
Review Articles

Trigger Point Injections for Headache Disorders: Expert Consensus Methodology and Narrative Review

Matthew S. Robbins, MD; Deena Kuruvilla, MD; Andrew Blumenfeld, MD; Larry Charleston IV, MD; Michael Sorrell, MD; Carrie E. Robertson, MD; Brian M. Grosberg, MD; Steven D. Bender, DDS; Uri Napchan, MD; Avi Ashkenazi, MD

Objective/Background.—To review the existing literature and describe a standardized methodology by expert consensus for the performance of trigger point injections (TPIs) in the treatment of headache disorders. Despite their widespread use, the efficacy, safety, and methodology of TPIs have not been reviewed specifically for headache disorders by expert consensus.

Methods.—The Peripheral Nerve Blocks and Other Interventional Procedures Special Interest Section of the American Headache Society over a series of meetings reached a consensus for nomenclature, indications, contraindications, precautions, procedural details, outcomes, and adverse effects for the use of TPIs for headache disorders. A subcommittee of the Section also reviewed the literature.

Results.—Indications for TPIs may include many types of episodic and chronic primary and secondary headache disorders, with the presence of active trigger points (TPs) on physical examination. Contraindications may include infection, a local open skull defect, or an anesthetic allergy, and precautions are necessary in the setting of anticoagulant use, pregnancy, and obesity with unclear anatomical landmarks. The most common muscles selected for TPIs include the trapezius, sternocleidomastoid, and temporalis, with bupivacaine and lidocaine the agents used most frequently. Adverse effects are typically mild with careful patient and procedural selection, though pneumothorax and other serious adverse events have been infrequently reported.

Conclusions.—When performed in the appropriate setting and with the proper expertise, TPIs seem to have a role in the adjunctive treatment of the most common headache disorders. We hope our effort to characterize the methodology of TPIs by expert opinion in the context of published data motivates the performance of evidence-based and standardized treatment protocols.

Key words: trigger point injection, myofascial pain, headache, local anesthetic, tension-type, migraine

(*Headache* 2014;54:1441-1459)

From the Montefiore Headache Center, Department of Neurology, Albert Einstein College of Medicine, Bronx, NY, USA (M.S. Robbins, D. Kuruvilla, and B.M. Grosberg); Neurology, The Headache Center of Southern California, Encinitas, CA, USA (A. Blumenfeld); University of Michigan Health System, Ann Arbor, MI, USA (L. Charleston IV); Tufts University School of Medicine, Springfield, MA, USA (M. Sorrell); Springfield Neurology Associates, Springfield, MA, USA (M. Sorrell); Department of Neurology, Mayo Clinic, Rochester, MN, USA (C.E. Robertson); North Texas Center for Head, Face & TMJ Pain, Dallas, TX, USA (S.D. Bender); Texas A&M University Baylor College of Dentistry, Dallas, TX, USA (S.D. Bender); Headache Clinic, Middletown Medical, Middletown, NY, USA (U. Napchan); Doylestown Hospital, Doylestown, PA, USA (A. Ashkenazi).

Address all correspondence to M.S. Robbins, Montefiore Headache Center, 1575 Blondell Avenue, Suite 225, Bronx, NY 10461, USA.

Accepted for publication July 18, 2014.

Trigger point injections (TPIs) have been performed to treat a variety of musculoskeletal and neurological disorders featuring pain for years.¹ Trigger points (TPs) are the hallmark physical examination sign of myofascial pain,^{2,3} which may be present in both primary and secondary headache disorders.⁴ The pathophysiological mechanisms underlying TPs are poorly understood. It has been suggested that TPs are formed as a result of abnormal endplate potentials that in turn lead to excessive acetylcholine release in the neuromuscular junction.^{2,5} This process may result in the formation of a taut band. The sustained muscle contraction may lead to local ischemia, hypoxia, and the release of algogenic substances that sensitize peripheral nociceptors (peripheral sensitization). Shah et al found significantly elevated concentrations of various pain mediators (eg, substance P, calcitonin gene-related peptide [CGRP], bradykinin, serotonin) and other neurochemicals in the trapezius muscle of patients with active TPs, as compared with those who had latent TPs or healthy controls.⁶ When the process of peripheral sensitization is sustained, it may lead to long-term electrophysiologic changes in dorsal horn neurons and supraspinal structures, resulting in central sensitization. This will manifest clinically with hyperalgesia and allodynia.

TPs in head and neck areas have been associated with various headache disorders.⁷⁻¹⁰ In one study, an association was found between active TPs in the upper trapezius, sternocleidomastoid, and temporalis muscles, and chronic tension-type headache (CTTH).⁸ The presence of active TPs was associated with greater intensity and longer duration of headache in that study. Calandre et al examined the prevalence of TPs in migraine, which were found in 94% as

compared with 29% of controls.⁹ The number of TPs was related to both attack frequency and disease duration. The majority of TPs were found in the temporal and sub-occipital areas. In another study by the same group, TPs were found in all 12 patients with cluster headache (CH) who were examined.¹⁰

The treatment of TPs includes both non-invasive (eg, manual therapy, transcutaneous electrical nerve stimulation [TENS]), and invasive modalities, including TPIs and dry needling. Of these therapies, TPIs are employed frequently to treat headache disorders to alleviate head and neck pain. In a 2010 survey of American Headache Society (AHS) membership, 75.3% of responders reported performing TPIs in the treatment of headache.¹¹ However, there has been no consistent methodology proposed in this commonly utilized therapy, and practice patterns may feature marked heterogeneity in terms of indications, medications used, and technique.

The Peripheral Nerve Blocks & Other Interventional Procedures for Headache & Facial Pain Section of the AHS (AHS-IPS) contains a membership that shares a goal of standardizing “the approaches, techniques, and indications of peripheral nerve blocks and other interventional procedures in headache management.”¹² Despite the widespread use by practitioners of such procedures,¹¹ the overall level of evidence is not strong, and until more randomized controlled trials are performed, the support for their use relies on experiential evidence, few controlled studies, and expert opinion.

In pursuit of this goal, members of the AHS-IPS reached a consensus for the methodology in the performance of peripheral nerve blocks (PNBs) in the treatment of headache disorders.¹³ As there is even

Conflict of Interest: There are no conflicts of interest directly related to the content of this manuscript. The full author disclosures are reproduced here: Matthew S. Robbins, MD: received honoraria from the American Headache Society, Prova Education, American College of Physicians, Medlink, North Shore-LIJ Hofstra School of Medicine, and SUNY Downstate College of Medicine, and receives book royalties from Wiley; Deena Kuruvilla, MD: none; Andrew Blumenfeld, MD: has received research grants and/or consulting fees from Allergan, Pfizer, Merck, GSK, Posen, Forrest, Keller Labs, Medtronic, Impax, Supernus, Zogenix, Nautilus, Depomed, and Transcept; Larry Charleston IV, MD: has received consulting honoraria from Allergan, Inc.; Michael Sorrell, MD: has received speaker honoraria from Allergan, Inc.; Carrie E. Robertson, MD: has received financial compensation as an author for UpToDate®; Brian M. Grosberg, MD: served on a scientific advisory board for Kowa Pharmaceuticals America, Inc., and Tribute Pharmaceuticals; has received speaker honoraria from Zogenix; receives research support from Allergan, Inc., Boston Scientific, and ElectroCore; and receives book royalties from Wiley; Steven D. Bender, DDS: none; Uri Napchan, MD: has received speaker honoraria from Zogenix, and consulting honoraria from Allergan, Inc. and MAP Pharmaceuticals; Avi Ashkenazi, MD: has received speaker honoraria from the American Academy of Neurology.

less evidence for the use of TPIs,¹ members of the AHS-IPS aimed to also reach a consensus on their performance in the treatment of headache disorders.

METHODS

This consensus statement was preceded by a systematic literature review that summarized the evidence for the performance of TPIs in the treatment of headache.¹ A follow-up literature review was also conducted to survey the literature in discussing potential primary and secondary headache disorder indications for TPIs. A parallel study demonstrated the need for a standardized methodology by practitioners treating headache.¹¹

The consensus was reached after a series of discussions at AHS-IPS meetings, including a preliminary review at the 2012 AHS annual scientific meeting in Los Angeles, CA, a detailed point-by-point discussion at the 2013 International Headache Congress in Boston, MA, and a workshop of the consensus statement at the 2013 AHS Scottsdale Headache Symposium for finalization. The discussions explicitly addressed nomenclature, indications, contraindications and precautions, procedural details, outcomes, and adverse effects. Throughout the process a majority rule was required and achieved for consensus. The manuscript was drafted and revised by a subcommittee of the AHS-IPS (manuscript authors) between and after the 2013 meetings.

RESULTS

Nomenclature and Definitions.—A myofascial TP as described by Travell and Simons² is a “hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band.” The spot will normally be painful to compression and produce a stereotypical referral pattern to distant structures. Travell and Simons have divided TPs into subtypes, including active, associated, attachment, central, key, latent, primary, secondary, and satellite. For this discussion, only active and latent myofascial TPs will be described.

A myofascial TP is considered active when it produces a clinical pain complaint. It will consistently be tender to palpation and often cause motor dysfunction. An active myofascial TP will often elicit a “twitch

Table 1.—Headache Diagnoses Treated With Trigger Point Injections by American Headache Society Members in a 2010 Survey (Adapted from Blumenfeld et al¹¹)

Headache Diagnoses	Percent of Respondents
Chronic tension-type headache	81.5
Chronic migraine	67.7
New daily persistent headache	47.6
Status migrainosus	46.8
Episodic tension-type headache	41.1
Chronic cluster headache	30.6
Migraine without aura	29.8
Hemicrania continua	29
Migraine with aura	25
Episodic cluster headache	23.4

response” when stimulated. In some cases, along with the characteristic referral pattern, autonomic responses may be observed when the site is stimulated. Also, active TPs may elicit spontaneous pain.

A myofascial TP is considered latent when it produces pain only with palpation, and it typically lacks the characteristic referral pattern seen in active TPs. A latent myofascial TP will share many of the clinical characteristics of an active TP, such as motor dysfunction, and will reside in a taut band, but should not produce spontaneous pain.¹⁴

Indications: Headache Disorders.—TPIs are often used to treat headache and myofascial pain disorders, though with limited evidence for efficacy. In a study surveying the use of PNBs and TPIs in headache treatment among American Headache Society members,¹¹ 75.3% (122 practitioners) of the respondents used TPIs as part of their headache management (Table 1). The most common headache diagnoses in which TPI were performed were chronic tension-type headache (CTTH) (81.5%) and chronic migraine (CM) (67.7%).

Strong evidence to indicate superiority of one injectable agent (eg, local anesthetic, corticosteroid, alpha-adrenergic antagonist, neurotoxin, non-steroidal anti-inflammatory agent) over another when performing TPIs for primary or secondary headache disorders is lacking. A variety of agents, doses, and volumes of agents have been used for various headache diagnoses, though the use of local

anesthetics for TPIs in the literature predominates and is consistent with our clinical experience. As such, local anesthetics are our recommended medication class for TPI in the treatment of headache disorders. Recent data with respect to headache diagnoses and therapy with TPIs are herein reviewed (Table 2).

Episodic Tension-Type Headache (ETTH).—In a double-blinded, placebo-controlled randomized study, lidocaine injections in the TPs of pericranial muscles reduced the frequency and severity of pain at 2, 4, and 6 months compared with placebo (saline) injections in patients with frequent ETTH. There was greater improvement in patients receiving repetitive lidocaine injections than in the single lidocaine injection or placebo groups.¹⁵

Chronic Tension-Type Headache (CTTH).—OnabotulinumtoxinA has been used in patients with CTTH with cervical myofascial TPs. In a randomized, double-blind, placebo-controlled pilot study, patients with CTTH who had onabotulinumtoxinA injected into myofascial TPs initially showed a decreased headache frequency; however, there was no difference in frequency at week 12 or in headache intensity at any time point.¹⁶ Despite the lack of response at this time frame, it may be plausible that the effects of the toxin may subside before 12 weeks, and therefore serial injections may be required for a more definitive response, or a shorter ascertainment period is needed to capture the appropriate duration of response. In another randomized, double-blind, placebo-controlled study, the effect of TPIs using lidocaine on headache and other symptoms was examined in 48 patients with CTTH, though supraorbital, infraorbital, mental nerve and superior cervical ganglion injections were also administered. After 3 treatment sessions each 3 days apart, the number of headache days per month, headache severity, analgesic use, depression and anxiety all decreased, with lidocaine showing a more robust effect than saline injections after 3 months.¹⁷

Episodic and Chronic Cluster Headache.—One case series identified at least one TP in 12 patients with episodic and chronic cluster headache (CH). Patients were injected in an uncontrolled fashion with mepivacaine at 3 different circumstances: (1) during an attack; (2) in an attempt to prevent an imminent

attack; (3) as a prophylactic therapy, with success rates of 85%, 86%, and 88%. Seven of the 8 patients with chronic CH had significant improvement with TPIs when combined with prophylactic drug therapy.¹⁰

Episodic and Chronic Migraine.—Fernandez-de-las-Peñas et al identified TPs in neck and head muscles in subjects presenting with unilateral migraine and healthy controls, and a greater number of patients with migraine had active TPs, though rates of latent TPs were the same.¹⁸ Ashkenazi and Young¹⁹ examined the effect of greater occipital nerve block (GONB) with TPI (in most patients) on the reduction of head pain and brush allodynia in patients with episodic and transformed migraine. Acute headache reduction was achieved in 89.5%, and the allodynia scores were reduced in both trigeminal and cervical dermatomes in all patients.

Garcia-Leiva et al²⁰ evaluated TPIs in 52 patients with migraine, of whom 61.5% had CM and 53% had medication overuse, with prospective headache diaries at baseline and during the 12-week treatment period. Weekly injections of ropivacaine 10 mg (1 mL) were performed for 12 weeks. In 9 (17.3%) patients the frequency of attacks was reduced $\geq 50\%$. There was 11%–49% reduction in the frequency of attacks in 19 (36.5%) patients. A total of 31 (59.6%) patients reported to be much or very much improved after finishing the injection period. Rescue medication intake was reduced $\geq 50\%$ in comparison with baseline period in 11 (21.2%) and the attacks of severe intensity decreased significantly. Eight (26.6%) out of 30 patients suffering from CM reverted to episodic migraine, though specific data regarding the patients with CM and medication overuse were not provided.

Cervicogenic Headache and Vestibular Migraine.—Vestibular migraine (VM) has been included in the appendix of the International Classification of Headache Disorders, 3rd edition beta.²¹ Baron et al reported improvement in patients with suspected “cervically mediated” headache and dizziness that received both GONB and TPIs in a retrospective review.²² Improvements were seen in 57% of patients with headache and 46% percent of patients with dizziness. Other symptomatic improvement

Table 2.—Evidence for Trigger Point Injections in the Treatment of Primary and Secondary Headache Disorders

Headache Diagnosis	Study Design	Subjects	Injection Method	Operator(s)	Agents	Injection Frequency	Outcomes	Blinded Outcome Evaluation	Reference
Episodic tension-type headache	Randomized, double-blind, placebo-controlled study	108	TPI	Multiple	Normal saline single injection (group 1) vs lidocaine single injection (group 2) vs normal saline multiple injections (group 3) vs lidocaine multiple injections (group 4)	1 single injection or 5 injections on alternate days	Monthly pain days improved significantly in groups 2 (10.8 ± 1.8 to 8.4 ± 2.4), 3 (11.0 ± 1.6 to 9.9 ± 1.3), and 4 (11.0 ± 2.2 to 6.6 ± 2.2) at 2 months post-treatment VAS scores improved significantly in group 2 (70.4 ± 8.4 to 59.8 ± 13.6) and 4 (71.1 ± 10.2 to 48.5 ± 13.7) at 2 months but improvement remained at 6 months only in group 4 (58.7 ± 12.7) Group 2 had better VAS scores and monthly pain days than group 1 only at 2 and 4 months Group 4 had better monthly pain days and VAS scores than group 3 at 2, 4, and 6 months Initial decrease in headache frequency (peak weeks 5-8, d = 0.56) but no effect by week 12 No reduction in pain intensity at any time point	Yes	Karadas et al ¹⁵
Chronic tension-type headache	Randomized, double-blind, placebo-controlled pilot study	23	TPI	Multiple	Onabotulinumtoxin A vs normal saline	1 single series of injections		Yes	Harden et al ¹⁶
Chronic tension-type headache	Randomized, double-blind, placebo-controlled study	48	TPI, supraorbital, infraorbital, mental nerve blocks, and superior cervical ganglion injections	Multiple	Lidocaine vs normal saline	Injection series every 3 days for 3 sessions	<p>Injections with lidocaine vs placebo decreased painful days (-12.7 ± 3.6 vs -1.5 ± 3.1, <i>P</i> < .001) and VAS scores (-38.8 ± 10.5 vs -6.2 ± 9.0, <i>P</i> < .001) at 3 months</p> <p>87.5% with CCH had reduction of attack frequency over several months</p>	Yes	Karadas et al ¹⁷
Episodic and chronic cluster headache	Case series	12	TPI	Single	Mepivacaine	1 to 5 series of injections per week (cumulative maximum 32 injections in 8 months)		No	Calandre et al ¹⁰
Episodic and transformed migraine	Prospective study	19	GONB, TPI	NA	Lidocaine + triamcinolone (GONB) lidocaine (TPI)	1 single series of injections	Headache improved in 89.5% of patients 20 minutes after treatment (pain intensity 6.53 vs 3.47)	No	Ashkenazi and Young ¹⁹
Chronic migraine (including medication overuse)	Prospective study	52	TPI	Single	Ropivacaine	1 series of weekly injections over 12 weeks	59.6% of patients reported being much or very much improved after 12 week treatment period	No	García-Leiva et al ²⁰
Cervicogenic headache Vestibular migraine	Retrospective study	147	GONB, TPI	Multiple	Bupivacaine + betamethasone	1 series of injections	Improvement seen in 57% of patients with headache	No	Baron et al ²²

CCH = chronic cluster headache; GONB = greater occipital nerve block; NA = not available; TPI = trigger point injection; VAS = visual analog scale.

included neck range of motion (71%), neck pain (52%), ear discomfort (47%), and tinnitus (30%). The authors concluded that cervically mediated symptoms may exist by influence of trigeminocervical and vestibular circuitry through cervical afferent neuromodulation.

Post-Traumatic Headache.—Packard reviewed the data that suggest a plausible relationship of neck injury and post-traumatic headache.²³ The development of TPs was included as a mechanism that may contribute or lead to the development of post-traumatic headache, and suggested that TPIs may be a treatment for this headache disorder, though no studies have specifically addressed this question.

Indications: Physical Exam, Posture.—The key to injection treatment of most primary headache disorders is the identification of areas on the head and neck that reproduce the patient's headache pain.^{24,25} Most patients having migraine without aura, some having migraine with aura, and most who have tension-type headache (TTH) have myofascial TP tenderness of muscles, ligaments, and tendons of the head and neck which, when pressed, reproduce their typical headache pain both during and between headache episodes.^{2,26-28} Local anesthetic injection to these tender areas can stop pain arising from them for hours to months.¹³

The examiner can reliably identify taut bands in muscles and the tender myofascial TPs within them that reproduce the pain by pressing the symptomatic areas with enough pressure to blanch the fingernail or to elicit a report of discomfort.²⁹ The pressure needed to reproduce an element of the patient's typical headache may vary from patient to patient and from site to site.^{2,3,30} Clinicians should exclude structural causes of headache by examining eye grounds, cranial nerves, and other parts of the nervous system covered in the standardized neurologic exam. The examiner should press or squeeze the trapezius at the shoulder, sternocleidomastoid, splenius and semispinalis capitis and cervicis, temporalis muscles and the trochlear, masseter, and temporomandibular areas to try to reproduce all aspects of the patient's headache pain.^{25,31-34} This technique may also elicit non-headache symptoms of migraine, such as nausea, photophobia, and phonophobia.²

Forward head posture, as measured by a variety of techniques, occurs in many patients who have TTH and also migraine without aura; it may be associated with continuous myofascial strain of the neck extensors, which could be a chronic source of local and referred pain.^{18,35} Correction of forward head posture may be an important but relatively unexplored aspect in the treatment of primary and secondary headache disorders.³⁶

Contraindications and Precautions.—Contraindications to TPI (Table 3) would include a local or systemic infection, an open skull defect beneath the injection site, or an allergy to anesthetic agents. For patients on anticoagulation, we would recommend checking an international normalized ratio (if taking warfarin) before the procedure, spending extra time palpating for and avoiding neighboring arteries, and compressing over the injection site for 5–10 minutes. If the patient is pregnant, lidocaine (FDA Category B) would be preferred over bupivacaine (FDA Category C) as an anesthetic, akin to PNBs.¹³ Pneumothorax has been reported rarely after TPIs in the cervicothoracic region, especially the trapezius.^{37,38} In patients with unclear anatomical landmarks because of body habitus, it may be reasonable to have trapezius injections performed with electromyographic³⁹ or ultrasound⁴⁰ guidance.

Muscle Selection.—For the treatment of headache disorders, the most common muscles selected for TPIs include the trapezius (Fig. 1), sternocleidomastoid (Fig. 2), and temporalis (Fig. 3) muscles.^{2,8} Other muscles that may also feature TP-induced referred pain to the head and neck include the cervical paraspinal muscles (Figs. 4–6), masseter (Fig. 7), levator scapulae (Fig. 8), frontalis, and occipitalis (Fig. 3).^{2,41} The areas that the patient reports as painful can be used as a marker for identifying the muscles to be selected for TPI.

The trapezius (Fig. 1) is the most common TP site encountered in headache disorders.² It can produce pain in the temporal, jaw, occipital, and upper neck areas.⁴² This pain is usually ipsilateral to the TP and can follow a hemicranial distribution. The sternocleidomastoid (Fig. 2) may be the second most common TP site in headache disorders. It can refer pain to the vertex, frontal, temporal, occipital, anterior neck,

Table 3.—Potential Precautions and Contraindications in the Performance of Trigger Point Injections for Headache

Patient Population	Concern	Action
Local anesthesia allergy	Allergic reaction, including anaphylaxis	Local anesthetic contraindicated Use saline, corticosteroids, or other agents only
Vulnerability to neurally-mediated syncope or hypotension	Near syncope Syncope	Reduce concentration of anesthetic ⁶⁴ Limit number of total TPIs in a single session Perform TPIs in supine or prone position, where feasible Allow for extra time in the supine position after the procedure as a precaution
<ul style="list-style-type: none"> • First TPI series • Pregnancy • Elderly • History of vasovagal events • Dehydration • Protracted headache attack with nausea and/or vomiting 		
Pregnancy	Teratogenicity	Local anesthetics may be preferable Use lidocaine (FDA Category B) over bupivacaine (FDA Category C)
Open skull defect Craniotomy	Intracranial diffusion of anesthetic agent	TPI may be contraindicated, but if benefits > risks inject at a distance or contralateral from defect
Local or systemic infection Immunocompromise	Abscess, cellulitis, myositis	Avoid TPI
Anticoagulation therapy Antiplatelet therapy	Hematoma	Recent INR should be available if taking warfarin, and avoid TPI if >3.0 Extra attention to palpate for (and avoid) neighboring arteries Minimize total number of injection sites Only perform TPI in superficial and easily compressible sites Compress at each TPI site for 5-10 minutes
Cosmetic concerns	Cutaneous or muscle atrophy Alopecia	Avoid corticosteroids
Obesity or thin body habitus Unclear anatomical landmarks	Pneumothorax	Avoid TPI in those regions, especially trapezius Use a smaller needle (27 gauge) Use EMG or sonographic guidance

EMG = electromyography; FDA = Food and Drug Administration; INR = international normalized ratio; TPI = trigger point injections.

supraorbital areas, and temporomandibular joint, depending on sternal vs clavicular involvement.⁴² Sternal division involvement may produce referred pain to the unilateral, contralateral, or bilateral frontal regions. If upper neck and occipital pain predominate, the cervical paraspinal muscles (Figs. 4–6) should be examined for a TP and can be considered for injection as well.⁴³ Although a levator scapulae TP (Fig. 8) is more likely to produce pain in the inter-scapular area and around the trapezius, rather than cranially, a failure to inject the muscle can cause the trapezii to remain active and cause difficulty with contralateral sternocleidomastoid relaxation.² The temporalis

(Fig. 3) often produces pain to the teeth, as well as more distant temporal and supraorbital areas. If temporomandibular joint dysfunction and tooth pain are the predominant features of the patient's pain, one can consider injecting the masseter muscle (Fig. 7) along with the temporalis. Although less common, the frontalis and occipitalis muscles (Fig. 3) can be injected if pain is found at these corresponding areas.

Muscle Injections.—Before starting a TPI, the patient should be seated in a chair or in a recumbent position to prevent a vasovagal reaction. The injection site should be cleaned with an alcohol solution. A 22-, 25-, or 27-gauge, 1.5-inch needle can be used

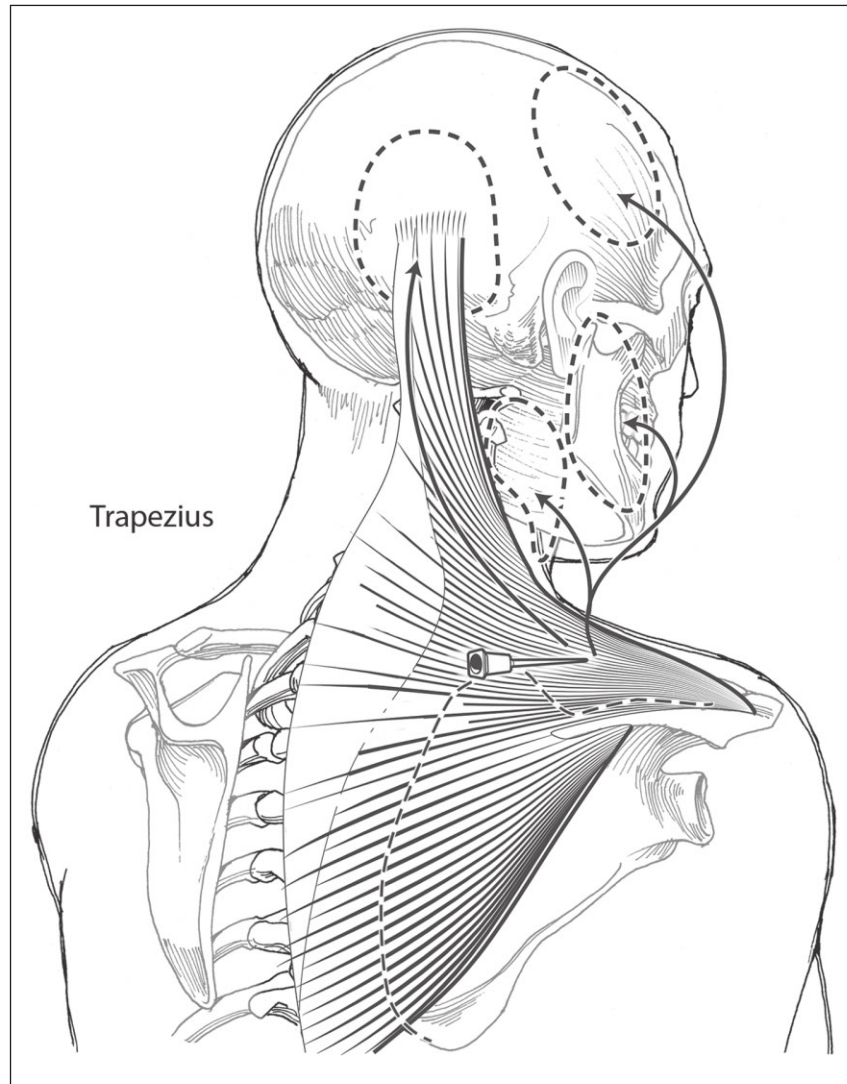


Fig 1.—Trigger point injections in the trapezius. The needle indicates a common trigger point and injection site. Arrows indicate pain referral trajectories, with destinations outlined by dashed lines.

for most superficial injections. A 21- or 22-gauge, 2- or 2.5-inch needle may be more favorable for deeper muscles. Once the palpable band is correctly identified, the overlying skin is held and stabilized between the thumb and index finger. This stabilizes the TP and prevents it from rolling from the needle. The needle is inserted 1 to 1.5 cm away from the TP and then slowly advanced at a 30-degree angle into the TP. Once the TP site is entered, the needle should be gently aspirated to ensure that a blood vessel has not been punctured. Subsequently, 0.1–0.3 cc of 1% lidocaine or 0.5% of bupivacaine should be injected.¹⁵ The needle can be withdrawn slightly and then redirected to sur-

round the target, enabling reinjection multiple times for a total volume of 2 to 4 mL. Hemostasis is obtained by compression of the site for 2 to 4 minutes. These injections may typically be performed monthly,^{2,44} though there is some evidence for safety and efficacy at more frequent intervals for at least short spans of time in some studies.^{10,15,20} “Dry needling” or injection without the use of anesthetic medication has also been proposed to be useful in treating TPs, as the needle itself may cause mechanical disruption of abnormally formed tissues that cause pain.^{2,45}

Specific Muscle Injection Considerations (Adapted From Simons et al²).—Trapezius.—The most

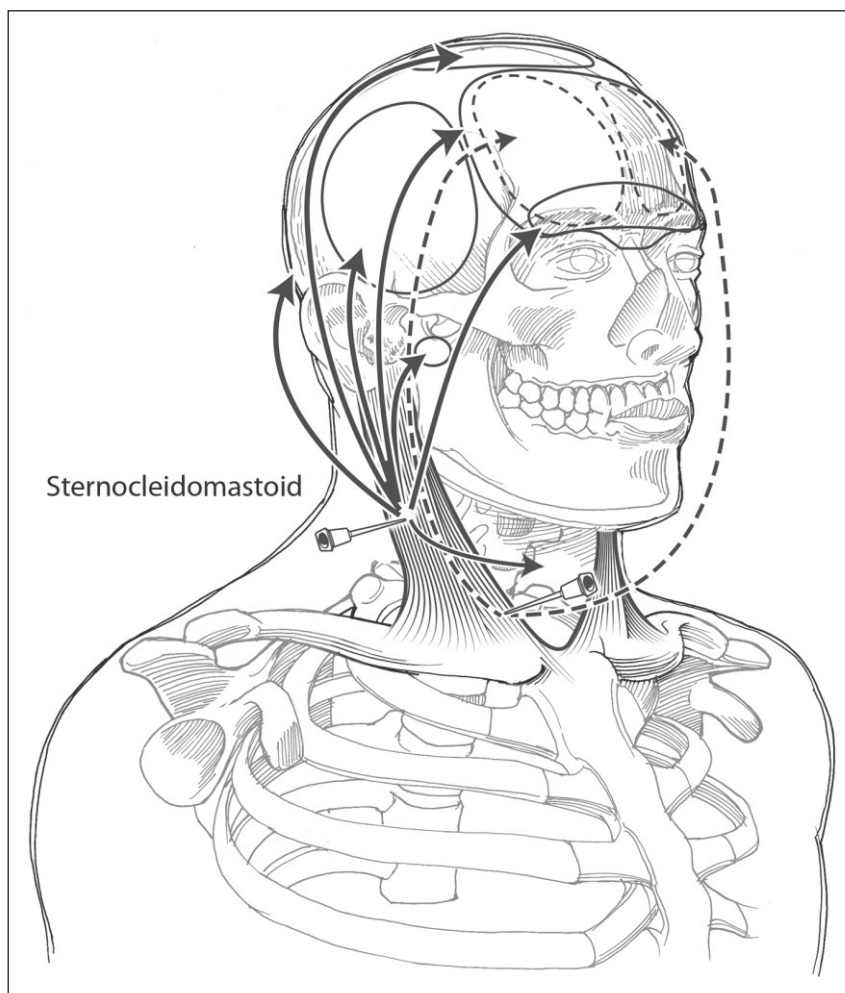


Fig 2.—Trigger point injections in the sternocleidomastoid. Needles indicate common trigger points and injection sites. Arrows indicate referred pain trajectories including trigger points in the sternal division (dashed arrows) and the clavicular division (solid arrows), with destinations outlined by dashed (sternal division) or solid (clavicular division) lines.

common TPI site is the supra-clavicular region (Fig. 1). For the injection, the patient may be positioned prone or leaning forward on a padded table with the head resting on a pillow. The patient should be positioned such that they are facing away from the examiner. The upper trapezius can be injected posteriorly and superior to the scapula. For both sites, the muscle is gripped between the thumb and the fingers and pulled away to isolate it from the supraspinatus muscle, the apex of the lung and also to prevent the muscle from rolling under the needle. To accurately identify the TP, the muscle is palpated for taut bands, local twitches are activated, and the point of pain is identified. The needle is inserted and directed upward to avoid puncturing the lung.

Sternocleidomastoid.—For injection of the sternal and clavicular divisions of the sternocleidomastoid muscle (Fig. 2), the patient may be seated or lay supine. The head should be turned opposite to the side being injected and tilted slightly down toward the side of injection. A 22- or 25-gauge, 1.5-inch needle is used for this injection. The bulk of the muscle is grasped between the thumb and forefingers in order to isolate it from underlying vessels. Before insertion of the needle and anesthetic, the external jugular vein must be located and avoided. To accurately identify the TP, the muscle is palpated for taut bands, local twitches are activated, and the point of pain is identified. The needle is then injected once and adjustments may be made without withdrawing the needle.

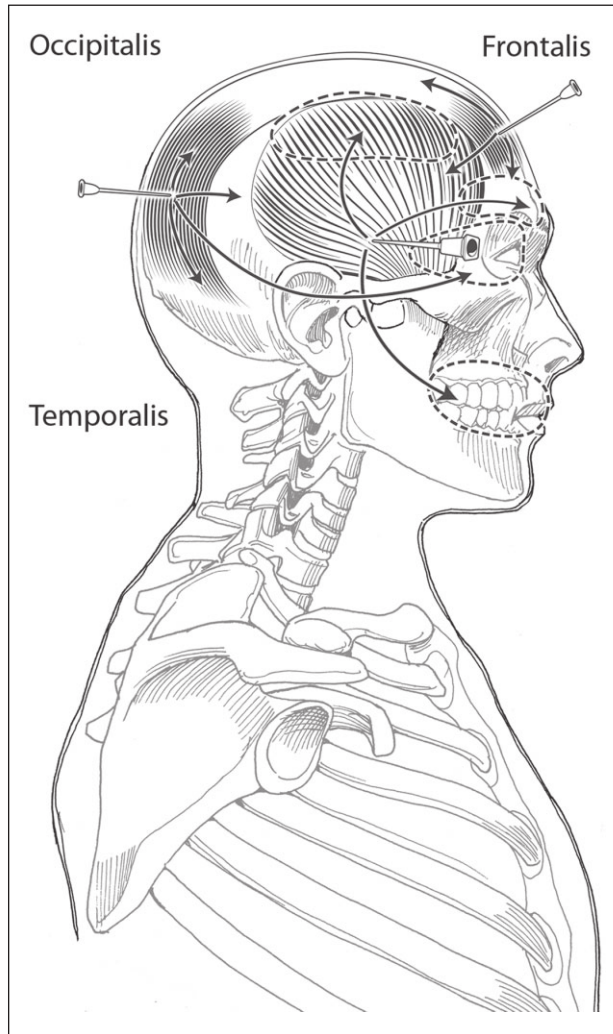


Fig 3.—Trigger point injections in the occipitalis, frontalis, and temporalis. Needles indicate common trigger points and injection sites. Arrows indicate pain referral trajectories, with destinations outlined by dashed lines.

The sternal division is typically injected first because of its easy accessibility medially. The clavicular division is injected second, given that it is more posterior and lateral. Only one side is typically injected at a visit, to monitor the patient's tolerability and pain relief. Injecting the inferior part of the clavicular division may be very challenging because it overlays the apex of the lung and a pneumothorax could result. The patient should be advised to rest in a recumbent position for a few minutes after the procedure.

Cervical Paraspinal Muscles.—The cervical paraspinal muscles consist of a layer of muscles with the trapezius, splenius capitis, and splenius cervicis

being superficial and the semispinalis capitis, and semispinalis cervicis being deeper. TPs in the splenius capitis (Fig. 4) may refer pain to the vertex of the head while TPs just inferior to this muscle in the splenius cervicis (Fig. 5) can refer pain to the occipital, temporal, and retro-orbital areas as well as down into the neck and shoulders. A safe injection site in the splenius cervicis is just above the angle of the neck, between the lower end of the splenius capitis and the levator scapulae muscles, at the C7 vertebral level. The needle is guided under the anterior border of the trapezius to reach the TP while taking caution to stay posterior to the transverse processes.

TPs in the semispinalis capitis (Fig. 6) and one layer deeper in the semispinalis cervicis (Fig. 4) can refer pain to the head, neck, and shoulders, usually manifesting as suboccipital and upper back pain. A safe site to inject the semispinalis capitis muscle is located just above the base of the neck at the C4/C5 vertebral levels. The patient should be lying on the side with head and neck support to keep the neck straight; slight flexion of the neck may assist in TP palpation, which is usually found 1–2 cm lateral to the midline and 2–5 cm beneath the skin, trapezius, and splenius capitis at the C4/C5 vertebral levels. Another TP site can often be found just below the occipital ridge at the insertion of the semispinalis capitis. This site can refer pain around the head, focusing in the temples and forehead. For this injection, the needle should be angled upward toward the occipital bone to avoid the vertebral artery. The semispinalis cervicis is just under the semispinalis capitis and may also contain TPs amenable to injection. The needle must be advanced further (>5 cm) at the site described above to reach this TP, but TPIs into this region must be performed extremely cautiously and only by experienced practitioners because of the risk for potential complications.

The vertebral artery is several centimeters deeper than the cervical paraspinal muscles and is protected by the transverse processes of the vertebrae. If the needle is advanced too far for any cervical paraspinal muscle TPI, especially from an extreme lateral or medial approach, there is a risk of puncture. To avoid this complication, it is helpful to visualize and outline the suboccipital triangle and avoid

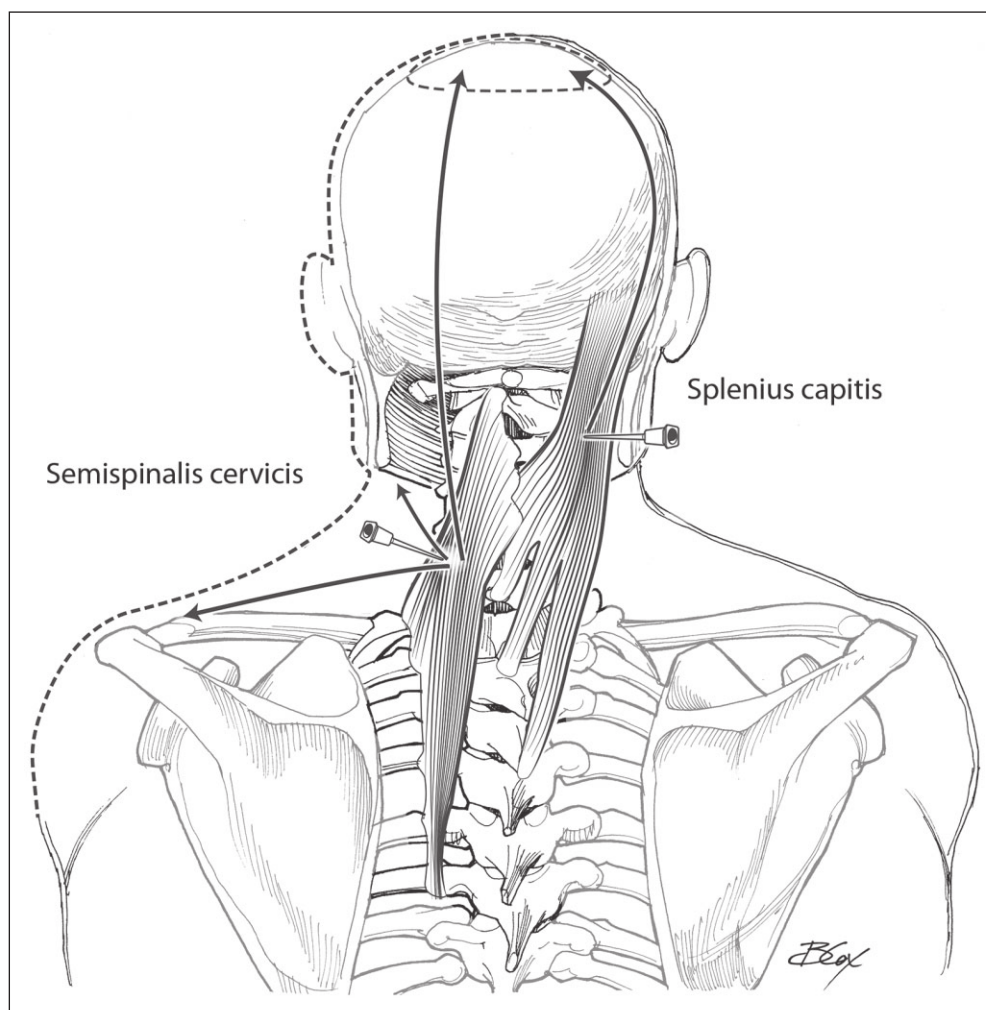


Fig 4.—Trigger point injections in the semispinalis cervicis (left) and splenius capitis (right). Needles indicate common trigger points and injection sites. Arrows indicate pain referral trajectories, with destinations outlined by dashed lines.

injection within this region. Furthermore, deep injections can lead to puncture of the epidural space in the region of the foramen magnum, placing the patient at risk for high epidural block.

Levator Scapulae.—The levator scapulae muscle (Fig. 8) may be responsible for frequent tension in the neck. TPIs of the muscle can help relieve this discomfort. The patient may be positioned sitting or lying on their opposite side. The levator scapulae is inferior to the trapezius and can be palpated underneath it. Once the palpable band is correctly identified by the methods described, the overlying skin is held and stabilized between 2 fingers and the needle is directed upward and medially toward the transverse process of the vertebrae. This injection should be shallow to

avoid puncturing deeper structures (ie, thoracic cage). If the patient continues to have soreness in the levator scapulae referral pattern, have the patient round the shoulders inward and palpate the superior angle of the scapula, moving the fingers from side to side to identify the TP. This injection should be shallow, and directed upward to avoid causing a pneumothorax.

Temporalis.—Injection of the temporalis muscle (Fig. 3) can be complementary to injections in the upper trapezius and sternocleidomastoid muscles because they can all refer pain to the temples. The patient is positioned lying supine with the head turned to the opposite side. The temporal artery must be palpated to avoid puncture, and then the muscle

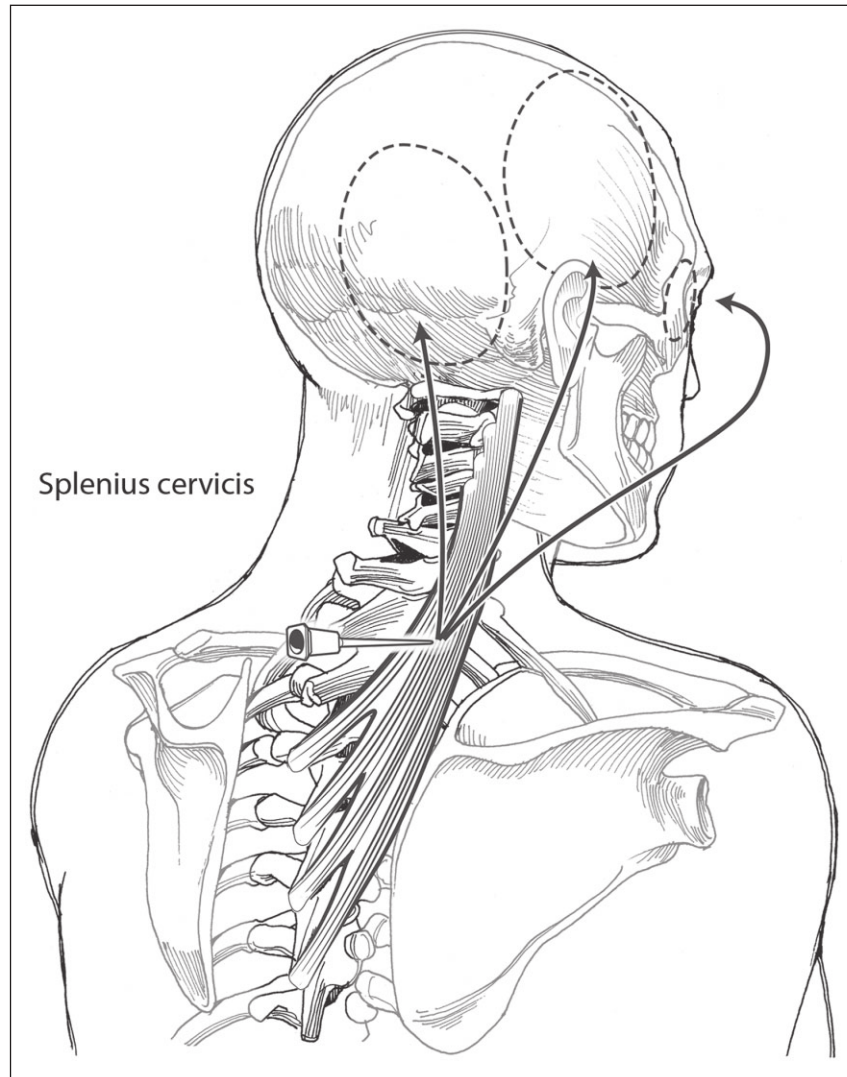


Fig 5.—Trigger point injections in the splenius cervicis. The needle indicates a common trigger point and injection site. Arrows indicate referred pain trajectories, with destinations outlined by dashed lines.

can be identified by having the patient open and clench the jaw closed. Once the TP is identified, maintain one finger over the temporal artery and inject between 2 fingers, angling the needle upward. If TPs remain, or there is restriction of jaw opening, injections may be repeated over 3 sites in the temporalis muscle.

Masseter.—In order to inject the masseter (Fig. 7), the patient should be positioned supine with the head slightly tilted to the opposite side. The middle belly of the masseter muscle may be identified by opening and closing the jaw and palpating below the lower border of the zygoma. The affected area is held between 2

fingers. The needle should be directed toward the posterior portion of the ramus of the mandible.

Frontalis and Occipitalis.—The frontalis muscle (Fig. 3) may be injected in addition to the sternocleidomastoid and temporalis muscles if pain is predominantly in the frontal area. A continuation of the frontalis muscle posteriorly over the top of the head is the occipitalis muscle (Fig. 3). It may be additionally injected if pain is predominantly in the occipital area. This muscle can be identified by having the patient raise the eyebrows and palpating the back of the head.

Outcomes.—Several outcomes can be potentially measured when performing TPIs for the treatment of

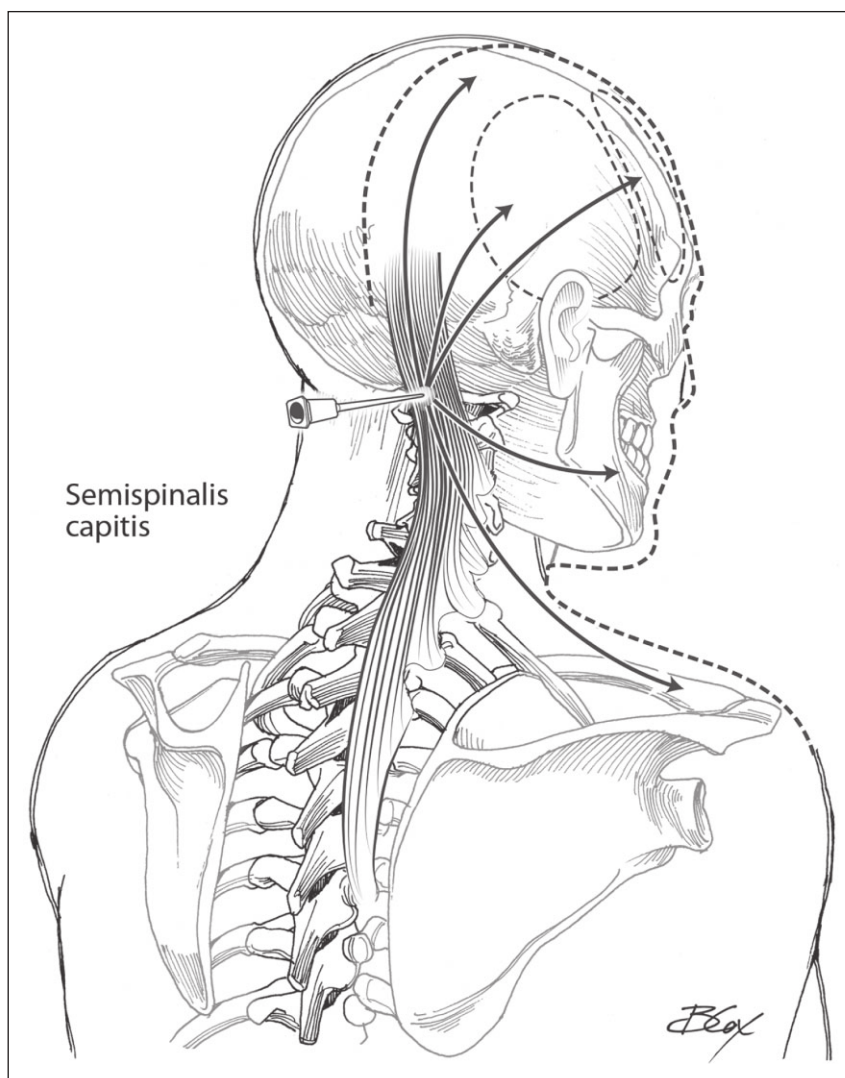


Fig 6.—Trigger point injections in the semispinalis capitis. The needle indicates a common trigger point and injection site. Arrows indicate referred pain trajectories, with destinations outlined by dashed lines.

patients with acute exacerbations of a primary headache disorders. Acutely, pain relief and pain freedom can be measured at several time points up to 2 hours. Sustained pain freedom can be measured at 24 hours as recommended by the guidelines published by the International Headache Society.⁴⁶ The effect of pain reduction is often immediate, within minutes of the performance of TPIs.

If TPIs are being used for the prophylaxis of headaches, the number of headache days, headache attacks, disability scores, and days of medications used after TPIs can be measured on a weekly or monthly basis. On follow-up, one can also examine for the transformation of an active TP into a latent TP.

Pain reduction at the local TP site and the referred head pain site can also be measured. Pressure algometry is an objective tool that can also be used to measure the benefit of TPIs.^{47,48}

In clinical practice, TPIs are often performed in concert with other therapies, including the addition of acute or prophylactic therapies, PNBs, cessation of medication overuse, and non-pharmacological techniques including stretching exercises and physical therapy. Therefore, disentangling the influence of TPIs in isolation may be challenging and will require future study.

Adverse Effects.—There are 5 primary potential adverse effects (AEs) to TPIs, namely (1) direct nerve

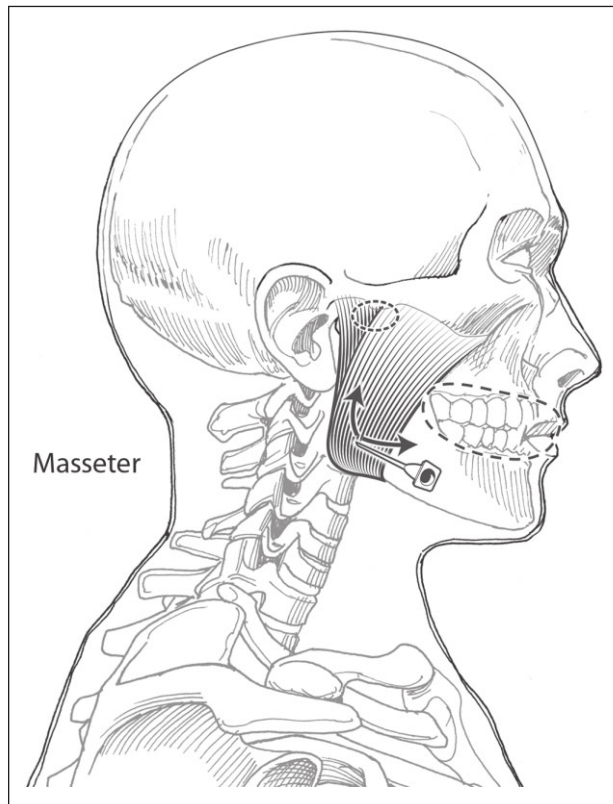


Fig 7.—Trigger point injections in the masseter. The needle indicates a common trigger point and injection site. Arrows indicate referred pain trajectories, with a destination outlined by a dashed line.

or muscle injury; (2) syncope; (3) anaphylaxis; (4) hemorrhage; (5) infection.^{2,49,50} Direct damage to nerve fibers can be minimized by using a small needle (25 gauge or smaller) if feasible, performing straight needle movements, and avoiding lateral movements. Symptoms of nerve injury include new onset burning pain, numbness, and paresthesia. Direct injury to the muscle fiber can be avoided by staying away from areas that appear inflamed or edematous. Careful avoidance of vascular structures and minimization of hematoma formation can also help reduce injury to muscle fibers.

Intramuscular injection with local anesthetics can cause reversible myonecrosis, especially in patients receiving serial or high potency injections. Bupivacaine is the most likely local anesthetic to cause this effect, while procaine is the least likely to be associated with this AE. Once treatment is discontinued, muscle regeneration typically occurs in 3 to 4 weeks.⁵⁰

Patients receiving TPIs are at risk for neurally-mediated syncope, similar to other procedures featuring injections. For this reason, patients should be positioned in the recumbent position to decrease the risk of falling, and also to minimize unexpected movements. This is especially important in patients who are not used to the procedure, or patients who are volume depleted due to headache-associated nausea and vomiting, are anxious, pregnant, or elderly.

In order to minimize toxicity, doses should be limited to less than 300 mg of lidocaine and 175 mg of bupivacaine in a single injection series.⁵¹ Though there is no clear weight-based consensus in the anesthesia literature for adults, maximum doses of 4.5 mg/kg lidocaine and 2 mg/kg bupivacaine have been suggested.^{51,52} For the practitioner treating a 70 kg patient, such maximum volumes equate to 16 mL of 2% lidocaine and 28 mL of 0.5% bupivacaine.

Anaphylaxis must be suspected if the patient experiences hemodynamic instability shortly after a low dose of local anesthetic. As noted previously, patients should be screened for anticoagulant use and worked up appropriately in order to avoid unanticipated hemorrhage. Excessive bleeding may also be lessened by avoiding vascular structures and by applying pressure to the injection site for several minutes after the injection. As with most transcutaneous procedures, TPIs can be associated with skin infections and cellulitis. The site of injection must be cleaned with alcohol prior to the injection. Immunocompromised patients are at higher risk for infection; as such, both patients and clinicians should proceed with caution.

Although the aforementioned 5 adverse risk factors are the most commonly seen in clinical practice, it is important to note that there are serious AEs reported in the current literature including pneumothorax,^{37,38} intrathecal injection,⁵³ and epidural abscess formation.⁵⁴

DISCUSSION

TPIs are commonly used by headache clinicians. In a recent AHS survey, the most common indications for performing TPIs were CTTH and chronic

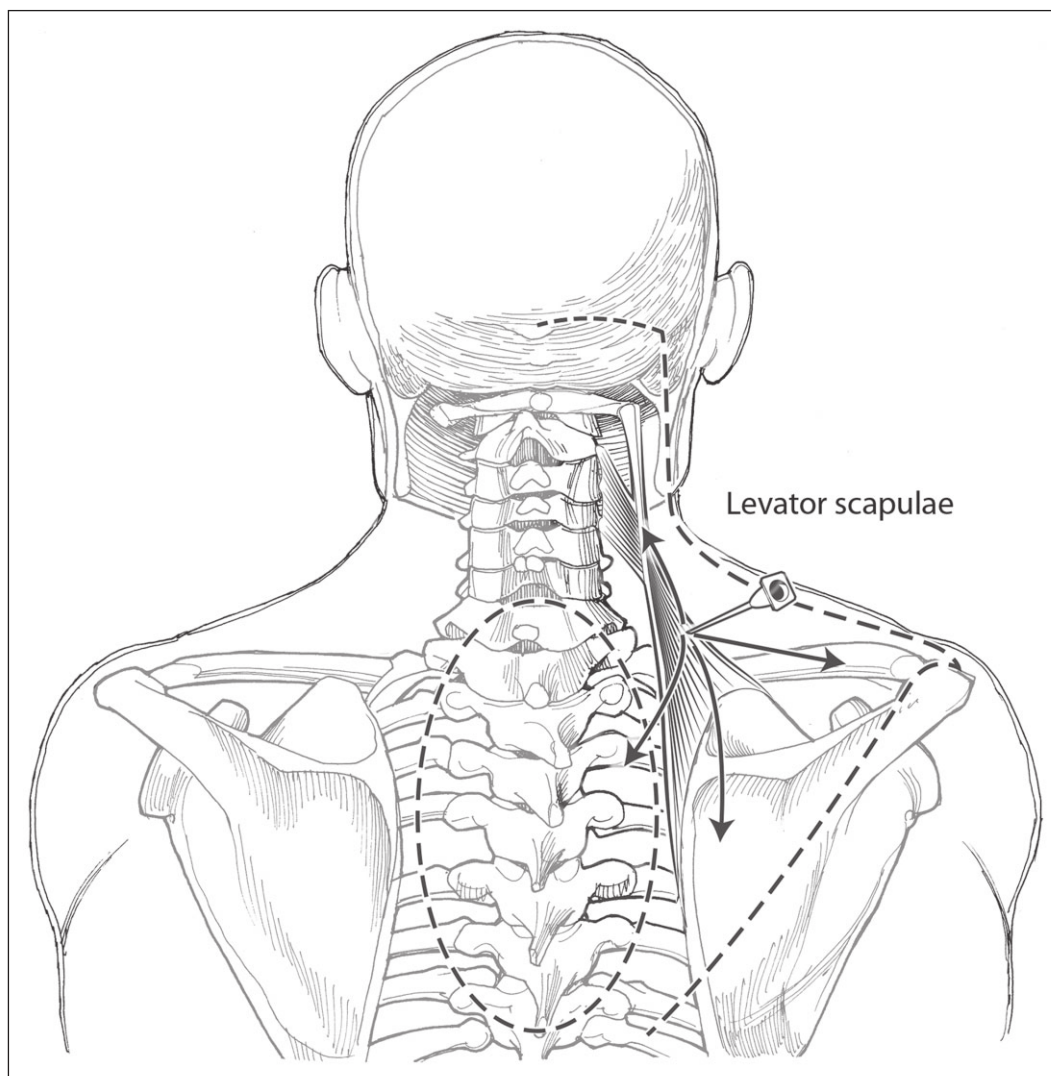


Fig 8.—Trigger point injections in the levator scapulae. The needle indicates a common trigger point and injection site. Arrows indicate referred pain trajectories, with destinations outlined by dashed lines.

migraine.¹¹ TPIs were also reported to being used fairly often for a variety of other headache disorders in this survey. Despite the wide use of TPIs for headache, data to support their use for this indication are very limited.

TPIs have been mostly associated with myofascial pain syndromes, and there are more data on their use for these indications than for headache disorders.⁵⁵ The major objectives of TPIs are to reduce pain and to restore function. In this setting, TPIs have been performed using a variety of drugs (local anesthetics, corticosteroids, non-steroidal anti-inflammatory drugs, onabotulinumtoxinA), vitamins,

or fluids (eg, saline). Alternatively, inserting the needle into or around the TP without injecting any drug or fluid (“dry needling”) has been performed in an attempt to relieve pain.^{55,56} However, data to support the efficacy of this technique are scarce.⁵⁷ Scott et al reviewed the efficacy of TPIs in various musculoskeletal pain syndromes, finding that TPIs relieved symptoms in patients with whiplash injury, as well as in chronic head, neck, shoulder, and back pain.⁵⁵ However, the variability of study quality, techniques used, and data reporting precluded any conclusions regarding the optimal method of injection or drugs used. In general, TPIs were felt to be safe

procedures by the authors although, as outlined previously, rare serious AEs have been reported. For headache disorders, our consensus is to use local anesthetics for TPIs and not other agents such as corticosteroids based on our experience, available literature reports to date, and the lack of a clear biological rationale. For a related therapy, PNBs, the addition of corticosteroids to local anesthetics does not lead to added efficacy in migraine,¹⁹ though they have efficacy in treating CH.^{58,59}

There are several theories as to the mechanisms by which TPIs may alleviate pain. It has been shown in an animal study that needle insertion to the TP region decreases muscle spontaneous electrical activity if a local twitch response is elicited.⁶⁰ This in turn may disrupt the sequence of events that leads to the formation of a taut band. Another theory is that TPIs may prevent local ischemia and hypoxia in the treated area. This may be attained through the local trauma by the needle that results in the release of vasodilators such as substance P and CGRP.⁶¹ Other investigators suggested that needle insertion to a TP area activates A β and A δ fibers, which in turn suppress pain transmission centrally, based on the gate control theory.⁶² In addition, some studies suggest that needle insertion into a TP, combined with electrical stimulation, may activate the endogenous opioid system resulting in enkephalin release.⁶³ Needle insertion into a TP area may exert an analgesic effect through the phenomenon called diffuse noxious inhibitory control (DNIC), also known as conditioned pain modulation (CPM). In this paradigm, a noxious stimulus applied remotely may exert inhibitory effect on pain at the original site of pain.⁵⁶ It should be noted that the above theories focus on the effect of the needle insertion at the TP area, rather than that of any drug or fluid injected, on pain.

More studies are needed to assess the effect of TPIs on headache disorders, independent of the effect of PNBs, which are often performed contemporaneously in clinical practice. In these future studies, the patient population should be as homogenous as possible with regard to their headache diagnosis. In addition, the treatment protocols (indications for treatment, location of injections, type, dose, and

volume of injected drugs) should be predetermined and standardized. Outcome measures should be predetermined and be assessed in a blinded fashion. Blinding may be challenging when local anesthetics are used, as some patients will experience numbness in the injection area after drug injection, whereas those injected with placebo will not.

We hope our effort to characterize the methodology of TPIs by expert opinion in the context of published data motivates the performance of such trials. After obtaining and analyzing the results of such studies, we may be able to design more rational, evidence-based, and standardized treatment protocols for the use of TPIs in various headache disorders. In the interim, when performed in the appropriate setting, patient selection, and with the proper anatomic knowledge and technical expertise, TPIs seem to have a role in the adjunctive treatment of the most common headache disorders.

Acknowledgment: The authors are grateful to Birck Cox of Birck Cox Medical Illustration, Philadelphia, PA, for the design and creation of the figures for this manuscript.

STATEMENT OF AUTHORSHIP

Category 1

(a) Conception and Design

Matthew S. Robbins, MD

(b) Acquisition of Data

Matthew S. Robbins, MD; Deena Kuruvilla, MD; Andrew Blumenfeld, MD; Larry Charleston IV, MD; Michael Sorrell, MD; Carrie E. Robertson, MD; Brian M. Grosberg, MD; Steven D. Bender, DDS; Uri Napchan, MD; Avi Ashkenazi, MD

(c) Analysis and Interpretation of Data

Matthew S. Robbins, MD; Deena Kuruvilla, MD; Andrew Blumenfeld, MD; Larry Charleston IV, MD; Michael Sorrell, MD; Carrie E. Robertson, MD; Brian M. Grosberg, MD; Steven D. Bender, DDS; Uri Napchan, MD; Avi Ashkenazi, MD

Category 2

(a) Drafting the Manuscript

Matthew S. Robbins, MD; Deena Kuruvilla, MD; Andrew Blumenfeld, MD; Larry Charleston IV,

MD; Michael Sorrell, MD; Carrie E. Robertson, MD; Brian M. Grosberg, MD; Steven D. Bender, DDS; Uri Napchan, MD; Avi Ashkenazi, MD

(b) Revising It for Intellectual Content

Matthew S. Robbins, MD; Deena Kuruvilla, MD; Andrew Blumenfeld, MD; Larry Charleston IV, MD; Michael Sorrell, MD; Carrie E. Robertson, MD; Brian M. Grosberg, MD; Steven D. Bender, DDS; Uri Napchan, MD; Avi Ashkenazi, MD

Category 3

(a) Final Approval of the Completed Manuscript

Matthew S. Robbins, MD; Deena Kuruvilla, MD; Andrew Blumenfeld, MD; Larry Charleston IV, MD; Michael Sorrell, MD; Carrie E. Robertson, MD; Brian M. Grosberg, MD; Steven D. Bender, DDS; Uri Napchan, MD; Avi Ashkenazi, MD

REFERENCES

- Ashkenazi A, Blumenfeld A, Napchan U, et al. Peripheral nerve blocks and trigger point injections in headache management – a systematic review and suggestions for future research. *Headache*. 2010; 50:943-952.
- Simons DG, Travell JG, Simons LS. *Myofascial Pain and Dysfunction: The Trigger Point Manual*, Vol. 1, 2nd edn, *Upper Half of Body*. Baltimore: Lippincott Williams & Wilkins; 1999.
- Sorrell MR. The physical examination of migraine. *Curr Pain Headache Rep*. 2006;10:350-354.
- Graff-Radford SB. Myofascial pain: Diagnosis and management. *Curr Pain Headache Rep*. 2004;8:463-467.
- Ge HY, Fernandez-de-Las-Penas C, Yue SW. Myofascial trigger points: Spontaneous electrical activity and its consequences for pain induction and propagation. *Chin Med*. 2011;6:13.
- Shah JP, Phillips TM, Danoff JV, Gerber LH. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *J Appl Physiol (1985)*. 2005;99:1977-1984.
- Alonso-Blanco C, de-la-Llave-Rincon AI, Fernandez-de-las-Penas C. Muscle trigger point therapy in tension-type headache. *Expert Rev Neurother*. 2012;12:315-322.
- Fernandez-de-Las-Penas C, Alonso-Blanco C, Cuadrado ML, Gerwin RD, Pareja JA. Myofascial trigger points and their relationship to headache clinical parameters in chronic tension-type headache. *Headache*. 2006;46:1264-1272.
- Calandre EP, Hidalgo J, Garcia-Leiva JM, Rico-Villademoros F. Trigger point evaluation in migraine patients: An indication of peripheral sensitization linked to migraine predisposition? *Eur J Neurol*. 2006;13:244-249.
- Calandre EP, Hidalgo J, Garcia-Leiva JM, Rico-Villademoros F, Delgado-Rodriguez A. Myofascial trigger points in cluster headache patients: A case series. *Head Face Med*. 2008;4:32.
- Blumenfeld A, Ashkenazi A, Grosberg B, et al. Patterns of use of peripheral nerve blocks and trigger point injections among headache practitioners in the USA: Results of the American Headache Society Interventional Procedure Survey (AHS-IPS). *Headache*. 2010;50:937-942.
- Peripheral Nerve Blocks and Other Interventional Procedures for Headache and Face Pain Section. In: American Headache Society. http://www.americanheadachesociety.org/members/special_interest_sections/peripheral_nerve_blocks_and_other_interventional_procedures_for_headache_and_face_pain. Accessed August 9, 2013.
- Blumenfeld A, Ashkenazi A, Napchan U, et al. Expert consensus recommendations for the performance of peripheral nerve blocks for headaches – a narrative review. *Headache*. 2013;53:437-446.
- Celik D, Mutlu EK. Clinical implication of latent myofascial trigger point. *Curr Pain Headache Rep*. 2013;17:353.
- Karadas O, Gul HL, Inan LE. Lidocaine injection of pericranial myofascial trigger points in the treatment of frequent episodic tension-type headache. *J Headache Pain*. 2013;14:44.
- Harden RN, Cottrill J, Gagnon CM, et al. Botulinum toxin A in the treatment of chronic tension-type headache with cervical myofascial trigger points: A randomized, double-blind, placebo-controlled pilot study. *Headache*. 2009;49:732-743.
- Karadas O, Inan LE, Ulas U, Odabasi Z. Efficacy of local lidocaine application on anxiety and depression and its curative effect on patients with chronic tension-type headache. *Eur Neurol*. 2013;70:95-101.
- Fernandez-de-Las-Penas C, Cuadrado ML, Pareja JA. Myofascial trigger points, neck mobility and

- forward head posture in unilateral migraine. *Cephalalgia*. 2006;26:1061-1070.
19. Ashkenazi A, Young WB. The effects of greater occipital nerve block and trigger point injection on brush allodynia and pain in migraine. *Headache*. 2005;45:350-354.
 20. Garcia-Leiva JM, Hidalgo J, Rico-Villademoros F, Moreno V, Calandre EP. Effectiveness of ropivacaine trigger points inactivation in the prophylactic management of patients with severe migraine. *Pain Med*. 2007;8:65-70.
 21. Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalalgia*. 2013;33:629-808.
 22. Baron EP, Cherian N, Tepper SJ. Role of greater occipital nerve blocks and trigger point injections for patients with dizziness and headache. *Neurologist*. 2011;17:312-317.
 23. Packard RC. The relationship of neck injury and post-traumatic headache. *Curr Pain Headache Rep*. 2002;6:301-307.
 24. Marcus DA, Scharff L, Mercer S, Turk DC. Musculoskeletal abnormalities in chronic headache: A controlled comparison of headache diagnostic groups. *Headache*. 1999;39:21-27.
 25. Tfelt-Hansen P, Lous I, Olesen J. Prevalence and significance of muscle tenderness during common migraine attacks. *Headache*. 1981;21:49-54.
 26. Jensen K, Tuxen C, Olesen J. Pericranial muscle tenderness and pressure-pain threshold in the temporal region during common migraine. *Pain*. 1988;35:65-70.
 27. Olesen J. Some clinical features of the acute migraine attack. An analysis of 750 patients. *Headache*. 1978;18:268-271.
 28. Ferguson LW, Gerwin R. *Clinical Mastery in the Treatment of Myofascial Pain*. Philadelphia: Lippincott Williams & Wilkins; 2005.
 29. Gerwin RD, Shannon S, Hong CZ, Hubbard D, Gevirtz R. Interrater reliability in myofascial trigger point examination. *Pain*. 1997;69:65-73.
 30. Simons DG, Mense S. [Diagnosis and therapy of myofascial trigger points]. *Schmerz*. 2003;17:419-424.
 31. Leone M, D'Amico D, Moschiano F, Farinotti M, Filippini G, Bussone G. Possible identification of cervicogenic headache among patients with migraine: An analysis of 374 headaches. *Headache*. 1995;35:461-464.
 32. Sjaastad O, Fredriksen TA, Pfaffenrath V. Cervicogenic headache: Diagnostic criteria. *Headache*. 1990;30:725-726.
 33. Biondi DM. Cervicogenic headache: A review of diagnostic and treatment strategies. *J Am Osteopath Assoc*. 2005;105:16S-22S.
 34. Yanguela J, Pareja JA, Lopez N, Sanchez Del Rio M. Trochleitis and migraine headache. *Neurology*. 2002;58:802-805.
 35. Fernandez-de-las-Penas C, Alonso-Blanco C, Cuadrado ML, Pareja JA. Forward head posture and neck mobility in chronic tension-type headache: A blinded, controlled study. *Cephalalgia*. 2006;26:314-319.
 36. Abboud J, Marchand AA, Sorra K, Descarreaux M. Musculoskeletal physical outcome measures in individuals with tension-type headache: A scoping review. *Cephalalgia*. 2013;33:1319-1336.
 37. Shafer N. Pneumothorax following "trigger point" injection. *JAMA*. 1970;213:1193.
 38. Paik NC, Seo JW. CT-guided needle aspiration of pneumothorax from a trigger point injection. *Pain Med*. 2011;12:837-841.
 39. Botwin KP, Patel BC. Electromyographically guided trigger point injections in the cervicothoracic musculature of obese patients: A new and unreported technique. *Pain Physician*. 2007;10:753-756.
 40. Botwin KP, Sharma K, Saliba R, Patel BC. Ultrasound-guided trigger point injections in the cervicothoracic musculature: A new and unreported technique. *Pain Physician*. 2008;11:885-889.
 41. Fernandez-de-Las-Penas C, Ge HY, Alonso-Blanco C, Gonzalez-Iglesias J, Arendt-Nielsen L. Referred pain areas of active myofascial trigger points in head, neck, and shoulder muscles, in chronic tension type headache. *J Bodyw Mov Ther*. 2010;14:391-396.
 42. Fernandez-de-Las-Penas C, Simons D, Cuadrado ML, Pareja J. The role of myofascial trigger points in musculoskeletal pain syndromes of the head and neck. *Curr Pain Headache Rep*. 2007;11:365-372.
 43. Giamberardino MA, Tafuri E, Savini A, et al. Contribution of myofascial trigger points to migraine symptoms. *J Pain*. 2007;8:869-878.
 44. Alvarez DJ, Rockwell PG. Trigger points: Diagnosis and management. *Am Fam Physician*. 2002;65:653-660.
 45. Weiss LD. *Easy Injections*. Philadelphia: Butterworth-Heinemann/Elsevier; 2007.

46. Tfelt-Hansen P, Pascual J, Ramadan N, et al. Guidelines for controlled trials of drugs in migraine: Third edition. A guide for investigators. *Cephalalgia*. 2012;32:6-38.
47. Reeves JL, Jaeger B, Graff-Radford SB. Reliability of the pressure algometer as a measure of myofascial trigger point sensitivity. *Pain*. 1986;24:313-321.
48. Fischer AA. Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. *Pain*. 1987;30:115-126.
49. Hong C-Z. Considerations and recommendations regarding myofascial trigger point injection. *J Musculoskelet Pain*. 1994;2:29-59.
50. Cheng J, Abdi S. Complications of joint, tendon, and muscle injections. *Tech Reg Anesth Pain Manag*. 2007;11:141-147.
51. Rosenberg PH, Veering BT, Urmev WF. Maximum recommended doses of local anesthetics: A multifactorial concept. *Reg Anesth Pain Med*. 2004;29:564-575, discussion 524.
52. Becker DE, Reed KL. Local anesthetics: Review of pharmacological considerations. *Anesth Prog*. 2012; 59:90-101, quiz 102-103.
53. Nelson LS, Hoffman RS. Intrathecal injection: Unusual complication of trigger-point injection therapy. *Ann Emerg Med*. 1998;32:506-508.
54. Elias M. Cervical epidural abscess following trigger point injection. *J Pain Symptom Manage*. 1994;9:71-72.
55. Scott NA, Guo B, Barton PM, Gerwin RD. Trigger point injections for chronic non-malignant musculoskeletal pain: A systematic review. *Pain Med*. 2009;10:54-69.
56. Cagnie B, Dewitte V, Barbe T, Timmermans F, Delrue N, Meeus M. Physiologic effects of dry needling. *Curr Pain Headache Rep*. 2013;17:348.
57. Tough EA, White AR, Cummings TM, Richards SH, Campbell JL. Acupuncture and dry needling in the management of myofascial trigger point pain: A systematic review and meta-analysis of randomised controlled trials. *Eur J Pain*. 2009;13:3-10.
58. Leroux E, Valade D, Taifas I, et al. Suboccipital steroid injections for transitional treatment of patients with more than two cluster headache attacks per day: A randomised, double-blind, placebo-controlled trial. *Lancet Neurol*. 2011;10:891-897.
59. Ambrosini A, Vandenhede M, Rossi P, et al. Suboccipital injection with a mixture of rapid- and long-acting steroids in cluster headache: A double-blind placebo-controlled study. *Pain*. 2005;118:92-96.
60. Chen JT, Chung KC, Hou CR, Kuan TS, Chen SM, Hong CZ. Inhibitory effect of dry needling on the spontaneous electrical activity recorded from myofascial trigger spots of rabbit skeletal muscle. *Am J Phys Med Rehabil*. 2001;80:729-735.
61. Sato A, Sato Y, Shimura M, Uchida S. Calcitonin gene-related peptide produces skeletal muscle vasodilation following antidromic stimulation of unmyelinated afferents in the dorsal root in rats. *Neurosci Lett*. 2000;283:137-140.
62. Chu J, Schwartz I. The muscle twitch in myofascial pain relief: Effects of acupuncture and other needling methods. *Electromyogr Clin Neurophysiol*. 2002;42:307-311.
63. Niddam DM, Chan RC, Lee SH, Yeh TC, Hsieh JC. Central modulation of pain evoked from myofascial trigger point. *Clin J Pain*. 2007;23:440-448.
64. Sahai-Srivastava S, Subhani D. Adverse effect profile of lidocaine injections for occipital nerve block in occipital neuralgia. *J Headache Pain*. 2010;11:519-523.