Review Article

Trigger point injections for orofacial muscle pain: a narrative review

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Background and Objective: Masticatory muscle pain (MMP) is a common condition. Though various treatments exist, a portion of patients achieve inadequate relief. One treatment option is trigger point injections (TPIs). We here review the pathophysiology and clinical presentation of trigger points (TP), and the indications, contraindications, technique, and positive and negative outcomes of TPIs.

Methods: PubMed was searched using the terms "trigger point injection AND (TMD OR masticatory OR jaw OR head)". Our inclusion criteria were articles of any type on the topic of TPIs for temporomandibular disorders (TMDs).

Key Content and Findings: The literature search retrieved 80 potential articles. Fifty-two articles were excluded based on the abstracts. The full texts of 28 articles were retrieved, of which 12 articles were included in this review. Two more articles were identified through the references of the included articles. Numerous theories have been proposed for the pathophysiology of TP, and their referral patterns. Treatments ideally target the causative factors, but when they are inadequate, TPIs may be considered. Small randomized blinded trials have demonstrated efficacy for TPIs with procaine, lidocaine, saline, and no substance. Small non-blinded or non-randomized studies have demonstrated efficacy with mepivacaine, dexamethasone, triamcinolone, and platelet-rich plasma. Combination therapy, such as with paracetamol, methocarbamol, levosulpiride, and stabilization splints (SS) may be more effective than TPIs alone.

Conclusions: TPI may improve MMP symptoms and jaw function, though the evidence is not strong. The evidence also does not conclusively indicate any injected substance, or any substance at all, to be superior. Larger randomized, blinded, controlled studies are needed to determine whether TPIs are effective, and if so, the specific methods and substances that are effective.

Keywords: Trigger point injections (TPIs); temporomandibular disorder (TMD); masticatory muscle pain (MMP)

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Introduction

It is estimated that 9.7% of the United States fits the diagnostic criteria for masticatory muscle pain (MMP) (1). It is most prevalent in females of 30–40 years in age (1,2). It has been demonstrated to cause impairment in the form of physical pain, psychological discomfort, and psychological disability (3).

MMP is usually a multifactorial condition, with different

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Table 1 Search strategy summary

Items	Specification
Date of search	10/19/2021
Databases and other sources searched	Source material obtained from PubMed search, and references of selected studies
Search terms used	Trigger point injection AND (TMD OR masticatory OR jaw OR head)
Timeframe	No limit
Inclusion and exclusion criteria	Inclusion criteria: studies of any type with comparison group(s) on the topic of trigger point injections for TMD
	Exclusion criteria: studies that studied solely botulinum toxin
Selection process	Selection performed by Andrew Young

TMD, temporomandibular disorder.

combinations of factors for each individual. Ideally, the factors are identified and eliminated, reduced, or addressed. Jaw parafunction is a significant risk factor for temporomandibular disorders (TMDs) (4), and reducing daytime parafunction through habit awareness and postural training, or addressing nocturnal parafunction with a night guard, can be effective. Stress, anxiety, depression, and distress can be causes of daytime or nocturnal parafunction, and managing those conditions or emotions through psychological therapy can be effective. Inadequate sleep can also aggravate TMD, and should therefore be addressed if present.

Often not all causative factors are identified, they cannot be reduced sufficiently to fully eliminate pain and dysfunction, or the pain has become partially centralized. Numerous treatments can still contribute to pain reduction. Stretching with self-massage, cold compresses, warm compresses (5), and cold sprays (6) are also done. Muscle relaxants and non-steroidal anti-inflammatory drugs (NSAIDs) can provide symptom relief, but should generally not be taken for more than a few months. Physical and psychological therapy (7) is also used.

When MMP is resistant to the aforementioned treatments, botulinum toxin (BTX) injections are sometimes used. The twin-block technique has also been reported as a promising option (8). Finally, trigger point injections (TPIs) can be done.

A search of the literature since 2000 revealed only one narrative review article on the topic of TPI for TMD (9). A systematic review was published in 2018, specifically comparing different injected substances and dry needling for TMD (10). As a narrative review, this manuscript aims to give the reader a broad overview of TPI for TMD by describing the pathophysiology of TPs, and the protocol, indications, contraindications, efficacy, and adverse outcomes of TPIs. We present the following article in accordance with the Narrative Review reporting checklist (available at https://joma.amegroups.com/article/view/10.21037/joma-22-5/rc).

Methods

For the section of this article on "Positive Outcomes", source material was obtained through a PubMed literature search using the terms "trigger point injection AND (TMD OR masticatory OR jaw OR head)" (*Table 1*). Our inclusion criteria were studies of any type with comparison group(s) on the topic of TPIs for TMD. Studies that studied solely botulinum toxin were excluded, since the mode of action of botulinum toxin, particularly with its long duration of action on motor endplates, is significantly different from other substances injected in TPIs. No language or date limits were set.

Results

The literature search retrieved 80 potential articles (*Figure 1*), which were screened. Based on the abstracts, 52 articles did not meet the inclusion criteria and were excluded from the review. The full texts of 28 articles were retrieved. Reading of the full texts resulted in the exclusion of 19 more articles, and inclusion of the remaining 9. Five more articles [von Lindern *et al.* 2003 (11), Venâncio Rde *et al.* 2008 (12), Fernández-Carnero *et al.* 2010 (13), Dıraçoğlu *et al.* 2012 (14), and Silva *et al.* 2012 (15)] were identified through the references of the 9 included articles. Therefore, a total

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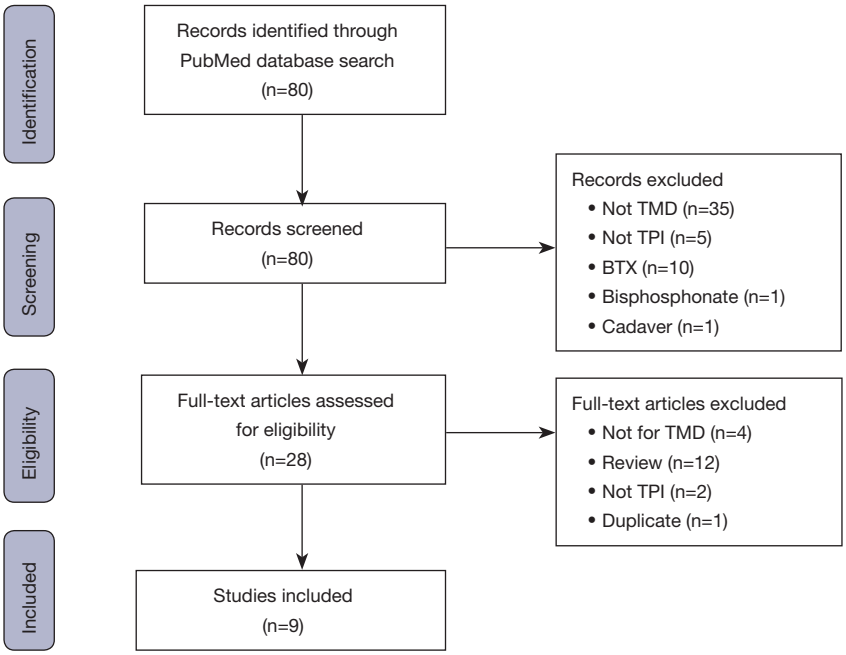

Figure 1 Flowchart of study selection process. TMD, temporomandibular disorder; TPI, trigger point injection; BTX, botulinum toxin.

of 14 articles were included in this review for the section of this article on "Positive Outcomes" of TPI.

Of the excluded articles, 40 were regarding TPI that was not used for TMD, 10 studied solely BTX, 7 did not involve TPIs, 8 were reviews, 1 was regarding bisphosphonates, 1 used only cadavers, and 1 was a case report. Of the included studies, 6 utilized both random allocation and blinding, 4 utilized random allocation but not blinding, 1 utilized blinding but not random allocation, 2 were retrospective and did not utilize random allocation or blinding, and 1 was prospective and did not utilize random allocation or blinding.

For the remainder of this article, the reviews, as well as their references, were used as source material.

Discussion

Trigger points (TP)

TPs were first described by Ralph Stockman in 1904 (16), though he did not name them as such. Since then, various authors have investigated and reported on TPs, with the most prolific being Janet Travell (17). Travell and Rinzler were the first to use the term "trigger points" in their 1952 publication (18).

TPs are taut bands of contracted or shortened muscle fibers (19), millimeters in diameter, which refer pain when palpated or aggravated by muscle activity (9). Typical symptoms are pain, stiffness, decreased range of motion (ROM) (20), weakness, and increased fatigability (6). Pain, usually dull or aching in quality, may occur only with aggravation, or may be continuous and worsened with aggravation. Aggravators include eating, opening, and oral parafunction. Severe pain may have a sharp quality (5).

During the physical examination, palpation of MMP patients may reveal taut and tender bands of muscle fibers 1–4 mm in diameter. Palpation along these bands may lead to the most tender point, usually no longer than several millimeters. When palpated, these TPs elicit pain and may twitch (21). The pain may be localized to the site of palpation, or may spread or refer. Spreading is the phenomenon in which pain spreads beyond the TP, but remains within the same muscle. In referral, pain is felt beyond the painful muscle (22). For example, a painful masseter muscle may refer to the molars. Referred pain is often felt in the eyes and ears. According to the Diagnostic Criteria for TMD, pressure of 1 kg for 5 seconds (22) must be applied to detect referral patterns, though some have recommended 2–4 kg/cm^2 for at least 6–10 seconds (23). Palpation of the TP may also decrease the pain in the referral zone (6).

The pathophysiology of TPs is still under investigation.

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Overload, damage, and stress may be causative factors (24). The tautness of TPs may due to a localized inflammatory mileu, which triggers the release of calcitonin gene-related peptide, inhibiting acetylcholine esterase or upregulating acetylcholinesterase receptors, and thereby increasing acetylcholine (ACh) activity. The localized acidic environment also increases ACh activity. Excessive ACh causes sustained contraction, resulting in taut bands. The tautness of the bands may inhibit blood flow locally, resulting in an energy crisis, decreasing the production of adenosine triphosphate (ATP). ATP is necessary for calcium reuptake, so a contraction is further sustained. The pain of TPs may be due to both peripheral and central sensitization. Damaged muscle tissue has been demonstrated to have inflammatory mediators and an acidic pH, both of which may sensitize peripheral nociceptors. Sustained peripheral noxious input is known to cause central sensitization (5).

The mechanism of referred pain is not entirely clear (25). According to the Convergent Projection Theory, peripheral neurons from various sites converge on common spinal neurons; the cerebral cortex thus can mistakenly assign pain to a healthy site. The Convergence-Facilitation Theory proposes that peripheral noxious input creates an irritable area in the central nervous system, which may alter the perception of non-noxious sensory input as noxious. According to the Hyperexcitability Theory, nociceptive input may open latent neural connections between sites.

The causes for TP formation are also not entirely clear. Acute trauma, or repeated microtrauma, has been proposed (26). Microtrauma, such as jaw parafunction, or occupations that require frequent use of the jaw, may be settings in which functional demands exceed the muscle's capacity to adapt (5).

Objective of TPI

The objective of TPI is to temporarily relax the TP, allowing for improved ROM, and thereby better perfusion, nourishment, and removal of metabolic waste, breaking the pain cycle (27,28). The limitation caused by TPs is considered a perpetuator of TPs (6).

Mechanism of TPI

It is theorized that the mechanism of action of TPI is through mechanical disruption of the TP (6), then breaking the vicious cycle of spasm-pain-spasm (29). The stimulation may also affect the somatosensory thresholds (30), or reduce electrical activity, to reduce pain (31).

Indications for TPI

Numerous treatments exist for MMP, so TPI are not necessarily the first line of treatment. Ideally, as stated earlier, causative factors are identified first, and reduced or eliminated. When MMP still remains, TPI may be considered, as well as the other treatment options previously mentioned. TPI is indicated in patients who have achieved inadequate pain relief or restoration of function from other treatments. It also may be indicated for those who have not complied well with those treatments, as less commitment is needed on the patient's part. Finally, it may be considered in patients who have severe pain in need of rapid relief.

Contraindications for TPI

Pregnancy is considered a relative contraindication; other non-pharmacological options can be considered or enhanced. If the patient is taking anticoagulants, including Aspirin, or has a history of keloid formation, the decision to inject or not inject should be made with the patient's physician. If the TP is near a high-risk structure, the TPI may need to be reconsidered (32).

An active infection at the site of injection (32), or acute trauma to the muscle are absolute contraindications to TPI (33). TPIs should also be avoided in those with a bleeding disorder or allergy to anesthetic.

Technique of TPI

Measurement of the ROM before TPI can be helpful in gauging efficacy on an individual basis (21). A 1.25–1.5 inch needle is preferred, to ensure adequate length to reach the TP from an acute angle, though ¾ inch needles are sometimes used (34). The needle thickness should be 25 or 27 gauge (33); narrower needles may be deflected by firm the TP, rather than penetrate it (21), though they are used (34). Various contents for the syringe have been reported: anesthetic without epinephrine, sodium bicarbonate 5% buffer, dextrose 5% in water, sterile saline, triamcinolone, and dexamethasone (9).

The patient may be sitting or lying down for the injection, depending on the muscle being injected (9), but should be comfortable (33). The injection site is cleansed

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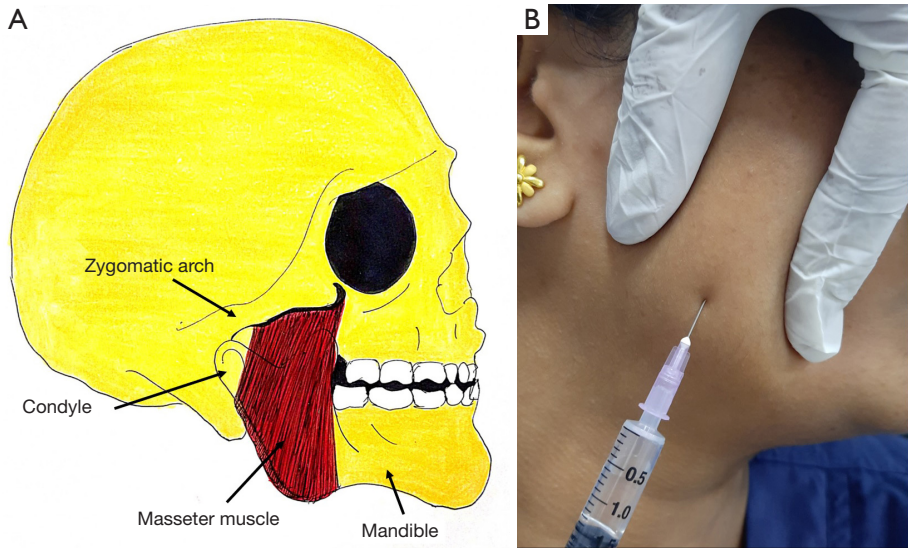

Figure 2 Masseter muscle. (A) Anatomy of the masseter muscle, which originates at the zygomatic arch and inserts at the mandible. (B) Injection of the masseter muscle, with the trigger point stabilized by pincer palpation, and the needle inserted at a 30-degree angle, approximately 1–2 cm from the trigger point.

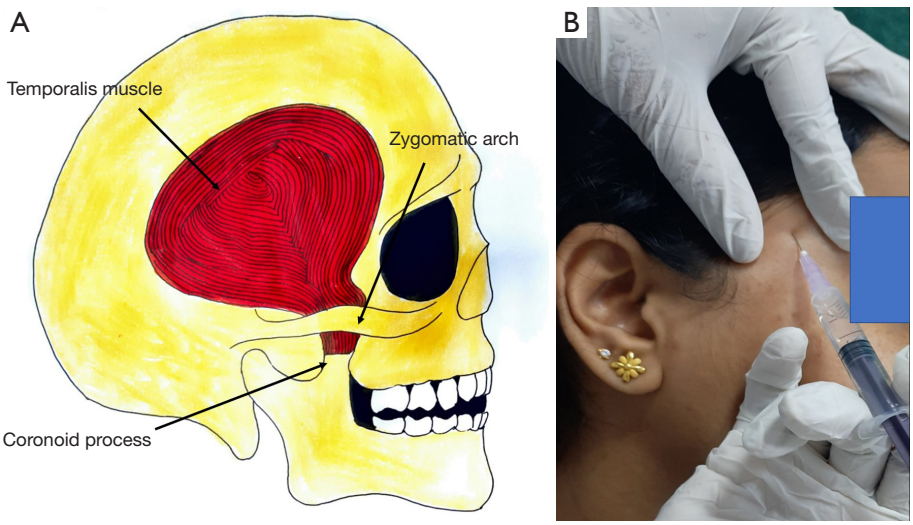

Figure 3 Temporalis muscle. (A) Anatomy of the temporalis muscle, which originates at the temporal fossa, and inserts on the coronoid process of the mandible, as well as the anterior border of the mandibular ramus. (B) Injection of the temporalis muscle, with the trigger point stabilized by pincer palpation, and the needle inserted at a 30-degree angle, approximately 1–2 cm from the trigger point.

with alcohol or chlorhexidine. The TP is then located by palpation, ideally by a twitch, then held in a pincer fashion with one hand (9); holding the TP reduces the likelihood of it rolling aside when contacted by the needle (33). Both during localization and injection of TP in the masticatory muscles, the jaw should be propped widely open (21). The TP should then be cleansed with alcohol, betadine, or 4% chlorhexidine. The needle is then inserted at a 30 degree angle toward the TP; this usually means the skin would be penetrated approximately 1–2 cm from the TP (33) (*Figures 2,3*).

The needle is inserted and retracted repeatedly, without

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fully removing tip from skin, from multiple directions in a fan shape (21,33), slowly delivering the contents throughout, until the twitching has subsided, or muscle adequately relaxed. Crunching or pulling may be felt in the beginning of the process (9). Approximately 1–2 mL of solution is typically used at each TP (33). Once the injection is complete, the patient should gently move the affected structure in its full ROM in all directions (21,33), to initiate perfusion of the muscle. It is advised that this movement is done three times during the appointment.

Following the injection, the patient may apply ice to the TPI areas for a few hours, and should be told that soreness may persist for 3–4 days. The structure should continue to be routinely move the jaw gently and fully in the days following the appointment, to maintain the ROM gained and to allow fuller perfusion of the muscle, the muscle should not be over-used for 3–4 days (33).

Some details when injecting masticatory muscles warrant mention (*Figures 2,3*). The medial pterygoid muscle rarely requires TPI, since it responds well to spray and stretch. When the medial pterygoid or lateral pterygoid muscles are injected, the approach can be extraoral or intraoral. Extraoral access is from below the zygomatic arch and between the mandibular condyle and coronoid process (21). Intraoral access is through the pharyngeal wall for the medial pterygoid muscle. For the lateral pterygoid muscle, intraoral access starts distal to the second maxillary molar, and proceeds in a distopalatal direction (35). When injecting the temporalis muscle, the temporal artery should be first identified and avoided (21).

Positive outcomes

In a randomized, double-blind study, Sabatke *et al.* compared a single TPI with 2% lidocaine LA (n=21), and single TPI with 0.9% saline (n=26), and no TPI (n=23), evaluating their effects 15 days post-injection. Both local anesthetic (LA) and saline TPI statistically significantly decreased facial pain, headache pain, and headache frequency, but there was no significant difference between groups. The control group had no significant improvement in any of the parameters (36).

A systematic review by Machado *et al.* (10) assessed the effect of different TPI techniques, and TPI versus other treatments. In a randomized, double-blind, double-placebo study, McMillan *et al.* compared 1% procaine and simulated dry-needling (n=10), dry needling and simulated LA using saline (n=10), and simulated LA and simulated dry needling (n=10). Injections were performed in the masseter muscle, once a week for 3 weeks, and assessment was performed before and after each session. None of the groups experienced an increase in PPT. All groups had statistically significant decreases in pain intensity and pain unpleasantness, but there were no significant differences between groups (30).

When dry needling (once per week for 3 weeks) in 24 subjects was compared to a combination of 760 mg methocarbamol and 600 mg paracetamol (every 6 hours for 3 weeks) in 24 subjects, in a randomized blinded clinical trial, both groups demonstrated statistically significant reductions in pain at rest and with mastication, when assessed 2 and 8 weeks after completion of treatment. The dry needling group also performed statistically significantly better than the methocarbamol and paracetamol group for improving pain at rest and with mastication, latertrusive range, and protrusive range (11).

Whether TPI was performed with injection of a substance, or without injection of a substance ("dry needling"), pain intensity, frequency, and duration decreased. However, there were no statistically significant differences between TPI with and without injected substances (12,15).

When Fernández-Carnero *et al.* compared dry needling to false needling (using a short needle) in a randomized double-blind cross-over study of 12 subjects, dry needling demonstrated a statistically significant improvement in the PPT and maximum mouth opening (MMO) at the 5 minute post-injection assessment (13). When Dıraçoğlu *et al.* compared dry needling in 25 subjects to false needling (needling a region without a TP) in 25 subjects in a randomized double-blind parallel trial, with needling being performed 3 times at 7-day intervals, dry needling demonstrated a statistically significant improvement in palpation pain threshold (PPT), but not MMO or pain measured on the visual analog scale (VAS), at the 1-week assessment (14).

Non-randomized and non-blinded studies have also been done, though their findings should be interpreted with consideration for their weaker study design. Ozkan *et al.* compared SS (worn for 3 months) in 25 subjects to SS and TPI (first two injection sessions with 0.5 mL lidocaine and 0.5 mL saline; third injection session with 0.1 mL triamcinolone acetonide) in 25 subjects in a randomized non-blinded trial at 2, 4, and 12 weeks after treatment was completed. Both groups saw statistically significant reductions in the frequency and intensity of pain, number

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of TP, number of patients with myofascial orofacial pain at rest, and number of patients with myofascial orofacial pain with movement. The group that received stabilization splints (SS) and TPI also had statistically significantly less pain with movement than the SS group (37).

Gupta *et al.* compared TPI (given once, using 0.5% bupivacaine) in 37 subjects to TPI and levosulpiride (50 mg twice a day for 2 weeks, then increased to a maximum of thrice daily for a maximum of 6 weeks) in 36 subjects, in a randomized non-blinded trial. Assessment was performed at weeks 1, 4, 6, and 12. VAS and depression significantly decreased for both groups at all points. The group that received TPI and levosulpiride had significantly more VAS reduction than the group that received only TPI, at the 4-, 6-, and 12-week points, and significantly more depression reduction at the 6- and 12-week points (38).

Bilici *et al.*, in a non-blinded, non-randomized retrospective cohort study, compared SS (29 subjects) to SS + TPI 3 times, on alternate days (12 subjects), and to SS + TPI 3 times, once a week (15 subjects). The group that received injections on alternate days performed significantly better than the other groups for VAS reduction (29).

Okada-Ogawa A, in a single-blinded study compared TPI done three times, one week apart (10 subjects) to massage and stretch at home for 2 weeks (10 subjects). TPI with massage & stretch significantly decreased VAS, but only massage & stretch significantly decreased taut band hardness. Patients may feel immediate pain reduction, which may wane by 2 weeks later (39).

A non-blinded retrospective cohort study by Yilmaz *et al.* compared TPI with mepivacaine 3% LA (n=21), BTX (n=26), and platelet-rich plasma (PRP) (n=29). Injections were performed once, and assessed 1 month, 3, and 6 months post-injection. At 1 month and 3 months, all groups had statistically significant improvements in pain, jaw function limitation, and self-reported discomfort, and disability due to oral conditions. Groups receiving LA and BTX performed better in those parameters than the group receiving PRP at 3 months. The BTX group performed better in those parameters than the LA group at 3 months. By 6 months, only the BTX group still had significant improvement in any of those parameters (40).

Sakalys *et al.*, in a randomized, non-blinded prospective study, compared TPI in the masseter muscle using 2% lidocaine (n=25) and PRP (n=25). Both groups had statistically significant improvement in VAS at 4 weeks post-injection, but not at 2 weeks post-injection. The PRP injection also performed statistically significantly better than the LA injection for VAS reduction at 4 weeks post-injection, but not at 2 weeks post-injection (41).

Taşkesen *et al.* compared the masseteric nerve block (n=15), TPI with 2% lidocaine (n=15), and dry needling with acupuncture needles (n=15) in a non-blinded study. The random allocation methodology was not clear enough to determine its adequacy. At 12 weeks, all groups had significant increases in maximum opening, but the dry needling and TPI with lidocaine groups had significantly more improvement in pain on function compared to masseteric nerve block group. However, while the dry needling and lidocaine TPI were performed twice with a 7-day interval, it is unclear how many times the masseteric nerve block was administered (34).

The positive outcomes are summarized in *Table 2*.

Negative outcomes

Hammi *et al.* listed adverse effects that can occur with TPIs, which include pain, bleeding, infection, allergy or systemic toxicity to the anesthetic, hematoma, vascular injury, and a vasovagal and syncopal response (9). Severe cramping in the general region of the injection may occur, and is usually due to shortening activation of the antagonist muscle; it can be avoided by also injecting the antagonist muscle. Pain occurs during the injection initially; hyperalgesic patients may have less pain with a smaller gauge needle, though if it is too narrow, it may deflect off the TP, rather than penetrate it (21).

However, it should be noted that none of the studies included in this review reported negative outcomes. Machado *et al.*, in a systematic review, likewise noted no adverse effects in any of the included studies, except those studied Botox injections (10).

Conclusions

There is evidence that TPI may improve MMP symptoms and jaw function, though the evidence is not strong. The evidence also does not conclusively indicate any injected substance, or any substance at all, to be superior. Larger randomized, blinded, controlled studies are needed to determine whether TPIs are effective, and if so, the specific

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Table 2 Characteristics of included studies

Study	Study design	Groups	Follow-up after treatment complete	Effect
McMillan *et al.* 1997 (30)	Randomized, double-blind, double-placebo	Once a week for 3 weeks: 1% procaine and simulated dry-needling (n=10); dry needling and simulated LA using saline (n=10); simulated LA and simulated dry needling (n=10)	Immediately after injection	All groups: no increase in PPT; significant decreases in pain intensity and unpleasantness; no significant differences between groups
von Lindern *et al.* 2003 (11)	Randomized, blind	Dry needling once per week for 3 weeks (n=24); 760 mg methocarbamol and 600 mg paracetamol every 6 hours for 3 weeks (n=24)	8 weeks	Both groups: significant reductions in pain: at rest; with mastication Dry needling: significantly better improvement in: pain at rest; pain with mastication; laterotrusive range; protrusive range
Venâncio Rde *et al.* 2008 (12)	Randomized	Single injection session: dry needling (n=15); 0.25% lidocaine (n=15); 0.25% lidocaine and 4 mg/mL dexamethasone (n=15)	30 days	All groups: significantly decreased: pain intensity; pain frequency; pain duration, local post-injection sensitivity; pain relief obtainment time; duration of relief; use of rescue medication Lidocaine and Decadron group: significantly less local post-injection sensitivity than other groups
Fernández-Carnero *et al.* 2010 (13)	Randomized, double-blind cross-over	Single injection session: false needling (n=12); dry needling (n=12)	5 minutes	Dry needling group: significant improvement in PPT; MMO
Ozkan *et al.* 2011 (37)	Randomized, non-blinded	SS worn 3 months (n=25); SS and TPI (first two injection sessions with 0.5 mL lidocaine and 0.5 mL saline; third injection session with 0.1 mL triamcinolone acetonide) (n=25)	12 weeks	Both groups: significant reductions in: pain intensity; pain frequency; number of trigger points; number of patients with myofascial pain at rest; number of patients with myofascial pain with movement SS and TPI group: significantly less pain with movement
Dıraçoğlu *et al.* 2012 (14)	Randomized, double-blind	3 times at 7-day intervals: false needling (n=25); dry needling (n=25)	1 week	Dry needling: significant improvement in PPT
Silva *et al.* 2012 (15)	Randomized, double-blind	0.25% lidocaine (n=8); dry needing (n=8)	30 days	All groups: significantly increased PPT; significantly decreased VAS; no significant difference between groups
Sabatke *et al.* 2015 (36)	Randomized, double-blind	Single TPI with 2% lidocaine LA (n=21); single TPI with 0.9% saline (n=26); no TPI (n=23)	15 days	LA and saline TPI groups: (I) significantly decreased: facial pain; headache pain; headache frequency; (II) no significant difference between groups No TPI group: no significant improvement in any parameter

Table 2 (*continued*)

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Table 2 (*continued*)

Study	Study design	Groups	Follow-up after treatment complete	Effect
Gupta *et al.* 2016 (38)	Randomized, non-blinded	TPI given once, using 0.5% bupivacaine (n=37); TPI and levosulpiride 50 mg twice a day for 2 weeks, then increased to a maximum of thrice daily for a maximum of 6 weeks (n=36)	12 weeks	TPI and levosulpiride group: significantly more reduction in: VAS; depression
Bilici *et al.* 2018 (29)	Non-randomized, non-blinded, retrospective	SS (n=29); SS + TPI 3 times, on alternate days (n=12); SS + TPI 3 times, once a week (n=15)	3 months	SS + TPI 3 times, on alternate days, had significantly more VAS reduction than other groups
Sakalys *et al.* 2020 (41)	Randomized, non-blinded	TPI in the masseter muscle using: 2% lidocaine (n=25); PRP (n=25)	4 weeks	Both groups: significant VAS reduction PRP group: significantly more VAS reduction
Taşkesen *et al.* 2020 (34)	Random allocation method unclear, non-blinded	Masseteric nerve block (n=15); TPI with 2% lidocaine (n=15); dry needling (n=15)	12 weeks	All groups: significant increase in maximum opening Dry needling and TPI with lidocaine groups: significantly more improvement in pain on function compared to masseteric nerve block group
Okada-Ogawa 2019 (39)	Non-randomized, single-blinded	TPI done three times, one week apart (n=10); massage and stretch at home for 2 weeks (n=10)	On last day of TPI or massage & stretch	Both groups: significantly decreased VAS Massage & stretch group: significantly decreased taut band hardness
Yilmaz *et al.* 2021 (40)	Non-randomized, non-blinded, retrospective	TPI with: mepivacaine 3% LA (n=21); BTX (n=26); PRP (n=29)	6 months	All groups, at 1 month and 3 months, had significant improvements in: pain; jaw function limitation; self-reported discomfort; disability due to oral conditions LA and BTX groups performed better in these parameters than PRP group at 3 months BTX group performed better in those parameters than the LA group at 3 months By 6 months, only the BTX group still had significant improvement in any of those parameters

LA, local anesthetic; PPT, palpation pain threshold; MMO, maximum mouth opening; SS, stabilization splints; TPI, trigger point injection; VAS, visual analog scale; PRP, platelet-rich plasma; BTX, botulinum toxin.

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methods and substances that are effective.

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Footnote

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