

TRIGGER POINT INJECTIONS: LESSONS FROM THE LITERATURE

TRIGGER POINT INJECTIONS (TPI) HAVE BEEN USED BY CLINICIANS TO TREAT MYOFASCIAL PAIN SYNDROME (MPS) FOR DECADES. HOWEVER, MOST OF THE HIGHEST-QUALITY RESEARCH IN THIS AREA HAS BEEN CONDUCTED ONLY IN THE LAST TWO DECADES, SHEDDING NEW LIGHT ON INDICATIONS FOR THIS PROCEDURE, ITS MECHANISM OF ACTION, GUIDANCE ON INJECTATE AND NEEDLING TECHNIQUE, AND ITS COMPARISON TO OTHER MODALITIES. THIS PRESENTATION WILL REVIEW THE LATEST MEDICAL LITERATURE PERTAINING TO THIS COMMONLY USED PROCEDURE.



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- Dr. Shelley has **NO** conflicts of interest related to the content of this presentation.

OBJECTIVES



- After this presentation, the attendee will be able to :
 - Describe the mechanism of action of TPI.
 - Compare different injectate options and needling techniques.
 - Identify patient candidates for trigger point injections (TPI).
 - Evaluate how TPI compares with other modalities for the treatment of Myofascial Pain Syndrome (MPS).

ABBREVIATIONS

- MPS Myofascial Pain Syndrome
- MTrP Myofascial Trigger Point
- TPI Trigger Point Injection
- DN Dry Needling
- SP Substance P
- BK Bradykinin
- 5-HT 5-hydroxytryptamine
- CGRP Calcitonin gene-rel. peptide
- IL Interleukin
- NE Norepinephrine
- TNF Tumor necrosis factor
- ACh Acetylcholine
- AChE Acetylcholine esterase

OBJ: DESCRIBE THE MECHANISM OF ACTION OF TPI.



ABOUT MPS AND MTRPS:

GERWIN RD. DIAGNOSIS OF MYOFASCIAL PAIN SYNDROME. *PHYSICAL MEDICINE AND REHABILITATION CLINICS OF NORTH AMERICA*. 2014;25:341–355.

- Myofascial pain syndrome (MPS) is common.
- MPS is due to TrPs which are a small region of hardness and tenderness in a taut band of muscle.
- TrPs refer pain.
- Diagnosis is made by palpation of TrPs along taut bands.
- Predisposing factors are important.

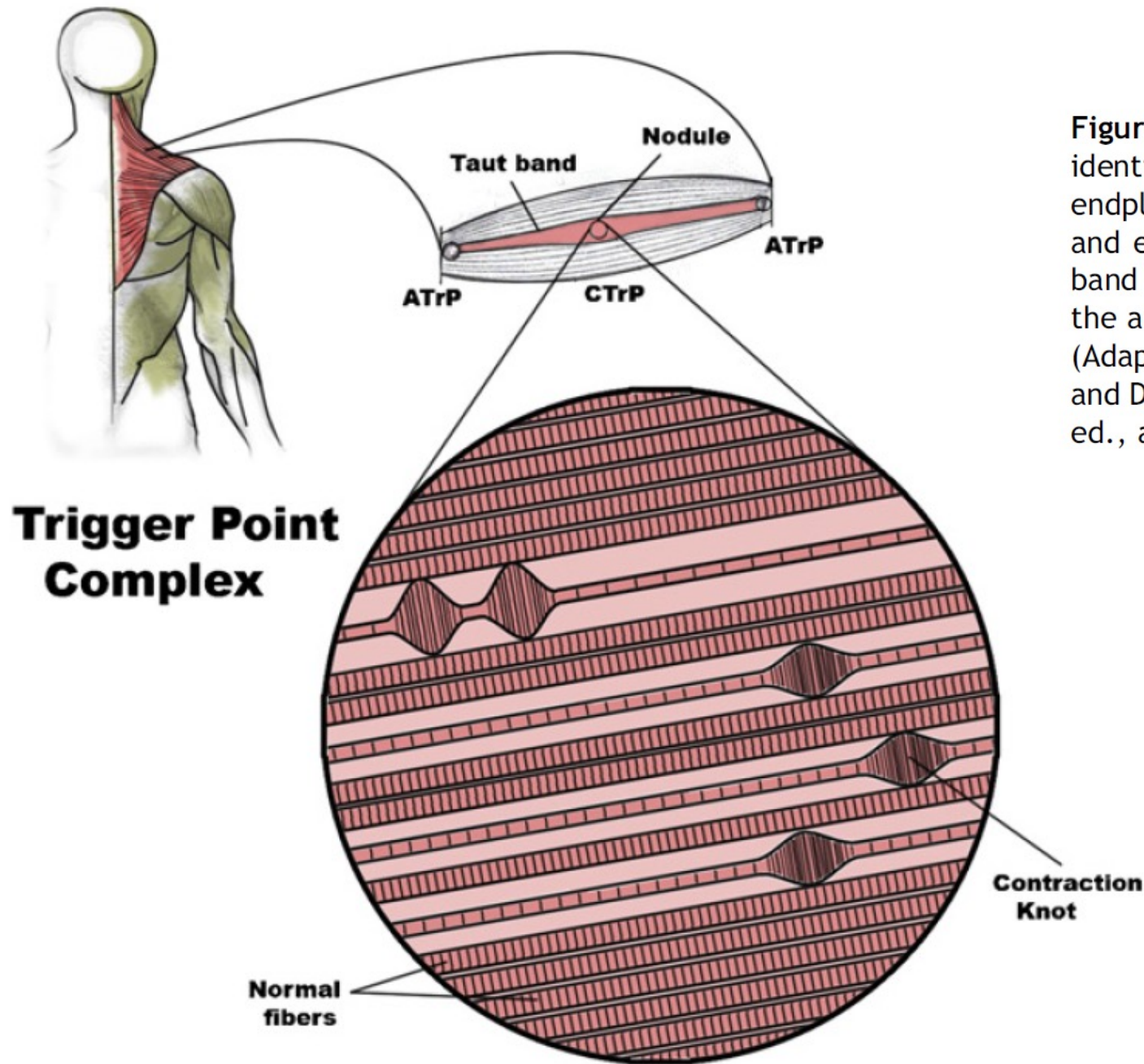


Figure 1 Schematic of a trigger point complex. CTrP identifies the central trigger point that is found in the endplate zone and contains numerous contraction knots and electrically active loci among normal fibers. A taut band of muscle fibers extends from the trigger point to the attachment (ATrP) at each end of the involved fiber. (Adapted from Simons, D.G., Travell, J.G. *Myofascial Pain and Dysfunction: The Trigger Point Manual*, vol. 1; second ed., and Användare: Chrizz.)

GERWIN: SX & ASSOCIATED CONDITIONS

Symptoms:

- Sharp or dull
- Muscular or visceral
- Achy or radicular

MTrP can activate **autonomic activity**:

- Vasodilation
- Vasoconstriction
- Piloerection
- Tearing

Associated with radiculopathy and joint pain

- MPS can persist long after primary cause of pain eliminated

EPI & DDX:

BORG-STEIN J, IACCARINO MA. MYOFASCIAL PAIN SYNDROME TREATMENTS. *PHYSICAL MEDICINE AND REHABILITATION CLINICS OF NORTH AMERICA*. 2014;25(2):357-374.

- Epidemiology:
 - 21-30% in internal medicine and ortho clinics;
 - up to 90% in some pain clinics;
 - men and women are equally affected.
- Differential diagnosis: see next slide

Table 1
Differential diagnosis for MPS

Joint disorders	Zygapophyseal joint disorders Osteoarthritis Loss of normal joint motion
Inflammatory disorders	Polymyositis Polymyalgia rheumatica Rheumatoid arthritis
Neurologic disorders	Radiculopathy Entrapment neuropathy Metabolic myopathy
Regional soft tissue disorders	Bursitis Epicondylitis Tendonitis Cumulative trauma
Diskogenic disorders	Degenerative disk disease Annular tears Disk protrusion or herniation
Visceral referred pain	Gastrointestinal Cardiac Pulmonary Renal
Mechanical stress	Postural dysfunction Scoliosis Leg length discrepancy
Nutritional, metabolic, and endocrine disorders	Vitamin deficiency (B ₁ , B ₁₂ , D, calcium, folic acid, iron, magnesium) Alcoholic and toxic myopathy Hypothyroidism
Psychological disorders	Depression Anxiety Disordered sleep
Infectious disease	Viral illness Chronic hepatitis Bacterial or viral myositis
Widespread chronic pain	Fibromyalgia

BORG-STEIN: OTHER TREATMENTS

Modalities:

- Education
- Exercise
- Stress reduction
- Acupuncture
- Massage
- TENS and other similar therapies
- Ultrasound with heat +/- NSAIDs
- (LLLT)
- (Manual therapies)

Medications:

- Topical NSAIDs (no data for oral NSAIDs in MPS *per se*)
- Cyclobenzaprine (for sleep)
- Tizanidine
- Amitriptyline
- SNRIs (no data for MPS *per se*)
- Tramadol (no data for MPS *per se*)
- Topical lidocaine

GERWIN: OBJECTIVE LOCALIZATION

MTrPs can now be identified by **imaging**:

- Magnetic resonance elastography
- Ultrasound with vibration sonoelastography

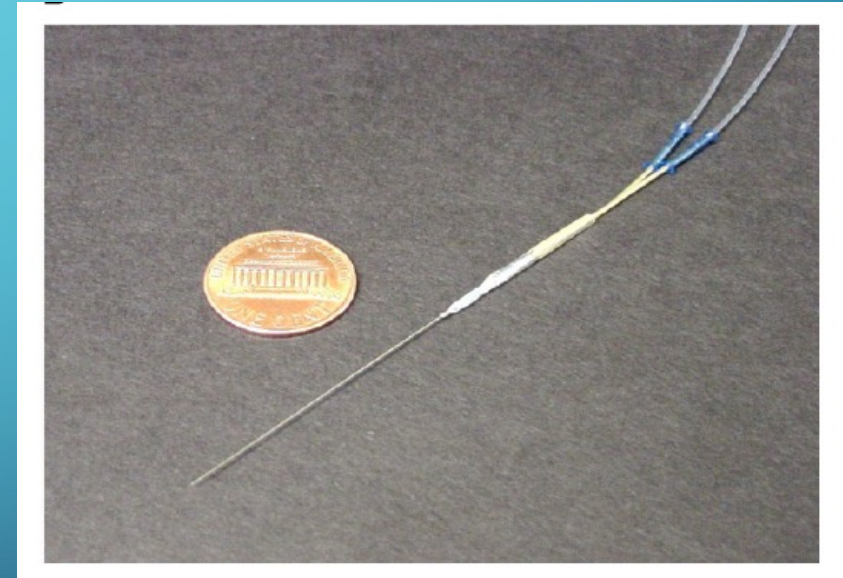
EMG detects low-amplitude high-frequency discharges in the MTrP

- Aka Spontaneous Electrical Activity (SEA) or “endplate noise”
- This probably arises from the motor endplate, caused by $\uparrow$ ACh
- The degree of SEA correlates with the degree of MTrP irritability

SHAH: BIOCHEMICAL MILIEU

Microdialysis sampling of MTrPs shows:

- ↓ pH
- Neuropeptides: SP CGRP BK
- Catecholamines: NE 5-HT
- Cytokines: TNF- α , IL-1 β , IL-6, IL-8



GERWIN: MTRP PHYSIOLOGY

Pain at MTrPs is due to the release of neuropeptides, cytokines, and inflammatory substances:

- Substance P (SP)
- Calcitonin gene-related peptide (CGRP)
- Interleukin-1 α (IL-1)
- Bradykinin (BK)
- Acidic protons

MTrPs refer to distant sites and lead to **CNS sensitization** (or vice versa?)

- CNS sensitization then →
- ↓ pain threshold
- ↑ tenderness
- Expansion of painful areas and the numbers of MTrPs
- (Rationale for neurologic disruption including anesthetics.)

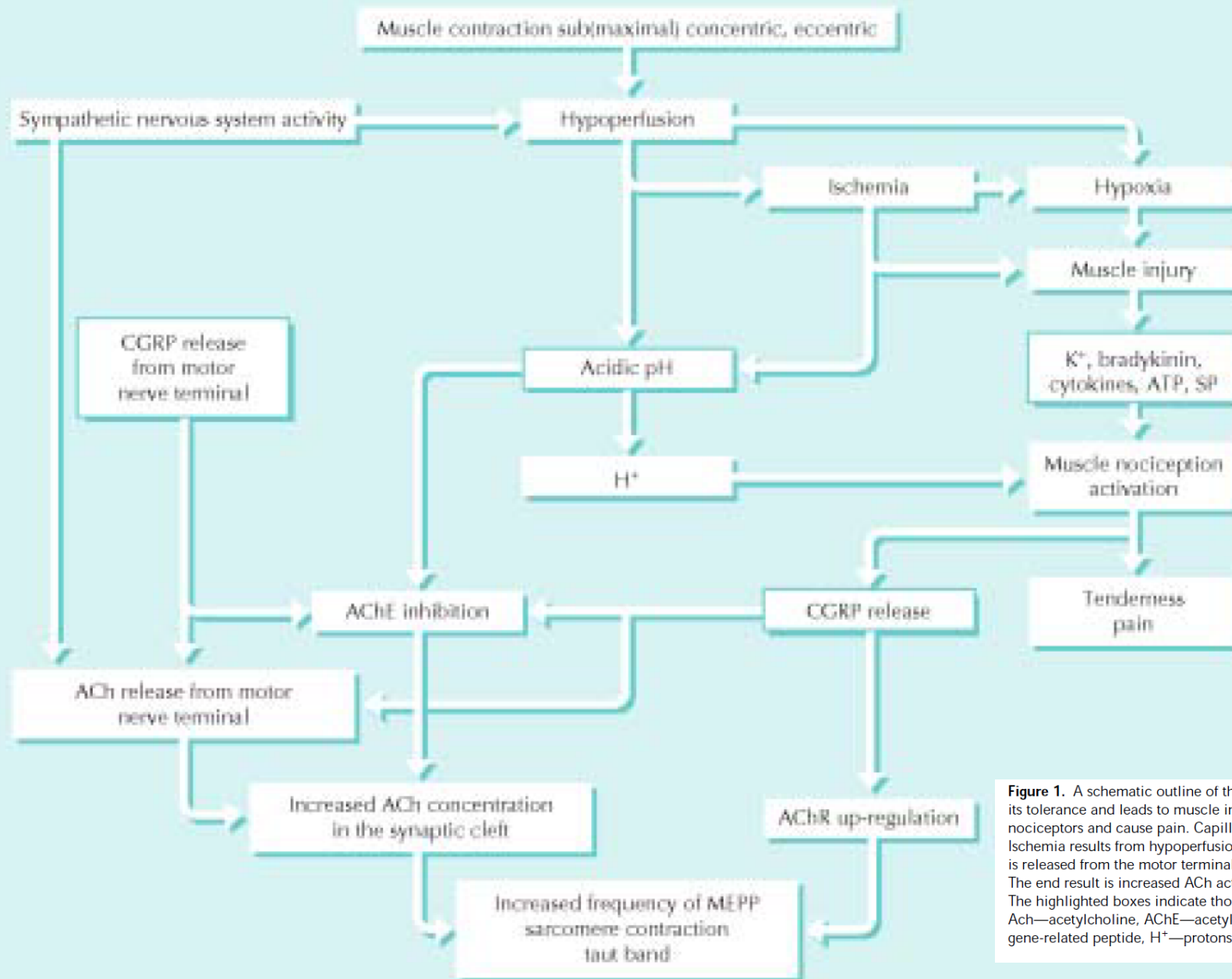
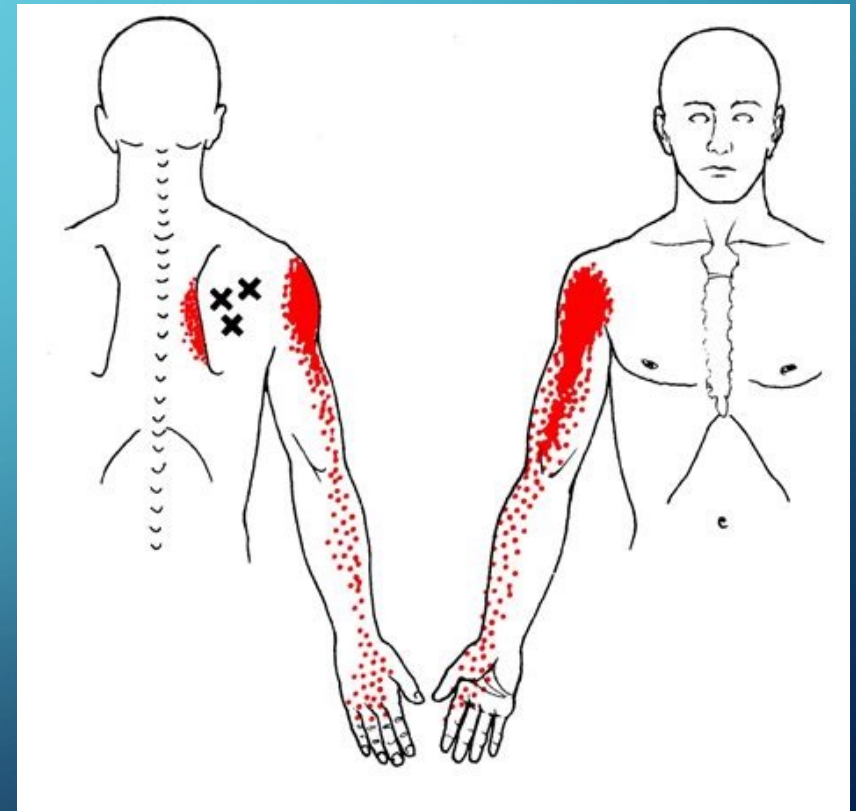


Figure 1. A schematic outline of the expanded trigger point hypothesis. The activating event is muscle activity that stresses muscle beyond its tolerance and leads to muscle injury and capillary constriction. Muscle injury results in the release of substances that activate muscle nociceptors and cause pain. Capillary constriction occurs as a result of muscle contraction and sympathetic nervous system activation. Ischemia results from hypoperfusion, which is caused by capillary constriction. The pH becomes acidic, inhibiting AChE activity. CGRP is released from the motor terminal and from injured muscle. CGRP inhibits AChE, facilitates ACh release, and up-regulates AChRs. The end result is increased ACh activity with increased frequency of MEPPs, sarcomere hypercontraction, and the formation of taut bands. The highlighted boxes indicate those events that have been identified or are supported by microdialysis studies of the trigger point. ACh—acetylcholine, AChE—acetylcholinesterase, AChR—acetylcholine receptors, ATP—adenosine triphosphate, CGRP—calcitonin gene-related peptide, H⁺—protons, K⁺—potassium, MEPP—miniature endplate potentials, SP—substance P. Adapted from Shah *et al.* [13•].

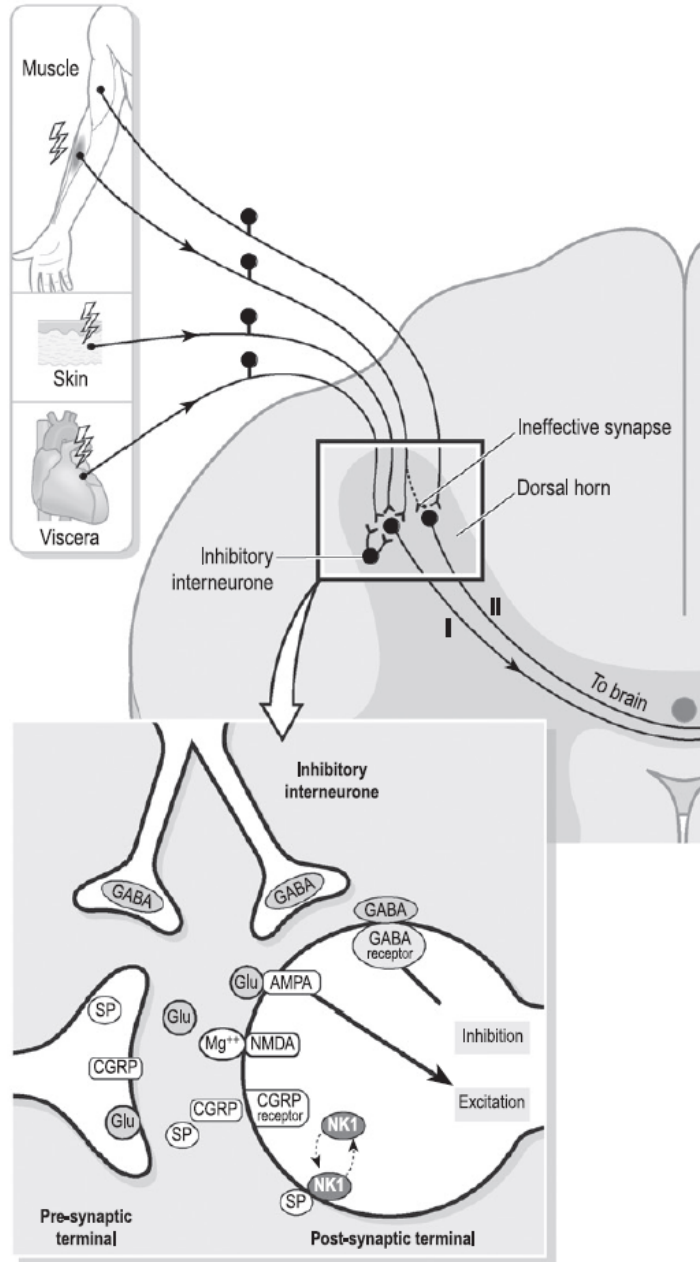
GERWIN: CNS SENSITIZATION

- The LTR is elicited by mechanical stimulation of the taut band causing local contraction (not a DTR). This requires an intact spinal cord reflex.
- Referred pain represents central sensitization, usually in the nerve which innervates the muscles:
- Infraspinatus is mostly innervated by C5
- Can spread to C4 and C6
- Can continue to spread unchecked.



A

Acute Nociceptive Transmission



B

Centrally Sensitized Nociceptive Transmission

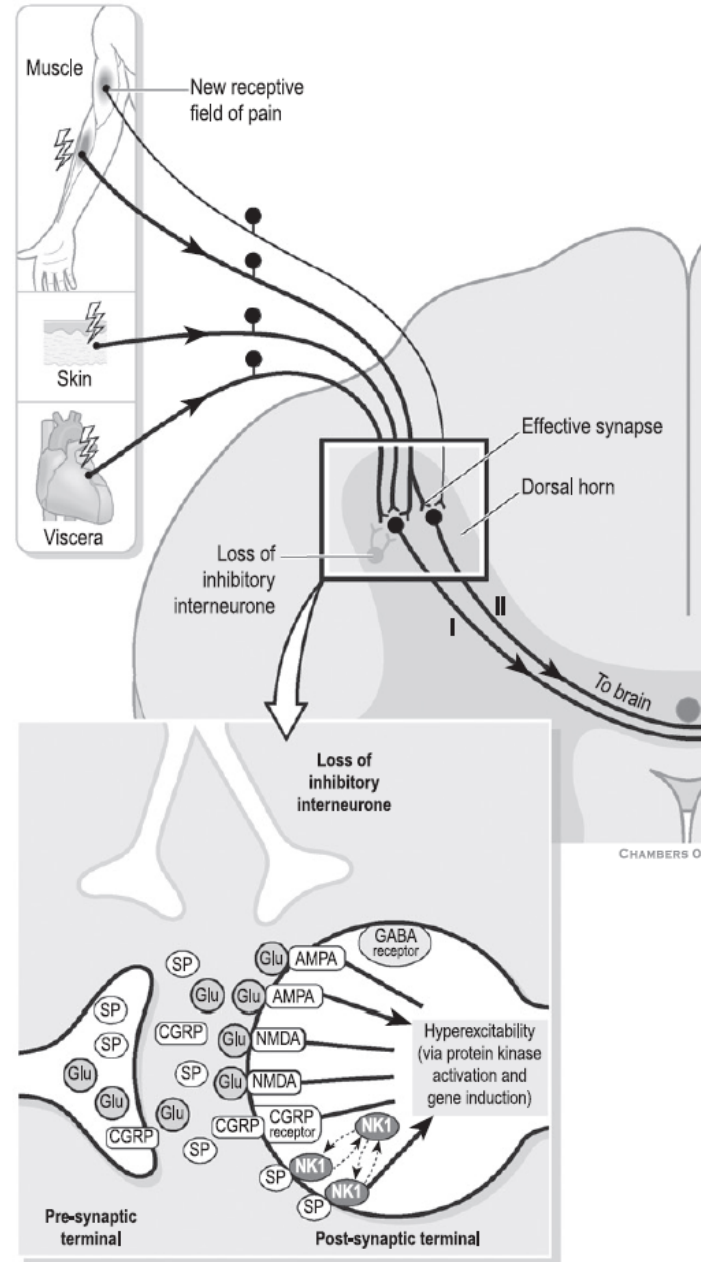


Figure 3 Transition from normal to pathological pain perception, via central sensitization at the spinal cord dorsal horn. Insets of central synaptic transmission. (A) Acute nociceptive transmission. Nociceptive signals may originate from muscle, cutaneous, or visceral afferent neurons. (B) Centrally sensitized nociceptive transmission. Convergence of muscle, cutaneous, and visceral afferents can be responsible for referred pain patterns. Activation of ineffective synapses in the dorsal horn may create additional receptive pain fields of pain. For example, input from the extensor carpi radialis longus normally activates neuron I. With intense and/or continuous noxious input from the extensor carpi radialis longus, another previously ineffective (silent) synapse may be converted into an effective (active) synapse. Here, a synapse to neuron II, which normally receives input from the biceps brachii, becomes effective. The expansion of effective spinal connections at neuron II can create a new receptive field of pain at the biceps brachii, where no nociceptor is being activated and the tissue is normal. Increased neurotransmitter release at the dorsal horn alters receptor ion channel activity. All of these factors contribute to central hyperexcitability via protein kinase activation and gene induction. Glu: glutamate, SP: substance P, CGRP: calcitonin gene-related peptide, GABA: gamma-aminobutyric acid, AMPA: α -amino-3-hydroxy-5-methyl-4-isoxazole propionate, NMDA: N-methyl-D-aspartic acid, NK1: neurokinin 1. (Adapted from Mense, 2003; Vadivelu, N., Sinatra, R., 2005. Recent advances in elucidating pain mechanisms. *Current Opinion in Anaesthesiology* 18, 540–547; Willard, 2008.)

GERWIN ET AL: HOW MPS DEVELOP

- Muscle strain/injury →
- muscle fiber damage and segmental hypercontraction →
- capillary constriction →
- hypoperfusion →
- ischemia/hypoxia →
- ↓ pH →
- ↓ AChE activity →
- ↑ ACh ↑ CGRP ↑ SP ↑ K⁺ ↑ 5-HT ↑ BK
- **Summary:** Insult → ↑ ACh → motor endplate activity → taut band → CNS sensitization

BORG-STEIN: NEEDLE MOA

Mechanism of action is theoretical:

- The needle disrupts and stops the dysfunctional activity of the motor endplate of the skeletal muscle motor neuron via the LTR
- Superficially: the needle possibly deactivates MTrPs by stimulation of cutaneous A- δ fibers *without* LTR
- Deep needling: targets muscle afferents and $\downarrow$ pain.
- Anesthetic: $\downarrow$ central sensitization and neurogenic component of MTrP
- TPI $\approx$ DN in efficacy but TPI is typically covered by payors for medical providers

PERREAU: OTHER NEEDLE MOA THEORIES

DN

- Mechanical disruption of muscle fibers and nerve endings might stimulate some α motor neurons in spinal cord via afferent fibers
- DN reduces motor endplate activity – LTR may $\downarrow$ ACh
- DN results in $\uparrow$ β -endorphins, $\downarrow$ CGRP and $\downarrow$ SP
- Physiologic mechanism unknown
- DN might just cause vasodilation to “wash out” noxious substances

PERREAU: OTHER NEEDLE MOA THEORIES

Acupuncture can:

- ↑ CGRP and SP *without* DTR → vasodilation
- Or: needling causes ↑ CGRP/SP which creates a negative feedback loop
 - CGRP in high concentrations stimulates inflammation
 - CGRP in low concentrations is anti-inflammatory

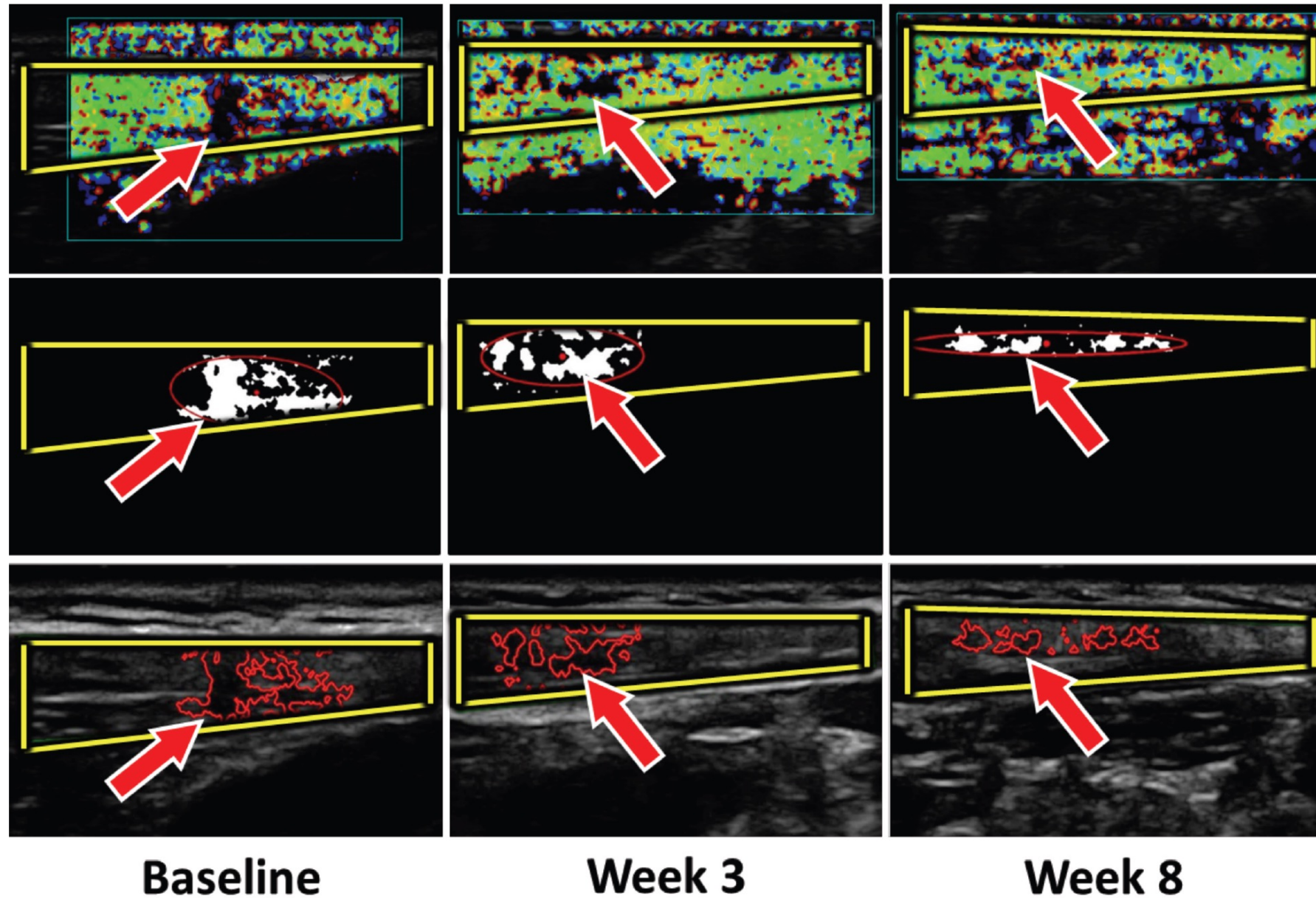
Winding (rotating) the needle may:

- ↑ cellular ATP
- Stimulate C fibers, mechanoreceptors, stretch receptors

TURO: NEEDLING EFFECTS

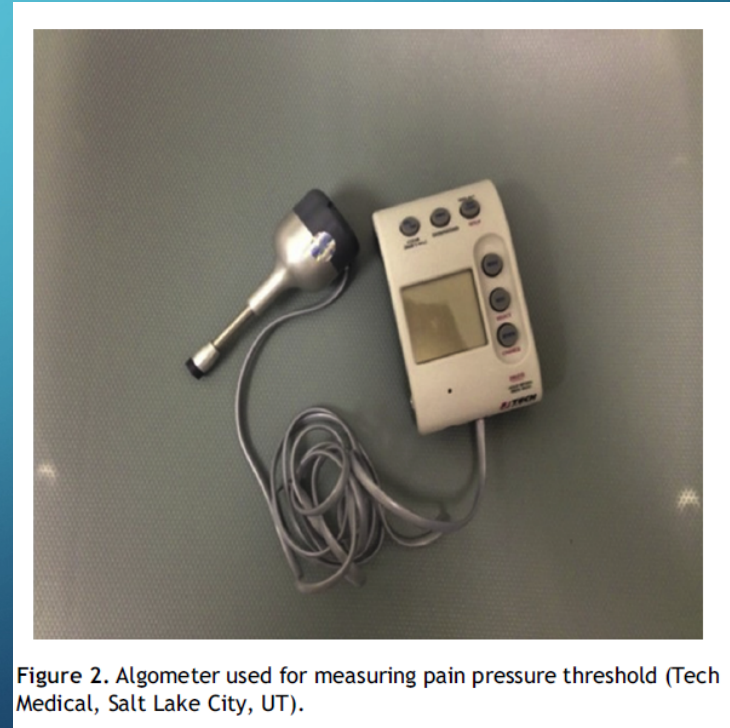
- DN mechanism of action not known
- Not just MTrPs but surrounding tissue too, which can have a “heterogeneity of **stiffness**”
- Dry needling TrPs in the upper trapezius muscle resulted in:
 - Significant decreases in the MECHANICAL HETEROGENEITY INDEX
 - Correlated with decreased pain
 - Demonstrates that the pain reduction is due to changes in the TrP @ 3 & 8 wks
 - See U/S pics...

Figure 3. Myofascial trigger point (arrows) size evolution. Top row, Color Doppler variance images. Middle row, Binary images of the mechanical heterogeneity region. Bottom row, Yellow borders indicate B-mode images of the upper trapezius muscle, and red borders indicate regions with a lower vibration amplitude (red line).



GERBER: NEEDLING EFFECTS

- Needling TrPs in the upper trapezius resulted in:
 - Single study, $n = 52$, average age 36 yo (young)
 - Pain score reductions
 - Improved cervical range of motion
 - Decreased disability scores
 - Improved **pain pressure threshold** (using algometer)



MECHANISM OF ACTION: SUMMARY

- TPI with anesthetic breaks the cycle of afferent nerve transmission to the CNS
- DN (no anesthetic) also provides mechanical disruption
- TPI or DN result in:
 - ↓ ACh
 - ↑ Blood flow
 - ↓ SEA



DISAPPOINTMENT

MECHANISM OF ACTION: SUMMARY

- BUT the exact mechanism of how the needle does this is not yet known.
- However, there are many well-known physiologic and clinical correlations with needling.
- Note, the MOA of several common drugs is also not completely known, including:
 - Acetaminophen
 - Cyclobenzaprine
 - Metformin

https://en.wikipedia.org/wiki/Category:Drugs_with_unknown_mechanisms_of_action

**OBJ: COMPARE DIFFERENT INJECTATE OPTIONS AND
NEEDLING TECHNIQUES.**



LOCAL TWITCH RESPONSE MAY NOT BE ESSENTIAL

- Perreault (2017):

- LTR not consistent with pain response over time
- “Pistoning” to elicit TR may result in more post-injection soreness



GERBER: NEEDLING TRAPEZIUS MTRPS

- Trauma, muscle overload, muscle overuse →
- Release of noxious substances that bind to, sensitize, and/or activate nociceptors →
- Leading to “pain dysregulation” (peripheral and central):
 - ↑ afferent signaling of tissue damage and inflammation
 - ↓ descending inhibition
- ***The elicitation of the twitch response did not distinguish responders from non-responders.***

LIDOCAINE BETTER THAN SALINE, AND A SERIES IS BETTER THAN ONE

- Karadas (2013):
- N = 108 with frequent ETTH, split into 4 groups:
 - Group 1: 1 injection of saline (NaCl 0.9%) (n = 27)
 - Group 2: 1 injection of lidocaine (0.5%) (n = 27)
 - Group 3: 5 injections of saline (NaCl 0.9%) (n = 27)
 - Group 4: 5 injections of lidocaine (0.5%) (n = 27)
- On alternate days 2 mL for each muscle was injected bilaterally into these muscles:
 - frontal, temporal, masseter, sternocleidomastoid, semispinalis capitis, trapezius and splenius capitis
- Frequency of painful days per month (FPD) and visual analogue scales (VAS) were evaluated before treatment, and 2, 4 and 6 months after treatment.
- FPD scores improved significantly in group 2, 3 and 4 at 2 months posttreatment.
- Group 4 had better scores than group 3 at the 2, 4. and 6 months after treatment in both parameters (all $P < 0.05$)

RESEARCH ARTICLE

Open Access

Lidocaine injection of pericranial myofascial trigger points in the treatment of frequent episodic tension-type headache

Ömer Karadaş^{1*}, Hakan L Gül² and Levent E İnan³

COMBINATION WITH PSYCH MEDS MAY BE HELPFUL

GUPTA P, SINGH V, SETHI S, ET AL. A COMPARATIVE STUDY OF TRIGGER POINT THERAPY WITH LOCAL ANAESTHETIC (0.5% BUPIVACAINE) VERSUS COMBINED TRIGGER POINT INJECTION THERAPY AND LEVOSULPIRIDE IN THE MANAGEMENT OF MYOFASCIAL PAIN SYNDROME IN THE OROFACIAL REGION. *JOURNAL OF MAXILLOFACIAL AND ORAL SURGERY*. 2016;15:376–383.

- TPI combined with oral levosulpiride significantly helped people with facial MPS and moderate-severe depression (compared to TPI alone).
- Levosulpiride is a typical antipsychotic and a prokinetic agent of the benzamide class.
- It is a selective antagonist of the dopamine D2 receptors on both central and peripheral nervous systems.

LARGER NEEDLES MAY BE MORE BENEFICIAL

YOON SH, RAH UW, SHEEN SS, ET AL. COMPARISON OF 3 NEEDLE SIZES FOR TRIGGER POINT INJECTION IN MYOFASCIAL PAIN SYNDROME OF UPPER-AND MIDDLE-TRAPEZIUS MUSCLE: A RANDOMIZED CONTROLLED TRIAL. *ARCHIVES OF PHYSICAL MEDICINE AND REHABILITATION*. 2009;90:1332–1339.

- Three needle sizes were tested:
 - 21G
 - 23G
 - 25G
- No difference was seen in VAS or NDI (Neck Disability Index).
- SF-36 scores indicated that 21G or 23G needles were better.
- Presenter note: I use 27G needles—these were not included here.

OBJ: IDENTIFY PATIENT CANDIDATES FOR TRIGGER POINT INJECTIONS (TPI).



GOOD DATA FOR TPI IN HEADACHE

- Robbins (2014):

- Good review of MPS and general approach to TPI
- Review of lit by The Peripheral Nerve Blocks and Other Interventional Procedures Special Interest Section of the American Headache Society
- 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.

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

GOOD DATA FOR TPI IN HEADACHE

- Robbins:

- 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.

Review Articles

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TPI IS HELPFUL FOR ACUTE LOW BACK PAIN

- Kocak (2019):

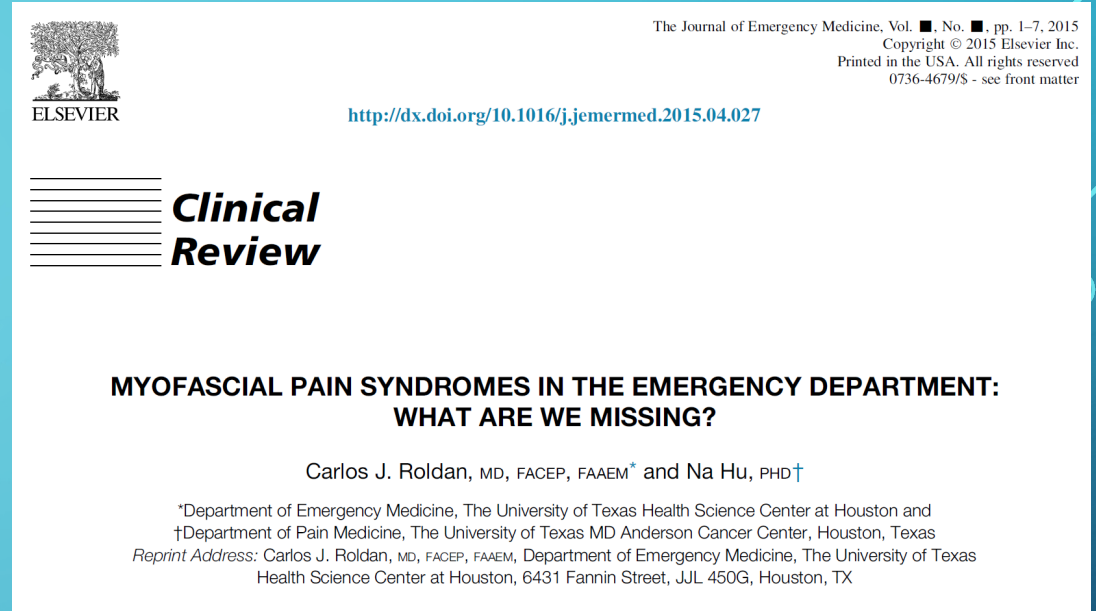
- N = 54
- TPI reduced average pain in ED from 7/10 to 1/10
- Better than IV NSAID (50 mg dexketoprofen)



TPI CAN BE DIAGNOSTIC AND THERAPEUTIC FOR ILIOCOSTALIS THORACIS-LUMBORUM MYOFASCIAL PAIN

- Roldan (2015):

- Anterior torso pain often resulted in extensive workups before ITL myofascial pain was diagnosed.
- 74% did not report LBP. Coughing was common in recent history.
- TPI were diagnostic and therapeutic of ITL myofascial pain.



Iliocostalis Thoracis-Lumborum Myofascial Pain

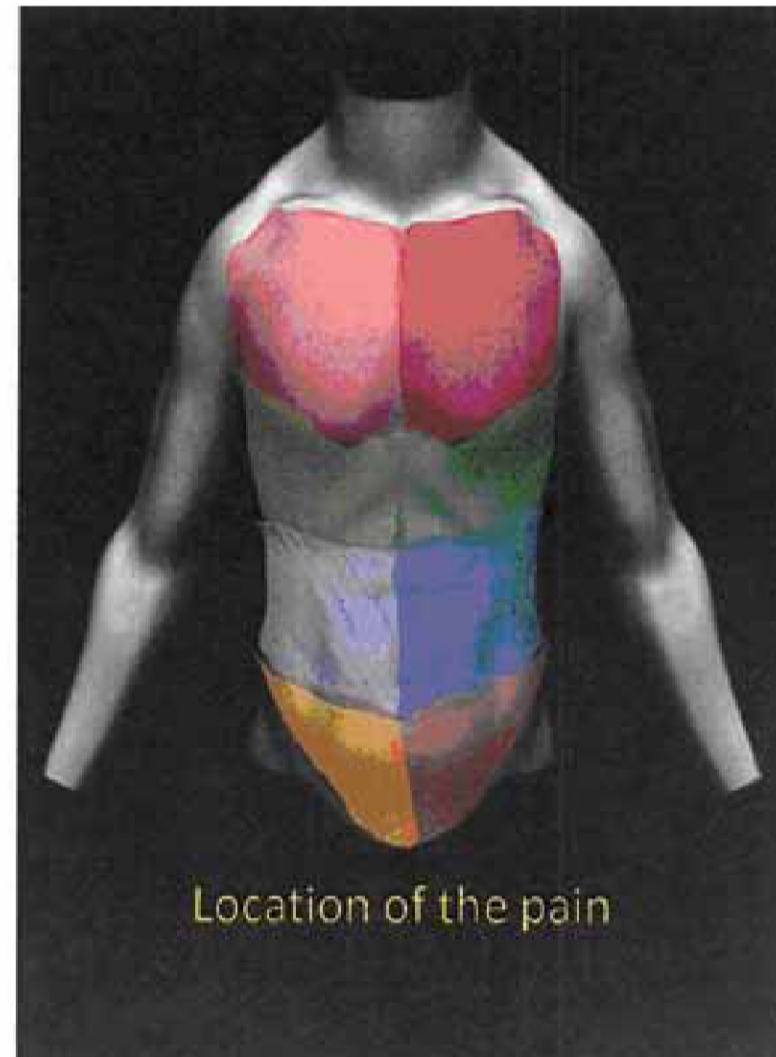
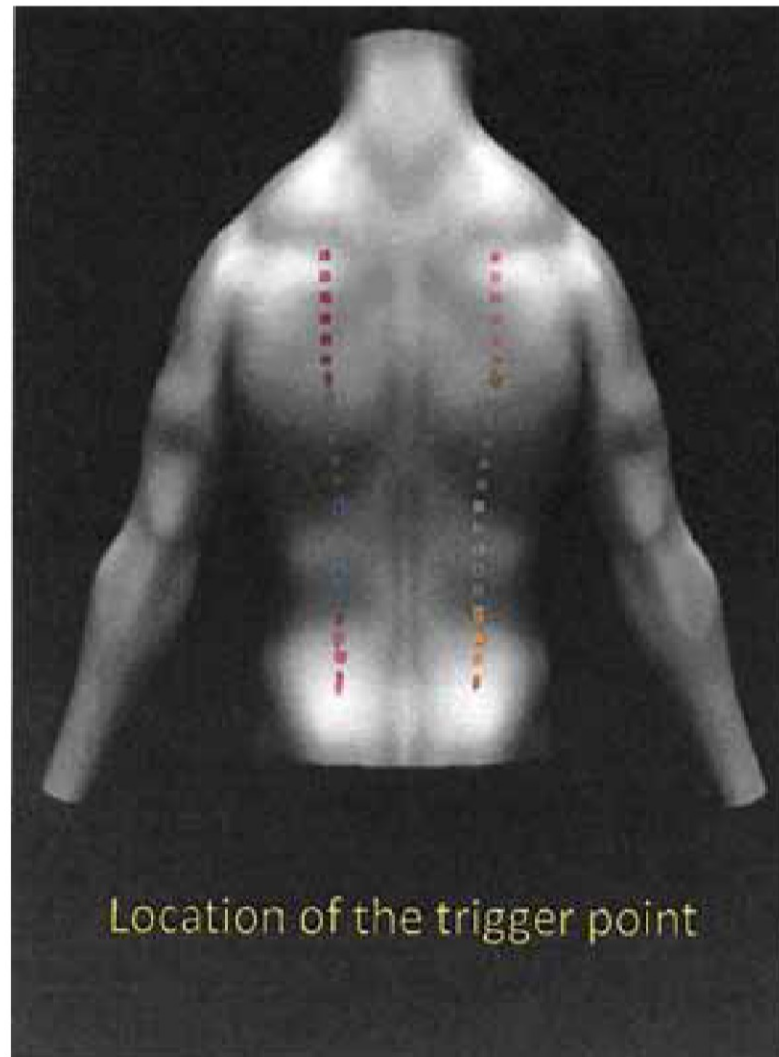


Fig. 1. *Graphic representation of the anatomical location of the iliocostalis-thoracis-lumborum muscle and the virtual location of the trigger points. Trigger points correlate with the area of referred pain in the anterior torso.*

PRESENCE OF MTRPS MAY INDICATE RADICULOPATHY

ADELMANESH F, JALALI A, SHIRVANI A, ET AL. THE DIAGNOSTIC ACCURACY OF GLUTEAL TRIGGER POINTS TO DIFFERENTIATE RADICULAR FROM NONRADICULAR LOW BACK PAIN. *CLINICAL JOURNAL OF PAIN*. 2016;32:666–672.

- TrPs in the superior-lateral quadrant of the gluteal are highly specific indicators for radicular LBP.
- Authors suggest using physical exam rather than expensive tests.
- Presenter's dissent: this is probably the gluteus minimus TrP, which is much more common than radiculopathy

TREATING MTRPS IN RADICULOPATHY MAY BE BENEFICIAL

- Saeidian (2014):

- $n = 100$
- Participants had LS radiculopathy, s/p 1 week of conserv. care; then checked for TrPs.
- Got TPI, one week later pain $7 \rightarrow 2$, SLR neg. (others $6.7 \rightarrow 4.06$)
- Muscles: LE muscles and lumbar paraspinals (gluteals and piriformis not mentioned)
- Conclusions: Results show that trigger point injection therapy in patients suffering from chronic lumbosacral radiculopathy with trigger points can significantly improve their recovery, and conservative therapy may not be adequate.

Effect of Trigger Point Injection on Lumbosacral Radiculopathy Source

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TPI FOR POST-MASTECTOMY SHOULDER PAIN

- Shin (2014):
 - Pilot study, $n = 78$
 - Subscapularis and pectoralis TPIs helped post- mastectomy shoulder pain

Application of Ultrasound-Guided Trigger Point Injection for Myofascial Trigger Points in the Subscapularis and Pectoralis Muscles to Post-Mastectomy Patients: A Pilot Study

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³Department of Physical and Rehabilitation Medicine, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea.



Fig. 1. Patient position for the subscapularis. The patient lay supine with the shoulder abducted, elbow flexed, hand supinated, and the scapula rotated as upwardly as possible to move the inferior angle away from the midline. The circles indicate the area of ultrasound examination for trigger point injection of the subscapularis compared with the square for the pectoralis muscle.

Application of Ultrasound-Guided Trigger Point Injection for Myofascial Trigger Points in the Subscapularis and Pectoralis Muscles to Post-Mastectomy Patients: A Pilot Study

Hyuk Jai Shin,¹ Ji Cheol Shin,² Wan Sung Kim,¹ Won Hyuk Chang,³ and Sang Chul Lee²

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²Department and Research Institute of Rehabilitation Medicine, Yonsei University College of Medicine, Seoul;

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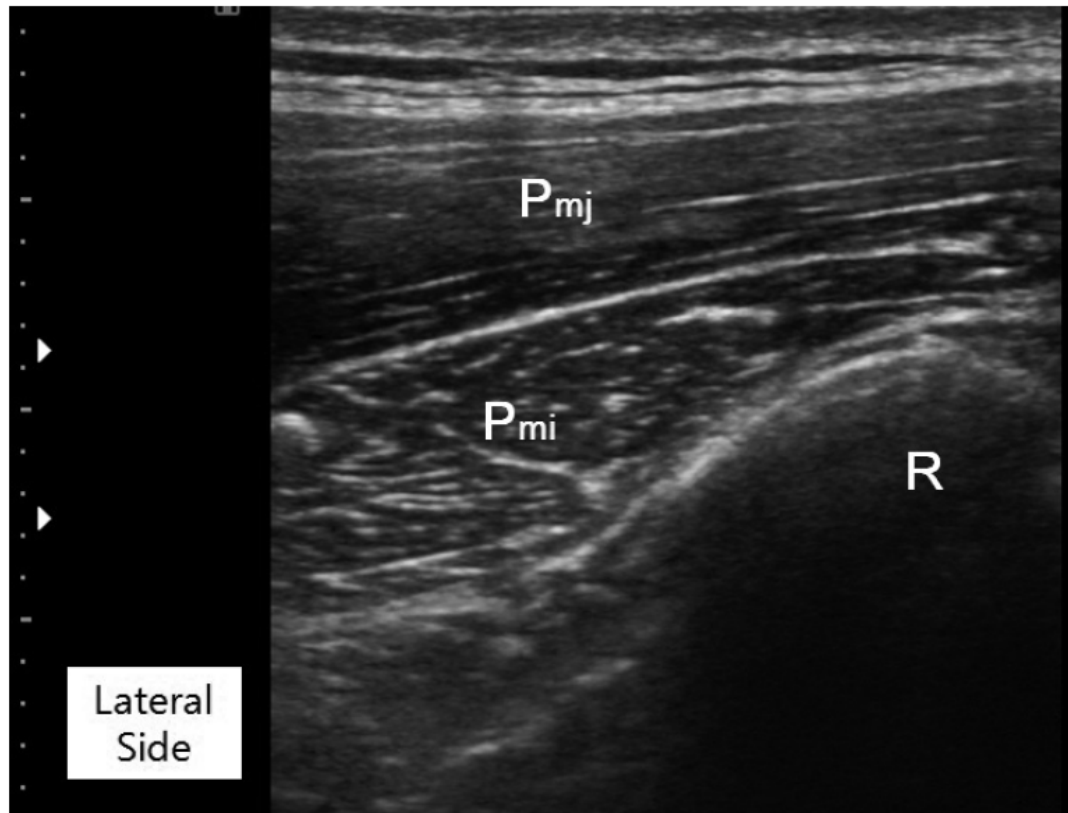


Fig. 2. A transverse plane ultrasound image of the pectoralis muscle. This superficial muscle was easily detected on ultrasound image. Pmj, pectoralis major; Pmi, pectoralis minor; R, rib.

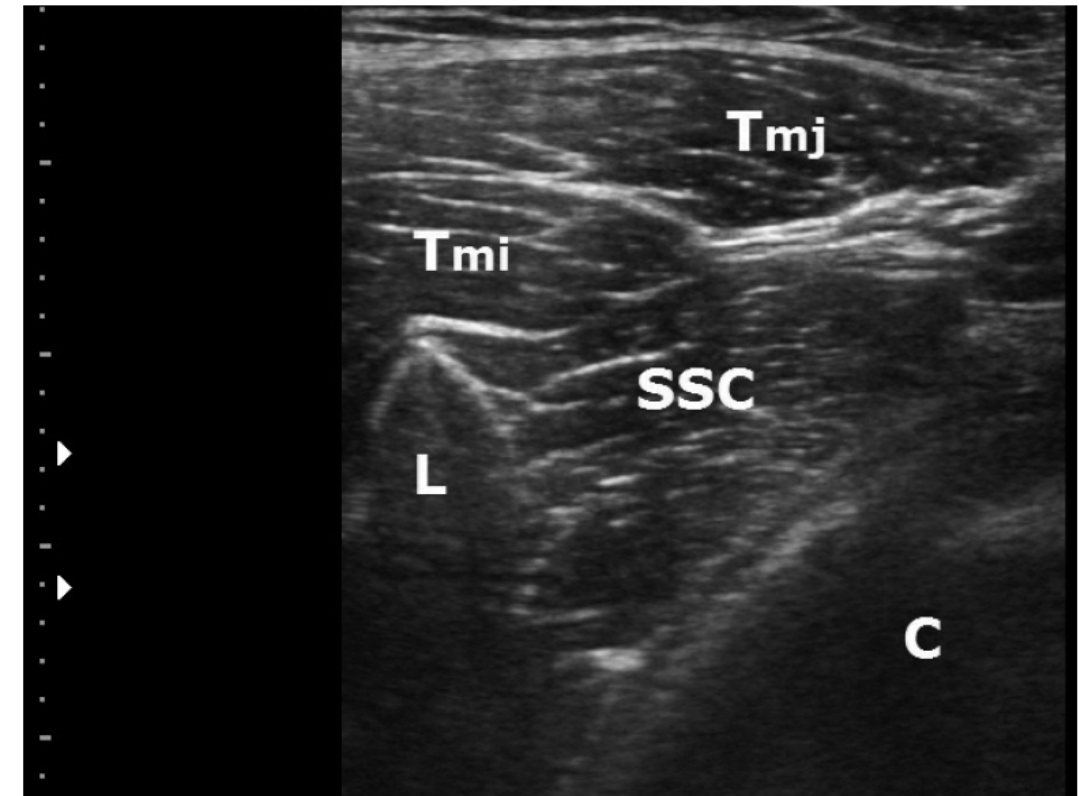


Fig. 3. A transverse plane ultrasound image of the subscapularis muscle. Before the trigger point injection, the lateral border of the scapula needs to be identified. C chest wall, L lateral border of the scapula. Tmj, Teres major; Tmi, Teres minor; SSC, subscapularis.

TPI FOR PRIMARY DYSMENORRHEA

- Huang and Liu (2014):
 - Abdominal muscle TPI with lidocaine 2 weeks prior to menses
 - Yielded improvement in primary dysmenorrhea.

Observation

Wet needling of myofascial trigger points in abdominal muscles for treatment of primary dysmenorrhoea

Qiang-Min Huang, Lin Liu

Huang Q-M, et al. *Acupunct Med* 2014;**32**:346–349. doi:10.1136/acupmed-2013-010509



Figure 1 A typical location pattern of trigger points for primary dysmenorrhoea in abdominal muscles from one patient: eight points on the rectus abdominus and four on the ventrolateral muscles.

OBJ: EVALUATE HOW TPI COMPARES WITH OTHER MODALITIES FOR THE TREATMENT OF MYOFASCIAL PAIN SYNDROME (MPS).



TPI VS DN

NAVARRO-SANTANA MJ, SANCHEZ-INFANTE J, GOMEZ-CHIGUANO GF, ET AL. DRY NEEDLING VERSUS TRIGGER POINT INJECTION FOR NECK PAIN SYMPTOMS ASSOCIATED WITH MYOFASCIAL TRIGGER POINTS: A SYSTEMATIC REVIEW AND META-ANALYSIS. *PAIN MEDICINE*. 2022;23(3):515-525.

- Comparison of TPI and DN for neck pain:
 - Six trials included
 - Pain intensity: $TPI > DN$.
 - Pain-related disability: $TPI = DN$
 - Pressure pain thresholds: $TPI = DN$
 - Cervical lateral-flexion: $TPI = DN$
 - Depression: $TPI = DN$

TPI VS DN

- Liu (2015):

- Review of wet vs dry needling for shoulder pain
- Wet needling a little better in medium term (9-28 days)
- Connelly lists other studies with similar results.

REVIEW ARTICLE (META-ANALYSIS)

Effectiveness of Dry Needling for Myofascial Trigger Points Associated With Neck and Shoulder Pain: A Systematic Review and Meta-Analysis



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ACUPUNCTURE FOR MPS

LI X, WANG R, XING X, ET AL. ACUPUNCTURE FOR MYOFASCIAL PAIN SYNDROME: A NETWORK META-ANALYSIS OF 33 RANDOMIZED CONTROLLED TRIALS. *PAIN PHYSICIAN*. 2017;20:E883-E902.

- Acupuncture is effective for decreasing pain compared to other methods.
- TPI with bupivacaine had a much higher risk of adverse events.
- Studies were unclear about treatment periods and other outcomes.

TPI VS BTXA

AHMED S, SUBRAMANIAM S, SIDHU K, ET AL. EFFECT OF LOCAL ANESTHETIC VERSUS BOTULINUM TOXIN-A INJECTIONS FOR MYOFASCIAL PAIN DISORDERS. *CLINICAL JOURNAL OF PAIN*. 2019;35(4):353-367.

- Meta-analysis; n = 33 studies.
- TPI > BTXA for pain intensity @ 16 weeks post-treatment
- Multiple injections sessions with local anesthetics were more effective than a single session.
- Local anesthetics inhibit afferent and efferent nerve signals within the MTrP region
- BTXA inhibits ACh activity which promotes relaxation.

MULTI-MODAL CARE

SEGURA-PÉREZ M, HERNANDEZ-CRIADO T, CALVO-LOBO C, ET AL. A MULTIMODAL APPROACH FOR MYOFASCIAL PAIN SYNDROME: A PROSPECTIVE STUDY. *JOURNAL OF MANIPULATIVE AND PHYSIOLOGICAL THERAPEUTICS*. 2017;40(6):397-403.

- Followed 150 patients with MPS in different regions
- Multimodal care included:
 - Dry needling
 - Spray and stretching
 - Kinesio-taping
 - Eccentric exercise
 - Patient education
- VAS scores ↓ 30 mm or more in 79% of cases.

META POINTS

- MPS is not just limited to NM or US – international, universal
- Young and old patients
- Multiple options, including LLLT (now available at UNM)
- Different needles and injectates (or not) OK
- Multi-modality approach probably better for more severe or chronic cases
- DDX: common co-travelers are OA, facet arthropathy, canal stenosis, neuropathy
- Many studies are in muscles we see commonly (trapezius, paraspinals, gluteals, gastrocnemius)

PRACTICE NOTES

- TPI $\approx$ DN in efficacy
 - Hybrid: “dry needling plus a drop”
- LTR vs. involuntary response (jump sign, gasp, grunt)
- Further training in TPI:
 - The UNM Myofascial Institute
 - TPI course November 3-4, 2022
 - <https://hsc.unm.edu/medicine/education/cpl/learn/cme-conferences-index.html>



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LAST SLIDE!

- NOW you can:
 - Identify patient candidates for trigger point injections (TPI).
 - Describe the mechanism of action of TPI.
 - Compare different injectate options and needling techniques.
 - Evaluate how TPI compares with other modalities for the treatment of Myofascial Pain Syndrome (MPS).
- **THANK YOU**

