The Concise Book of Dry Needling

A Practitioner's Guide to Myofascial Trigger Point Applications

Exam Edition

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Preface

This text concerns the treatment of myofascial trigger points through the exclusive use of *dry needling*, a term coined by Dr. Janet Travell in the famous "big red books" (Simons, Travell, and Simons 1999, pp. 154–155). Dry needling (with the use of a fine filament needle) is the ideal tool for therapists, of every stripe, involved in treating myofascial trigger points in adults, provided it falls within their scope of practice.

The book is written in a concise manner as a "quick-to-hand" reference guide regarding key issues around safe, effective, and appropriate dry needling. It is intended to be the ideal accompaniment to course notes and the perfect in-office tableside reference guide. Accurate and essential criteria are provided for the identification and subsequent treatment of myofascial trigger points through the exclusive use of a fine, filiform needle. Skilled palpation, supported by the ability to visualize and observe anatomical landmarks, is essential in order to avoid neurovascular and other vital structures that could result in insult, injury, or additional pain.

A description of the origin, etiology, and pathophysiology of the myofascial trigger point is offered. Indications and contraindications for myofascial trigger point dry needling are noted, and standards and guidelines are presented. Images concerning correct needle application/insertion are supplied for many muscles in the body, while guidelines are provided for muscles that are in

close proximity to the ones chosen for inclusion in the photographs. Two bespoke myofascial trigger point hypotheses concerning the mechanism of myofascial trigger points, along with the rationale for the success of dry needling, are also presented for further consideration and research.

This text does not deal with acupuncture or the many refined techniques and diverse approaches of that discipline; it focuses solely on the use of dry needling for the exclusive treatment of myofascial trigger points. The book is not a substitute for understanding the neurophysiological mechanisms of local or referred pain, or for completing an appropriate course of study in dry needling. Such a course should be delivered by a recognized, competent training provider who understands their responsibility to learners, which includes legal, ethical, and professional knowledge.

This book is intended for use by suitably and adequately qualified therapists as a quick-reference tool. This book is not about personal opinions but is about best practice.

Special Note

Images of dry needling application have been provided throughout this book for the reader's convenience. Images are not necessary for all muscles, especially for muscles in the forearm, as the needling procedures are so similar. The guidelines for needle application may be much the same for a muscle within close proximity of the ones chosen for image capture. Care is therefore required when changing the angle or needle direction, keeping in mind the need to ensure appropriate depth while recognizing anatomical location. For the purpose of clarity, in a selected number of the photographs a delivery tube or black line replaces the needle in order to more clearly show the needle application.

Do not attempt to offer dry needling unless you have received appropriate training from a recognized training provider. You do not need to have received training, however, to enjoy the book or to learn about myofascial trigger points. Please share this book with other therapists, medical practitioners, movement therapists, and others.

Introduction

No One Left to Lie to

Pain is a liar; however, research has changed the way we think about pain (Moseley 2012). Pain is a child of the brain—to fully understand it we must meet the entire pain family. Peripheral tissues are close relations and could be viewed as brothers and sisters, while tissues such as muscle fibers and sarcomeres would be first cousins. Mechanoreceptors, proprioceptors, and nociceptors might be the irritated family members, constantly taking information to and from mom and dad concerning irritating older siblings. These various members of the family can have a tendency to exaggerate or distort the truth. For example, mom and dad can overreact, underreact, or misread the situation, dishing out a response that is disproportionate and not appropriate to the reality; this is ultimately referred to as *allodynia*.

Chronic pain states are defined by significant changes in neuronal activity; such changes are profoundly influential in pain matrix mechanisms. Neuroplastic changes occur in the spinal cord, thalamic nuclei, cortex, and limbic system, and can alter pain thresholds, degree of sensitivity to pain, and the overall pain experiences of our patients (Woolf 2010).

Research by Staud (2011) describes spinal segmental sensitization (SSS) as being caused by heightened dorsal horn activity, brought about by constant bombardment of nociceptor impulses from the periphery (due to damaged or sensitized somatic or visceral tissues). Clinical experience of thousands of practicing therapists across the globe identifies pain referral patterns that cannot be of nerve origin. Travell and Simons (1992) reported myofascial trigger points within the soleus muscle that refer deep pain to the ipsilateral sacroiliac joint. Additional myofascial trigger points in the soleus refer exceptional pain to the face and jaw. Some mechanism or mechanisms other than nerve pathways must be at play in such situations, as nerves exclusively refer pain inferiorly (the face is the exception).

Far too few therapists and medical doctors are aware of the perpetuating role of myofascial trigger points as a combining source of sensory bombardment (Shah and Gilliams 2008), with the possible result of chronic pain in various guises. Myofascial pain, according to Fogelman and Kent (2015), is an "eminently treatable condition" yet almost "universally underdiagnosed by physicians and undertreated by physical therapy modalities." Constant noxious bombardment of the dorsal horn neuron causes a release of glutamate and substance P at the segmental level. By binding to their respective receptors on post-synaptic neurons, these chemicals induce sensitization of wide dynamic range (WDR) neurons, thus further sensitizing adjacent spinal segments. The sustained release of glutamate and substance P leads to apoptosis (programmed cell death) of inhibitory neurons. This perturbation leads to a sustained sensitized state, which in turn lowers neuronal pain thresholds, activates previously inactive synapses (expansion of the receptive field of pain), and leads to allodynia and hyperalgesia (Shah and Gilliams 2008).

Central sensitization that is maintained by myofascial trigger points and other peripheral sources can be reversed over time. Myofascial trigger point dry needling has been shown to be effective in that regard and therefore a worthwhile therapeutic intervention (Srbely et al. 2010). A more recent study measuring the concentrations of a diversity of biochemicals—including β-endorphin, substance P, tumor necrosis factor-α, cyclo-oxygenase-2, hypoxia-inducible factor 1-alpha, inducible nitric oxide synthase, and vascular endothelial growth factor—found that dry needling of trigger points modulates the concentrations of these noxious chemicals in a dosage-dependent manner (Hsieh et al. 2011).

Pain—Present, Past, Future

Pain can have a crepuscular aspect to it. Time can also be a healer. However, chronic myofascial pain patients know all too well the feeling of misery and despair when neither time nor medication reduces the untiring relentlessness of pain or changes in sensation.

With its clear images concerning the correct use of needles, needle placement, and needle direction, this book will help ensure safe, effective, and appropriate clinical applications of myofascial trigger point dry needling, eradicating pain in the present and into the future; pain will become a past and distant memory, or be reduced to manageable levels.

A paper in 1979 by Karl Lewit investigating the needle effect in the relief of myofascial pain reported that "dry needling is highly effective in the therapy of chronic myofascial pain. Immediate analgesia without hyperesthesia (the needle effect) can be produced by needling precisely the most painful spot."

Note: When I refer to *pain*, this is taken to mean "pain and changes in sensations."

"X" Does Not Mark the Spot

The reader will notice that the artwork in this book has no "X" to mark the position of the myofascial trigger point. This is because it is not appropriate to place an X in a specific position to identify the location of the myofascial trigger point. The point of view that the X represents a common location specific to a particular muscle is, in my opinion, a flawed argument, as thousands of therapists worldwide working daily with myofascial trigger points will attest.

Using an anatomical image with an X to identify the location of the myofascial trigger point is a poor substitute for excellent palpation skills. Such skills are essential for identifying the myofascial trigger point(s) that could be located *anywhere* in the hundreds of thousands of myofibrils in any one muscle. Appropriate palpation skills and knowledge of the cardinal signs are used to seek out the accompanying tense bands and nodules associated with myofascial trigger points, which are housed in the microscopic sarcomeres.

Biotensegrity and Dry Needling

My work in anatomy, physiology, and bodywork therapy has been significantly influenced by my mentor Stephen Levin M.D., an orthopedic surgeon who coined the term *biotensegrity*. Dr. Levin was the first to promote the biotensegrity model as the new biomechanics for all biological structures. I describe biotensegrity as "anatomy for the 21st century."

Biotensegrity has emerged as the most significant development in human anatomy in recent years, with important ramifications for a wide range of medical practitioners, including surgeons, bio-engineers, and human movement specialists. Bespoke Thiel soft-fix dissection techniques are providing a new vision and understanding of the continuity of the human form. A fresh look at human fascia highlights its role in providing continuous tension throughout its network.

The term *tensegrity* was coined by Buckminster Fuller by combining the words "tension" and "integrity." Fuller's student Kenneth Snelson built the first floating compression structure of tensegrity in 1949, while Dr. Stephen Levin was the pioneer of "biotensegrity," which was born out of his publications on the topic in the early 1970s. As a clinical anatomist, I have investigated this model and the role of fascia in my dissections in order to better understand the mechanisms of human movement and chronic pain, while providing new anatomical knowledge and awareness, leading to less invasive surgical and non-surgical therapeutic interventions.

Borrowing from Fuller's term *tensegrity*, Levin added the prefix *bio*, which refers to all living structures. Biotensegrity is the application of Fuller's tensegrity concepts to biological structure and physiology. In the biotensegrity model the limbs are not a collection of rigid body segments: the upper and lower limbs are semi-rigid, nonlinear, viscoelastic bony segments. These segments are interconnected by nonlinear, viscoelastic connectors, including cartilage, joint capsules, and ligaments, and have an integrated nonlinear, viscoelastic active motor system—the muscles, tendons, and fascia (connective tissue).

Biotensegrity counters the notion that the skeleton provides a frame for the soft tissues to hang upon; instead, biotensegrity structures are integrated, pre-tensioned (self-tensioned), continuous myofascial networks with floating discontinuous compression struts (the skeleton) contained within them. A column whose center of gravity is constantly changing while its base is rapidly moving horizontally would require forces too great to consider. The forces become incalculable if the column comprises several rigid bodies, hinged together by flexible, virtually frictionless, joints.

Daniele-Claude Martin, a pioneer in the world of biotensegrity and a member of the Biotensegrity Interest Group (B.I.G.), co-authored a chapter with Dr. Levin in the excellent book *Fascia: The Tensional Network of the Human Body* (Schleip et al. 2012). The title of the chapter was "Myofascia as the tensioner in the biotensegrity model," and Levin and Martin made the following important points regarding tension:

"Central to this concept is the understanding that the fascia imparts a continuous tension to the system. Fascia displays the nonlinearity characteristic of all biologic tissues. In nonlinear tissues, the stress/strain relationship never reaches zero (a characteristic of linear materials); and there is always tension inherent in the system. It gives the 'continuous tension,' an essential component of tensegrity, that helps set the tone of the organism. There are active contractile elements in fascia (Schleip et al. 2012) and the fascial network is intimately bound to muscle (Passerieux et al. 2007). Muscle also has intrinsic 'tone' and is never completely lax, and the entire fascial network is continually tensed, by both intrinsic tension and active contractions that can be 'tuned.'"

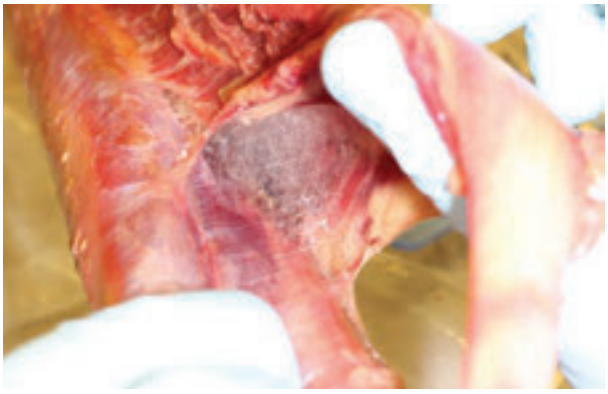
This concept of tuning the fascia blends well with the fascial response to needles, as described by the neuroendocrinologist Helene Langevin. In her 2006 paper, entitled "Connective tissue: A body-wide signaling network?", Langevin detected a mechanical response whereby the connective tissue wrapped itself around the needle, with a resulting electrical signal being transmitted to the surrounding connective tissue

cells via mechanotransduction. The change in tissue tension is obvious as the connective tissue swirls around the needle, creating a redistribution of tension and compression (biotensegrity).

The Cellular Level

At the cellular level, using fluoroscopic imaging Guimberteau et al. (2010) provided strong visual evidence that fascia contains a water-filled vacuolar system that is capable of sliding (I suggest "gliding") independently of the rate of contraction of muscle. In turn, it is capable of facilitating and supporting capillaries throughout the fascia. Sharkey (2015) provided fresh-frozen cadaver imagesof the fascia profundus at the macro level which reflect this fractal microvacuolar structure while revealing an icosahedron-like (tensegrity) composition in which fractal elements inter-relate, creating a body-wide framework or network.

This structure is able to change or maintain shape and form within a fluid base, allowing deformation and a subsequent return to its original state while maintaining volume. This creates the stable, yet flexible, environment necessary for fascia to act as a medium for force transmission (Huijing 2009).



A stretching force applied to the tissues of the anterior forearm, on a fresh-frozen cadaveric specimen highlights the fractal, chaotic arrangement of the deep fascia. (Photo by J. Sharkey 2010)

This new model for biological structures that is based on the concept of biotensegrity identifies fascia as the tensional, continuous member. In a tensegrity model, continuous tensile forces (from the myofascial tissues) provide an "ocean" within which the struts float (in the human body, the "struts" are the bones, which are continuous, as they are fascia, yet virtually separated and do not directly transmit compression forces to each other). The tensional members are continuous and directly distribute their tensional load to all other tensional members, as described by Fuller in 1961.

This amazing image illustrates the omnidirectional tensional network that is fascia. Even the muscle fibers are a specialized form of fascia. The absence of one precise vector allows maximum adaptation of the structure in a continuous evolution of balance through tensional and compressional forces. This is the fascial chaos of which my colleague Jean Claude Guimberteau speaks so eloquently. (Photo by J. Sharkey 2010)

The fascial oceans become seas, lakes, rivers, streams, and brooks; skin and bone represent opposite seashores. Newtonian, Hookian, and linear mechanical properties are the basis for the building of all things non-biological (Levin 1995). This description supports the more recently accepted image of a continuous tissue, ubiquitous in nature, connecting left to right, front to back, and top to bottom, embracing and permeating the entire body. Mesenchymal-derived connective tissues provide a body-wide network of communication (Schleip and Muller 2013).

This powerful image of the superficial fascia, removed as one continuous structure, allows the dry needling myofascial trigger point therapist to imagine the fascial continuity and ubiquitous nature of this all-embracing tissue. (Photo by J. Sharkey 2010)

Myofascial trigger point dry needling can furnish a mechanism to restore the fascial tone, so vital to the biotensegrity, providing a return to homeostasis, and thereby normalizing the tissues associated with, and connecting to, the myofascial trigger point. In fact, the muscle fiber within which is housed the myofascial trigger point is itself a specialization of fascia. Muscle fibers are part of a continuum of specialty that includes all the tissues of what Stephen Levin and Graham Scarr have called the *mesokinetic system*.

Setting the Scene

As a clinical anatomist, I cannot overstate the need for an excellent understanding of anatomy, including surface, topographical, and gross anatomy. Taking time to become accustomed to the various lengths of the needles, coupled with the ability to visualize the needle once it has been placed in the tissue, will prove to be a vital skill. With an excellent knowledge of anatomy, I suggest it is virtually impossible to cause harm or injury to a patient. With a poor knowledge of anatomy, on the other hand, it will be only a matter of time before harm and injury comes to the patient. Know your anatomy.

As an exercise physiologist, I struggled with Travell and Simon's proposal that common myofascial trigger points can have their location identified by placing an "X" over specific locations of a given muscle. For example, in Volume 1 of the famous red books (Simons, Travell, and Simons 1999, p. 331), Barbara Cummings' images of the masseter muscle include several chunky Xs marking the positions of specific myofascial trigger points. The size of each X is such that if one superimposes all the images, the Xs cover the entire muscle. One X alone is covering tens of thousands of superficial to deep fibers.

In my daily work I speak to therapists who tell me that they could not find a myofascial trigger point in the middle of the most vertical fibers of the upper part of the trapezius muscle, as described on p. 279 of Simons, Travell, and Simons (1999). With appropriate palpation skills and the reassurance that the myofascial trigger point can form anywhere within the muscle, these same therapists further investigate and report they found the culprit distal, lateral, or superior to the location identified by the "X."

Using the Thiel soft-fix method of cadaveric dissection, I have demonstrated what I call *muscle islands*. Muscle islands are small, isolated but regular, patches of muscle fibers or long thin cords of muscle fibers found on, or running in series with, muscle tendons. These are, I believe, why therapists find and can irritate what are termed attachment trigger points. In fact, muscle islands can be found almost anywhere, especially in the subcutis, and represent what some researchers may call a panniculus effect. Dissection provides a very different reality of anatomy—one that is not as uniform or as ordered as one sees in the classical anatomy textbooks.

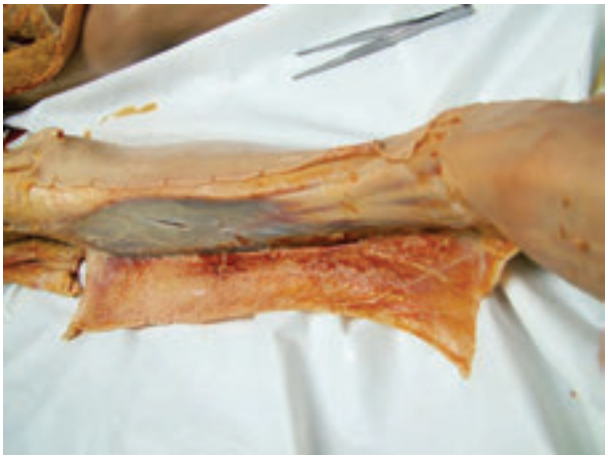


Image of a lower limb (anterior view) with the skin reflected, showing isolated muscles fibers just beneath the skin, or "muscle islands." (Photo by J. Sharkey 2015)

Why Consider Myofascial Trigger Point Dry Needling in the Treatment of Chronic Myofascial Pain?

Myofascial pain arises from muscle and its connective tissue (Shah and Heimur 2012). According to Simons, Travell, and Simons (1999), and supported by numerous researchers over the preceding years (Mense 2010), myofascial trigger points are responsible for, or play a role in, as much as 85% of musculoskeletal pain. Myofascial trigger points constitute commonly overlooked or ignored causes of common musculoskeletal pain conditions, chronic or acute. My colleague and researcher Jay Shah has demonstrated that active myofascial trigger points have a noxious biochemical milieu—including substance P, bradykinins, and other substances—which is at the root of the pain. Many drug-based therapies have in fact been demonstrated to be no better than a placebo.

A double-blind controlled dry needling study by Mayoral et al. (2013) demonstrated that the treatment of myofascial trigger points was superior to a placebo. In clinical practice, pain management or the eradication of pain is the primary focus for many patients and health care practitioners. It is worth noting that changes in sensation such as a constant itch, numbness, tingling, burning, crawling, or feelings of water running on the skin are all components across the spectrum of pain. These are real sensations that patients feel on an on-going daily basis—for some, twenty-four hours a day, every day. Not necessarily a pain per se, a change in sensation is rather a variation on the theme of pain.

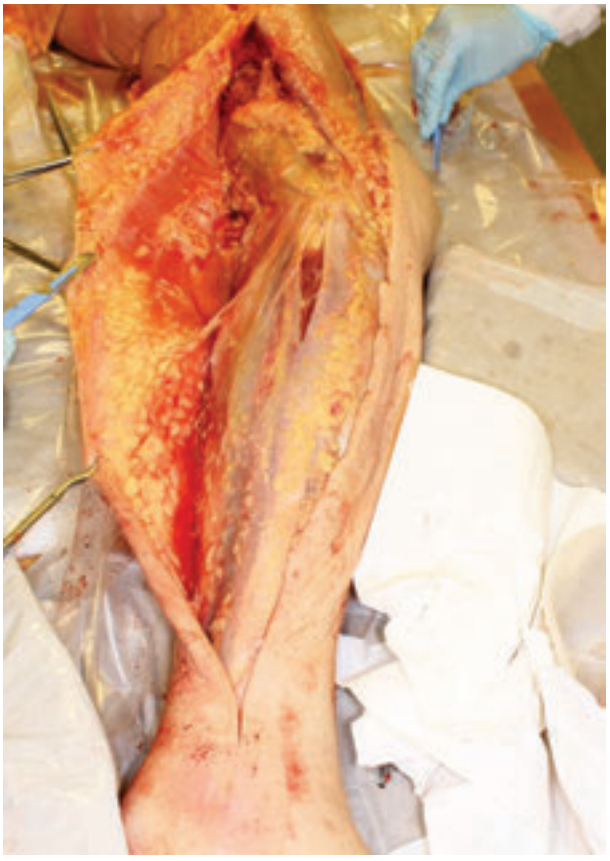
A pain experienced radiating down the anterior upper limb and terminating in the wrist and palm connotes a brachial nerve insult. When all avenues of traditional medical assessment have been exhausted without identifying any underlying pathophysiological cause, or etiology, then soft tissue myofascial trigger points must be considered. Myofascial trigger points can mimic or play significant contributing roles in migraines, cervicogenic headaches, frozen shoulder and associated pain issues, carpal or tarsal tunnel syndromes, frozen or lower back pain, sciatic pain, radiculopathies, knee and ankle pain, and a host of other conditions. Put simply, myofascial trigger points can mimic anything. For those patients who have "tried everything" with little or no therapeutic benefit, myofascial trigger point dry needling is worth considering.

Can Acupuncturists Perform Myofascial Trigger Point Dry Needling?

Although acupuncture practitioners already possess excellent needle-handling skills, they will additionally require knowledge of the pathophysiology, etiology, and pain referral patterns of myofascial trigger points. They will also need to develop the palpation skills required to feel for and find and locate myofascial trigger points in order to ensure accuracy during dry needling application. Myofascial trigger points are not acupuncture points, tender points, or ah shi points, and can form anywhere in the millions of muscle fibers throughout the body. Overlapping acupuncture points with myofascial trigger points and then removing all but the points that coincide would obviously lead the uninitiated to see a corresponding relationship.

Central Versus Attachment Trigger Points

As a clinical anatomist, I have investigated the idea of attachment trigger points and have arrived at the following proposal/suggestion. During dissection it is obvious that small clusters of isolated muscle fibers are found lying on the body of a tendon or on the undersurface of the skin throughout the body. These muscle islands can by their very nature develop myofascial trigger points, thus leading to the notion of *attachment trigger points*.

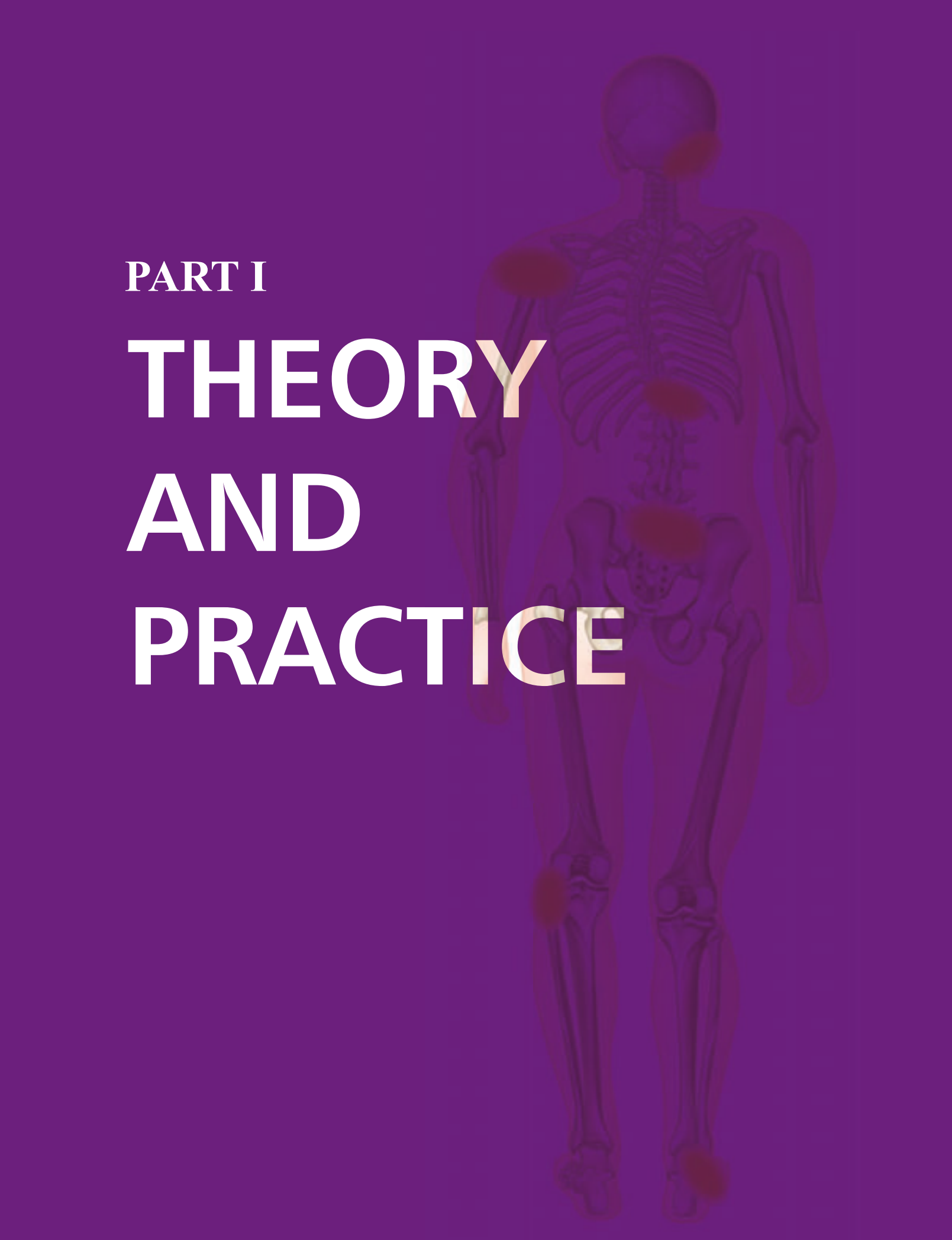


The red cylindrical protein we all know as muscle fibers can be found as muscle islands beneath the skin or on the outer surface of a tendon, and can house myofascial trigger points. (Image by J. Sharkey 2010)

Recommendation: treat myofascial trigger points within the gasters of the muscles and then re-examine the attachment trigger points to see if they have dissipated after the primary source has been treated. Attachment trigger points are most often the offspring of the primary parent trigger points.

Myocardial Myofascial Trigger Points

Much research in this field still needs to be done. For example, no research currently exists concerning the possible existence and consequence of myocardial myofascial trigger points. I therefore call for and encourage research on this topic. I suspect that many people, convinced they have had or are having a coronary episode, suffer unnecessary stress/strain, when in fact they may be experiencing a myocardial myofascial trigger point. As the heart is a striated muscle, it seems logical that it conceivably harbors myofascial trigger points, and I would welcome research into this uncharted territory.



PART I

THEORY AND PRACTICE

1 Genesis of the Myofascial Trigger Point

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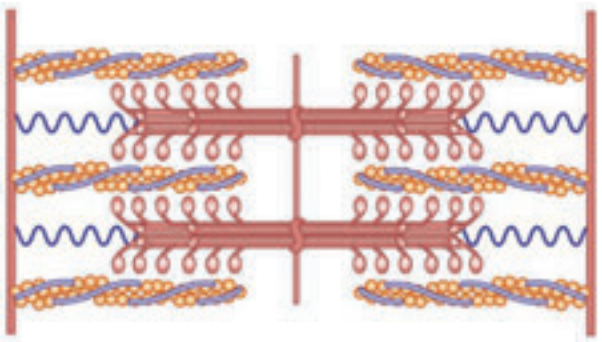
Myofascial trigger points are hyperirritable localized spots found in taut bands within the muscle sarcomeres (Simons, Travell, and Simons 1999). The hollow sarcoplasmic reticulum (SR) functions to store calcium ions that are constantly being pumped into it from the cytoplasm of the cell. When muscle fibers are not contracted, a high concentration of calcium is located in the sarcoplasmic reticulum, and low concentrations exist within the sarcoplasm. Special calcium gates can remain closed, blocking calcium from escaping and moving into the sarcoplasm. When an impulse travels along the membrane of the sarcoplasmic reticulum, these calcium gates open and allow a flood of calcium ions to rush out of the sarcoplasmic reticulum and into the sarcoplasm of the sarcomere, where the myofilaments are located. This is a key step in the normal sequence leading to muscle contractions.

Myofibrils consist of three types of myofilaments: *myosin* the thick protein, *actin* the thin protein, and *titan* the sticky protein. These myofilaments are arranged in a very precise pattern. The thick myofilaments are surrounded by six thin spiraling myofilaments, while the titan proteins act as tails to anchor the myosin to the Z disk. In figure 1.1, the thin actin myofilaments can be seen above and below each thick myofilament; in reality, however, they spiral around the thick proteins in a snakelike fashion.

Within each sarcomere the myofilaments overlap, similarly to when the bristles of two brooms are pressed into each other. When viewed under a microscope, the ends of a sarcomere appear lighter than in the center; this is because the thick myofilaments are situated in the center, while the thin myofilaments are located toward the ends. The name *striated muscle* was used for this very reason. *I band* is the name given to the light areas, while the dark areas are called *A bands*. Near the center of the I band is a thin dark line known as the *Z line* or *Z disk*: the Z line is where sarcomeres come together and the thin myofilaments of adjacent sarcomeres overlap slightly. The thick myofilament *myosin* has a core with

heads that stick out like the head of a golf club (two heads actually); these are also referred to as *myosin cross bridges*. These bridges or heads have a number of important characteristics:

- Adenosine triphosphate (ATP) binding sites
- Actin binding sites
- A hinge that allows a swiveling action so that the head can move the thin proteins, resulting in a contraction



Sliding filament theory (this should be the gliding filament theory).

Note the spherical shape of the long chains of actin molecules (also called *G actins*). The thin protein actin is constructed of two chains (*H* and *G proteins*) spiraling around each other. A smaller associated protein called *tropomyosin* in turn coils around the actin, as shown. Yet another protein, *troponin*, attaches itself at specific intervals to the tropomyosin. As these proteins are electromagnetically attracted to each other, once the troponin moves it will in turn move the attached tropomyosin with it. Here is the important point: tropomyosin covers the binding sites on the actin, and when the tropomyosin is attracted away by the movement of those electrically charged proteins acting on the troponin, the sites become free for the cross heads (or bridges) of the electromagnetically charged thick myosin to attract and associate (glide). This is how a muscle contracts.

Contractions—Pulling It All Together

When muscles are working normally they require a nerve impulse; this is the very first step leading to a contraction. This nerve impulse will travel along the sarcolemma and into the T tubules. From there the nerve impulse will travel to the sarcoplasmic reticulum, resulting in the active opening of the calcium gates, allowing calcium to diffuse into the sarcomeres, where the myofilaments are located. Calcium now binds itself to the troponin molecule, altering the shape of the protein and causing it to move, thereby moving the attached tropomyosin. Now that the tropomyosin has moved, the myosin binding sites become free, permitting the myosin heads to attach to and pull the actin. As the heads contact the actin, the myosin cross bridges hinge and swivel, thus pulling the myofilament actin; this does not happen all at once, but in a way similar to a tug of war team pulling a rope. The pulling action occurs in a synchronized manner: some myosin heads attach while others disassociate—a collective effort that leads to a concentric contraction. Should the external force overcome the pulling action, or should a person consciously allow the muscle to be overcome, the result is a lengthening of the muscle while it is pulling on the myofilaments; this is known as an *eccentric contraction*. Remember, this means that muscles can only pull—they cannot push.

For muscles to work effectively, energy is required; this energy is supplied by the breakdown of ATP. As long as calcium remains in the presence of the myofilaments, the sarcomeres will remain shortened. Under normal circumstances, when the nerve impulse stops, the membrane of the sarcoplasmic reticulum is no longer permeable to calcium, and the calcium gates now act in reverse, allowing the calcium to escape from the sarcomere back into the sarcoplasmic reticulum. As the calcium disassociates from the troponin, it now pulls the tropomyosin back into its resting place covering the myosin binding sites. Tropomyosin then, once more, blocks the cross bridges from touching the thin actin protein, thus preventing a contraction from taking place.

From the above description of muscle contraction, the reader can appreciate that calcium is the "key" that turns on a contraction, or for that matter turns it off. If, for some reason, calcium ions cannot escape from the sarcomere (or, as proposed by Gerwin, Dommerholt, and Shah (2004), a damaged sarcoplasmic reticulum leads to a flood of calcium concentrations), then the myofilaments will remain shortened. A dysfunctional endplate activity occurs, commonly associated with a strain (resulting from, for example, unaccustomed

physical activity) or other soft tissue insult. Stored calcium is released at the site; acetylcholine (ACh) is released through calcium-charged gates at the synapse, leading to an abundant and constant presence of this neurotransmitter. More energy may be required in order to rectify this situation than to maintain it, and so the muscle fiber remains short, thereby increasing tension. This leads to a reduction in oxygen and increased anaerobic metabolism, resulting in abnormal depolarization of the post-junctional membrane of the motor endplates.

Resultant ischemia develops and creates an oxygen/nutrient deficit, accompanied by a local energy crisis. Energy (ATP) is needed in order to remove the excessive calcium. ATP availability is decreased by the ensuing tissue tightness, which in turn restricts local blood supply. The persistent high calcium levels maintain ACh release. A vicious cycle results.

ACh transmission causes the actin and myosin elements of myofibrils to glide into a shortened position, leading to the formation of contractures (involuntary, without action potential). More energy is required for removing excessive calcium than for sustaining a contracture, and so the contracture remains.

Contractures are sustained by the chemistry at the innervation site, not by action potentials; they are to be differentiated from contractions (voluntary with action potentials) and spasms (involuntary with action potentials). The actin/myosin filaments glide into a fully shortened position (a weakened state) in the immediate area around the motor endplate (positioned at the center of the fiber). As the sarcomeres shorten, a contracture *nodule* forms—a palpable characteristic of a trigger point. The remainder of the sarcomeres on either side of this nodule within that fiber are lengthened, thereby creating a palpable taut band—another common trigger point characteristic. Other characteristics are spot tenderness of a nodule in the taut band, and the patient's recognition of pain or sensation when pressure is applied to the tender nodule.

Additionally, there may be:

1. Visual/tactile/autonomic evidence of local twitch response (LTR)
2. Pain or altered sensation in the target zone associated with that trigger point when provoked
3. An EMG demonstration of spontaneous electrical activity (SEA) in the nidus (nucleus) of the trigger point
4. A painful limit to full stretch and reduced range of motion
5. A positive test of weakness of the muscle housing the trigger point
6. Changes in cutaneous humidity (dry or moist), temperature (cool or hot), or texture (rough)
7. A "jump sign" or exclamation by the patient because of extreme tenderness of palpated tissues

Myofascial trigger points are often associated with the feeling of ropy bands beneath the palpating fingers. Locating and identifying these bands requires excellent palpation skills and knowledge of the cardinal signs. Placing the muscle in a lengthened position may exaggerate the ropy bands and should make them more noticeable to "listening" fingers. Contraction knots can be small or large, depending on a variety of factors, such as the number of myofascial trigger points making up the contraction knots, the tissue consistencies, and the amount of fluid infiltration involved.

When a muscle is burdened with multiple myofascial trigger points, there is pain when that muscle is lengthened or the myofascial trigger point is compressed. Pain occurs at the end range of motion (EROM) of the muscle in question, restricting ease of movement. The myofascial trigger point, in each muscle, causes a recognizable referral pattern. Sometimes those patterns are in the locality of the myofascial trigger points, but they may also cover several muscles. The patterns may not even include the muscle that holds the myofascial trigger points at all, as myofascial trigger points can *refer* pain and can alter sensations. Imagine having a constant itch you cannot scratch, ever. Imagine a noise in your ear that will not go away, ever. You have to find the myofascial trigger points that cause the symptoms and treat them. Myofascial trigger points mimic everything.

Each myofascial trigger point has its own recognizable pattern—a portrait of pain or changes in sensations. Simons, Travell, and Simons (1999) highlighted the difference between what are known as *active* myofascial trigger points and *latent* myofascial trigger points. Pain and changes in sensations from active myofascial trigger points are recognized by the patient as "their pain." Latent myofascial trigger points, on the other hand, cause pain that is not necessarily recognized by the patient, but may be contributing to the patient's problems. Latent and active myofascial trigger points provoke motor dysfunction and impaired muscle activation patterns (Lucas et al. 2004, 2009), weakness,

and muscle imbalances. It is vital to appreciate that latent myofascial trigger points can develop into active myofascial trigger points. The pain patterns in this book do *not* have an "X" to "mark the spot" of the myofascial trigger point, as may be found in other texts and books. It is important for everyone to understand that myofascial trigger points can occur *anywhere* in *any* muscle fiber.

Dry needling is an effective treatment for chronic pain of neuropathic origin and has been demonstrated to have very few side effects. This technique is unequalled in eliminating neuromuscular dysfunction of myofascial trigger point origin that results in pain, functional adaptations, and neuromuscular deficits.

Uniquely, this text provides all suitably qualified therapists with safe, effective, and appropriate clinical applications as part of a multidisciplinary approach, since only rarely can a single modality offer the intervention required for therapeutic success. Dry needling alone as a means of treating local myofascial trigger point pathology will almost certainly fall short of what is required for a complete rehabilitation.

If a muscle is sensitive and shortened or has active myofascial trigger points within it, the patient may feel a combination of sensations and pain. On compression, or needling, the patient can often feel a reproduction of "their" pain (active trigger point). This is a helpful diagnostic indicator for the practitioner when attempting to identify the true cause of the patient's symptoms.

A New Hypothesis

I wish to offer a new hypothesis while making a stark differentiation between myofascial trigger points and muscle spasm. A muscle spasm requires neural input (monosynaptic reflex arc) and ATP. Wearing my clinical anatomist's hat, I appreciate the fact that, on death, muscles take on a contracted state we call *rigor mortis* (Latin *rigor* means "stiffness," and *mortis* means "of death"). My hypothesis is that some, if not all, myofascial trigger points are a rigor contraction—an electromagnetic entity requiring no neural input and no need for ATP. This is an issue arising at the microscopic level, where the sarcomeres involved are only doing what they have evolved to be excellent at doing—contracting. To differentiate this "out of the normal" type of contraction, we refer to it as a *contracture*. The all-or-none principle would have all the sarcomeres contracting, or none contracting.

In the case of a myofascial trigger point, approximately 100 sarcomeres are in a state of contracture within a cluster of myofascial trigger points. Higher up than the microscopic level, at the gross level, what is occurring would require manual therapies, such as positional release, soft tissue release, or other. Regarding the microscopic level, I propose that the needle only has to come into close proximity to the myofascial trigger point in order to elicit a change in polarization; this causes a resulting twitch response. Research to confirm or disprove this hypothesis is called for. Once normal electromagnetic activity has been restored, tensional and compressional forces also return to normal, and regular cellular activity can resume.

2 Standards and Guidelines in Myofascial Trigger Point Dry Needling

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Anatomical Excellence

The therapist must pay scrupulous attention to anatomical detail. Before inserting the needle, the therapist is advised to check and recheck anatomical landmarks to ensure that they will avoid neural (or other) structures that could suffer insult. The use of needles is an invasive technique that carries a risk of infection to both the patient and the therapist. While it is important that therapists follow local, state, national, or international best practice, some additional standards and guidelines are presented in this chapter.

General Standards and Guidelines—Pre-treatment

- Ensure you have your patient's signed consent before dry needling.
- Hands must be properly washed, with soap and lukewarm water, and be clean before beginning every treatment.
- Nails should be smooth and short.
- Single-use disposable paper towels are recommended.
- Single-use gloves should always be worn by the therapist when handling a needle and for applied compression of the needled area following removal of the needle.
- The skin over which the treatment will be applied should be cleaned; however, in accordance with the WHO recommendations, it need not be disinfected.
- Single-use needles contained in delivery tubes are essential and must be used before their expiry date (check packaging for details).
- Needle thickness varies and should reflect the patient's needs and the anatomical location of the treatment (e.g. hands and face versus thigh).
- Avoid touching the needle shaft.
- Therapists should follow national guidelines and standards provided by a competent authority within their own geographical location, or follow international best practice if such standards are not locally in place.
- Therapists must carry out a full medical health screening in advance of any treatment to ensure the patient's suitability for the procedure, and to provide a comprehensive description of the procedure, including all potential risks.
- Target the four most significant symptoms. Identify all of the known and suspected perpetuating factors: control the known ones and investigate the suspected ones. Include tests (such as a sleep study), exercise regimens (including correct breathing technique), and dietary changes.
- Therapists providing dry needling should also be qualified in emergency first aid (first responder), and, although not essential, I recommend courses that include defibrillation.

General Standards and Guidelines—Post-treatment

- Single-use needles should be disposed of in a sharps container, following local disposal regulations for blood-contaminated needles.
- Any disposables—such as cotton wipes or similar—should be disposed of in an appropriate manner.
- Allow the adult patient appropriate time on the treatment table before they return to standing.
- If myofascial trigger point dry needling causes the eyes to water post-treatment, advise your patient to allow appropriate time before driving.
- Encourage your patient to take it easy over the following day or so, and to avoid repetitious or stressful movements. The patient will require energy to accommodate and facilitate change post-treatment. Dry needling can also cause some muscle soreness, similar to delayed onset muscle soreness, which can last from one to three days. Therapists should ensure that their patients are aware of this.
- Patients should avoid very cold (ice) applications, hot baths or showers, saunas, or steam rooms for a number of days following treatment.
- Cool water is the post-treatment of choice.

General Standards and Guidelines—During Treatment

- Take appropriate steps to ensure patient comfort.
- To ensure the best possible treatment outcomes, it is advisable to avoid treating too many muscles in a single treatment: it is recommended to limit treatment to between three and five muscles in any one treatment. Keep in mind, however, that this could constitute many hundreds of myofascial trigger points. The patient must have the capacity to facilitate changes as a result of the treatment. Less in this case is more. Practitioners can complement the treatment with other non-invasive modalities.
- The uncovered hand and fingers can be used to locate the myofascial trigger point and to identify key anatomical landmarks; however, to ensure the safe application of this technique, gloves must be worn when handling or using the needle or whenever there is a risk of contamination.
- On removal of the needle, ischemic pressure should be applied for a suitable length of time to minimize any blood loss.
- Any blood on the skin should be wiped with an alcohol swab, which should then be appropriately discarded.
- Regular communication with the patient is advised during the treatment, ensuring feedback and information is received from the patient.
- Look for non-verbal signs, such as facial expression, breath holding, and clenching.
- Treatment is generally not recommended with the patient in the seated position (because of the risk of fainting).

Wearing Gloves

While writing this book I received feedback regarding the recommendation that gloves should be worn when carrying out dry needling. The reviewer stated: "It is practically impossible to perform dry needling while wearing gloves." Wearing gloves can bring issues of reduced kinesthetic awareness and awkwardness in handling the needles. However, while opinions may differ, my responsibility as an author is to provide the safest recommendations to the therapist. I realize, of course, that palpation awareness and tactile feedback will be somewhat reduced with the wearing of latex or similar gloves.

The main reason for the use of gloves is that when a needle is withdrawn from a muscle, the most common side effect is bleeding. Whenever there is a possibility that the therapist's skin could come into contact with the patient's blood, care must be taken to provide a barrier and appropriate protection for the therapist. Of secondary benefit is the fact that gloves provide an additional (albeit minimal) layer of "skin" to the therapist, and therefore the risk of needle stick is further (if only fractionally) reduced. Gloves provide such protection and in that regard they must be recommended. In fact, it is worth noting that a number of insurance companies will not provide cover unless the therapist demonstrates that they wear gloves during the dry needling application.

Risks and Cautions in Myofascial Trigger Point Dry Needling

1. Some patients may experience an allergic reaction to the needle.
2. Fainting (vasodepressor syncope). This reaction can be caused by emotional stress and a fear of pain.
3. Hematoma (muscle bruising). Appropriate post-needling ischemic pressure will in general significantly reduce the possibility of developing a hematoma.
4. Prosthetic implants and implanted devices must be declared, since dry needling is contraindicated for such patients because of the risk of infection.
5. Nerve damage/injury or nerve block is rare but can occur.
6. Damage to a vein or artery is rare but can occur.
7. Insult to the spinal cord or brain is rare but can occur.
8. Inserting a needle into any of the internal viscera or through a fenestrated bone (a bone with small holes in it) is rare but can occur.
9. When infection is present, dry needling must be avoided.
10. Increased muscle spasm, increased pain, and muscular edema can occur.

Myofascial trigger point dry needling will not be suitable for all adult patients. If, for any reason, a patient is not able to give consent to the procedure, or if they seem confused in any way, then dry needling must not be applied. Check the patient's skin for signs of swelling or possible lymphedema, as the application of dry needling could increase the risk of infection. In fact, there may be particular days when patients who normally receive this treatment may be better advised to avoid it. Care should be taken to discuss the suitability of this technique with each patient, considering each case individually and on its own merits.

Explaining to patients in advance of the application the entire procedure and what to expect during treatment and post-treatment will help to ensure that your patients are well informed. Needles identical to those used in this unique application have been used for many years on a daily basis by thousands of therapists across the world. In appropriately qualified hands, needling is a very safe technique.

Contraindications to Myofascial Trigger Point Dry Needling

1. Open wounds or broken skin should be avoided.
2. Malignancies. Should a patient state they have a malignancy, their GP or specialist must give written permission before any treatment can be given.
3. Aneurysm.
4. Hematomas. These should never be pressed, massaged, or stretched.
5. Arteriosclerosis. Because of the risk of blood clot formation, a GP's or medical specialist's approval must be given in writing. Information regarding all medications should be provided.
6. Osteoporosis. This is particularly serious if using dry needling techniques because of the risk posed by fenestrations (e.g. small holes in the scapula, through which the needle could pass and contact the lungs if care is not taken).

Ruling Out Visceral Pain—"When in Doubt, Refer"

Visceral pain has a temporal evolution, and in its early stages can be insidious and difficult to identify. Because of the low density of sensory innervation of viscera, and the extensive divergence of visceral input within the central nervous system (CNS), what is called *true visceral pain* is a vague, diffuse, and poorly defined sensation, regardless of the specific internal organ of origin. This type of pain is usually perceived in the midline, at the level of the lower sternum or upper abdomen. Whether it originates from the heart, esophagus, stomach, duodenum, gall bladder, or pancreas, visceral pain in the early phase is perceived in this same general area.

Additional stimuli, such as local compression, applied to this area fail to worsen the pain. True visceral pain can easily be overlooked, partly because of the fact that the patient cannot clearly describe the pain. It is often described as a vague sense of discomfort, malaise, or oppression. The pain is typically associated with marked autonomic phenomena, such as pallor, profuse sweating, nausea, vomiting, changes in blood pressure and heart rate, gastrointestinal disturbances (e.g. diarrhea), and changes in body temperature. Strong emotional reactions are commonly present, including anxiety, anguish, and sometimes even a sense of impending death. Visceral pathology may occasionally manifest principally through vegetative and emotional reactions, with minimal pain and discomfort. A typical example is painless myocardial infarction, which may produce a sense of gastric fullness, heaviness, pressure, squeezing, or choking.

As a general rule, in the early stages the intensity of visceral pain bears no relationship to the extent of the internal injury. Visceral pain should always be suspected when your patient presents with vague midline sensations of malaise. This is even further compounded when the patient is elderly.

As visceral pain continues to progress (over a few minutes to several hours), it may refer to dermatomes whose innervations enter the spinal cord at the same level as the visceral organ involved. This can be misinterpreted by the brain as joint, muscular, or nerve pain manifesting itself as sharp, localized, deep somatic pain. For example, liver pathology can lead to referred pain in the upper right shoulder. Peripheral nerve pathology, such as irritation of the C7–C8 spinal nerves, presents as pain in the fourth and fifth digits (ulnar nerve). This type of pain can be accompanied by hyperalgesia (increased sensitivity, pain on light stimulation) or hypoalgesia (decreased sensitivity, numbness).

Detailed questioning of a patient is necessary in order to clarify their suitability for the treatment and their level of discomfort. During this stage (i.e. assessment), the therapist must determine the characteristics of the pain, the pathways of pain radiation or referral, and the form and dependency of the pain on active, active resisted, or passive movements. Feedback from the patient concerning neurological signs, skin sensitivity, pain, and referral, as well as other symptoms, including heat, cold, tingling, itch, and mood swings, are all vital ammunition in the war on pain. This information will ensure that the patient is referred to the appropriate medical practitioner if warranted. If you are in any doubt at all, refer the patient to their GP, who will respect you for your professional approach and concern for the patient's health and wellbeing.

Once your patient returns to you, they must supply a letter from their GP or medical specialist stating that pathology is not suspected and has been ruled out. As time can be such a crucial factor in pathology, referral without delay is always in the patient's best interests.

Before Starting—The Ten-Point Guidelines

The following safety-first guidelines are applicable in the post-screening and pre-needling stage:

1. Check your anatomical landmarks. Check again and be sure.
2. Use your palpation skills to: a) identify autonomic responses, and b) identify the myofascial trigger point by digital application.
3. Put on your protective gloves.
4. Check your anatomical landmarks once again, and then place the needle within the delivery tube over the area to be treated. Release the needle.
5. Insert the needle with a firm fast "tap" (observe for any reaction).
6. Treat the myofascial trigger point with straight in-and-out motions.
7. Allow the patient to control the situation: encourage them to breathe slowly through the nose on the inhale and with pursed lips on the exhale.
8. Deactivate all the myofascial trigger points in any one muscle (treat the muscles which are most superior and medial first), and avoid treating more than five muscles in any one treatment.
9. After removing the needle, apply ischemic pressure to the tissue and return the needle backward (handle first) into the delivery tube.
10. Discard all used needles and contaminated items safely in the sharps container or other appropriate waste disposal unit, which should be close to hand.

A Few Words About Dietary Influences

Adequate quantities of minerals and vitamins are essential for healthy muscles and tissues. Many patients presenting with chronic pain are found to be deficient in a number of vitamins and minerals. Vitamins B_1, B_6, and B_{12}, along with vitamin C and folic acid, are important in the war on pain; the minerals calcium, magnesium, iron, and potassium are critically important.

All too often, people are confused as to why they are deficient in these important minerals and vitamins, because they will report that they eat well and have normal dietary habits compared with other family members. The problem may not be their diet but rather their personal health choices, such as smoking and drinking alcohol or caffeine. Smoking, for example, annihilates vitamin C, while oral contraceptives affect vitamin B_6 levels. Antacid medication can leave many individuals with the symptoms of chronic fatigue; even writing their signature becomes an effort.

Patients with vitamin and/or mineral deficiencies may report feeling unusually cold, bouts of diarrhea, restless leg syndrome, headaches, disturbed sleep, and trigger point pain. Other symptoms include feeling fatigued, muscle cramping, and depression. Metabolic disorders should be ruled out, particularly thyroid problems and hypoglycemia. Referral of patients with vitamin/mineral deficiencies is recommended.

Needle Application

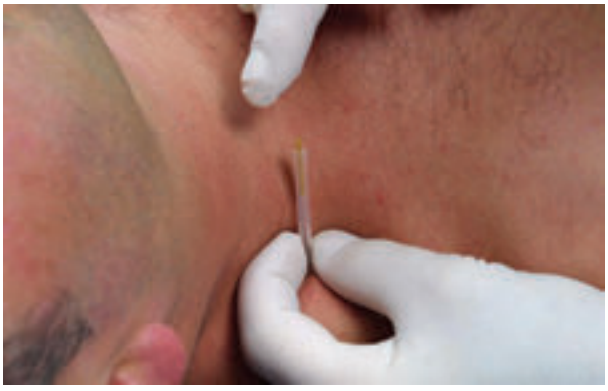
After removing the delivery tube from its packaging, place the delivery tube against the skin and release the needle. Quickly tap the needle into the tissue over the target muscle, ensuring that the needle is securely inserted just beneath the skin. Remove the delivery tube and place it between your fingers. Use straight in-and-out motions to direct the needle to its target.

Elicit a twitch response and proceed to eliminate all twitch responses. If the patient experiences burning or undue pain, immediately withdraw the needle and reposition the needle when the patient is ready and relaxed. *Never* bend the needle, especially when inserted, and avoid inserting the needle completely, while keeping a firm grip on the needle handle.

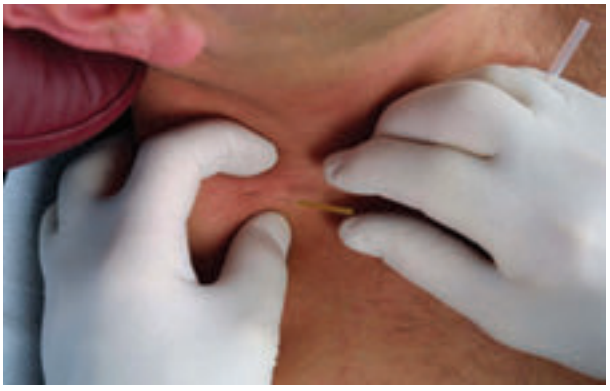
Returning the needle to the tube via the opposite end to the handle *must be avoided*, as this significantly increases the risk to the therapist of a needle stick. In other words, needles must be returned to the delivery tubes *handle first*. Wearing gloves during the needling application is recommended.

Direct the Needle with Straight In-and-Out Motion

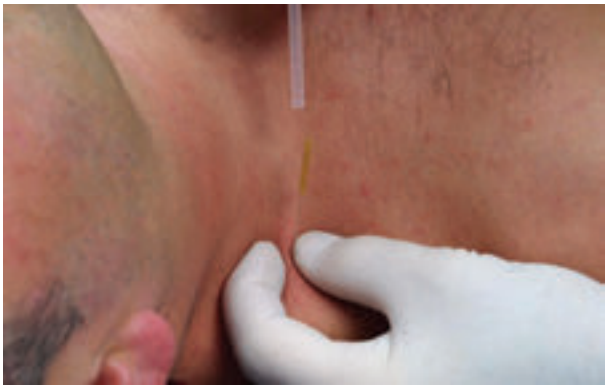
Remember, *never* try to bend the needle or change needle direction when it is already in the muscle.



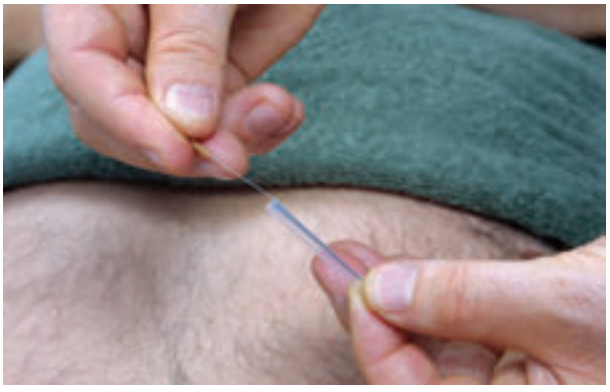
(a) After removing the delivery tube from its packaging, place the delivery tube against the skin and release the needle. Quickly tap the needle into the tissue over the target muscle, ensuring that the needle is securely inserted just beneath the skin.



(c) Place the delivery tube between your fourth and fifth fingers. Use straight in-and-out motions to direct the needle to its target. Elicit a twitch response and proceed to eliminate all twitch responses. If the patient experiences burning or undue pain, immediately withdraw the needle and reposition the needle when the patient is ready and relaxed. Never bend the needle, especially when inserted, and avoid inserting the needle completely, while keeping a firm grip on the needle handle.



(b) Remove the delivery tube.



(d) Returning the needle to the tube via the opposite end to the handle must be avoided, as this significantly increases the risk to the therapist of a needle stick. In other words, needles must be returned to the delivery tubes handle first. Wearing gloves during the needling application is recommended.

Health and Safety Considerations

Who (and What) Should Not Receive Dry Needling?

- Avoid dry needling patients who are clinically obese or whose body fat is below a safe level. Likewise, avoid needling patients who are intoxicated or fatigued, have an acute medical condition (such as epilepsy), or are pregnant. Patients with a needle phobia, or those who are unwilling, confused, or unable to give consent, should not receive myofascial trigger point dry needling.

- Nerves, blood vessels, and areas of lymphedema should be avoided. Patients who have bleeding tendencies or problems with blood clotting, or those taking blood-thinning (anticoagulant) medication, should avoid dry needling unless they are under the strict guidance of a medical practitioner. Patients with compromised immune systems are more susceptible to infection, as are patients with vascular disease and artificial joints and implants; in such cases, seek medical advice.

- Patients with pacemakers should avoid dry needling.

- Cancer patients should not receive dry needling, while for those with epilepsy more caution is required because of their low tolerance to strong sensory stimulation.

- Dry needling should be avoided when there is knowledge or evidence of infection, ulcers, osteoporosis, trauma/open wounds, aneurysm, or malignancy.

- Patients with psychological disorders may not be potential candidates for dry needling: emotional stress and anxiety may render dry needling unsafe. In such cases, medical advice must be sought.

Informing Patients

Dry needling is an invasive technique: needles break the skin and are placed either superficially or deep in the tissues, which brings with it associated risks. As a therapist, your responsibility is to clearly inform your patients of the risks involved and receive their written permission before placing a needle in their body. Patients must also be informed about possible side effects and needle effects, both immediate and longer term. Patients have the right to refuse a treatment or to cease a treatment at any time.

Patient Responsibilities

Each adult patient has the responsibility to provide information accurately and honestly to the therapist. Patients should notify therapists about any conditions that they have which can be transferred by blood, or about conditions that require blood anticoagulants or that may be adversely affected by needle punctures.

Pain During Needle Insertion

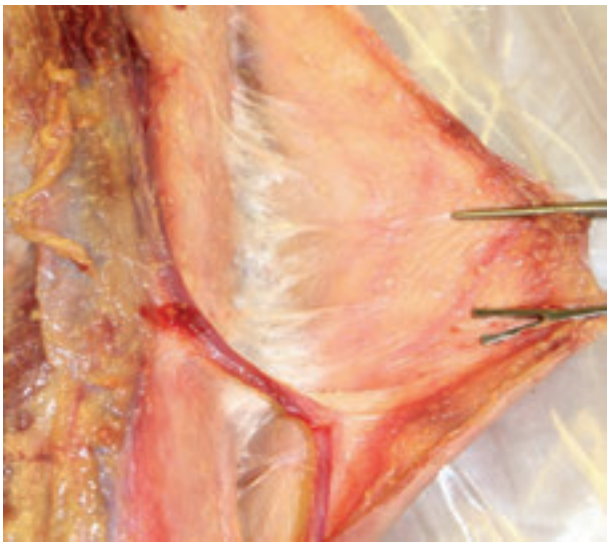
Pain experienced during insertion of the needle will most likely be due to a therapist's heavy-handed technique or awkwardness, usually because of lack of experience. Needle thickness and length must be appropriate to the target muscles or body part; for example, the hands, feet, and face are extremely sensitive and require a fine needle gauge. A blunt or hooked needle must never be used and will naturally cause the patient unnecessary pain. Pain may also occur in highly sensitive patients.

In most cases, skillful insertion of the needle through the skin with the appropriate level of controlled speed results in painless penetration of the deeper tissues. Participation in a formal course of studies under the watchful eye of a qualified and properly experienced tutor is essential. A qualified tutor will ensure that each therapist learns the correct technique while utilizing the optimum degree of force.

Avoiding Nerves and Blood Vessels

Nerves and blood vessels are literally everywhere in the human body, and so it is impossible to totally avoid one or the other. The largest blood vessels and nerves can be identified in several ways; to this end, knowledge of surface and gross anatomy is key.

Major veins are often obvious to the eye, while deeper arteries can be pinpointed by palpation and therefore, as in the case of nerves, avoided. Blood vessels and nerves travel together as neurovascular bundles as they course through the septal divisions and the fascial conduits of the human body.



Blood vessels and nerves, many of which can be seen just below the skin, are avoided through excellent knowledge of anatomy. (Image by J. Sharkey 2008)

Guided Delivery Tube

This book recommends the use of a guided delivery tube to facilitate the smooth delivery and rapid penetration of the needle. The use of a needle guide tube allows the therapist to hold the needle steady over the skin while it is then tapped into place using the pad of the second digit (see the images showing needle insertion).

Pain After Needle Insertion

Several issues can arise while the needle is being inserted, resulting in the patient experiencing pain. Patients should be made aware of this in advance so that they are better prepared to deal with pain should it occur. The most obvious reason for pain when the needle is inserted is contact with nerve tissue; in this case, the needle should be returned to just below the surface of the skin and redirected. Needle grasping should also be considered: the needle may become interwoven and twist within the connected tissues, requiring a de-rotation of the needle or a gentle tapping on the top of the needle handle until the needle releases.

The therapist should look for any needle grasping and "tenting" (visible raising-up of the tissues and skin as a consequence of increased resistance to removing the needle) as the needle is removed. Work slowly with the patient and encourage controlled breathing, pursing the lips on exhaling. The therapist should never bend a needle: the needle motion is straight in and straight out.

Should the patient move during the application, this could have an effect on the needle. Precautions should therefore be taken to minimize this possibility: the patient should be carefully positioned to provide the therapist with the greatest amount of control with regard to unexpected reactive movement.

Post-needling Soreness

Soreness can occur within the first 24 hours following myofascial trigger point dry needling. This typically feels like delayed onset muscle soreness (what should be called *delayed onset myofascial soreness*), as if the muscle had been subjected to unaccustomed exercise or physical activity. Application of a cool pack or cold water to the area (ice is *not* recommended) should reduce any discomfort, which should normally not last more than a day. Applying contrast cold and heat therapy can also be useful, starting and ending with a cold application.

3 Central Sensitization and Control of Perpetuating Factors

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Chronic pain syndromes display significant neuroplastic changes, altered neuron activity, and excitability and adaptations affecting pain matrix structures, specifically the spinal cord, thalamic nuclei, cortical areas, amygdala, and periaqueductal gray areas. In essence, central sensitization is characterized by an amplification of normal neurological activity (Giamberardino et al. 2011a).

Continuous bombardment of the dorsal horn by noxious afferent activity leads to a release of glutamate and substance P; this in turn leads to the activation of previously inactive synapses in the wide dynamic range (WDR), resulting in central sensitization. In normal circumstances, there is a balance between inhibitory and facilitatory neuronal activity in terms of pain management and control (Willard 2008). This results in *spinal segmental sensitization (SSS)*—a hyperactive state of the dorsal horn caused by constant noxious afferent bombardment, originating from damaged or sensitized tissues (e.g. myofascial trigger points or other soft tissue/connective tissue trauma, or visceral structures, such as a gall bladder that has become inflamed because of gallstones). A diagnosis of SSS includes observation of dermatomal allodynia, hyperalgesia, soft tissue pain/tenderness upon palpation, and myofascial trigger points (Giamberardino et al. 2011b).

Hypersensitivity initially occurs at the local segmental level. However, through the process of sensitization of adjacent spinal segments (spillover), a state of "wind-up" caused by *temporal sensory summation (TSS)*—an increased rate of nociceptive pulsing at the dorsal horn—facilitates widespread segmental sensitization, leading to body-wide peripheral pain. TSS is caused by increased C-fiber input at the dorsal horn and can maintain a state of hyperalgesia in chronic pain patients (Staud, 2011).

The stimuli that activate and sensitize the WDR neurons ascend the spinothalamic tract to reach the higher brain centers, where the thalamus and limbic systems are activated (anterior cingulate gyrus, insula, and amygdala). The limbic system is involved in modulating muscle pain, but it also modulates fear, anxiety, and distress. Therefore, increased activity in the limbic system, influencing the perpetuation of pain syndromes, can contribute to the fear or emotional stress associated with chronic pain syndromes (Niddam et al. 2007).

The rostral ventral medulla (RVM), acting as a relay point for descending activity from the periaqueductal gray (PAG), contains a number of "on" and "off" cells that can increase or decrease levels of pain. In the acute phase of injury, the "on" cells provide a protective mechanism—significant pain is evoked, thereby preventing undue movement/activity that might cause more damage. In chronic pain mechanisms, "on" cells remain active, and there appears to be an "on" cell dominance, rather than a balance of "on" and "off" cells that would maintain a balance between facilitation and inhibition (Willard 2008). Additionally, normal descending pain-inhibiting signals are disrupted, leading to a further sensitization of muscle tissue (Niddam et al. 2007).

Spinal Facilitation

Spinal facilitation is an increase in spinal cord neuron activity as a result of noxious peripheral nociceptor bombardment. In normal circumstances, this noxious stimulus is modulated by local mechanisms, or by descending pathways from the cerebral cortex and brainstem. Abnormal constant bombardment leads to cell apoptosis, wind-up, and segmental sensitization (Bishop, Beneciuk, and George 2011). As a consequence, the dorsal, ventral, and lateral horn circuits in the spinal cord may become more readily activated by lower intensity stimuli.

Spinal facilitation is characterized by: 1) increased ventral horn output, which results in increased muscle tone (corresponding to the segmental level); 2) increased lateral horn output, which increases nociceptive activity (reflex mechanisms); and 3) increased dorsal horn activity, resulting in an increase in the production of neuropeptides, which can increase inflammatory activity in the affected tissues.

The result is increased hyperalgesia, local tissue tenderness, and spillover, which affect adjacent spinal segments (Camanho, Imamura, and Arendt-Nielsen 2011).

Note: Too often doctors and therapists fail to consider the role of myofascial trigger points in chronic pain patients. They therefore fail to treat what may be, at least, a significant underlying perpetuating factor.

Many therapists deactivate myofascial trigger points as they find them, without giving due consideration to the mechanisms that caused them. In such cases, patients enjoy temporary relief but continue to have recurring pain issues that never fully resolve. Therapists can identify symptoms of spinal segmental sensitization and spinal facilitation by evaluating presenting symptoms of allodynia, hyperalgesia, pain pressure sensitivity, and motor and sensory responses (reflex tests, dermatome assessment, and local muscle endurance assessment). Continuous nociceptive bombardment of the spinal cord leads to increased peripheral sensitivity and a state of central sensitization (Shah and Gilliams 2008).

Active and latent myofascial trigger points are found in the tissues of both symptomatic and asymptomatic individuals. Dorsal horn neurons may manifest neuroplastic changes as a result of nociceptive bombardment if left unresolved.

Cortical changes amplify the pain state, creating a pain cycle that may be difficult to break, as in the case of chronic pain conditions such as fibromyalgia, chronic fatigue syndrome, and myalgic encephalomyelitis (Camanho, Imamura, and Arendt-Nielsen 2011). Many people do not appreciate that stress is a normal part of living. It is how our body deals with stress and how we cope and deal with our sensory impressions, and how they stack up against our internal view of our world, that results in distress or eustress. This is part of our fight or flight system, or our ability to confront, avoid, or submit. Failure to resolve a stressful situation by one of these means results in high sympathetic tone, increased cortisol production, increased resting muscle tone, and the possible formation of myofascial trigger points. Myofascial trigger points are more likely to develop in tissue which has neurological deficits that have been caused by compression, tension, disc dysfunction, facet joint dysfunction, vascular compression, metabolic stress, biomechanical stress, postural stress, etc.

When muscles develop myofascial trigger points, they remain tight, causing local compression of vascular, neurological, and joint/biomechanical structures, thereby hampering the normal function of that tissue. All tissues distal to the nerve involved will likely be affected. Dry needling can release the muscle tension in order to resume normal function, with improved neurological conduction and vascularity. Dry needling should be supported by other appropriate soft tissue manipulation modalities and suitable physical activity.

Keys to Symptom Management

The following ten key aspects should be considered when treating myofascial trigger points:

1. Differentiate the myofascial trigger points from pain points by using the cardinal signs, which must include palpable nodule and taught band, jump sign, twitch response, painful EROM, referred pain, and autonomic responses.

2. Treat the myofascial trigger points that are most superior and medial first.

3. The deltoid seldom develops its own active myofascial trigger points. Instead, most are "baby" or "satellite" myofascial trigger points; therefore treat associated muscles within the functional units of the deltoid first.

4. The upper trapezius is the "Grand Central Station" of myofascial trigger points and is a major contributor to neck, shoulder, upper back, and head pain.

5. Active myofascial trigger points, when irritated by a competent therapist, will result in referred pain or changes in sensation that the patient recognizes.

6. Latent myofascial trigger points generally result in pain or change in sensations that the patient does not recognize. These myofascial trigger points may be contributing to, but are not the true source of, a patient's problem.

7. Myofascial trigger points can form in any muscle fiber (Sharkey 2008) and not just in the center of a muscle, or where the "X" marks the spot (which is misleading) on so many myofascial trigger point charts. Identify and remove/change the perpetuating factor(s).

8. Excellent palpation skills are necessary for locating myofascial trigger points.

9. Upper or lower limb tension tests should be administered in order to rule out nerve insults, including compression and/or inflammation.

10. Any patient suffering from unresolved pain or changes in sensations should have the possibility of myofascial trigger point involvement ruled out as a primary or secondary cause or contributor.

Initiating, Aggravating, and Perpetuating Factors

Anything that perpetuates a myofascial trigger point is called a *perpetuating factor*. What initially activates a myofascial trigger point may be different from what aggravates (worsens) or perpetuates (maintains) it, but they are all commonly called perpetuating factors. The key to controlling any symptom is the control of as many perpetuating factors as possible.

An appropriate medical history will indicate whether pain patterns are stable or evolving. Chronic myofascial pain (CMP) is not progressive. The development of satellite myofascial trigger points that worsen symptoms, and the appearance of new symptoms, are indicators that there are perpetuating factors at play. To control symptoms, first identify and control perpetuating factors.

Controlling perpetuating factors is vital. Perpetuating factors include whatever impairs muscle function, such as anything that diminishes the cells' access to oxygen and nutrients, hampers the removal of cellular wastes, or adversely affects the metabolism of the neurotransmitter acetylcholine (ACh). Anything that enhances the formation of myofascial trigger points is a perpetuating factor. For instance, anything that constricts the flow of blood to the area will lessen its supply of oxygen and nutrients, adding to the energy crisis. A perpetuating factor can be anything that increases energy demand (trauma, overwork), decreases energy supply (inadequate nutrition, insulin resistance), sensitizes the CNS (pain, noise), decreases oxygen supply (congestion), enhances the release of sensitizing substances (allergies, infections), or increases endplate noise (increased ACh release, reduced acetylcholinesterase).

Perpetuating Factor Types: A Long Short War

We are fighting a war on pain. The foot soldiers of the enemy are those perpetuating factors such as mechanical stressors, including paradoxical breathing, body disproportions, myofascial or connective tissue abuse, and articular dysfunctions. Metabolic perpetuating factors include impairments to energy metabolism, and coexisting conditions, such as pain and a lack of restorative sleep. Environmental perpetuating factors include pollution, medications, trauma, and infections.

Psychological perpetuating factors are also an important area to investigate. The remedies for lifestyle perpetuating factors are often the least expensive, but may be among the most difficult to maintain. To further complicate matters, perpetuating factors often have perpetuating factors of their own. Cognitive therapy and mindfulness can be useful interventions to help us change the way we, and our patients/clients, think about and perceive pain.

Examples

Paradoxical Breathing

Paradoxical (or *abdominal*) *breathing* is a term used to describe an abnormal chest movement, with the patient's chest moving inward (or not moving at all) during inhalation rather than outward or forward. This means that your patient cannot take a functional breath and is most likely a shallow breather. Paradoxical breathing is a common perpetuating factor, but is easy to check if a patient is presenting with this breath rhythm issue.

To assess for correct rhythm, place one hand on your patient's abdomen. As the patient breathes in, their abdomen should swell as the abdominal cavity extends after the lungs expand. On breathing out, the patient's abdomen should come back in. When this occurs, it indicates that their respiratory muscles are healthy: the patient can move through their physiological range to accommodate the air required and expel residual air.

If the patient's chest is moving in as the breath comes in, and is moving out as the breath goes out, then this is paradoxical breathing. This inconsistent breathing includes mouth breathing, which is inefficient and shallow. Paradoxical breathing may indicate that your patient's body is not getting the oxygen it needs; it can occur temporarily during a time of congestion, such as a cold, and then may be maintained out of habit, or because myofascial trigger points have formed in the diaphragm or other respiratory muscles, thus inhibiting their function.

Training and awareness of proper breathing technique are important, but they are only part of the remedial process. Is adequate air coming in through the nose, or is there congestion? If so, why? Check into the possibility of allergies, low-grade sinusitis (sometimes caused by fungal infection), or other problems. A myofascial trigger point assessment is also needed, as myofascial trigger points can cause congestion, and their presence in respiratory muscles prevents these muscles from working properly. An assessment for myofascial trigger points includes accessory respiratory muscles, such as the scalenes and the serratus muscles.

Body Disproportion Examples

A structural leg inequality of 3/16" (0.5cm) can significantly tilt the body.

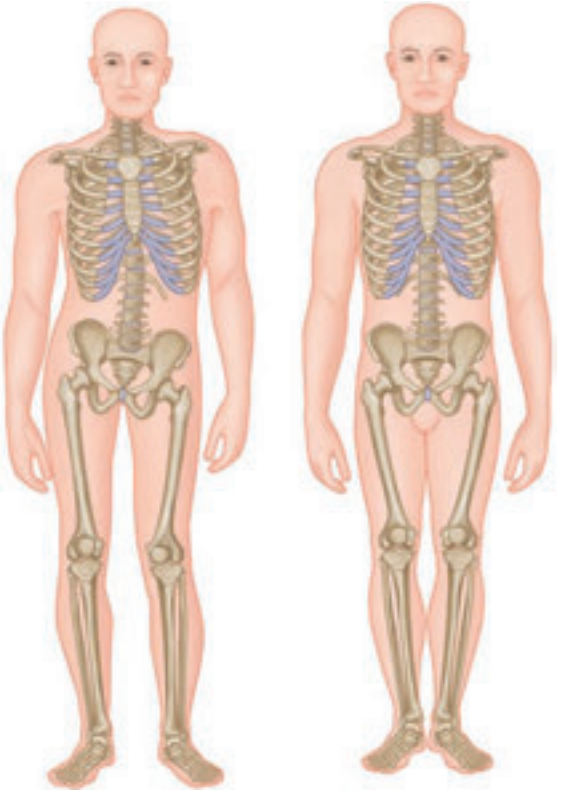


Figure 3.1: A structural leg inequality can significantly tilt the body.

What is more common, however, especially in people with myofascial trigger points, is pelvic torsion, where one thigh is drawn higher into the pelvis, thereby creating a functionally short leg. In this case, if one adds a heel lift to the shorter leg, the problem is compounded and reinforced.

True leg length inequality is a clue to check the whole body for proportional shortness on that side and for compensation on the opposite side. When the horizontal core stabilizers, including the deep ligaments and tendons, are not at healthy lengths, a spiral compensating effect can occur, called *rotoscoliosis*; this is a twisting of the tissues around the spinal column and can begin anywhere, with an end result of torsion of the feet, ankles, knees, hips, and shoulders. One area rotates right and the other rotates left to compensate. The body seeks harmony and balance, but this compensatory twisting can create functional hypermobility or restriction of numerous areas.

Another consideration is a one-sided small hemipelvis. The right and left hemipelvis should match. When the hemipelvis is smaller on one side, if a patient is sitting on a flat surface, the upper curve of one hip is higher than the other.

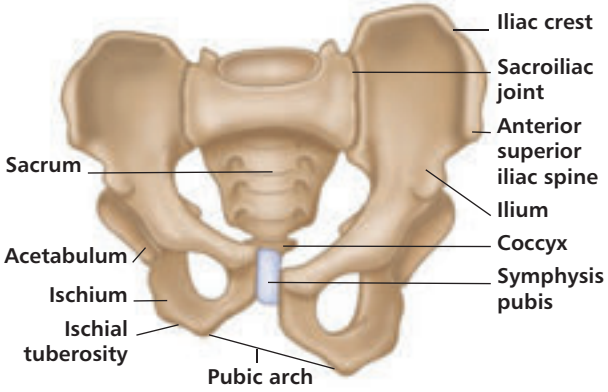


Figure 3.2: Hemipelvis.

Scoliosis and pelvic rotation can develop as other muscles struggle to compensate. The quadratus lumborum muscle is greatly affected by an asymmetrical hemipelvis, with the sternocleidomastoids and scalenes struggling to adjust to the overload from tilted thoracic muscles.

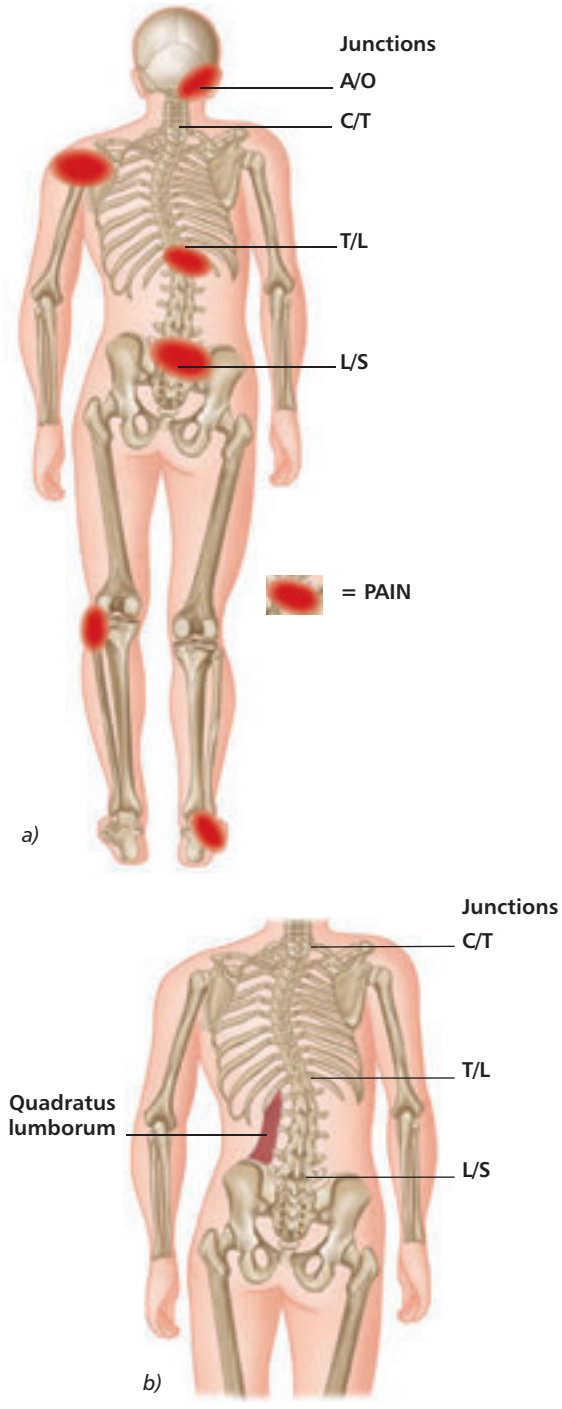


Figure 3.3: Functional scoliosis compensation.

Muscle Abuse

Any postural habit that causes prolonged muscle fiber contraction, repetitive low-intensity overload, and muscle stress (such as high-intensity muscle contractions) can cause myofascial trigger points (Edwards 2005). Posture affects respiration and can interact with vestibular dysfunction, thus compounding the symptoms (Yates, Billig, and Cotter 2002). Close attention must be paid to standing, sitting, and sleeping postures.

Figure 3.4: Adopt an ergonomic computer station to avoid postural problems from work-related activity.

Any cervical pillow must fit the curvature of the neck in order to support it without stressing coexisting myofascial trigger points. The size and shape of the pillow may need changing as the neck and shoulders respond to treatment.

Once correct neuromuscular balance has been achieved, many bad posture habits can be identified and corrected; these include head-forward posture, bracing the arms on the knees, crossing the legs, side leaning, and crossing the arms to prop up weak muscles. Bad postural habits are often clues to the locations of myofascial trigger points or other perpetuating factors, such as facet pathology.

One of the most preventable types of perpetuating factor is inappropriate physical activity (exercise). It is almost impossible to strengthen muscles that have myofascial trigger points or that are inhibited without first resolving the hypertonic muscles. It is essential to initially restore neuromuscular balance between muscle units (targeting the short spastic muscle first) and then to encourage neuromuscular efficiency, rather than strength. Developing muscular strength of the inhibited muscle comes later in the sequence.

Articular Dysfunction

Joint dysfunction can interact with myofascial trigger points. Any mechanical stress affecting joint position can initiate the process of osteoarthritis (AO) (Solomon, Schnitzler, and Browett 1982). Treating myofascial trigger points improves neuromuscular function and coordination, and anything that improves neuromuscular function can prevent or slow the progression of OA (Loeser and Shakoor 2003). Any arthritis treatment and prevention program needs to include the treatment of coexisting myofascial trigger points (Cummings 2003).

Myofascial trigger points can cause uneven contraction of muscles. An uneven contraction can cause or contribute to temporomandibular joint dysfunction (TMJD) and may cause bone misalignment (Koolstra and van Eijden 2005). Uneven muscle contraction may also be sufficient to cause jaw articular disc erosion (Liu et al. 2000).

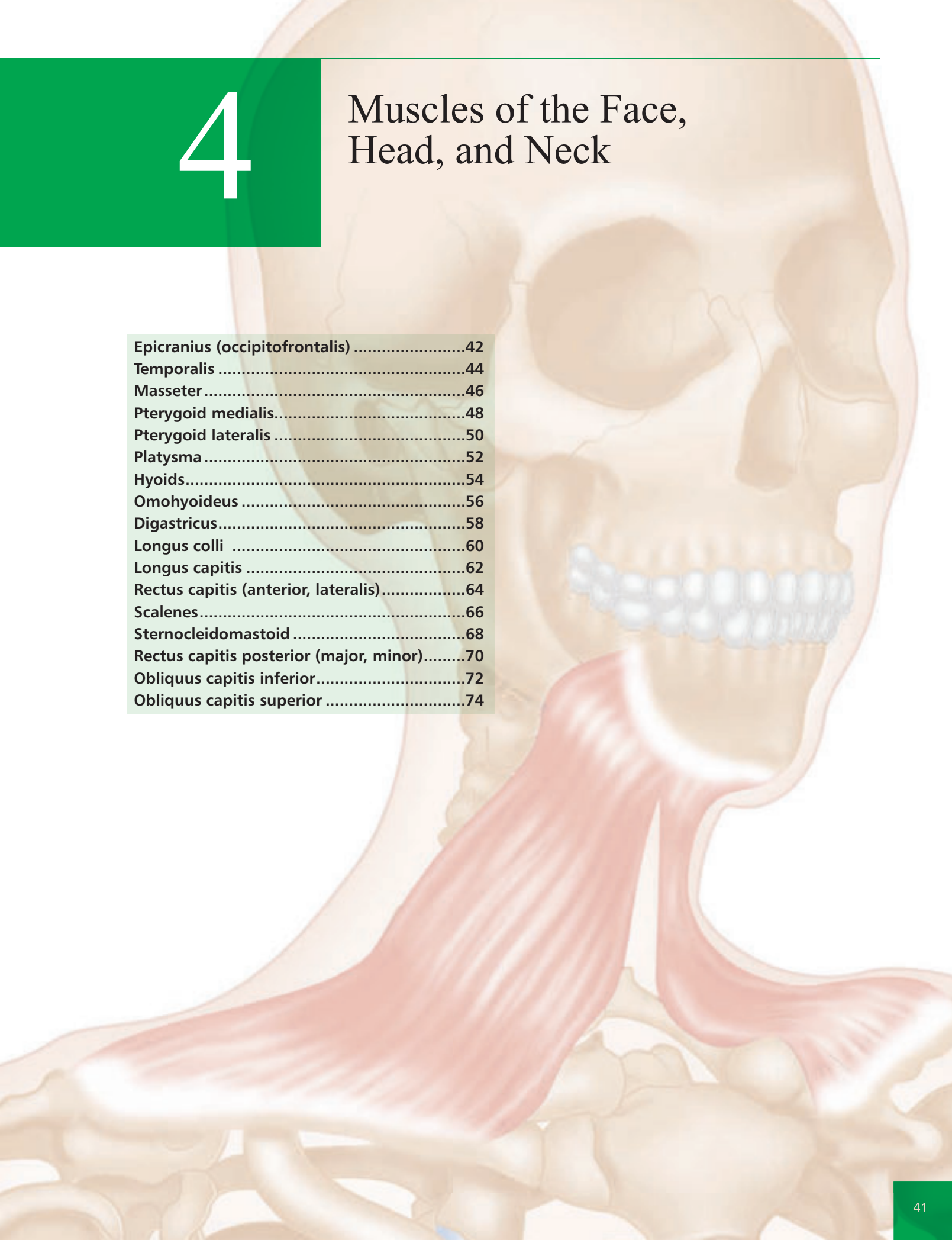
Vertebrae and myofascial trigger points interact. Active myofascial trigger points are associated with neck vertebral-disc lesions (Hsueh et al. 1998). As surrounding soft tissues are unevenly contractured because of myofascial trigger points, vertebrae may shift slightly out of alignment; this misalignment irritates the intervertebral discs. Intervertebral disc adjustments, and their associated ligamentous attachment compensations, cause changes in the angular motion of the body, which further stresses the inferior and superior intervertebral discs of the cervical spine (Kumaresan, Yoganandan, and Pintar 1999).

Soft tissue is often neglected. Disc deterioration may further alter motion and muscle compensation, which can contribute to additional pathologies in facet joints, muscles, and ligaments, resulting in a chronic pain state (Brisby 2006). Disc deformities or bone spurs that show up on imaging, however, may not be the cause of pain. Surgery performed without soft tissue evaluations can result in failed back surgery (Dubousset 2003). Subsequent surgical scars, adhesions, and postsurgical tightening of soft tissue can cause added stress to adjacent vertebrae, leading to the formation of myofascial trigger points.

PART II

MEET THE CULPRITS

4 Muscles of the Face, Head, and Neck



Epicranius (occipitofrontalis) 42
Temporalis 44
Masseter 46
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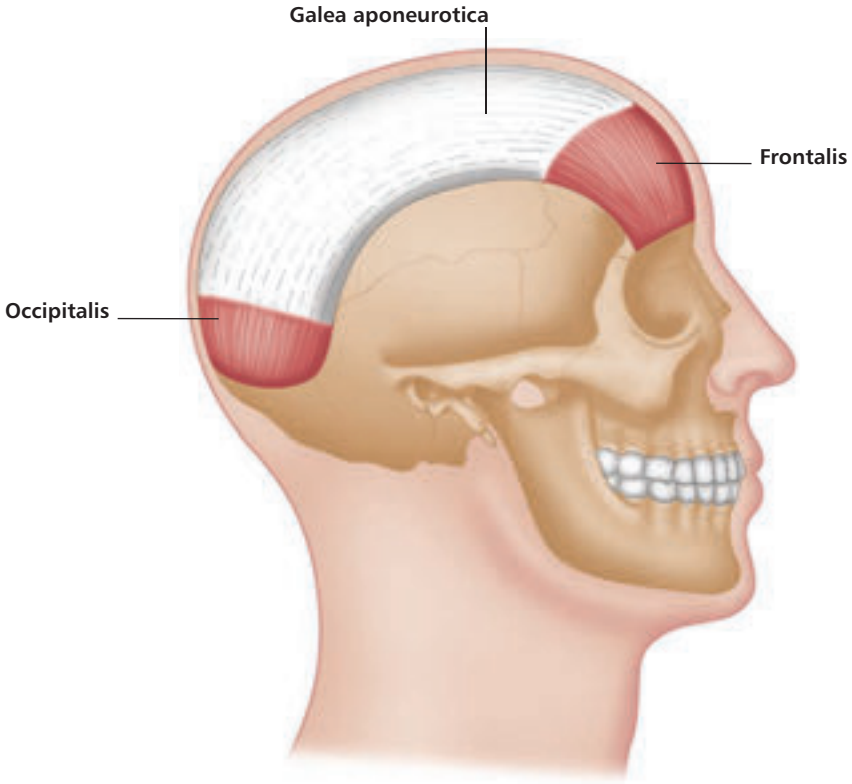
EPICRANIUS (OCCIPITOFRONTALIS)



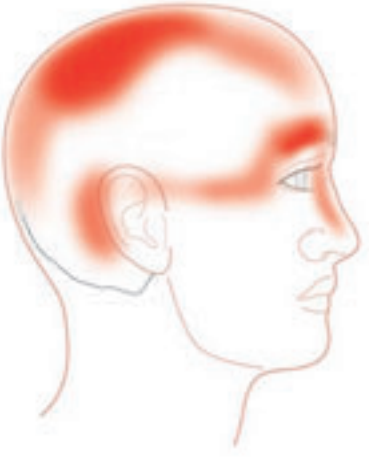
Latin, *occiput*, back of the head; *frons*, forehead, front of the head.

Origin
Occipitalis: Lateral two-thirds of the superior nuchal line of the occipital bone, and mastoid process of the temporal bone.
Frontalis: Galea aponeurotica.

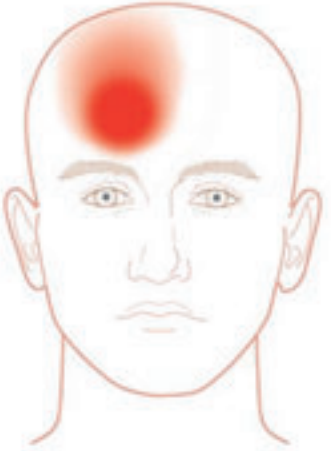
Insertion
Occipitalis: Galea aponeurotica.
Frontalis: Fascia and skin above the eyes and nose.

Action
Occipitalis: Moves the scalp backward. Assists the frontal belly to raise the eyebrows and wrinkle the forehead.
Frontalis: Moves the scalp forward and wrinkles the skin of the forehead horizontally.

Nerve
Facial VII nerve.



Occipitofrontalis.



Frontalis.

EPICRANIUS (OCCIPITOFRONTALIS)

KINETIC CHAIN COMMENT

The epicranius is essentially two muscle gasters with a strong fascial connection between them called the *galea aponeurotica*. Spasm in muscles such as the hamstrings (e.g. biceps femoris) or the plantar fascia can cause tightness through this area, ultimately causing tension in the head and neck, or headaches. Tension anywhere along the posterior back-line kinetic chain can lead to shortening of the galea aponeurotica, resulting in tension headaches and a hyperextended cervical spine. This can result in a posteriorly tilted pelvis in order to provide a level eye view when walking or running, and is a recipe for myofascial trigger point formation.

MYOFASCIAL TRIGGER POINT COMMENT

Pain is referred upward from the frontalis over the forehead on the same side. The occipitalis can refer pain into the eyeball or behind the eye. Pain can travel down behind the ear and into the nose. Sensitivity to sound and light are reported, with a resulting increase in experienced pain. I have had patients who complained of severe pain "inside their head"; on investigation, myofascial trigger points in the occipitalis reproduced a recognizable pain.

PRACTITIONER GUIDELINES

Patient positioning

Patient is supine to access frontalis. For access to occipitalis, the head is rotated to the opposite side or the patient can be positioned prone.

Needle type

Use 0.25 to 0.30mm × 30mm needle.

Needling directions

Locate and secure the myofascial trigger point within the palpable taut band using flat palpation. Insert the needle and direct it tangentially into the myofascial trigger point. These are very slim/thin muscles and are best approached at a 45-degree angle.

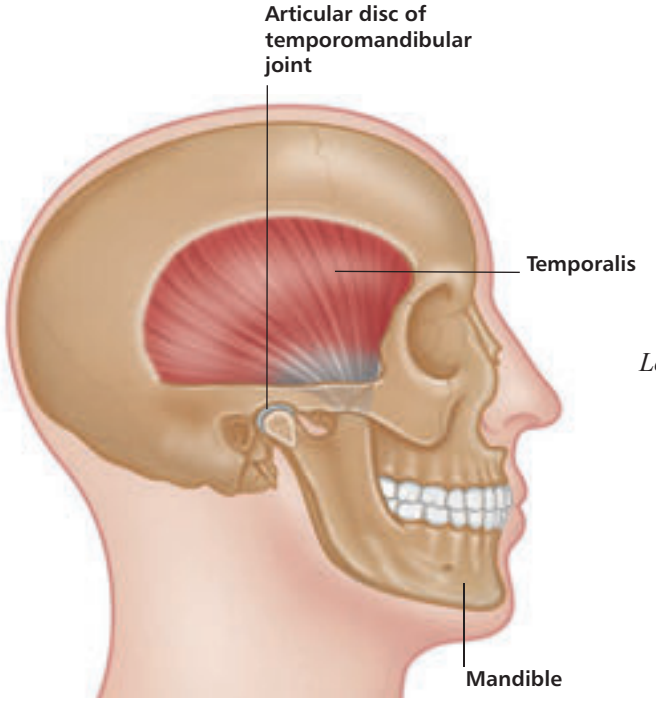
Precautions

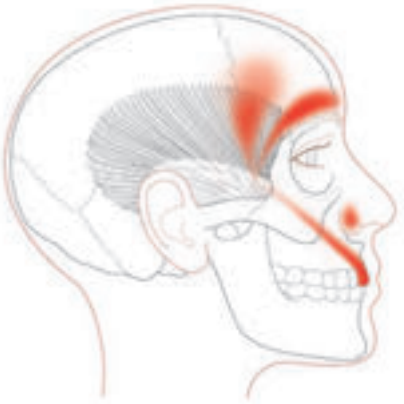
No special precautions.

Occipitalis, side lying or supine, with head rotated to opposite side.

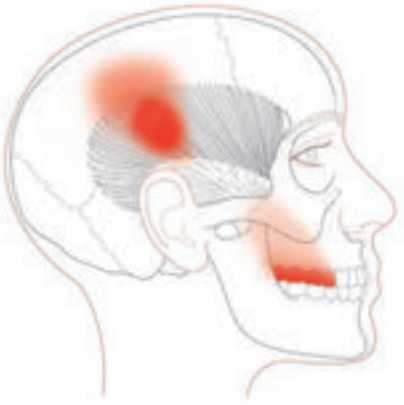
Frontalis, supine.

TEMPORALIS





Lower front attachment TrP.



Lower rear attachment TrP (in front of ear area).

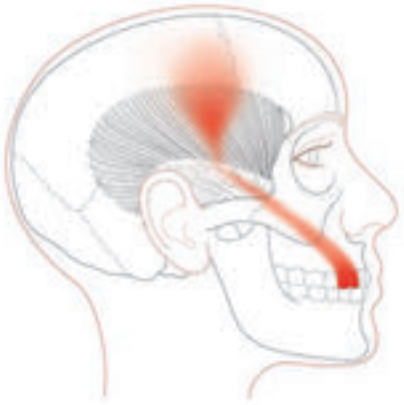
Latin, *temporalis*, relating to the side of the head.

Origin
Deep surface of the temporal fascia, and the entire fossa. The floor of the fossa is made up of the zygomatic, frontal, parietal, sphenoid, and temporal bones.

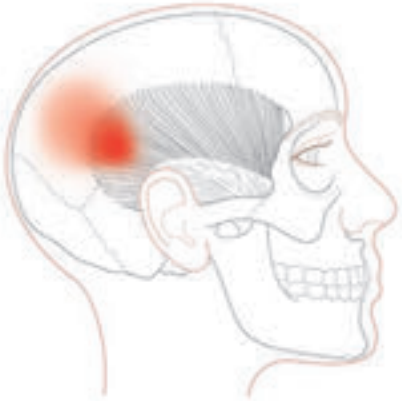
Insertion
Medial/lateral apex and deep surfaces of the coronoid process of the mandible, and anterior border of the ramus of the mandible.

Action
Closes the jaw (elevates the mandible), assists side-to-side deviations of the mandible and clenching of the teeth. Pulls the ears up to create tension across the scalp.

Nerve
Anterior and posterior deep temporal nerves from the trigeminal V nerve (mandibular division).



Lower center attachment TrP.



Central temporalis TrP (behind ear point).

TEMPORALIS

KINETIC CHAIN COMMENT

The temporalis and masseter are synergists. An overdeveloped upper trapezius can be an overlooked contributor to problems associated with these muscles. A short temporalis leads to teeth clenching, which can damage the sensitive proprioceptive covering on the teeth. Temporal dysfunction can ensue, with loss of balance, vertigo, nausea, hearing difficulties, tinnitus, trigeminal neuralgia, and optical problems. The neck, face, and head muscles are as important to global muscle function as the core (lumbopelvic-hip complex). Habits such as chewing gum can cause repetitive stress and strain.

MYOFASCIAL TRIGGER POINT COMMENT

One must appreciate the chain effect that an inhibited masseter could have on this muscle. The temporalis and masseter may develop myofascial trigger points in an effort to provide much-needed tension. A forward-head posture is most likely the evident posture. Pain passes upward and over the forehead on the ipsilateral side. Pain spills over just above the ear and into the nuchal line of the occiput. The temporalis should be considered in all headache patients. Pain in the upper or lower teeth and gums is the most common pain pattern with this muscle. A deep pain has also been reported over the eyebrow and occasionally into the same side and back of the head. The treatment of other muscles on the basis of their pain referral patterns, if associated with this area, should also be carried out as part of the myokinetic chain.

PRACTITIONER GUIDELINES

Patient positioning

Patient is supine or side lying, with the head rotated to the opposite side.

Needle type

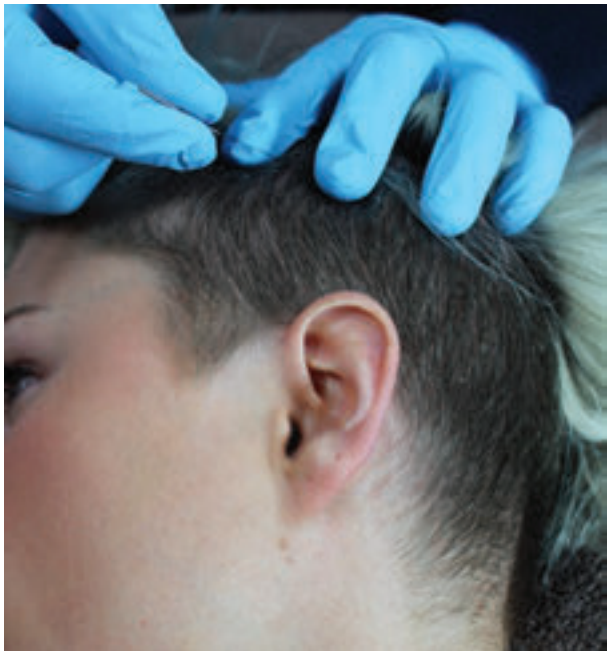
Use 0.14 to 0.16mm × 15mm needle.

Needling directions

Locate and avoid the temporal pulse. Locate and secure the myofascial trigger point within the palpable taut band using flat palpation. Insert the needle and direct it toward the temporal fossa at a shallow angle.

Precautions

Palpate the temporal artery, which bifurcates into a frontal and a parietal portion. A shallow angle of needle insertion is recommended in order to avoid the deep anterior and posterior temporal arteries.



Temporalis, supine with the head turned to the opposite side.