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Clinical Consideration of Trigger Point Injection/Dry Needling Therapy: A Narrative Review

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Myogenous temporomandibular disorder is a collective term for pathologic conditions of the masticatory muscles, mainly characterized by pain and dysfunction associated with various pathophysiological processes. Among the subtypes of myogenous temporomandibular disorder, myofascial pain is one of the most common muscle disorders, characterized by the presence of trigger points (TrPs). Various modalities, such as ultrasound, manipulative therapy, spray-and-stretch technique, transcutaneous electrical nerve stimulation, injection/dry needling, and low-level laser therapy are used to inactivate TrPs. Needling/injection on the TrPs is one of the most common treatments for myofascial pain. Despite the evidence, there is continued controversy over defining the biological and clinical characteristics of TrPs and the efficacy of injection/dry needling. This review discusses the current concept of injection/needling to relieve TrPs.

Key Words: Dry needling; Injections; Temporomandibular disorders; Trigger points

INTRODUCTION

Orofacial pain can be categorized according to origin into odontogenic pain and non-odontogenic pain. Pain originating from the masticatory muscles and temporomandibular joints is the most frequent cause of non-odontogenic orofacial pain.[1] Among the types of masticatory muscular pain, myofascial pain is one of the most common causes. A previous study reported that 55% of patients with head or neck pain were diagnosed as having myofascial pain syndrome (MPS).[1] MPS is defined as a regional pain syndrome of non-inflammatory musculoskeletal origin, presenting with muscular pain, referred pain and tenderness, motor dysfunction, and autonomic hyperexcitability. It is also characterized by the presence of palpable hyperirritable spots in the skeletal muscles, termed as trigger points (TrPs). A TrP is defined as a localized tender point in the firm band of skeletal muscles, tendons, or ligaments. TrP is also known as an origin site for MPS-associated symptoms. Accordingly, effective removal of TrPs is considered the most important clinical target to treat MPS. While various modalities, including spray-and-stretch, ultrasound, thermotherapy, ischemic compression, low-level laser therapy, and medication have been used to inactivate TrPs, TrP injection/needling is still considered one of most effective method for MPS because it mechanically disrupts the TrPs.[2] This review summarizes the clinical application of TrP injection/needling with special emphasis on various clinical procedures and related scientific findings.

DEFINITION AND DIAGNOSIS OF MPS

Since MPS was first reported in the 1950s by Travel, intense controversy has been provoked over its existence and

characteristics. Despite half a century of research on MPS, the controversy over defining MPS has continued because of the diversity of the experimental findings.[3] MPS is typically characterized by the presence of TrPs and associated symptoms. TrPs are defined as hyperirritable nodules in a taut band of muscle, and their stimulation or irritation causes not only regional pain in situ but also referred pain in other body parts.

The diagnostic criteria for MPS were first proposed by Simons et al.,[4] and include the following: the presence of TrP, referred pain immediately after the stimulation or irritation of a TrP, local twitch response to snapping palpation or needling on TrP, restricted range of motion and additional findings including muscular weakness, and various autonomic symptoms, such as pilomotor reaction, hypersecretion, and ptosis. However, a recent study proposed modified diagnostic criteria based on an international survey of pain management providers. The modified criteria were as follows: the presence of a tender spot with or without referred pain on palpation as the essential component.[5]

Despite an abundance of studies, many clinicians were confused by the lack of well-established and reliable diagnostic criteria based on experimental evidence. Diagnostic methods including electromyography, infrared thermography, and muscular biopsy have been investigated as possible diagnostic tools for visualizing and characterizing TrPs.[6] Recent studies have shown that non-invasive diagnostic ultrasonography might be used to distinguish hyperirritative muscular areas with TrPs from normal areas without TrPs as a hypoechoic signal area, indicating focal muscle contraction and, possibly, localized intramuscular ischemia.[7] However, further studies on the reliability of diagnostic methods are required for evaluating their practical application in pain clinics. A recent review indicated that while some clinicians consider all painful muscles as MPS, some diagnose MPS strictly only in case with the definite properties of TrPs, and other clinicians ignore the concept of MPS.[8] These confusions among clinicians are probably due to the absence of objective items in the diagnostic criteria and the complexity of the clinical manifestations of MPS.

ETIOLOGY AND PATHOPHYSIOLOGY OF TrPs

Regarding TrP etiology, it has been accepted that they may be mainly caused by acute trauma, including contusion and sprain as well as repetitive microtrauma, probably caused by prolonged poor posture or parafunction.[2] The degenerative structural changes in the joints and the decreased elasticity of fascia with aging may contribute to TrP development. In addition, emotional conditions including prolonged anxiety and stress and physical conditions including sleep disturbances, chronic fatigue, electrolyte imbalance, vitamin deficiencies, and hyposecretion of female and thyroid hormones are possible causes of TrPs. However, many aspects of TrP etiology remain unclear. Moreover, one of the most important pioneers in MPS and TrP research stated that "the cause of MPS is often enigmatic to modern medicine".[9]

Several pathophysiological mechanisms have been proposed to explain the development and progression of TrPs.[10,11] Many experimental studies using molecular biological and/or histopathological analyses have been performed to verify these supposed mechanisms. However, owing to the conflicting results of these studies, the mechanisms remain inconclusive. Considering that TrP might be a pathological but reversible condition in the affected muscle bundle, the invasiveness of the abovementioned methods might affect the results, because these molecular and/or histological experimental procedures might probably alter the intramuscular condition.

It is generally thought that these aforementioned causes, alone or in combination, may cause excessive secretion of acetylcholine at the motor end plate and prolonged muscular contraction, subsequently leading to localized ischemia and accumulated vascular and neuroactive substances in a certain muscle fiber bundle, thus evoking muscular pain. Such muscular pain can, in turn, cause and aggravate contraction and pain as well as further secretion of acetylcholine in the affected muscle bundle, ultimately resulting in a vicious cycle.

CLINICAL MANIFESTATION OF TrPs

Patients with TrPs mainly experience recurrent or

persistent pain that is regional, but sometimes, it can be complex and diffuse.[2] Such a pain is usually aggravated by the usage of the muscle housing the active TrPs, and it does not completely disappear even at rest. The limited range of a certain motion is often attributed to the increased pain on the usage of the affected muscle. The application of pressure over TrPs or contraction causes heterotopic pain in sites distant from the affected muscle, and this is called referred pain. The referral pattern is not associated with anatomic patterns, such as the distribution of dermatomes and nerves, and is expressed as a complex and overlapped distribution of referred pain.[12] Referred pain elicited by TrP is generally assumed to be induced by a central sensitization mediated by a peripheral sensitization process.[13] In extreme cases of MPS, the patient often presents with secondary hyperalgesia and autonomic symptoms, such as sweating, abnormal tearing, abnormal salivation, increased vasomotor response, and increased pilomotor response.

Myofascial pain in the masticatory muscle is primarily similar to that in other body parts. However, myofascial pain entities are often too complicated and obscure to make an exact diagnosis, especially when they are accompanied by autonomic symptoms including facial blushing, blepharoptosis, and tinnitus. This might be attributed to the dense and overlapping innervation from the head, face, and oral structures as well as the complex trigeminal-brain stem pathway with abundant pain and somatosensory processing.[12]

In the masticatory system, myofascial pain is characterized primarily by pain and tenderness originating from the jaw-elevating muscles. The referred pain induced by TrP stimulation is characterized by the fact that it does not cross the body midline toward the contralateral side. In addition, referred pain is generally elicited cephalic to the nerve and not caudally, indicating that pain originating from the trigeminal area rarely refers to the cervical area.[14] Clinical identification of TrPs is mainly based on manual palpation without consistent pressure. Therefore, some researchers have supported the use of an algometer to improve the diagnostic subjectivity by standardizing the pressure of palpation for each masticatory muscle.[15,16]

Clinicians need to distinguish tender points, a term also used to describe muscular pain, from TrPs. Unlike TrPs, tender points are characterized by tenderness on palpation only and the lack of a palpable nodule or band.[2] Tender points are much more frequent than are TrPs in patients with masticatory myofascial pain.[17] Moreover, TrPs are classified into active and latent TrPs, depending on their clinical characteristics.[2] An active TrP presents with spontaneous pain. It also causes a referred pain at the site of palpation, which is a reproduction of the patient's pain symptoms.[2] A latent TrP does not cause any spontaneous pain, but instead causes muscle restriction or weakness at rest. A patient may recognize pain originating from a latent TrP only when elicited in response to compression.

THERAPEUTIC TECHNIQUES FOR TrP MANAGEMENT

The first and most important step to break the cycle of chronic myofascial pain is the identification and evaluation of the perpetuating factors. Thereafter, various therapeutic modalities could be applied as follows: pharmacologic treatments using nonsteroidal anti-inflammatory drugs, antidepressants, and muscle relaxants, and non-pharmacologic treatments including ultrasound, acupuncture, massage, acupressure, spray and stretch, transcutaneous electrical nerve stimulation, thermotherapy or cryotherapy, dry needling, and TrP injection.[2] While the clinical efficacy of these different modalities remains undetermined because of the lack of well-designed experimental studies, dry needling and TrP injection are considered the most effective treatments with a relative abundance of scientific evidence and supportive investigations.[18]

TrP INJECTION AND DRY NEEDLING

1. History and Classification

After the concept of TrPs and injection therapy was popularized since the mid-20th century by Travel and her colleague Simons, dry needling has been utilized by physiotherapists in several western countries since the 1980s and in the United States since 1984 after it was introduced by Karel Lewit of Czechoslovakia.[19,20] However, scientifically important findings had already been reported in the early 20th century by several pioneers, including Kellgren and his

colleague Sir Thomas Lewis. They had already discovered that the intramuscular injection of hypertonic saline elicited pain in a certain region distant from the injection point and named this phenomenon pain referral. Furthermore, they suggested that clinicians should focus more on pain origin than on the pain site after observing that the injection of local anesthetics into tender points also resulted in the alleviation of referred pain within several days.[21]

Needling therapies could be categorized into dry and wet needling, also known as TrP injection, depending on the presence or absence of injectates.[11] Wet needling uses hollow-bore needles to deliver various injectates including anesthetics, steroids, botulinum toxin, or other agents. It was proposed by Travel and Simons, 2 important researchers who coined the term and established the concept of myofascial TrP pain syndrome.[22] Contrary to wet needling, dry needling uses thin non-hollow needles used for acupuncture therapy without the injection of any injectate.[11] While dry needling was empirically used to treat musculoskeletal disorders, it started to be widely used after Lewit's publication[23] supporting that the clinical effect of needling mainly originated from the mechanical effect of the needling rather than the pharmacological effects of the injectate. Dry needling has been primarily established by physiotherapists in western countries including the Canadian physician Dr. Chan Gunn since the 1980s.[20,22] Sometimes, some clinicians have used conventional hollow needles without the injection of injectates, because western clinicians find them easier to acquire and use.

In addition, needling techniques could also be subcategorized into traditional and conventional needling according to the angle of needle insertion.[24] Traditional needling technique is defined as the insertion of a needle perpendicular to the surface of the skin. In contrast, conventional needling is defined as the insertion of a needle at an angle of 30 degrees, which is more preferred by clinicians.[24] While traditional needling seems technically similar to and possibly influenced by traditional Chinese acupuncture (TCA) therapies, the 2 therapies have different underlying therapeutic concepts. TCA is exclusively based on the unique concept of "Qi", which has been hypothesized to circulate between organs through channels called meridians rather than anatomic structures, such as vessels and nerves.[20] However, it is interesting that TCA has also used non-meridian acupoints, termed as "ah-shi" points.[25] Moreover, while a recent study found a meaningful correspondence between TrPs and ah-shi points, the correspondence between TrPs and meridian acupoints was as low as 18%–19%.[26]

2. Clinical Procedures

First, various relaxed postures, including supine, prone, or side lying, are determined to expose the targeted muscles. Pillows and bolsters may be utilized to help the patients position themselves properly. However, the completion of dry needling in a seated position is not recommended in order to reduce the risk of vasovagal reaction.[20] For the masticatory muscles, the reclined position is usually applied during the procedure.[24] Thereafter, clinicians should disinfect the skin over the affected muscle with a cotton sponge soaked in a disinfectant containing 70% isopropyl alcohol and povidone-iodine.[18,20] Clinical methods currently used for identifying TrPs are subdivided into the flat, pincer, and deep palpation techniques.[18] Flat palpation refers to sliding a fingertip or thumb tip with pressure, like plucking a taut string of a stringed instrument, across the muscle fibers of the affected muscle. Pincer palpation refers to gripping a particular muscle by using the thumb and index finger. Deep palpation refers to placing the fingertip over the muscle attachment of the area suspected to include the TrP, when direct palpation of the TrPs would be difficult because of the overlying tissues.[18,27] The first technique is applied for superficial masticatory muscles including the temporalis and masseter muscles that are firmly attached to the underlying bone, and the second technique is more suitable for cervical muscles including the sternocleidomastoid (SCM) and upper trapezius muscles. Whenever tenderness is reported on palpation by the patient, the clinicians should try to identify the exact location of the TrPs based on the apparent elicitation of reproducible pain with the jumping sign and local twitch response with involuntary rhythmic visible contraction through additional meticulous palpation around the initial tender point and adjacent areas. TrPs would be identified as palpable, hyperirritable nodules within the affected muscle.

The detailed procedure for generally applied TrP needling is as follows. After the localization and identification of the

TrP, it should be pinned down by grasping TrP between the index and middle fingers or between the thumb and index finger, which prevents the taut band from rolling away from the advancing needle. Before advancing the needle into the TrP, the patient should be warned regarding the possibility of injection pain, local twitching response, or an irritable sensation when the needle hits the taut band. The needle should be inserted approximately 1 to 1.5 cm away from the TrP, depending on its depth, and finally advanced toward the palpable nodule of the TrP at an acute angel of about 30 degrees.[18] A "fast-in, fast-out" technique is usually used to identify the local twitch response-eliciting points, which is useful to predict precise localization and to exhibit better outcome of TrP injection.[28] If the injectates are injected using a syringe, the plunger should be withdrawn to ensure that the blood vessel is not punctured before injecting a small amount (about 0.2 mL per site) of anesthetic.[2] The needle is then withdrawn to the level of the subcutaneous tissue, and then the needling and injection processes are repeated in the re-oriented direction until further twitch response is exhibited.[2] After completing dry needling or injection, pressure should be manually applied over the injected/needling area for over 2 minutes before confirming hemostasis. Thereafter, an adhesive bandage may be used to protect and secure the punctured skin. Simons et al.[4] suggested that patients perform a rehabilitative self-exercise by fully stretching and contracting the injected muscle 3 times immediately after the injection. In addition, patients should be advised to avoid vigorous usage of the affected muscle for a week as post-injection soreness may deteriorate for 1 to 3 days after the injection.[4] Dry needling or injection is considered successful when the patient reports relief of the previously referred pain, which gradually increases again, even though without returning to the pre-injection pain intensity after lasting for about 1-week.[24] After re-evaluation, several sessions of dry needling or injection are accordingly performed to achieve lasting effects. However, re-injection or needling of TrP is not recommended until the post-injection soreness resolves. Moreover, additional injection is not recommended if 2 or 3 prior trials have been unsuccessful.[24]

A needle with adequate thickness and sufficient length should be selected for better safety and effectiveness, depending on the location of the targeted muscle. A 1.5-inch (3.81-cm)-long 22-gauge needle is usually recommended for most superficially located TrPs, while deeply located muscles may be reached using a 2- or 2.5-inch (5.08- or 6.35 cm)-long 21-gauge needle.[29] A 25-gauge needle is sometimes used in very painful zones or in patients with capillary fragility.[24] The needle recommended not to be inserted to the level of the hub in order to avoid the risk of needle fracture, because the hub is known as the structurally weakest point.[18]

It was generally believed that the usage of a thick needle is more likely to result in accurate penetration of the TrP with highly dense nodular contraction, because thick needles are stiff enough to arrive at the targeted TrP. Studies have also shown that a thick needle provides better tactile sensation when penetrating the overlying cutaneous and subcutaneous tissue.[18] Moreover, some researchers have reported the clinical efficacy of mechanical insertion of an acupotomy in MPS, which is a thick needle with a blade-like flat-edged end.[30] In contrast, a thin needle might cause less irritation and tissue damage during operations. Previous studies have revealed that needle diameter did not appear to influence the effects of dry needling or TrP injection in the treatment of MPS.[31,32] Simons et al.[4] indicated that the diameter of the needle is a matter of individual preference and skill established through personal practice. Therefore, dental clinicians might utilize the dental needle and syringe for the treatment of MPS in masticatory muscles if deployed with care.

Injectates, such as local anesthetics, steroids, nonsteroidal anti-inflammatory drugs, and botulinum toxin have been therapeutically injected in various concentrations and combinations. In particular, injection with botulinum toxin has recently been attempted as an alternative to the conventional strategies to manage intractable pain.[33] However, local anesthetics including 1% lidocaine or procaine are still the most frequently used injectates. Procaine has the least myotoxicity of all local anesthetics and is relatively selective to small, unmyelinated fibers responsible for nociception rather than motor control.[18] However, lidocaine might also be frequently selected because it is more commonly used for clinical practice.[18] Moreover, no experimental comparisons of the popular anesthetics have been previously conducted. After selecting the type of injectate, its proper

volume and concentration are carefully determined according to the type of injectates. About 0.2 mL per site has been recommended as the effective volume for local anesthetics.[2] The application of less than 1 mL per site has been generally advised for injectates.[18] A review suggested 0.5% as the ideal concentration of local anesthetics after considering the therapeutic advantages.[24] Other studies have reported that the most effective concentration for local anesthetics ranged between 0.2% and 0.25% rather than 1%.[34,35] Regarding additive use with local anesthetics, any additives including vasoconstrictors and bisulfite should not be included because they cause post-injection sensitivity and pain.[24]

Regarding the additional effects of injectates, many studies have shown no significant differences among groups of dry needling and wet needling using steroid, procaine or lidocaine, and botulinum toxin.[23,36,37] These findings implied that the main mechanism of the analgesic action of dry needling and even injection might be the disruption of TrPs through mechanical rather than pharmaceutical intervention.[23] Therefore, previous studies have concluded that despite the usage of various types of injectates and their interactions, the injectate does not affect the outcome of TrP injection. Moreover, TrP injection is not therapeutically superior to dry needling in terms of pain relief.[38] However, some therapeutic advantages have been found with the injection of local anesthetics in terms of post-injection discomfort. Post-injection soreness was reported to be more intense and longer in dry needling than in injections with lidocaine.[37,39,40] Furthermore, some studies have proposed botulinum toxin as an emerging therapeutic agent for the care of chronic orofacial TrP.[41,42] A recent study concluded that botulinum toxin injection did not guarantee complete resolution of masticatory myofascial pain despite some beneficial effect and, therefore, should be considered as an alternative treatment for refractory myofascial pain resistant to conservative methods.[41]

Many studies have suggested repetitive in-and-out motions, also known as pistoning or sparrow-pecking motion, as the conventional technique for needling. The in-and-out technique was defined as a therapeutic motion during which the needle is repeatedly inserted into and withdrawn from the TrP with about 1-2 strokes per second; the direction of the needle is slightly changed without completely withdrawing it out of the skin during dry needling or injection.[19] However, a systematic review showed that irrespective of the type of needling/injecting motion, for better relief of pain, the needle should be left in situ for about 10 minutes after its insertion.[11,43]

The absolute contraindications for dry needling/TrP injection are as follows: 1) a patient with needle phobia; 2) patient unwillingness due to patient beliefs, fear, etc.; 3) patient unable to give consent because of communication problems; 4) a patient with a history of abnormal or allergic reactions to injectates or needling; 5) a patient with serious bleeding diseases/conditions that prevent hemostasis establishment using digital pressure; and 6) a patient with uncontrolled susceptibility to infection because of lymphedema and extensive lymphectomy.[20] Once these absolute contraindications are ruled out, the practitioners would need to consider the possible risk of relative contraindications. Relative contraindications include the patients with abnormal bleeding tendency, immunocompromised immune conditions (e.g., bacterial endocarditis, valve replacements, human immunodeficiency virus infection, hepatitis, or cancer), vascular disease, an altered psychological status, pregnancy, uncontrolled diabetes mellitus, debilitating conditions, epilepsy, allergy to equipment material (e.g., metals or latex), local or systemic infection, and intake of significant prescriptive medications (e.g., immunosuppressive drugs, mood-altering medication, or blood-thinning agents); children are also not considered candidates. Additional cautious considerations include a decreased tolerability for these procedures, needling near the surgical site within 4 months, and needling near vulnerable anatomical structures (e.g., nerves, blood vessels, the pleura and lungs, other internal organs, joints, prosthetic implants, implanted electrical devices, or tumors).[20] Orofacial pain specialists should perform needling and TrP injection into the SCM with caution because of the adjacent carotid artery. Unexpected rupture of the carotid artery by the needle might cause rapidly expanding hematoma, thereby resulting in the serious condition of airway compromise. Before needle insertion, clinicians should grab the SCM up by using their thumb and index finger in order to isolate it from adjacent anatomical structures, thereby avoiding the inadvertent rupture of

the carotid artery. The Australian Society of Acupuncture Physiotherapists also introduced risky acupoints that should be approached with caution during needling/injection. When TrP needling/injection is needed around these risky acupoints, clinicians should be aware of their locations during the procedure.

3. Outcome

While the duration of action of these procedures has been reported to vary across studies, a recent systemic review with a meta-analysis found that dry needling resulted in decreased pain immediately and at 4 weeks after the procedure when compared with placebo or sham treatment.[44] A recent study showed that the intensity of pain decreased for as long as 26 days after injection (Dhadwal N 2013 1). Similarly, Gomez-Tames et al.[45] reported that the decreased pain lasted for up to 6 months in patients undergoing dry needling for TrPs on the lateral pterygoid muscle. A previous study provided neurophysiological findings for the long-term efficacy of TrP needling/injection. This study found that the afferent A-delta nerve fibers were stimulated for as long as 72 hours after dry needling and suggested that the prolonged activation of A-delta nerve fibers (group III) might attenuate the muscular pain by activating the enkephalinergic inhibitory interneurons in the dorsal spinal horn.[46] Studies have long been suggesting that a predictive factor for effectiveness is the accurate localization of TrP as indicated by the occurrence of the local twitch response.[28] However, previous studies have indicated that the reliability of TrP diagnosis varied widely for each diagnostic criterion and each affected muscle.[47-50] Lucas et al.[49] revealed that the diagnostic reliability was generally higher for subjective signs, such as tenderness within a taut band and the reproduction of familiar pain but, unfortunately, lower for objective signs, such as the presence of taut band and local twitch response. Lew et al.[48] found that the accurate location of latent TrP was found only in 10% of subjects without referred tenderness and 21% of subjects with referred tenderness. Sciotti et al.[50] reported in the trapezius muscle that the overall amount of locational errors in locating the TrP ranged from 3.3 to 6.6 cm. In addition, the therapeutic outcome of dry needling might depend not only on the adequacy of dry needling procedure, but also on host and disease characteristics. A recent study showed that chronic and severe pain, disturbed sleep, and repetitive stress might be the predictive factors for poor outcomes of dry needling.[51]

Although several investigations have been conducted on the application of TrP injection and needling in the masticatory muscles, only few studies were well designed. Dıraçoğlu et al.[52] found in a double-blind, randomized, placebo-controlled study that dry needling was more effective over sham dry needling on reducing myofascial pain intensity in masseter and temporalis muscles, as measured by higher mean algometric values. However, they also reported the absence of differences in visual analogue scale (VAS) scores of pain intensity and unassisted jaw-opening range without pain between the 2 groups. Gonzalez-Perez et al.[53] reported significant differences in pain intensity and mandibular movement before and after intervention. They also found that dry needling on TrPs in the lateral pterygoid muscle showed better efficacy than did the application of methocarbamol/paracetamol in alleviating pain and increasing the maximum range of mouth opening, as well as lateral and protrusive movements.[54] Ozkan et al.[55] revealed that TrP injection combined with stabilization splint therapy was more effective than stabilization splint therapy alone in relieving myofascial temporomandibular pain. In contrast, a previous study revealed no significant differences in pain pressure thresholds and pain VAS scores among patients receiving dry needling, procaine injection, or sham treatment.[56]

CONCLUSION

Although many aspects related to the application of TrP injection/needling remain unclear despite numerous studies, TrP injection/needling is still considered a clinically effective therapeutic modality. Although TrP injection/needling is a minimally invasive procedure, clinicians should be well aware of the technical procedures, indications, contraindications, and outcomes to ensure effective treatment with few or no complications. Further randomized controlled trials are necessary to determine their underlying mechanisms and to establish clinically optimized procedures.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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