TRAVELL, SIMONS & SIMONS'

Myofascial Pain and Dysfunction

THE TRIGGER POINT MANUAL

THIRD EDITION

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Chapter 72

Trigger Point Injection and Dry Needling

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1. INTRODUCTION

Two different treatment methods use the insertion of needles to inactivate trigger points (TrPs): wet needling and dry needling (DN). Wet needling refers to the injection of a substance into a TrP through a hypodermic needle, whereas DN refers to the insertion of a filiform needle into a TrP without the introduction of any substance. Both needling therapies can effectively manage myofascial pain, but it seems that DN results in increased postneedling soreness in some muscles.[1] Generally, Simons et al[2] recommend TrP injections (TrPI) of a local anesthetic without corticosteroid and no adrenalin. Effective treatment using either TrPI or DN depends on mechanical disruption and inactivation of the active loci in the TrP.[1] Under particular circumstances, the injection of Botulinum toxin A (Botox A) has been proposed. Inactivation of TrPs by injecting Botox A depends on its specific pharmacologic effect on the motor endplate and may be an effective solution for those patients with myofascial pain who are not responsive to TrPI with local anesthetics or DN.

A single TrPI or DN treatment should be defined as the needling of one TrP site, despite the number of times some solution has been deposited within that one TrP site. One TrP site has a highly variable number of active loci that must be inactivated, and all of the loci in one TrP can be needled with one skin penetration. Using a nonmyotoxic local anesthetic or DN, many needle movements within the TrP site are normally required. When a local anesthetic is used, clinicians should inject only a small amount (<1 mL) at any one location within the TrP site. The clinician must obtain twitch responses from all of the remaining active loci in that TrP in order to ensure effective treatment.

Some clinicians depend on the injection of large amounts of extremely myotoxic drugs like Botox A or concentrated long-acting local anesthetics in the general vicinity of a point of tenderness, hoping to inactivate TrPs. When myotoxic drugs are considered unavoidable for injection of TrPs, it is much better to inject small amounts precisely where the contracture knots of the TrPs are located. Selective injection of small amounts of these substances wherever the needle elicits a local twitch response (LTR) in a TrP is much less damaging to the muscle as a whole, and is just as effective as a large amount.

When documenting TrPI or DN, clinicians should specify the muscle needled, any adverse event, and the postneedling outcomes in regard to pain, activity limitations, and participation restrictions. These outcomes will be directly related to postneedling care which should include instruction in proper resting and sleeping postures, activity modification, self-stretching exercises, and a carefully preplanned therapeutic exercise prescription. Section 5 of each muscle chapter of this textbook contains vital information for patient self-management to improve the efficacy of manual therapy, TrPI, or DN treatments. Manual therapy, TrPI, or DN used in isolation will typically not result in an effective therapeutic outcome for the patient.

2. MANUAL THERAPY OR NEEDLING THERAPY

The decision whether to treat TrPs by manual therapy, TrPI, or DN depends strongly on the training and skill of the clinician. Ideally, all approaches should be equally available to the patient and used when indicated. In fact, some studies conducted in the cervical spine reported no differences between manual therapy of upper trapezius active TrPs.[3-5] Manual methods are noninvasive, adaptable for the patient to use for self-treatment, and can be used to release multiple TrPs at the same time in one muscle or a group of TrPs in several muscles that function synergistically. Manual therapy may also address the associated articular (joint) dysfunctions that could be activating or perpetuating the TrPs. However, manual methods are more likely to require several treatments, and the benefit of treatment may not be as fully apparent for a day or two, when compared with TrPI or DN. Manual methods are specifically indicated when the TrP is acute, when the goal is to train the patient in methods of home self-management, when the patient is needle-shy or has needle phobia, or when the TrPs in the muscle belly are not readily accessible to TrPI or DN.

One well-performed TrPI or DN treatment can fully inactivate TrPs quickly, which is reassuring to both the patient and the clinician. Identification and needling of active TrPs can produce impressive results. Success depends strongly on the recognition of the referred pain pattern, accuracy of the clinician's palpation, and the ability to direct the needle accurately. This accuracy depends strongly on the precision with which the TrPs are localized and on the manual skill of the clinician.

Trigger point injection or DN is indicated when a few TrPs remain that are unresponsive to manual methods, when skilled manual TrP therapy is not available, when there are only a few relatively acute TrPs and the treatment time is limited, and when the patient has hyperuricemia and symptoms of gout. Needling can be helpful when the muscle cannot be directly stretched for mechanical reasons or when stretch should be limited because of hypermobility.

Patients with both fibromyalgia and myofascial TrPs are much more sensitive to painful therapeutic techniques than are patients with TrPs without fibromyalgia. Clinically, patients with both conditions could respond to TrPI or DN, but they do not respond as well as patients with myofascial TrPs alone.[6]

It is a serious mistake to judge the efficacy of TrP treatment by manual methods or by needling if the clinician was not adequately trained and experienced in the techniques used for the muscles and dysfunction treated. When patients give a history of being treated for TrPs without benefit, careful questioning makes it clear that treatment was given without adequate manual examination for TrPs or was not performed in a manner that one would expect to be effective. One of the main reasons for treatment failure, seen clinically, is the lack of postneedling treatment care and instructions or failure to recognize or address systemic perpetuating factors (see Chapter 4).

3. WET NEEDLING OR DRY NEEDLING

Clinicians may consider the potential risks and benefits of utilizing TrPI with solutions versus application of DN techniques. Trigger point injections are administered using hypodermic needles, a hollow needle through which a solution is injected at the TrP site.

Local anesthetics, corticosteroids, and neurolytics are substances commonly used in pain management[7] and are also used in solutions for TrP management. Trigger point injection involves depositing approximately 0.2 mL of solution at the TrP.[2] Some studies have used larger volumes up to several milliliters. For example, Ay et al[8] used 2 mL of 1% lidocaine injection for comparison with DN in conjunction with exercise and found no significant differences between the groups relative to pain level, cervical range of motion, and depressive mood; both groups benefited.

Hypodermic needles used for TrPI have a beveled, cutting edge. Filiform needles have a conical point. This difference should be considered when treating areas close to visceral anatomy such as pleural, vascular, or neural structures. The use of a noncutting needle may reduce the risk of laceration of these tissues that could lead to complications such as bleeding, pneumothorax, or nerve injury. DN may be preferable if injecting close to a nerve to avoid possible anesthetic block. Also, the use of DN prevents inadvertent intervascular deposition of treatment solution. It is prudent to aspirate, especially in the vicinity of large vessels, prior to injecting to assess that the needle has not entered a vessel. Accurate injection at deep sites, near neurovascular structures, or in patients who are obese may be improved by the use of ultrasonic or electromyography (EMG) guidance.[9,10]

As with DN, the goal is to accurately locate and deactivate the TrP loci. Obtaining LTRs is similarly desirable. Mechanical disruption of the TrPs is effective by DN as well as needling in conjunction with TrPI of solution. Good technique and provocation of an LTR are important in obtaining a positive result, whether using DN or TrPI of a local anesthetic.[11] Nevertheless, the topic of the LTR during DN is currently under debate.[12]

Myofascial pain often has a chronic component, frequently requiring serial treatments. Clinicians may adjust their approach, including needle or solution options, at subsequent sessions to assess the best individualized treatment approach. Adequate treatment adjustment trials are appropriate prior to determining that TrPI or DN is not effective for a particular patient as long as effective postneedling care and home self-management is employed.

Practical considerations may also drive the decision to use DN rather than TrPI solutions. Some clinicians may not have the option to utilize injection therapy within professional licensing parameters. Reimbursement issues and price point of supplies may also bias the clinician's choice.

Studies of TrPI have been limited by heterogeneity of subject groups and small sample sizes. Pain is complex, and multiple factors can affect fluctuations in pain intensity, quality-of-life measures, and perceived relief. Limited high-quality randomized controlled trials (RCTs) are available for review. A majority of the studies available involve treatment of the neck and upper quadrant.[13] Additional research would be helpful in assessing optimal treatment approach and response to specific patient types. Huang and Liu[14] found that abdominal muscle TrPI with lidocaine 2 weeks prior to menstruation yielded improvement in primary dysmenorrhea. However, no comparison with DN treatment was included in this study.[14]

There is little consensus regarding the superiority of either DN or TrPI. A meta-analysis demonstrated that both wet needling and DN were effective for neck and shoulder myofascial pain without clear differences between the needling therapies.[13] Another systematic review found no differences between DN and wet needling using lidocaine after treatment or at follow-up intervals of 1 month or at 3 to 6 months.[15] Nevertheless, some studies showed evidence that lidocaine injection may be more effective for pain reduction than DN at 4 weeks.[16,17] An older randomized, double-blind crossover study compared the use of 0.5% bupivacaine, 1% etidocaine, or physiologic saline. Pain categories measured prior to treatment and 15 minutes, 24 hours, and 7 days after treatment indicated the use of the local anesthetic injections to be preferable over saline.[18] Although the sample size was small (n = 15), the crossover component of this study limits heterogeneity of the comparison groups and compares response of different treatment solutions on the same patients.[18]

In clinical practice, patients are generally treated with more than one session. A double-blinded study of individuals with episodic tension-type headache compared TrPI using saline to 0.5% lidocaine and compared single injection with a series of five injections.[19] The results indicated that repeated lidocaine injections showed improvement at 2, 4, and 6 months, but only the group treated with a series of lidocaine injections showed significant changes at 6 months, suggesting multiple lidocaine injections may be more effective than a single treatment.[19] Treatment frequency and duration may be factors to consider when reviewing comparative studies.

4. SELECTION OF NEEDLE GAUGE

The clinician must determine an appropriate needle gauge and length to access the target tissue. Choice may vary greatly, from 30-gauge ½ in to treat the anterior border of the upper trapezius to ultrasound-guided 20-gauge 3.5-in spinal needle to address the piriformis muscle. In thick subcutaneous muscles, such as the gluteus maximus or paraspinal muscles, a 21-gauge, 5-cm (2-in) hypodermic needle, or 0.30 × 50 mm filiform needle is usually necessary. For TrPI or DN technique, the needle should be long enough to reach the TrP without inserting the needle to its hub. A 21-gauge, 6.4-cm (2.5-in) hypodermic needle or a 0.30 × 60 to 75 mm filiform needle is generally long enough to reach TrPs in the deepest muscles, such as the gluteus minimus, quadratus lumborum, and psoas muscles. The spinal needle is not as effective for TrP injection as the hypodermic type needle because the spinal needle may push the TrP aside, rather than penetrating it because of its flexibility and diamond-shaped tip.

It would seem intuitive that a smaller gauge needle would be less painful for the patient. When initiating TrPI, patients will often ask, "how big is the needle?" Some authors, including Simons, hypothesize that a larger gauge allows for more accurate localization and improved mechanical disruption in treatment of TrPs with TrPI or DN. There is limited data on response as related to needle gauge. Yoon et al[20] measured treatment efficacy comparing 21, 23, and 25 gauge hypodermic needles in individuals with TrPs in the upper trapezius muscle followed up to 14 days after treatment. All the groups improved and, interestingly, there was no difference in the pain perception of injection needle size. Nevertheless, although no difference in pain relief scores was noted, 21 or 23 gauge needles were more effective in SF-36 health survey scores.[20]

A knowledge of anatomy, the depth of target tissue, and the clinical status of the patient are all factors to be considered by clinicians when choosing needle gauge and length for TrPI or DN. Larger gauge needles may offer less flexibility and possibly more accurate localization and treatment of TrPs. Muscles that are deep or in close proximity to neurovascular structures may best be accessed utilizing ultrasound guidance. Consideration may be given to using a smaller gauge in patients being treated with capillary fragility, platelet disorders, or anticoagulant agents such as warfarin or novel oral anticoagulants.

Coagulopathy may be inherent or medically induced and should be included in patient evaluation prior to embarking on TrP management by invasive techniques, such as needling or even with some manual therapy techniques. It is prudent for patients on warfarin to check for current monitoring and stability of international normalized ratio (INR) within range. Consider consultation with the patient's hematologist in the setting of coagulopathy, such as platelet dysfunction. Also, consider the anatomy of the target site to assess that the treatment area is readily compressible (hemostasis) to limit bruising or bleeding.

Clinically, a 30-gauge needle is effective for TrPI with the effect of obtaining LTR and clinical response with less bleeding noted. A 30-gauge needle has a nominal outer diameter or 0.30 mm that corresponds to a fairly common filiform needle diameter choice for use in DN techniques. Ga et al[21] compared the efficacy of treatment with a 0.30 mm DN compared to TrPI using 0.5% lidocaine administered 25-gauge needle in elderly patients. Although the with a 25-gauge needle has a greater diameter, there was no significant difference in postinjection soreness or in improvement of cervical range or motion or myofascial pain.

5. CLINICAL GUIDELINE FOR APPLICATION OF NEEDLING THERAPIES

Prior to performing any needling technique (TrPI or DN), the clinician should consider patient positioning, medical history with regard to possible increased bleeding tendency, needle selection, proper cleansing, painless skin penetration, and the value of preinjection blocks.

The patient should be positioned in recumbence (supine, semisupine, side-lying, prone, or semiprone) for any TrPI or DN technique to avoid falling associated with psychogenic syncope. When the patient sits on a chair or is standing, needle therapies can be hazardous in susceptible individuals.[22,23] Recumbence also facilitates manual palpation of the TrPs because the patient is typically more comfortable and relaxed. It is also easier to adjust muscle tension so that the bands containing TrPs stand out in a background of relaxed muscle fibers.

Trigger point injection and DN are not sterile techniques. The area to be needled should be properly cleaned with alcohol or soap and water. The needles used are sterile, single use, and disposable. Nevertheless, there is an ongoing debate whether disinfection of the skin or the use of gloves is necessary or not, and guidelines vary in different countries and regions.[24,25]

Some patients are afraid of the skin pain caused by needle penetration. This fear of the needle is usually acquired in childhood and creates obstacles to the therapeutic alliance.[26] Most patients find the sharp skin pain more threatening than the deep aching and more severe pain of the needle contact with the TrPs. The skin pain is avoidable with the use of cold anesthesia for TrPI or in the case of DN holding the tube firmly against the patient's skin prior to tapping the needle through the skin.

In adults, vapocoolant spray can provide cold anesthesia[27,28] to effectively block nerve conduction when the skin temperature falls to 10°C (50°F). After carefully disinfecting the skin with alcohol, vapocoolant spray is applied from a distance of about 45 cm (18 in) for 5 or 6 seconds, and then the needle is inserted quickly after the spray has evaporated leaving the skin dry.[26,29] For young children who dislike the sudden cold impact of the vapocoolant jet stream, a sterile, fluffy, small cotton ball is saturated with vapocoolant until it is dripping wet. The wet cotton is held *lightly* against the skin for about 10 seconds and then removed. At the instant the skin dries, the needle is inserted painlessly.

Three less reliable, but more convenient, techniques that can be combined are to (1) insert the needle *very quickly* through the skin with a flick of the wrist or with a very firm tap with a DN, (2) place the skin under tension so that the additional tension of the needle penetration is hardly noticeable (this can be done by the clinician strongly spreading the fingers apart against the skin and inserting a needle between them), and (3) increase skin tension by pinching a fold of the skin between the thumb and fingers and inserting the needle through the tightly folded skin. When the skin has been cleansed with an alcohol wipe, a film of liquid alcohol can remain for seconds. If the needle is inserted through the wet alcohol, it produces a stinging sensation because the needle carries some of it into the skin. This can be avoided by simply waiting until the alcohol dries. The particular technique used is less important than the communication to the patient that the clinician cares and knows how to insert the needle painlessly.

Prior to TrPI or DN, the patient should be warned that successful needle contact with TrPs may produce a flash of distant pain and likely will cause the muscle to twitch. The patient should be asked to note where that pain is felt, permitting an accurate description afterward of the pattern of pain referred by the TrPs. In this way, the clinician can confirm the referred pain pattern of that TrP, and the patient can realize the connection between the pain and the TrP in that muscle (pain recognition or not). This reassures both the clinician and the patient as to the importance of inactivating it. Patients learn to welcome this painful harbinger of a successful TrPI or DN treatment and future relief.

A preinjection block may be used prior to the TrPI procedure. It is now well established that even brief exposure to considerable pain can cause neuroplastic changes in the spinal cord that tend to enhance sensitization and pain. For patients who are particularly pain-sensitive, or who have found the pain produced by needle encounter with TrPs seriously distressing, a preinjection block can be helpful. This procedure must be adopted with due caution. It is described in detail by Fischer[30] who presents two methods: one involves diffuse infiltration of a local anesthetic proximal to the area to be injected and the other involves infiltration of the entire TrP area with a local anesthetic before needling individual active loci. It is important, if a clinician does these infiltrations, to use 0.5% procaine because of its lower myotoxicity, its relative innocuousness if a vessel were accidently injected, and the more rapid recovery of normal nerve function.[30]

Although there are a number of alternate TrPI and DN techniques now in use, the following precision technique is the one that was presented by Simons et al.[2] It is a basic technique that is applicable to TrPs in any muscle location that can be reached with a needle.

Localization of a TrP is done mainly by the practitioner's sense of feel, assisted by patient expressions of pain, and by visual observation of LTRs.

The two methods of palpation (cross-fiber flat or pincer palpation) are utilized to identify TrPs. The more precisely the TrP is localized, the more effective the needling technique will be. When flat palpation is used to locate the TrP for needling, its position can be confirmed precisely by pushing the nodular TrP back and forth between two fingers (Figure 72-1A and B). The TrP can be fixed for needling by pinning it down midway between the index and middle finger tips (Figure 72-1C). This identifies for the clinician the plane that passes through the TrP perpendicular to the skin. The needle can be aimed and directed half way between the fingers precisely in that plane and angled to whatever depth is necessary to reach the TrP. The needle should be considered as an extension of the clinician's finger; the clinician, therefore, palpates the TrP with the tip of the needle and penetrates it. When pincer palpation is used to locate the TrP, the degree of tension placed on the muscle fibers can be fine-tuned by varying the distance that the muscle is pulled away from underlying tissues. The nodule is located by rolling sequential portions of the taut band between the clinician's digits (Figure 72-2). For needling, the TrPs are held tightly between the thumb, index, and middle fingertips; the needle is typically directed toward the TrPs; and the clinician's fingers on the underside of the tissue. The needling procedures for both flat and pincer palpation should

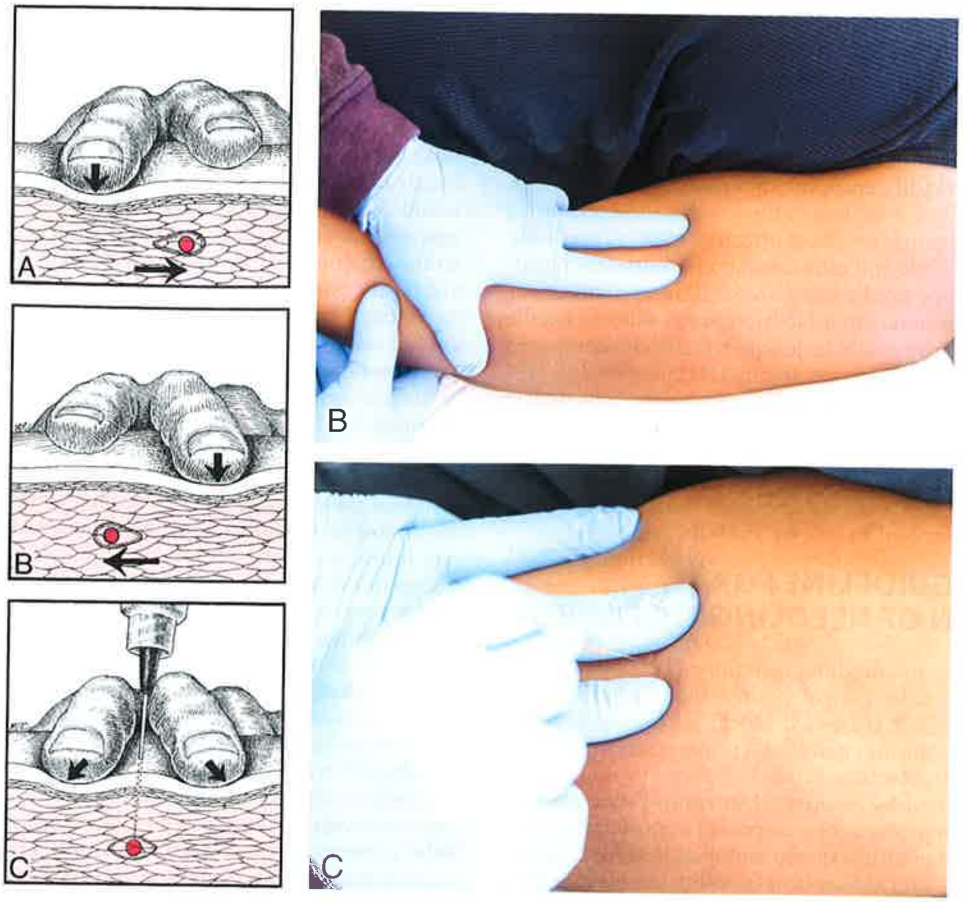

Figure 72-1. Cross-sectional schematic drawing of flat palpation to localize and hold the TrP (dark red spot) for TrP injections or dry needle. A and B, Use of alternating pressure between two fingers to confirm the location of the palpable TrP. C, Positioning of the TrP half way between the fingers to keep it from sliding to one side during the needling.

be conducted similar to manual palpation techniques for the identification of TrPs.

For TrPI or DN of TrPs when employing any method of palpation, the muscle fibers of the taut band are placed on sufficient stretch to take up any slack but not enough stretch to cause additional pain. This tautness is necessary to help hold the TrP in position. If the muscle is slack, there is a tendency for the dense contracture knots of the TrP to slide to one side as the needle tip encounters them.

To needle TrPs in superficial layers of muscle close to the skin, the needle tip can be brought precisely to the TrP by first carefully locating TrP with the finger and then, after inserting the needle subcutaneously, pressing it against the finger through the skin to accurately localize the TrPs. Finally, the needle tip is directed into the TrP by means of this "tactile vision" provided by palpating both the needle and the TrP at the same time.

The same technique is useful for TrPI or DN in the area of the muscle opposite the needling site when using a pincer grasp. The location of the needle and the TrP can be identified by palpation as the needle approaches the skin after penetrating most of the muscle.

Trigger point injection or DN TrPs is a full-time job for both hands of the clinician. The needling hand is busy placing the needle and controlling the plunger of the syringe for TrPI. The palpating hand constantly maintains hemostasis and often must fix the TrP to help the needle penetrate it. It also must be ready to detect any palpable LTRs. Hemostasis is important. Local bleeding irritates the muscle, causes postinjection soreness, and can produce unsightly ecchymosis. Ecchymosis is usually preventable; when it occurs, only time eradicates it.

To prevent bleeding, the fingers of the palpating hand of the clinician should be spread apart, maintaining tension on the skin (Figure 72-3A) to reduce the likelihood of subcutaneous bleeding where the needle has penetrated during TrPI. Bleeding is considered a minor adverse event when performing DN. Also, during TrPI or DN, the fingers exert pressure around the needle tip to provide hemostasis in deeper tissues. When the angle of the needle is changed, the direction of the pressure changes with it. The pressure should be applied throughout the needling procedure. As the needle is withdrawn, one finger slides over the track of the needle and instantly applies pressure where the needle was. If visible bleeding develops, prolonged hemostasis and a cold pack should be applied and the patient is warned of a possible "bruised" spot.

Blindly probing an area of diffuse tenderness where there is no palpable band or muscle attachment is futile. Such an area is most likely to be a pain reference zone, not TrPs. Injecting a local anesthetic in the reference zone may temporarily reduce the referred pain, but it does not eliminate the cause of the pain.

The importance of distinguishing between TrPs in the muscle belly, the myotendinous junction, or enthesis when TrPI or DN is applied was illustrated (Figure 72-4) by Fischer.[30] The precision required to penetrate the TrP with a needle is a skill that requires practice. When using flat palpation, as illustrated in Figures 72-1C and 72-3A and B, the needle is inserted between the fingers that have located the TrPs. The needle penetrates the skin 1 to 2 cm away from the TrP so that it can approach it at an acute angle of about 30° to the skin. Adequate tension of the muscle fibers is required to penetrate the TrP. The needle should explore both the deep and superficial fibers of the muscle. For

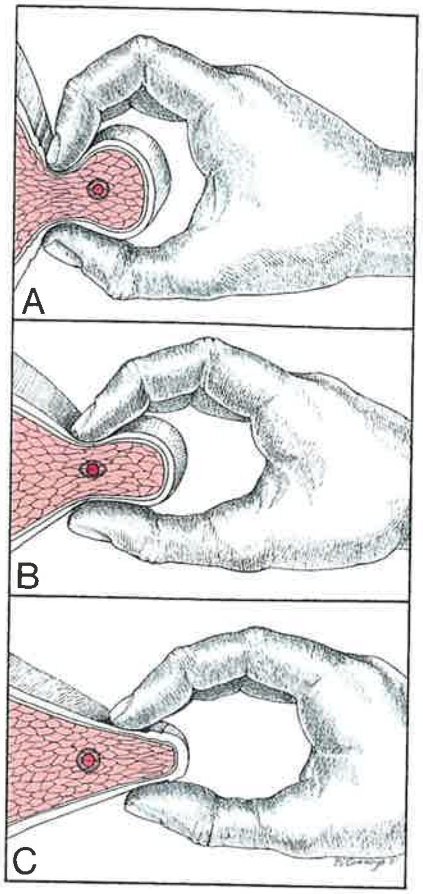

Figure 72-2. Cross-sectional schematic drawing showing cross-fiber pincer palpation of a taut band (black ring) at a TrP (red spot). Cross-fiber pincer palpation is used for muscles (light red) that can be picked up between the digits, such as the sternocleidomastoid, pectoralis major, and latissimus dorsi muscles. A, Muscle fibers surrounded by the thumb and fingers in a pincer grip. B, Hardness of the taut band felt clearly as it is rolled between the digits. The change in the angle of the distal phalanges produces a rocking motion that improves discrimination of fine detail. C, The palpable edge of the taut band is sharply defined, as it escapes from between the fingertips, often with a local twitch response.

TrPI, the syringe may be held between the fingers of the injecting hand, and thumb pressure used against the plunger, which is the method shown in most of the figures illustrating injection in this chapter. Thumb pressure on the plunger slowly introduces small amounts of 0.5% procaine solution as the needle advances within the muscle. This ensures that the procaine is present to relieve pain at the instant that the needle tip encounters an active locus of the TrP. The filiform needle can be easily redirected with the needling hand to induce and eradicate all the LTRs in the area.

The clinician should avoid inserting the needle to the hub where the needle is most likely to break off. Some additional depth of penetration can be safely obtained by compressing the skin and subcutaneous tissues with the palpating hand if needed. Once the skin and subcutaneous tissues have been compressed, the clinician should maintain the compression until the needle is withdrawn.

The dense contracture knots in TrPs often feel, to the practitioner, as if the needle tip has encountered hard rubber that is resistant to penetration and tends to slide to one side, as described by Gold and Travell many years ago. Using the needle as a probe, the TrP sometimes feels like a dense globule, 2 to 3 mm in diameter; resistance to penetration helps identify it.[31] Occasionally, when the needle makes contact with the TrP, it may feel "gritty." Adequate tension of the muscle helps stabilize the position of the TrP to permit precise penetration by the needle, especially for deep TrPs that cannot be easily fixed in position by manual palpation.

If an LTR and referred pain were elicited from the TrP prior to TrPI or DN, then both should be observed when the needle penetrates the TrP during the procedure. Hong[32] showed that when needle penetrations of a TrP produced LTRs, they were much more likely to result in subsequent pain relief than penetrations that did not elicit an LTR. Following effective needling, most TrP characteristics should have minimized or disappeared. The taut band is more relaxed following effective needling and may no longer be distinguishable by manual palpation. Nevertheless, the topic of the LTR during DN is currently under debate.[12]

Sometimes a cluster of TrPs are present in one part of the muscle. This fact is often recognized when the muscle is initially palpated for TrPs. When one of these TrPs has been inactivated, the area is peppered in a fan-like manner or in a full circle[32] in an effort to ensure that all remaining TrPs in the group are inactivated, as illustrated in Figure 72-3B. Following each probing movement, the needle tip must be withdrawn to the subcutaneous tissue and redirected before the next movement. When this probing search of the region is completed, the site is palpated for any remaining spots of tenderness. If another one is found, it is accurately localized with the fingers and needled. All potential tender spots in that region should be eliminated before withdrawing the needle through the skin, if possible. Box 72-1 describes a general guideline for TrPI or DN procedures. Boxes 72-2 and 72-3 identify absolute contraindications and precautions to TrPI or DN, respectively. Box 72-4 lists caveats from Travell and Simons regarding TrPI or DN.

Hong[32] introduced two needling techniques: one was a safer way to hold the syringe and the other was a different way to perform the injection itself, that is DN. When a clinician injects a TrP in locations that pose a hazard should the patient make a sudden unexpected movement (such as a startle reaction, sneeze, and/or cough), Hong[11,32] recommends a way to hold the syringe

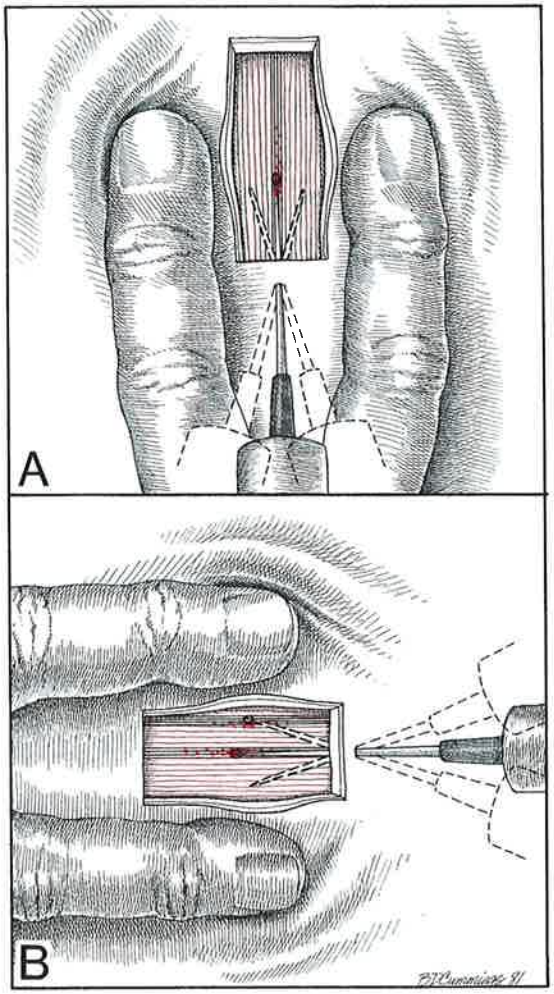

Figure 72-3. Schematic top view of two approaches to the flat needling of a TrP area (dark red spot) in a palpable taut band (closely spaced black lines). A, Needling away from fingers, which have pinned down the TrP so it cannot slide away from the needle. Dotted outline indicates additional probing to explore for additional adjacent TrPs. The fingers are pressing downward and apart to maintain pressure for hemostasis. B, Needling toward the fingers, with similar finger pressure. Additional TrPs are often found in the immediate vicinity by probing with the needle.

Box 72-1 General guidelines for TrP injection or dry needling

Palpate and identify anatomic landmarks
Palpate the taut band with cross-fiber flat or pincer palpation
Identify the TrP and fix with either a pincer grasp or flat palpation
Needle with straight in and out motions
Elicit a local twitch response (or referred pain)
Draw the needle back to the subcutaneous tissue and redirect the needle to treat other TrPs in the same or nearby areas
Provide hemostasis immediately upon withdrawal of the needle
Apply a postneedling intervention for reducing postneedle soreness

Box 72-2 Contraindications to TrP injection or dry needling

Inadequately trained practitioner
Needle phobia
Cognitive impairment
Patient's unwillingness to be treated
Patient's inability to give consent
Local skin lesions
Local or systemic infections
Needling directly over implants

Box 72-3 Precautions to TrP injection or dry needling

First trimester of pregnancy
Vascular disease
Abnormal bleeding tendency (anticoagulant therapy, thrombocytopenia)
Compromised immune system
Intercostal area
Needle aversion

that is safer than the usual way. His technique ensures that the syringe will move with the patient and not enter an unintended tissue, and that the finger on the plunger of the syringe will move with the syringe and not cause an accidental injection. The hand holding the syringe must be firmly supported by the patient's body; this is readily accomplished with his technique, as illustrated in Figure 72-5. The syringe is held between the thumb and lesser fingers, and the plunger is depressed with the index finger. This technique is particularly valuable when injecting over the lung or when the needle is directed toward major arteries or nerves.

6. NUMBER OF NEEDLING SESSIONS

Note the definition of one injection at the beginning of this chapter. The number of TrP sites that need to be injected per visit and the number of visits required are strongly dependent on the patient's condition and the practitioner's skill and judgment. To date, no medical specialty has adopted the diagnosis and treatment of myofascial TrPs as an official part of the training program, nor have *specialty* standards of training and practice been established for this diagnosis. The *International Association for the Study of Pain* has published recommended standards of TrP training.[33]

Active myofascial TrPs with no long-lasting perpetuating factors or additional tissue damage due to mechanical injury to other structures should resolve with one or two needling sessions. This estimate is especially true if the patient has been provided with adequate self-management techniques, which are discussed in Section 5 of each muscle chapter. When initial TrP therapy is delayed and symptoms have not subsided with time, the longer the period of delay before starting TrP therapy, the higher the number of TrPI or DN techniques that will be required over a longer period of time.[34] Some chronic TrP problems could involve several injections over a span of months of treatment. In this situation, the primary guideline is that the period of relief from TrP pain and dysfunction should become progressively longer

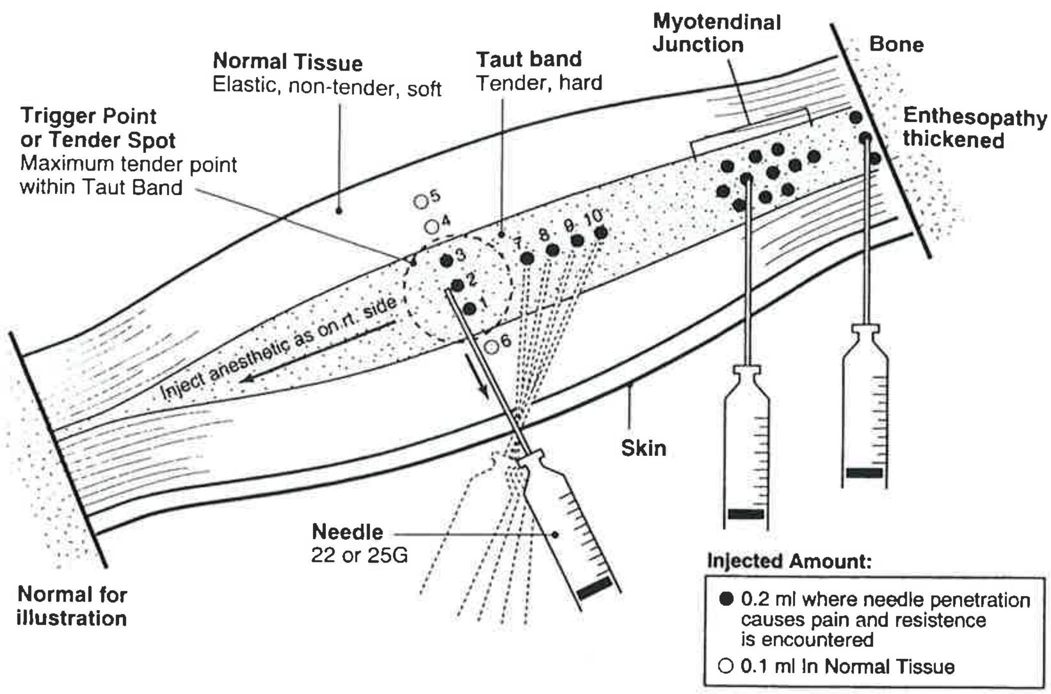

Figure 72-4. Diagrammatic representation of preneedling sites (open circles), and needling sites (solid circles) of local anesthetic in relation to the TrP (large broken circle). The taut band is represented by the enclosed stippled area. This diagram distinguishes the mid-muscle belly TrP within the broken circle from the myotendinous junction and at the attachment of the tendon to the bone. Each of these three TrP regions can be identified by the individual spot tenderness and anatomic locations. No rationale is apparent for needling the part of the taut band that lies between solid circles numbers 7 to 10. Reproduced with permission from Fischer AA. New approaches in treatment of myofascial pain: myofascial pain–update in diagnosis and treatment. *Phys Med Rehabil Clin North Am*. 1997;8(1):153-169.

with successive TrPI or DN treatments, and the patient should be performing a self-management program between sessions.

When a patient demonstrates multiple active TrPs in functionally related muscles, there is a distinct advantage to inactivating them as a group. Thus, 5 or even 10 needling treatments at one visit may be appropriate but could be more than tolerable. Because a properly performed and effective needling treatment produces an LTR that is often associated with considerable pain, there is a limit as to how many painful injections should be performed at one visit given the patient's emotional and autonomic distress level.

The presence of unrecognized perpetuating factors (see Chapter 4) will lead to unnecessary TrPI or DN. The presence of associated joint dysfunctions that need manipulation can cause poor response to needling and prompt recurrence of the TrP activity. After appropriate treatment of the joint(s), one or two more TrPI or DN sessions should resolve the problem. The presence of concurrent fibromyalgia will increase the number of needling sessions required and can justify recurrent TrPI or DN every 6 to 8 weeks because fibromyalgia acts as a perpetuating factor that has no cure. Inactivating TrPs can provide significant pain relief for many of these patients.

Box 72-4 Travell and Simons caveats for TrP injection or dry needling

By NEVER aiming the needle at an intercostal space, the clinician avoids the distressing complication of a *pneumothorax*. As a resident, Dr Travell found in her early experience of doing many pleural taps for pleural effusions, that patients consistently reported a salty taste in the mouth whenever the pleura was punctured. The patient might say, "Oh, I can taste the solution." When the lung is punctured and collapses, dyspnea, cough, and chest pain characteristic of a pneumothorax follow.

A needle is prone to break where it attaches to the hub. The needle should never be inserted solidly to its hub because of the difficult situation that would ensue should it break off at the hub and disappear under the skin. Recovering the needle can be a time-consuming, frustrating process for the clinician. A long enough needle should be used, or the skin compressed around it, to ensure that some of the needle projects above the skin surface.

The location of the needle tip can readily be misjudged when using a long slender needle. It is especially important to insert the needle straight and avoid any side pressure that might bend the needle.

When the tip of a needle contacts the bone, the impact frequently curls the tip to produce a "fishhook" burr that feels "scratchy" and drags as the needle is drawn through tissues; it causes unnecessary bleeding and pain and the needle should be replaced immediately. It is especially important to avoid using such a barbed needle for TrPI or DN TrPs in muscles like the scalene that lie near nerve trunks.

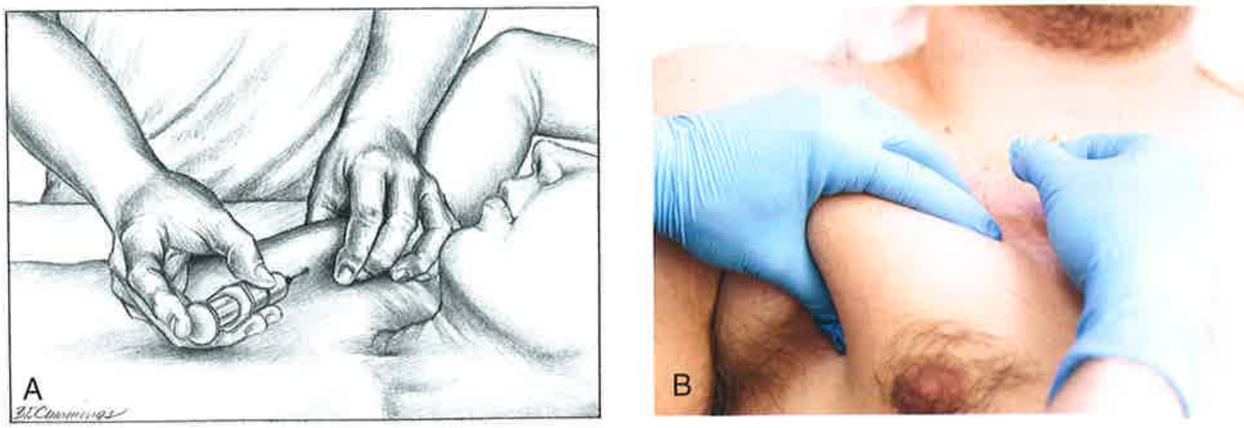

Figure 72-5. Trigger point injection using a technique for holding the syringe that minimizes the danger of accidentally inserting the needle farther than intended if the patient makes a sudden unexpected movement. A, Drawn from an original photograph, courtesy of John Hong, MD who first described this method. Hong C-Z. Myofascial trigger point injection. *Crit Rev Phys Med Rehabil.* 1993;5:203-217. B, Similar technique shown for dry needling.

7. POSTNEEDLING PROCEDURES

Postneedling-induced soreness is thought to be a consequence of the neuromuscular damage generated by needling insertions into the muscle.[35] The presence of postneedling soreness can be associated with a possible reluctance to receive further needling therapy by the patient, generating patient dissatisfaction and reduction in treatment adherence. In fact, it is highly recommended to advise the patient about the presence of soreness after any needling therapies.[36] Therefore, postneedling procedures should always be implemented into a multimodal treatment approach.

Postural education and positioning, activity modification, self-stretch, and neuromotor retraining following TrPI or DN is an integral part of that treatment. Immediately following TrPI or DN, hemostasis should be applied for a minimum of 30 seconds or longer, the patient should actively move each muscle needled through its full range of motion three to five times, reaching its fully tolerable shortened and fully tolerable lengthened position during each cycle. The muscle usually feels stiff toward the end of full stretch range of motion on the first cycle, less on the second, and begins to feel comfortable through its full range by the third to fifth cycle. It is important that the patient move the muscle slowly to explore the end range of motion for additional release. It is also vital that the patient is able to activate the muscles that have been needled volitionally to improve motor control. An ice pack immediately following the TrPI or DN procedure, stretch,[37] manual compression,[38] or low-load eccentric exercise[39] can be helpful in reducing postneedling soreness and enhancing recovery.

The postinjection stretch is important because it helps to again equalize sarcomere lengths throughout the length of the affected muscle fibers, which relieves the abnormal tension and can eliminate the palpable taut bands.[37] Voluntary movement also relieves residual stiffness at full range of motion, helps the patient appreciate the improved range of motion, and provides the patient stretches that will be incorporated in the home management program. Additionally, this range-of-motion activity establishes the patient's conscious awareness of normal function in that muscle, while reprogramming the cerebellum to incorporate the newly restored full-range capability of the muscle into the patient's daily activities. Establishing a home exercise prescription for the patient following TrPI or DN is vital for treatment success.

Lewit[40] noted muscle soreness after DN and after TrPI with a local anesthetic but made no mention of applying heat or cold as part of the treatment. The postinjection soreness, per se, is not unfavorable if the patient's related pattern of referred pain has been relieved. In fact, some clinicians consider postneedling soreness as a "natural" or "positive" effect indicating that TrP inactivation has been effectively achieved. However, it is wise to let the muscle recover completely from postinjection soreness, which ordinarily lasts at most 48 to 72 hours independent of the postneedling intervention used,[37-39] before needling its TrPs again. Soreness can also be caused by ineffectually needling close to, but not into, TrPs.

If two or three treatments of TrPI or DN fail to eradicate the TrPs in a muscle, repeated TrPI or DN is rarely the answer. The perpetuating factors that are causing recurrence of the TrPs must be identified and managed (see Chapter 4). Box 72-5 lists possible reasons for failure of TrPI or DN to effectively treat the myofascial pain and dysfunction caused by TrPs.

Box 72-5 Possible reasons for failure of TrP injection or dry needling

1. Disregarding perpetuating factors is probably the most important reason for failure
2. Needling a latent TrP, not an active TrP
3. Needling the area of referred pain and referred tenderness, not the TrPs, therefore providing incomplete or temporary relief
4. Failure to needle the TrP itself
5. Inappropriate needle gauge
6. Injecting a solution with an irritating or allergenic bacteriostatic preservative
7. Inadequate hemostasis followed by irritation of the TrP due to local bleeding
8. Overlooking other active or associated TrPs that are contributing to the patient's pain
9. Failure to provide postneedling care including hemostasis, full stretch range of motion, postural and positioning instruction, activity modification, and neuromotor retraining
10. Failure to establish a home exercise prescription that includes intensity, frequency, and duration of the home management program

WET NEEDLING: TRIGGER POINT INJECTIONS

1. LOCAL ANESTHETICS

This discussion is an overview summary in the use of the TrPI solutions for treatment of myofascial pain and TrP dysfunction. Certainly, more specific and extensive research, scientific detail, and clinical opinion are available than can be addressed within the confines of this chapter.

A variety of solutions and combinations have been utilized in TrPI. Local anesthetics have been the most common solution utilized. Lidocaine, bupivacaine, and procaine are most frequently used local anesthetics for TrPI. Simons et al[2] were of the opinion that DN is as effective, but it results in greater postneedling soreness than treatment with the injection of a local anesthetic.[2,11] In fact, Simons et al[2] recommended using small amounts of solution, a few 10ths of a mL at any single location.

Many patients have chronic pain and are candidates to receive serial injections in clinical practice. It is common to make alterations in treatment approach relative to needle and solution choice to identify the optimal response for an individual patient. Treatment approach may also be adjusted for changes in the patient's presentation, such as in episodes of exacerbation or alteration in pain distribution.

Local anesthetics have a favorable safety profile when appropriately administered, mostly in the doses and concentrations used in TrPI. Mechanism of action for local anesthetics involves blockade of nerve conduction by reversibly binding to sodium channels, thus preventing depolarization. Duration of action is determined by protein binding capacity to the sodium channel receptors. Onset, duration, and maximum doses are referenced within Table 72-1. For patients with multiple areas requiring treatment, a lower concentration of local anesthetic may allow more areas to be safely injected.

Local anesthetics fall in to the biochemical category of ester or amide, as determined by the linking bond of the substance. Lidocaine and bupivacaine are commonly used local anesthetics that fall under the amide group. Amide local anesthetics are metabolized in the liver. This mechanism may be a consideration in patients with impaired hepatic function, although the volumes and concentrations of local anesthetics used for TrPI are not large and can be adjusted for patient condition. Procaine is an ester local anesthetic. Ester local anesthetics are hydrolyzed and rapidly metabolized and, therefore, have lower toxicity. Rapid degradation reduces the potential for adverse reaction if inadvertently introduced intravascularly. Para-aminobenzoic acid is a metabolite that can act as an allergen in some patients, but rapid metabolization reduces the likelihood of reaction when used in TrPI. Metabolites of procaine are excreted in the urine.[7] The half-life of local anesthetics may be prolonged in the setting of renal failure,[41] but local anesthetics are routinely used for procedures in renal failure and patients requiring dialysis. The quantity and dilution of local anesthetics used in TrPI would generally not be a concern in patients with renal dysfunction.

True allergy to local anesthetics is rare, accounting for less than 1% of adverse reactions.[42,43] Some patients have sensitivity to the antimicrobial preservative methylparaben contained in multidose vials. Most allergic reactions to local anesthetic are type IV hypersensitivity and have small risk of anaphylaxis. Although allergy testing can be conducted to assess whether or not a specific patient is truly allergic to a local anesthetic, the lack of overwhelming data supporting superiority of wet needling may then predispose the clinician to opt for treatment with DN. A common adverse event that may occur with any injection is a vasovagal reaction, usually secondary to needle insertion rather than to the administration of local anesthetics.

Myotoxicity of local anesthetics is another factor that may influence treatment decisions regarding the type of solution used or the option to use DN technique. The review by Zink and Graf[44] found that the intramuscular injection of local anesthetics frequently results in local myonecrosis, although significant skeletal muscle toxicity is a rare side effect. Myonecrosis persists for 24 to 48 hours until phagocytes invade the area. Intracellular dysregulation of $Ca^{2}+$ appears to be an important element in myocyte injury. This effect is reversible and muscular regeneration occurs within 3 to 4 weeks. Muscle injury is noted to be least with use of procaine and most with bupivacaine. This effect appears to be dose and volume dependent as well as related to serial use.[44] Clinically, myotoxicity and resultant inflammation may be a consideration in patients who experience exacerbation within a few days of TrPI.

Some authors have proposed that the use of a local anesthetic may impair palpatory evaluation of response and treatment of

Table 72-1 Classification and Uses of Local Anesthetics

	Clinical Uses	Usual Concentration (%)	Usual Onset	Usual Duration (h)	Maximum[a] Single Dose (mg)	Unique Characteristics
			Aminoesters			
2-Chloroprocaine	Infiltration	1	Fast	0.5-1.0	1000 + EPI	Lowest systemic toxicity
	PNB	2	Fast	0.5-1.0	1000 + EPI	
	Epidural	2-3	Fast	0.5-1.5	1000 + EPI	Intrathecal route may be neurotoxic
Procaine	Infiltration	1	Fast	0.5-1.0	1000	Used for differential spinal
	PNB	1-2	Slow	0.5-1.0	1000	
	Spinal	10	Moderate	0.5-1.0	200	
Tetracaine	Topical	2	Slow	0.5-1.0	80	
	Spinal	0.5	Fast	2-4	20	
			Aminoamides			
Lidocaine	Topical	4	Fast	0.5-1.0	500 + EPI	
	Infiltration	0.5-1.0	Fast	1-2	500 + EPI	
	IV regional	0.25-0.5	Fast	1-3	500	
	PNB	1.0-1.5	Fast	0.5-1.5	500 + EPI	
	Epidural	1-2	Fast		500 + EPI	
	Spinal	5			100	

Table 72-1 Classification and Uses of Local Anesthetics (*continued*)

	Clinical Uses	Usual Concentration (%)	Usual Onset	Usual Duration (h)	Maximum[a] Single Dose (mg)	Unique Characteristics
Prilocaine	IV regional PNB Epidural	4 1.5-2.0 1-3	Fast	1.5-3.0	600 600 600	Least toxic amide Methemoglobinemia possible when >600 mg
Mepivacaine	PNB Epidural	1.0-1.5 1-2	Fast Fast	2-3 1.0-2.5	500 + EPI 500 + EPI	Duration of plain solutions Longer than lidocaine with EPI, useful when EPI contraindicated
Bupivacaine	PNB Epidural Spinal	0.25-0.5 0.25-0.75 0.5-0.75	Slow Moderate Fast	4-12 2-4 2-4	200 + EPI 200 + EPI 20	Exaggerated cardiotoxicity With accidental IV injection Low doses produce sensory > motor blockade
Etidocaine	PNB Epidural	0.5-1.0 1.0-1.5	Fast Fast	3-12 2-4	300 + EPI 300 + EPI	Motor > sensory blockade

[a]Maximum single dose is affected by many factors; this is only a guide.

EPI, epinephrine; IV, intravenous; PNB, peripheral nerve block.

Modified from Barash PG, Cullen BF, Stoelting RK. *Handbook of Clinical Anesthesia*. 2nd ed. Philadelphia, PA: Lippincott; 1993:206-207; Dreyer S, Beckworth W. Commonly used medications in procedures. In: Lennard TA, Vivian D, Walkowski S, Singla A, eds. *Pain Procedures in Clinical Practice*. 3rd ed. Philadelphia, PA: Elsevier-Saunders; 2011:5-12.

remaining TrPs. Limiting volume and concentration of local anesthetic injected at each site is a consideration. Also, clinicians should seek to localize the site of injection through skilled palpation and elicit an LTR before injecting solution.

Lidocaine without the vasoconstricting agent epinephrine is the most common solution utilized in TrPI. It is readily available and is relatively inexpensive. Lidocaine toxicity may cause central nervous system and cardiovascular effects. Toxicity is rare and dose dependent. It has a relatively immediate onset and duration of action from 30 to 90 minutes. Lidocaine is a category B drug for pregnancy, but is excreted in breast milk.

When reviewing the literature, there is considerable variation in the dosing of lidocaine used for TrPI. Volumes studied may range from 0.2 to 2 mL and concentrations from 0.25% to 2%, factors that may limit comparison between these studies. Lidocaine may be diluted with sterile water to a concentration of 0.25%, and it has been shown to be equivocal or better in effect to concentrations of 0.50% or 1% in a single study.[45] Iwama and Akama[46] showed diluted lidocaine to be less painful and have a longer duration of relief in a study of comparative treatment of trapezius TrPs.

Lidocaine has a pH of 6.3 to 6.4. Some clinicians will buffer lidocaine using 8.4% bicarbonate solution at a ratio of 10:1, with the goal of reducing burning discomfort. Conflicting results are noted in the studies of intradermal lidocaine injection. Matsumoto et al[47] noted that buffered lidocaine significantly reduced discomfort when compared with 10:1 dilution with normal saline, whereas Zaiac[48] found lidocaine with epinephrine diluted at 10:1 ratio with normal saline to be less painful than buffering with lidocaine. These studies were intradermal rather than intramuscular.

A study of the tolerability of intramuscular injection using 1% lidocaine as a diluent for ceftriaxone administration versus buffered 1% lidocaine as a diluent demonstrated no difference in pain or discomfort associated with the injection.[49] If this information may be extrapolated to intramuscular TrPI, there would be little benefit to buffering lidocaine for TrPI. Additional comparisons specific to comfort during TrPI would be helpful.

Procaine, similar to lidocaine, has a rapid onset and short duration of action. Procaine may have a lower myotoxicity effect and was the preferred local anesthetics recommended by Janet Travell, MD. Dr Travell recommended the use of the short-acting local anesthetic diluted to 0.5%, because a higher concentration showed no greater anesthetic effect.[50,51]

Bupivacaine or ropivacaine are longer-acting local anesthetics that are sometimes used alone or in combination for TrPI. Duration of action may last several hours. A comparison of intramuscular injection showed pain related to injection to be less with ropivacaine when compared with bupivacaine; however, this application was not assessed specific to TrP location.[52] Bupivacaine is the more common product cited in clinical use and in research for TrPI. Ropivacaine is more frequently used for procedural anesthesia such as spinal anesthesia or nerve block. Although it may be tempting for patients and clinicians to view longer-acting anesthetics as more powerful or giving longer postinjection soreness relief, longer-acting anesthetics have not been shown to be clearly superior to shorter-acting products for TrPI. There is potential for longer postinjection sensory or motor block if injected closer to a nerve as well as increased myotoxicity.

Finally, clinicians have empirically made additions to TrPI solutions, sometimes on theoretic basis and anecdotal information. Therefore, demonstrated efficacy and potential risks should be assessed before including additives to TrPI solutions.

2. CORTICOSTEROIDS

Corticosteroids have both anti-inflammatory and immunosuppressive effects. They are probably the most common additive to TrPI, although no clear overall benefit has been observed.

Table 72-2 Comparison of Commonly Used Glucocorticoid Steroids[a]

Agent	Anti-inflammatory Potency[a]	Salt Retention Property	Plasma Half-life (min)	Duration	Equivalent Oral Dose (mg)
Hydrocortisone (Cortisol)	1	2+	90	S	20
Cortisone	0.8	2+	30	S	25
Prednisone	4-5	1+	60	I	5
Prednisolone	4-5	1+	200	I	5
Methylprednisolone (Medrol, Depo-Medrol)	5	0	180	I	4
Triamcinolone (Aristocort, Kenalog)	5	0	300	I	4
Betamethasone (Celestone)	25-35	0	100-300	L	0.6
Dexamethasone (Decadron)	25-30	30	100-300	L	0.75

[a]Relative to hydrocortisone

I, intermediate; L, long; S, short.

Adapted from Lennard TA. Fundamentals of procedural care. In: Lennard TA, ed. *Physiatric Procedures in Clinical Practice*. Philadelphia, PA: Saunders; 1995; Dreyer S, Beckworth W. Commonly used medications in procedures. In: Lennard TA, Vivian D, Walkowski S, Singla A, eds. *Pain Procedures in Clinical Practice*. 3rd ed. Philadelphia, PA: Elsevier-Saunders; 2011:5-12.

Simons et al[2] advocated against the use of long-acting steroids for use in TrPI. Steroid preparations with predominantly glucocorticoid activity rather than mineralocorticoid activity are used in pain management procedures (Table 72-2). Adverse effects of corticosteroid injection include facial flushing, depigmentation, and muscle atrophy. Local administration may produce systemic effects such as hyperglycemia in patients with diabetes. Potential risks should be included when considering this option for TrPI. Generally, corticosteroids can be mixed in the same syringe with local anesthetics. Betamethasone should not be mixed with local anesthetics containing methylparaben as a preservative because flocculation of the solution may result[7] (Celestone Package insert).

Results regarding the addition of corticosteroid to TrPI solution are mixed, and therefore, studies should be assessed for the inclusion of a control group. For example, an ultrasound-guided TrPI for piriformis syndrome performed using lidocaine was equivalent to injection using a combination of lidocaine and steroid.[53] Some studies have suggested benefit from the addition of steroid. A study of patients with headache compared treatment with DN, use of 0.25% lidocaine, and 0.25% lidocaine with 0.2 mL decadron 4 mg/mL.[54] Less postinjection discomfort and ingestion of rescue medication was noted in the group injected with a combination of a local anesthetic and corticoid.[54] On the contrary, cortisone injection directed toward the bursa for the treatment of greater trochanteric pain syndrome is a common procedure and did not prove superior to a series of DN TrP treatments.[55]

A case report of treatment of serratus anterior muscle pain syndrome describes the use of 2% lidocaine and 0.5% bupivacaine and 1 mL (40 mg) triamcinolone with a total of 3 mL deposited at each site via ultrasound guidance to target muscle fibers at the TrPs, localized by palpation.[56] A small sample group was treated but showed a fairly robust response in seven of eight patients relative to medication use and pain scale at 3 months and beyond. This patient group was unique in that pain syndrome was triggered following surgical intervention. Also, no control group for treatment without steroid or with DN was involved.[56]

Additional research may show benefit in particular patient types or anatomic locations. Positive steroid response may also be a factor that is concomitant rather than specific to addition to TrPI solution. Steroids are used in nerve blocks such as intercostal or greater occipital neuralgia and TrPI in these areas may have overlapping effects. Steroids by oral or infusion route are often a component of headache exacerbation management. In reviewing the literature or case reports showing positive result from adding steroid to TrPI, consideration should be given to proximity of injections to nerve or systemic effects before concluding benefit from the addition to TrPI solution.

Serapin is a sterile aqueous solution of salts of the Pitcher plant (sarracenia purpurea). It has been used in injections for more than 50 years to treat pain of both muscular and neuropathic origin. Limited research is available on the use of serapin for myofascial pain, and most available information is old. A mechanism of action of selectively blocking C-fiber activity was proposed.[57] Bates[58] noted longer relief with injection of serapin when compared with novocaine or saline without sensory or motor block. No RCTs of its use in TrPI are noted at this time.

Clinically, serapin can be successfully used in a 50/50 combination with a local anesthetic to treat patients in exacerbation, with a significant component of aching pain or if limited response to local anesthetic alone. No adverse reactions to the addition of serapin to TrPI solution have been seen with this technique. Any improvement in response quality or duration is purely observational. Serapin in no longer available on the US market, and it is not certain when or if the product will be available.

Hyaluronate is a glycosaminoglycan found in the extracellular matrix, especially of soft connective tissues. Product forms are used for intraarticular viscosupplementation injections. Comparison was made of TrPI using 0.5% lidocaine with the same solution of lidocaine mixed with hyaluronidase 600 IU/mL. No significant differences at day 0, 4, 7, or 14 were reported. Patients receiving hyaluronidase showed less postinjection soreness on day 1.[59] Given the very limited benefit and associated cost, it would be difficult to recommend unless additional supportive data became available.

Dextrose solution has been used for proliferative injection techniques such as prolotherapy at concentrations ranging from 10% to 20% and in perineural injection treatment buffered at 0.5% in sterile water. A proposed mechanism for the use of dextrose in myofascial pain cites glycopenia as a potential trigger for C-fiber excitation, neurogenic inflammation, and neuropathic pain.[60] A single Korean study proposed dextrose as an energy supplement for impaired energy metabolism of the TrPs. In this study, comparison of the pain intensity and pressure pain thresholds showed lower scores indicating greater improvement at 7 days for the group treated with 5% dextrose water than for the one treated with 0.5% lidocaine or normal saline.[61] More research would be needed before recommending the addition of dextrose to TrPI solutions.

Clinicians have added intramuscular vitamin preparations such as Vitamin B_{12}, D, or C to TrPI solutions. Although the assessment of overall health and nutritional status is an important consideration when addressing perpetuating factors for myofascial pain, there is no evidence to recommend the addition of vitamins (see Chapter 4).

The anti-inflammatory ketorolac is a nonsteroidal anti-inflammatory drug in the family of propionic acids, often used as an analgesic and antipyretic. Ketorolac acts by inhibiting bodily synthesis of prostaglandins. Approved use includes both intramuscular and intravenous administration. Ketorolac may be utilized to treat pain exacerbations, including musculoskeletal pain. No RCT that is specific to the addition of ketorolac to TrPI solution is available.

Potential remains for other types of solution to be helpful additions to TrPI, such as 5-HT3 receptor antagonists,[62] tumor necrosis factor blockers,[63] or autologous serum conditioned for interleukin-1 receptor antagonists. Quality research would be needed before recommending additional solution components for routine clinical use.

3. NEUROTOXINS

The use of neurotoxins for the treatment of musculoskeletal pain has expanded[64] and requires mention, although a full review of this treatment intervention is beyond the scope of this text.

Botox is produced by *Clostridium botulinum*, an anerobic, gram-positive organism that can be found in soil and water. Botox, when injected, causes a degree of flaccid paralysis by blocking the release of acetylcholine at the presynaptic terminal of the neuromuscular junction. There are multiple neurotoxins designated as types A, B, C1, C2, D, E, F, and G. Table 72-3 is a summary of the neurotoxins that are Food and Drug Administration (FDA) approved. Botox is used to treat multiple painful conditions such as cervical dystonia and chronic daily migraine headaches. Its effects on the motor neurons are well known, causing a relaxation of hypertonic or spastic muscle. It has also been found to inhibit the release of neurotransmitters involved in pain transmission, such as glutamate and substance P.[65] A study performed on rats, showed significantly reduced glutamate release as well as reduced local edema and diminished signs of pain after peripheral injection of toxin. This may be the rationale for why Botox reduces pain in addition to its motor effects.[66]

Muscle weakness can begin to take effect in 2 to 5 days, with maximum effect at about 2 weeks. As a result of the toxin's effect on the neuromuscular junction, the motor endplate fails and the nerve ending dies back. A new nerve ending sprouts from the residual axon, eventually forming the new neuromuscular junction. The neuromuscular junction is usually reestablished in approximately 3 months. Thus, Botox injections last for 3 months on average.[67]

Table 72-3 Summary of FDA Approved Neurotoxins

Molecular Name	Pharmaceutical Name	Type
Onabotulinum	Botox	A
Abobotulinum	Dysport	A
Incobotulinum	Xeomin	A
Rimabotulinum	Myobloc	B

From Davids HR. Botulinum toxin in pain management. https://emedicine.medscape.com/article/325574-overview#a4. Accessed August 31, 2017.

The results reported in studies comparing the efficacy of TrPI with that of Botox are mixed. In reviewing the literature, the treatment response window studied often compared relatively short postinjection intervals. Given the neurotoxin duration of action, differences in response should have been evaluated 2 to 3 months from treatment rather than postinjection and at intervals of a few weeks after injection.[67,68] The Cochrane review found only four trials comparing the effects of Botox A against placebo in individuals with myofascial pain. The results were controversial because three trials reported no statistically significant differences between Botox A and placebo for reducing pain.[68]

A subset of individuals with chronic myofascial pain will have TrP reactivation despite repeated TrPI. This group of patients may be considered for botulinum injections. The injections can reduce the electrical activity of the TrPs and provide longer lasting response.[67] The method of identifying a locus for treatment may be different when evaluating for neurotoxin injection. Trigger point injection is directed by palpation, whereas evaluation for neurotoxin injection may be performed by needle EMG to appropriately identify and map out the muscles to be targeted. EMG mapping is generally used in the cervical region given the complexity of the anatomy. Manual palpation and clinical judgment are generally used in other regions of the body. Studies have shown that patients who have mapping studies tend to have a greater response to injections than those who do not.[69] Mapping may also include the use of ultrasound to evaluate deeper muscles that would ordinarily be avoided because of proximity to sensitive structures such as nerves, vessels, or pleura. Clinically, patients who have elevated activity of the muscles on testing tend to have better outcomes with neurotoxin injection than patients with minimal to no activity.

Neurotoxin injection may offer longer duration of relief of myofascial pain and may also offer a window of opportunity for rehabilitation efforts such as muscle reeducation, effective stretching, and postural correction. Longer duration of effect may also have negative consequences. Clinically, there are patients who tolerated TrPI well who experience significant exacerbation after neurotoxin treatment. Patients with underlying segmental or generalized hypermobility may be further destabilized by the weakness resulting from Botox injection. For example, shoulder pain may be worsened by a compromise of scapular stabilization by neurotoxin administration to treat TrPs at the medial scapular border.

Patients should be educated on the potential side effects from neurotoxin injections. As stated previously, the injections take about 2 weeks to begin taking effect. After 2 weeks, patients may notice a flare in the pain. If injecting neck muscles, they may also notice weakness in the neck with difficulty lifting the head up. Mild flu-like symptoms may also be reported. These side effects are self-limiting and should not last more than 2 weeks.

If a patient responds positively to neurotoxin injection, repeat injections should be considered at 3 months. If TrPs are deactivated and do not reactivate, patients may be able to stop treatment. Others may notice a return of symptoms after 3 months, requiring subsequent injections. Patients should be monitored after each injection to assess the need for further treatment.

4. SUMMARY OF TRIGGER POINT INJECTIONS

There continues to be a significant degree of empirical evaluation regarding the practice and efficacy of TrPI. This area of study offers many potential research opportunities. Comparisons are needed of treatment in matched patient types, solutions, and additives, as well as optimal frequency and duration regimens.

As noted by Simons et al,[2] patients may report a history of treatment by TrPI without benefit. Patients may also report a history of significant pain or postinjection soreness with previous treatments. Clinicians who perform TrPI should be both well-trained and experienced. With more states and countries acknowledging that DN is within the scope of physical therapist practice, patients will have greater access to qualified clinicians for the treatment of myofascial pain and dysfunction.

As the available literature does not overwhelmingly demonstrate superiority of TrPI over DN or of a particular solution for injection, it ultimately falls to the clinician to choose the initial treatment approach and individualize to each patient. Concomitant health issues, body habitus, and anatomy of treatment target are all factors to be considered when determining needle choice, solution, and possibly guidance assistance such as ultrasound or EMG. A combination of wet needling and DN may also be used. Subsequent treatments may be adjusted pending the patient's response.

There may be some benefit in the use of local anesthetics relative to postinjection comfort and possibly several weeks to months out from treatment. Lidocaine or procaine may be used at 1% or diluted with normal saline or sterile water down to a 0.5% or even 0.25% concentration. Longer-duration local anesthetic such as bupivacaine may be used but has not shown greater effect and may increase the component of myotoxicity and potential for longer neural blockade. Higher-concentration solutions have generally not shown better effect. Volumes greater than a few 10th of a milliliter at each site are generally not indicated. Limiting concentration, volume, and delay in the injection of local anesthetic may better allow the practitioner to localize TrPs and elicit LTRs.

Additives to TrPI solutions have been used empirically by clinicians but have limited or no support in the literature. Corticosteroids may have benefit in some patient types, but there is insufficient research to outline recommendations for use. Steroids carry additional local and systemic risks, especially if the dosage is higher, with multiple injection sites or with repeated use. A trial may be reserved for patients not responding to TrPI using only local anesthetic.

The use of neurotoxins in the treatment of myofascial pain remains controversial. It is possible that we have not yet fully teased out which patients are most likely to respond to the use of neurotoxin administration or which individual's pain may be temporarily exacerbated by its use. Given the higher costs associated for neurotoxin solution and administration, the use of local anesthetic TrPI should likely remain the first choice of intervention.[54] Evaluation for the use of neurotoxin should be reserved for cases in which response to TrPI or DN is limited or not of adequate duration. Positive response to the use of neurotoxin can reduce the frequency or eliminate the need for TrPI. Some patients benefit from the availability of TrPI between neurotoxin sessions to address the residual areas of myofascial dysfunction.

DRY NEEDLING

It is important to consider that filiform needles are used in acupuncture and DN. A discussion of the differences between both approaches is beyond the scope of this text. The American Physical Therapy Association defines DN as a "skilled intervention using a thin filiform needle (usually an acupuncture needle) to penetrate the skin that stimulates myofascial TrPs, muscles, and connective tissue for the management of neuromusculoskeletal disorders."[70]

Some authors have described different DN techniques. Probably the most expanded needling intervention is the one described by Hong.[11] Hong[11] described his "fast in, fast out" method of needling of a TrP that has been precisely located by palpation. The palpating finger should stay over or straddle the taut band in order to guide the needle insertion directly to the TrP area. The needle is held by the other hand. With the thin (27-gauge) needle remaining deep to the subcutaneous tissue, the muscle fibers of the TrP are explored with multiple needle insertions. The needle movement is rapid, "fast in" and "fast out." Hong has modified the technique as originally described by including a pause of 2 or 3 seconds between insertions. The pause following each insertion permits time to consider the tissue textures traversed by the needle and where to redirect the needle, the time for the identification of an LTR, and the time to immediately inject anesthetic solution into the same needle track when a twitch occurs. The needle is inserted deep enough to fully penetrate the TrP and then is pulled back to the subcutaneous tissue layer but not out of the skin. If the clinician is performing TrPI, a drop of 0.5% procaine (or lidocaine) is injected into the taut band following every LTR that is detected by the feeling of needle tip movement (from the hand holding the syringe), by palpating the twitch contraction (with the palpating hand), or by seeing, if the muscle is superficial, the movement of a visible twitch. The local analgesic agent should be injected only if an LTR accompanies needle insertion.

This rapid technique avoids muscle fiber damage from LTRs. Experience during research studies showed that LTRs are elicited more frequently when the needle is moved quickly rather than slowly. The track of needle insertion is usually very straight and the needle is less likely to be deflected by the dense contracture knots when the needle is inserted at high speed. For this reason, this "fast in, fast out" technique is well suited to the use of filiform (acupuncture) needles. Hong[11] originally proposed that an LTR should be obtained during the application of the technique to be effective. However, how many LTRs are needed to obtain a positive outcome is still debatable. A recent study has found no clinical differences in pain depending on the number of LTRs obtained during DN in the upper trapezius muscle in patients with neck pain.[71] Similarly, another study suggests that the LTR may not be as necessary as Hong[11] described for a successful outcome because no difference at 1 week was observed between patients experiencing LTR and those not experiencing LTR.[72] Discrepancies in the published studies have lead some authors to question the need of LTR during DN.[12]

Gunn[73] recommends identifying TrPs by spot tenderness in a taut band and then using DN techniques. He first identifies the TrPs as a spot of localized tenderness in a taut band and then identifies the precise skin location through which to insert the needle using a dermometer (point finder or skin resistance detector). He then inserts the needle through this location to the TrP where he feels a "grabbing" sensation at the needle tip, which is often associated with aching pain, as the needle enters into the TrP area. Gunn[73] defined this TrP needling technique as Intramuscular Stimulation.

The effectiveness of DN in many conditions is supported by systematic reviews and meta-analysis. For instance, it has been concluded that TrP-DN is effective, at least in the short term, for the management of pain conditions in the upper[74] and lower[75] quadrants, neck-shoulder pain,[13] low back pain,[76] and plantar heel pain.[77] An interesting meta-analysis found evidence suggesting that DN applied by physical therapists was superior to no treatment or sham, but it has been found to be equally effective as other physical therapy treatments for short- and mid-term follow-ups in individuals with musculoskeletal pain.[78] No clear evidence of long-term effects of DN is available. Nevertheless, the Canadian Agency for Drugs and Technologies in Health has accepted the use of DN following an appropriate clinical reasoning in the public health system.[79]

The underlying mechanism by which DN exerts its therapeutic effects is not understood, and both mechanical and neurophysiologic mechanisms are proposed.[80,81] From a mechanical point of view, disruption of the integrity of dysfunctional endplates, increase of sarcomere length, and reduction of the overlap between actin and myosin filaments are proposed.[1] From a neurophysiologic point of view, DN may reduce both peripheral and central sensitization by removing the source of peripheral nociception (TrP), by modulating spinal efficacy in the dorsal horn, and by activating central inhibitory pain pathways. Likely, DN acts simultaneously at different levels in this process.[82]