.

peziu TP₁ is observed the most often myofascial TPs in the body. It was y the most common in a survey of ealthy asymptomatic young adults.[48] TP₁ area makes a significant contri- n to the facial pain of the myofascial dysfunction syndrome, which is y recognized by the dental profes- , 11, 44, 64

r Trapezius Fibers

ı (Fig. 6.1). In our experience, TPs in area consistently refer pain unilat- upward along the posterolateral as- of the neck to the mastoid process, re a major source of "tension neck- ' (Fig. 6.1).[28, 66] The referred pain, ı intense, extends to the side of the centering in the temple and back of orbit,[25, 67] and also may include the of the jaw.[32, 56, 57, 59, 61, 69] Occasion- pain extends to the occiput, and



e 6.1. Referred pain pattern and location (×s) igger point 1 in the upper trapezius muscle. Solid nows the essential referred pain zone; stippling the spillover zone.

rarely, mild pain is referred to the lower molar teeth. Pain referred from TP₁ may appear in the pinna, but not deep inside the ear. Stimulation of this TP by needling and injection has initiated referred vaso- motor effects in the homolateral and op- posite ear.[56–58, 60]

Other authors describe a similar post- auricular pain pattern,[12, 23, 39] including one in children.[2] A shoulder component of the pain[13, 24] is to be expected when the under- lying supraspinatus muscle also harbors active TPs.[26] Occasional reports[12, 18] asso- ciate TP activity of the upper trapezius fibers with symptoms of dizziness or "vertigo," and with dizziness experienced momentarily when the TP is penetrated by a needle during injection. This postural dizziness may be referred directly from the trapezius or, we think more likely, it may result from reflex stimulation of ac- tive TPs in the clavicular division of the synergistically related sternocleidomas- toid muscle. A comparable secondary ex- tension of referred pain is sometimes seen between related muscle groups in other parts of the body.

When patients had both neck and shoul- der pain, Sola and Kuitert[47] found that levator scapulae and infraspinatus TPs were more frequently the cause than were trapezius TPs.

Experimental injection of the upper tra- pezius 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 tempera- ture that overlapped the area of referred pain in 6 subjects.[50]

TP₂ (Fig. 6.2). The referred pain pattern of this TP lies slightly posterior to the essential cervical reference zone of TP₁, blending with its distribution behind the ear (Fig. 6.2). The location of TP₂ is caudal and posterior to the free border of the upper trapezius.

Lower Trapezius Fibers

TP₃ (Fig. 6.2). This lower trapezius TP refers pain severely to the high cervical region of the paraspinal muscles, to the adjacent mastoid area and to the acromion (Fig. 6.2).[57] It also refers an annoying deep ache and diffuse tenderness over the su-

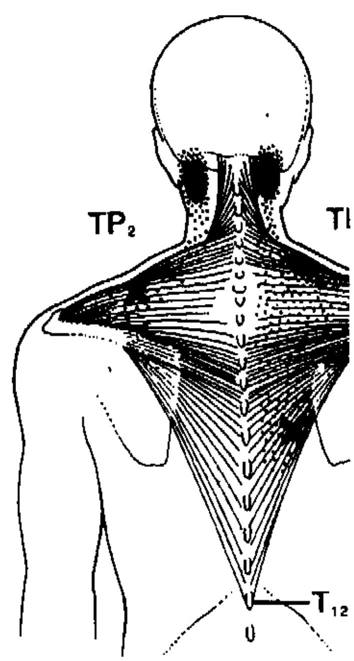


Figure 6.2. Referred pain patterns (×s) of trigger point 2 in the left upper of trigger point 3 in the right lower trap tions are as in Figure 6.1).

prascapular region.[67] This tend scribed by the patient as a "so the patient tends to rub the te Such referred diffuse tender not be mistaken for the focal t a TP. However, TP₁ and TP₂ velop as satellites within this and tenderness referred from secondary TPs can be disting simple referred tenderness b pable bands, local twitch sharply localized spot tender tion of referred pain, and by s tion of neck rotation to the op

TP₄ (Fig. 6.3). This TP produ burning pain referred down and medial to, the vertebral b scapula.

Middle Trapezius Fibers

TP₅ (Fig. 6.3). From this TI burning pain is referred media the TP and the spinous proces and T₁ vertebrae.

TP₆ (Fig. 6.4). A trapeziu found near the acromion r iscle

postural dizziness
ven more incapaci-
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toid attachment of the sternocleidomastoid. They reported inducing referred head pain by applying digital pressure to these tender muscles and by injecting hypertonic salt solution into them, location unspecified.

Sternal Division
(Fig. 7.1*A*)

An active TP in the *lower* end of the sternal division refers pain downward over the upper portion of the sternum (Fig. 7.1*A*). This is the only downward reference of pain from this muscle.[49,53] True trigeminal facial neuralgia is not accompanied by sternal pain, which, when also present, suggests the sternocleidomastoid myofascial syndrome.

When an unusual TP is activated in the lowest part of the sternal division, where that division may merge with a slip of the inconstant sternalis muscle, the TP is associated with a paroxysmal dry cough that can be precipitated by mechanical stimulation of the TP.

At the *mid-level* of the sternal division, TPs 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.[61] Pain may be referred on the same side to the external auditory canal.[46,62] The quality of the pain is described by patients to be aching as in the deep pain defined by Kellgren.[23] The TPs along the inner margin at the mid-level of this division refer pain to the pharynx and to the back of the tongue during swallowing,[5] which causes "sore throat," and to a small round area at the tip of the chin.[53] Marbach [28] shows a similar pattern that includes the cheek, temporomandibular joint and mastoid areas.

In the *upper end* of the sternal division, TPs 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.

Autonomic concomitants of TPs in the sternal division relate to the homolateral eye and nose.[49,53] 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 caused by increased excitability of motor units within the reference zone of sternal division TPs. The patient may have to tilt the head backward to look up, because of inability to raise the upper eyelid. Visual disturbances include not only blurring of vision,[47,49] but also dimming of perceived

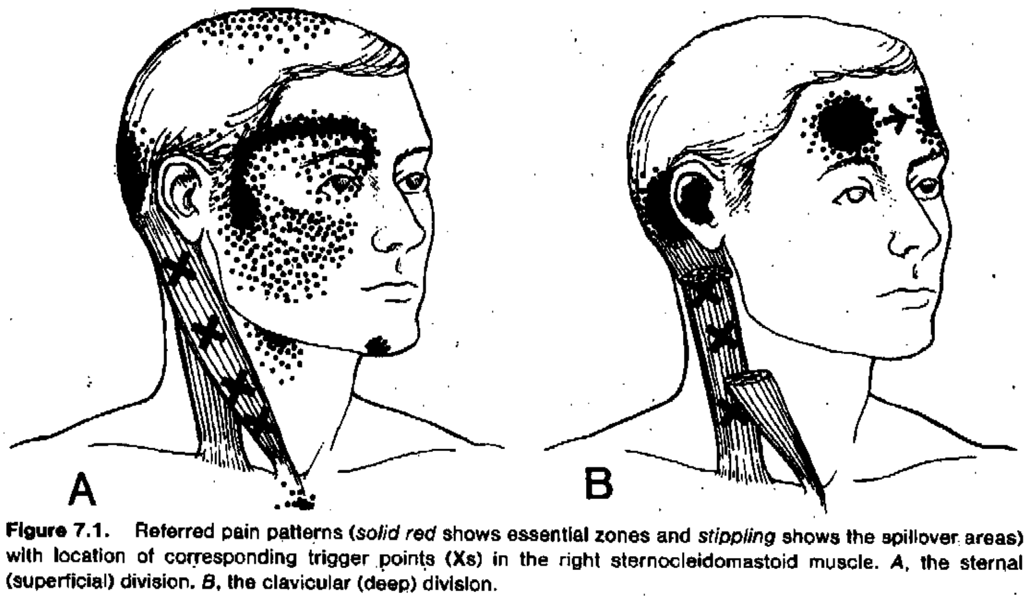

Figure 7.1. Referred pain patterns (*solid red* shows essential zones and *stippling* shows the spillover areas) with location of corresponding trigger points (Xs) in the right sternocleidomastoid muscle. *A*, the sternal (superficial) division. *B*, the clavicular (deep) division.

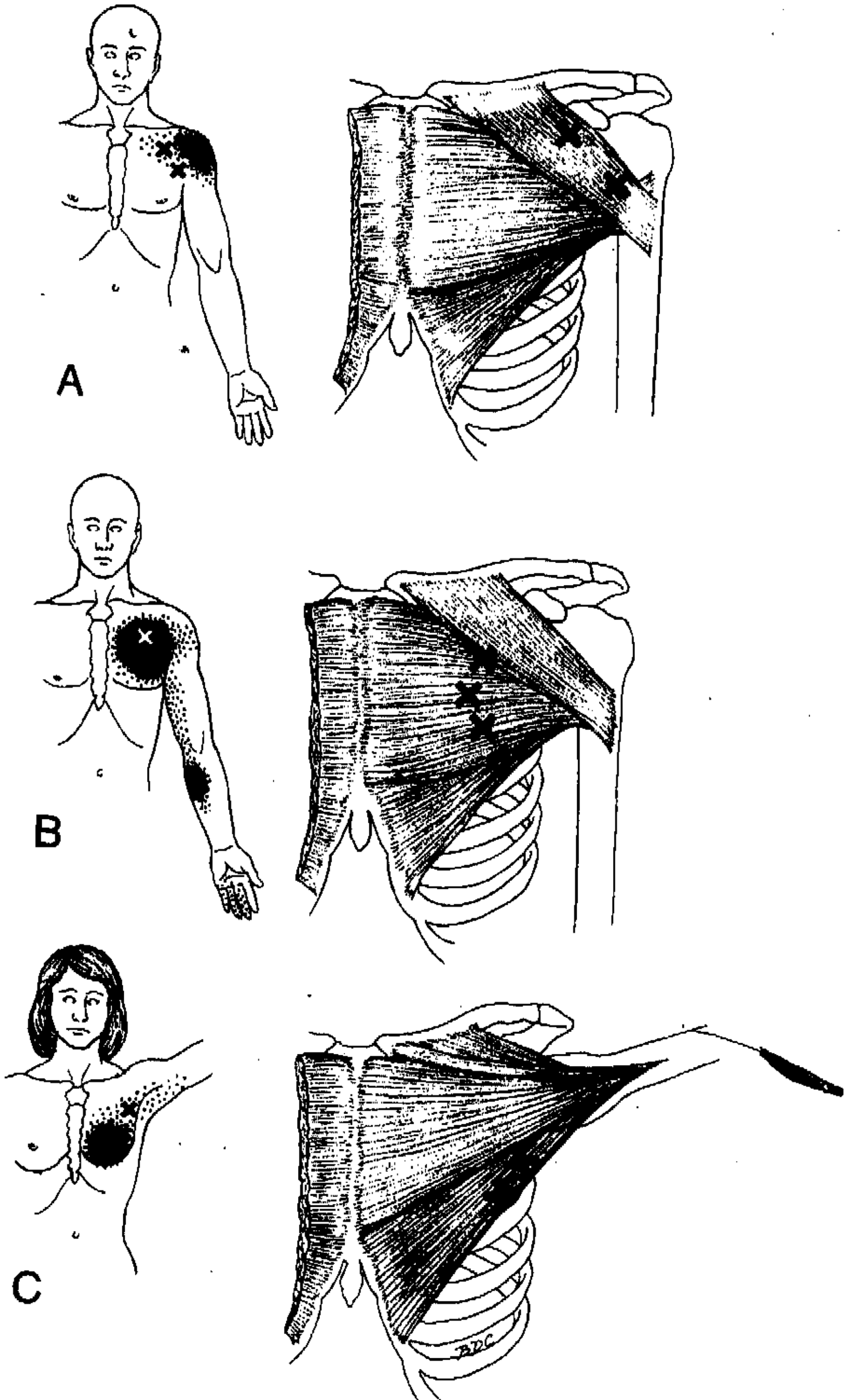

Figure 42.1. Referred pain patterns (*red*) and trigger points (×s) in the left pectoralis major muscle. *Solid red* shows essential areas of referred pain, and *stippled red* shows the spillover pain areas. *A*, the clavicular section. *B*, the intermediate sternal section. *C*, the lateral free margin of the muscle, which includes fibers of the costal and abdominal sections that form the anterior axillary fold.

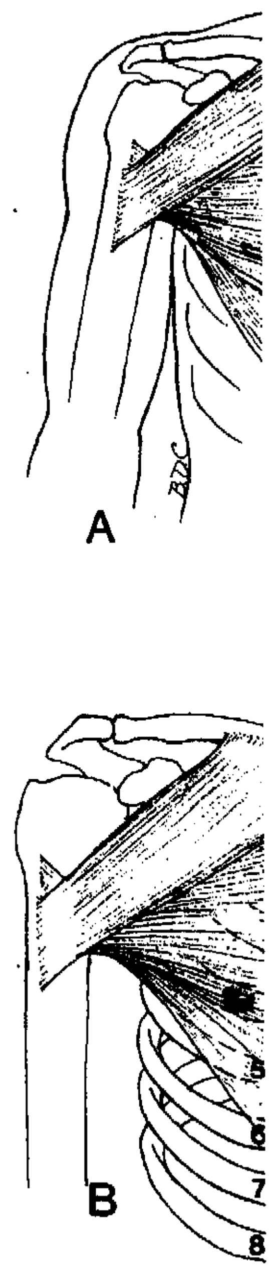

Figure 42.2. Right pecto
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ıry effort, as by coughing or :hing. **TRIGGER POINT EX-** res strong protraction and ab- ıpula to uncover the TPs and sible to palpation against the **ND SPRAY** require the seated vard and drop the head, hump ale deeply as the vapocoolant caudally along the muscle ompression is helpful. For **IN-** **'RETCH** the needle is directed ls pinned down against a rib. **:TIONS** include learning ab- ı and the home application of ısion.

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e posterior border of the long head of the triceps [14, 15, 16] It often covers the gion, with an accent on the yle of the elbow, and oc- des the ulnar side of the and all of the little finger. pectoral region may occa- ful.

ıL ATTACHMENTS **45.3)**

posterior superior muscle o the dorsal midline fascia, ı T_2 or T_3, and ***below*** and : digitations, to the cranial second through the fifth

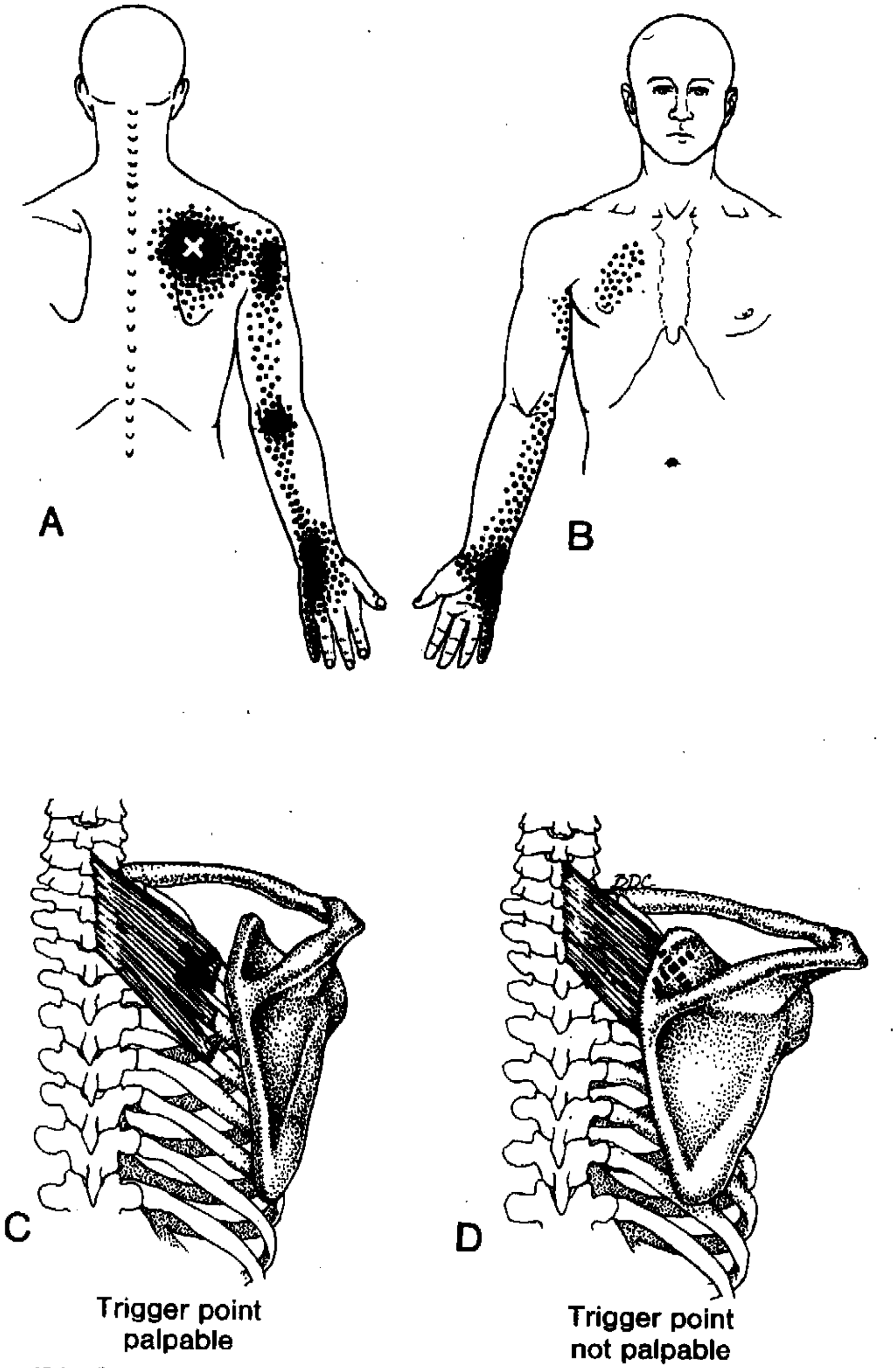

Figure 45.1. Referred pain pattern of a trigger point (×) in the right serratus posterior superior muscle. Essential pain is *solid red*, spillover pain is *stippled red*. *A*, back view of pain pattern. *B*, front view of pain pattern. *C*, scapula abducted, making the trigger point (×) accessible to palpation and injection. *D*, scapula in the normal rest position, and the trigger point (*dashed* ×) *inaccessible*.

VOLUME 2

Myofascial Pain and Dysfunction

The Trigger Point Manual

THE LOWER EXTREMITIES

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Illustrations by Barbara D. Cummings

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PART 1

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

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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.[266,277] Leriche[165] 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,

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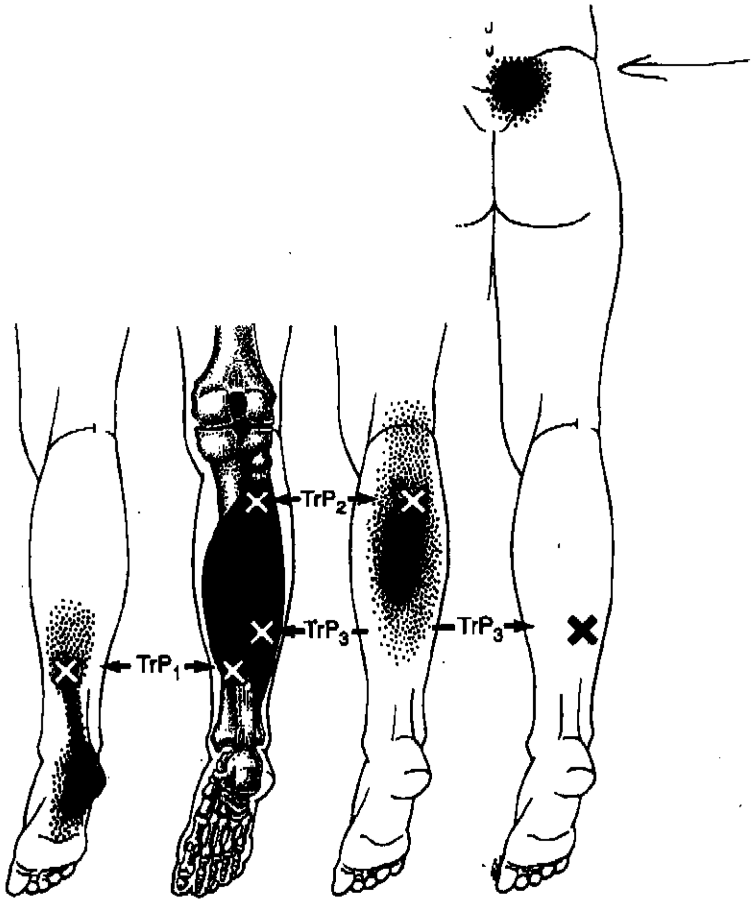

Figure 22.1. Pain patterns (*dark red*) referred from trigger points (*Xs*) commonly observed in the right soleus muscle (*light red*). The essential pain pattern (*solid red*) denotes the pain experienced by nearly everyone in whom these trigger points are active. *Red stippling* indicates the occasional spillover pain pattern. The most distal trigger point, TrP_1, causes heel pain and tenderness. The most proximal trigger point, TrP_2, is associated with calf pain (but not with nocturnal calf cramps). An intermediate and less common trigger point, TrP_3, slightly proximal and lateral to TrP_1, refers pain mainly to the region of the ipsilateral sacroiliac joint.

The very rare soleus TrP_3 (Fig. 22.1) is slightly more proximal and more lateral than TrP_1 and refers deep pain in the ipsilateral sacroiliac joint in an area about 2.5 cm (1 in) in diameter.[135] Less frequently, this TrP_3 may cause less intense spillover pain in the region of the TrP itself and over the posterior and plantar surfaces of the heel, mimicking the pattern of TrP_1.

An exceptional pain pattern referred to the jaw from the TrP_3 region has been observed twice (Fig. 22.2). In one patient, this TrP referred severe pain to the ipsilateral face deep in the jaws and temporomandibular joint with malocclusion ("Now my teeth don't meet," she said) whenever the ankle on that side was actively or passively dorsiflexed, but with no pain that is usually characteristic of the soleus muscle. The jaw pain and spasm were eliminated immediately by injecting soleus TrP_3. Occasionally, one sees such totally unexpected patterns of pain referred from TrPs in other muscles, which emphasizes the importance of obtaining a detailed and comprehensive pain history.

Other authors reported that TrPs in the soleus muscle cause heel pain[6] or pain in the heel and sole of the foot.[7]

The TrPs in the soleus muscle do *not* cause calf cramps, as do TrPs in the gastrocnemius muscle.

Plantaris
(Fig. 22.3)

Trigger points in the plantaris muscle (Fig. 22.3) refer pain behind the knee and downward over the calf as far as the midleg level. In some patients, a TrP in the vicinity of the plantaris refers pain to the ball of the foot and base of the big toe. However, it is not clear whether this pain arises from TrPs in the plantaris muscle

PART 3

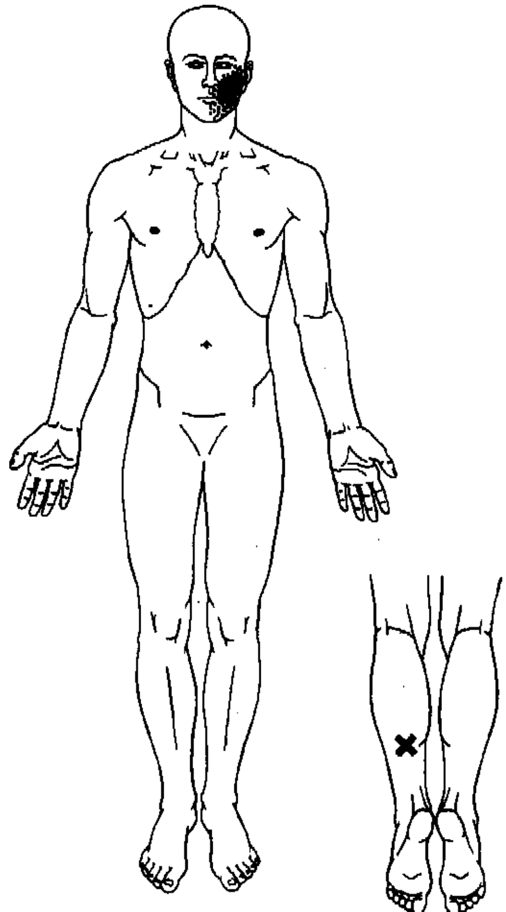

Figure 22.2. Exceptional pain pattern (*red*) referred to the left face and jaw from a rare trigger point (*X*) in the ipsilateral (left) soleus muscle.

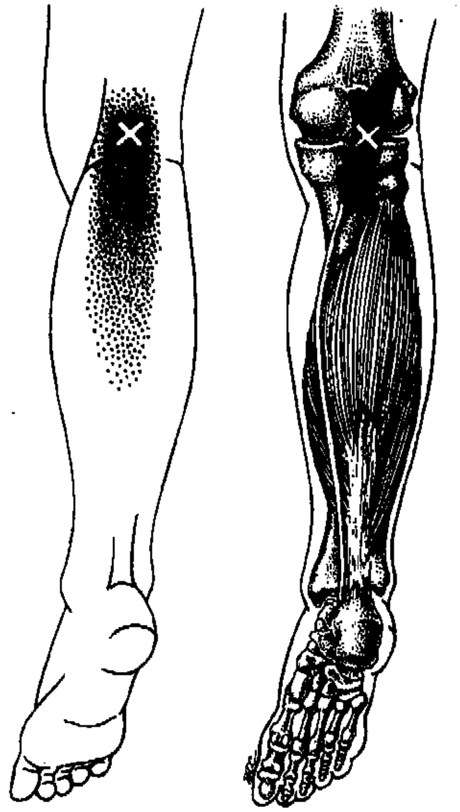

Figure 22.3. Pain pattern (*bright red*) referred from a trigger point (*X*) in the right plantaris muscle (*dark red*). The pain pattern back of the knee and usually extending down to midcalf locates the superficial diffuse pain experienced when this trigger point is active.

or in the fibers of the lateral head of the gastrocnemius.

2. ANATOMICAL ATTACHMENTS AND CONSIDERATIONS
(Figs. 22.4–22.7)

Soleus
(Figs. 22.4–22.7)

The soleus muscle crosses only the ankle joint region and not the knee joint, unlike the gastrocnemius. The soleus acts across the talocrural ('ankle') and the talocalcaneal (subtalar) joints.

The soleus muscle attaches ***proximally*** to the posterior surface of the head of the fibula and along the proximal third of the posterior surface of that bone (Fig. 22.4), to the middle third of the medial border of the tibia, and to the tendinous arch (Figs. 22.5 and 22.6) between the proximal tibia and fibula. This arch forms the roof of the soleus canal. The canal encloses the posterior tibial artery, veins, and tibial nerve. It is unusual that a tendinous arch for nerve and vessels should serve as a major attachment site for a muscle. ***Distally***, the soleus fibers attach to the underside of the aponeurosis that also provides an anchor for the gastrocnemius muscle. This aponeurosis forms the tendo calcaneus (Achilles tendon) that attaches to the posterior part of the calcaneus.

The soleus muscle is enclosed between two layers of unyielding fascia: the aponeurosis of the Achilles tendon superficially and a layer of tough fascia deep to the soleus, which is distinct from the thinner fascia that covers the deep poste-

PART 3

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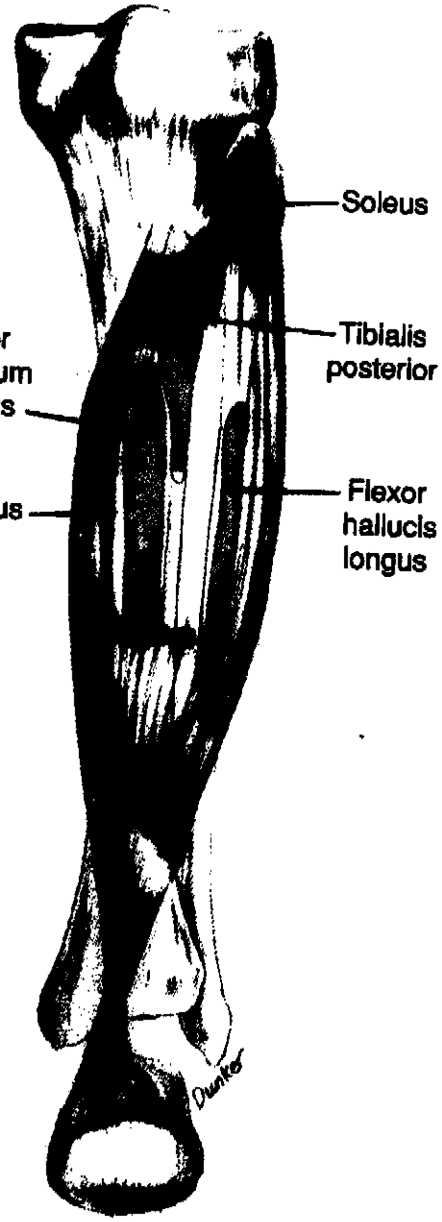

Figure 22.7. Attachment of the soleus portion of the right Achilles tendon to the os calcis, posterior view. Note the tendon's rotation of 90° and attachment to the medial one-third of the calcaneus. The gastrocnemius portion of the tendon (not shown) attaches to the lateral two-thirds of the os calcis. [Reproduced with permission.[98]]

and the vastus lateralis about 50%. The percentage of type 1 fibers in the soleus muscle was higher in six athletic young adult men (79%) than in six comparable women (67%).[140]

Elder and associates[37] found that the variability in the distribution of fiber types was so large that five sites must be sampled from the same muscle to keep the standard deviation below 5%.

Weber[137] reported that the muscle fibers of the soleus muscle weighed 335 g, about one-fourth the weight of the gluteus maximus muscle and nearly the same weight as the gastrocnemius muscle. The average fiber length of both the soleus and gastrocnemius muscles was quite short, 3.7 and 3.5 cm (1.5 in).

Plantaris (Fig. 22.4)

Because it attaches proximally beside the lateral head of the gastrocnemius muscle, the plantaris can be thought of as an accessory lateral head of that muscle. The

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Supplemental References

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