Spokane Pain Conference 10/27/17

Neuropathic-Myofascial Pain Syndromes & Intramuscular Stimulation Trigger Point Dry Needling

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Board Certified PM&R, Electrodiagnostic Medicine

2014 University of British Columbia Chan Gunn Lecturer

2015 Physical Therapy Association of Washington ‘Friend of PT Award’
COURSE OBJECTIVES:

1) Understand Types 1-3 Pain and Recognize the High Prevalence of Neuropathic-Myofascial Pain Syndromes: NMPS

2) Recognize the Physical Exam Findings of NMPS

3) Understand the Proper Treatment of NMPS
Disclaimer: No Financial Relationships or Affiliation to Disclose
NEUROPATHIC- MYOFASCIAL PAIN

Non-Articular Musculoskeletal Pain Identified by Motor, Sensory & Autonomic Findings including the presence of ‘Trigger Points’, Myotomally Localized Tender & Shortened Muscle Bands (‘Taut Bands’) that can often be located by palpation and that produce local and/or referred pain, parasthesias, restricted ROM and/or Autonomic Disturbance
"I'm afraid it's your body, Mr. Haskins."
C.C. Gunn, M.D. and S. Goodman, M.D.

1996

2017 2nd ed.

2014 UBC Chan Gunn Lecturer
International Association for the Study of Pain

“Pain is an unpleasant sensory & emotional experience associated with actual or potential tissue damage, or described by the patient in terms of such damage.”
Pathophysiological - Temporal

Pain Model

PAIN = THREE Sub-Types:

- **TYPE 1: NOCICEPTION**
  = ‘Noxious’: Neurophysiologic Specialized Peripheral Receptor/Nerve

- **TYPE 2: INFLAMMATION**
  = ‘CHEMICAL’
  Acute Tissue Damage: Trauma, Infection or Auto-Immune

- **TYPE 3: NEUROPATHIC**
  = Supersensitivity Dysfunction
TEMPORAL PROFILE

• TYPE 1: Nociception
  IMMEDIATE!!!

• TYPE 2: Inflammation
  Acute!

• TYPE 3: Neuropathic
  Chronic!!!!!!!
Descartes Describes Type 1 Nociceptive ‘Pain Pathway’ in 17th Century

Figure 25. Descartes’ (1664) concept of the pain pathway. He writes: “If for example fire (A) comes near the foot (B), the minute particles of this fire, which is you know move with great velocity, have the power to set in motion the spot of the skin of the foot which they touch, and by this means pulling upon the delicate thread (cc) which is attached to the spot of the skin, they open up of the same instant the pore (de) against which the delicate thread ends, just as by pulling at one end of a rope one makes to strike at the same instant a bell which hangs at the other end.”
### Type 1 PAIN = A-Delta Fibre

<table>
<thead>
<tr>
<th>Fiber Type</th>
<th>Function</th>
<th>Fiber Diameter (µm)</th>
<th>Conduction Velocity (m/s)</th>
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<td>A α</td>
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<td>12-20</td>
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#### Unmyelinated

<table>
<thead>
<tr>
<th>C Dorsal root</th>
<th>Pain; Reflex responses</th>
<th>0.4-1.2</th>
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</table>
Harry The Homunculus

(a) Somatosensory cortex in right cerebral hemisphere

(b) Motor cortex in right cerebral hemisphere
‘Type 1’ Pain: ‘Experimental Pain’ or ‘Monkey Business’ A-Delta Nerve Fiber
# CHARACTERISTICS of TYPES 1-3 PAIN

<table>
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<th>1 NOCI</th>
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<th>3 NEUROPATHIC</th>
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<td>Visceral/Referred Pattern</td>
<td>Mixed</td>
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<td>Chronic Unremitting</td>
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</tr>
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<tr>
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<td>Concern</td>
<td>Depression</td>
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<tr>
<td>Response</td>
<td>Care, Anxiety</td>
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‘Type 2’ Pain: Cellular Damage
Trauma or Immune Mediated
‘Chemical’ Inflammation:
DOLOR RUBOR CALOR TUMOR
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Figure 1 Different nociceptors detect different types of pain. a. Peripheral nerves include small-diameter (Aδ) and medium- to large-diameter (Aα, β) myelinated afferent fibres, as well as small-diameter unmyelinated afferent fibres (C). b. The fact that conduction velocity is directly related to fibre diameter is highlighted in the compound action potential recording from a peripheral nerve. Most nociceptors are either Aδ or C fibres, and their different conduction velocities (6–25 and ~1.0 m s⁻¹, respectively) account for the first (fast) and second (slow) pain responses to injury. Panel b adapted from ref. 75.
Total Body TYPE 2 PAIN
The Dinosaur Has ‘Tough Skin’

No A-Delta Fibres (Only Mammals Have)

Must Create Tissue Damage To Cause PAIN
## CHARACTERISTICS of TYPES 1-3 PAIN

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Cells Involved in Wound Healing

- Platelets
- Neutrophils
- Macrophages
- Lymphocytes
- Fibroblasts
- Capillaries

Days
Rate of Recovery After Injury

Disability (%)

Time (weeks)

0 4 8 12 16

0 25 50 75 100

34% 15% 9%

Rate of Recovery After Injury

Disability (%)

Time (weeks)
• TYPE 1: ‘NOCICEPTION’ = NOXIOUS Peripheral Receptor/Nerve

• TYPE 2: INFLAMMATION = ‘CHEMICAL’
  Acute Tissue Damage, Infection or Auto-Immune

• TYPE 3: NEUROPATHIC = Supersensitivity Dysfunction
International Association for the Study of Pain

Definition of Neuropathic Pain:

"Pain arising as a direct consequence of a lesion or disease affecting the somatosensory system"
‘Neuro’-pathy = Nerve - disease

Nerves ‘Gone Wild’ (erratically):

- **Ectopic d/c**: nerve and muscle fibers become receptive to chemical transmitters along length
- Ephaptic transmission = ‘CROSS TALK’ NON-SYNAPTIC NERVE TRANSMISSION
- Neural sprouting in both afferent & efferent fibers
- ‘Short circuits’: sensory/motor/autonomic
<table>
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<th>Central effects</th>
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<td>- Ectopic and spontaneous discharge</td>
<td>• Central sensitization</td>
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<tr>
<td>- Ephaptic conduction</td>
<td>• Spinal reorganization</td>
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<tr>
<td>- Alterations in ion channel expression</td>
<td>• Cortical reorganization</td>
</tr>
<tr>
<td>- Collateral sprouting of primary afferent neurones</td>
<td>• Charges in inhibitory pathways</td>
</tr>
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<td>- Sprouting of sympathetic neurones into the DRG</td>
<td></td>
</tr>
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<td>- Nociceptor sensitization</td>
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(A) Hyperalgesia
- An increased response to a normally painful stimulus

(B) Allodynia
- A painful response to a normally innocuous stimulus
Cannon and Rosenblueth’s ‘Law of Denervation Supersensitivity’

“When in a series of efferent neurons a unit is destroyed, an increased irritability to chemical agents develops in the isolated structure or structures, the effect being maximal in the part directly denervated.”
(1871-1945) was an American disciple of Sherrington, and Higginson Professor & Chairman of the Department of Physiology @ Harvard 1906-1945.

- ‘Fight-or-Flight’ Response
- ‘Homeostasis’ (Claude Bernard)

ALSO

- ‘Law of Denervation Supersensitivity’
ALL STRUCTURES RESPOND TO MOTOR DENERVATION by DEVELOPING ‘SUPERSENSITIVITY’

- Skeletal Muscle
- Smooth Muscle
- Spinal Neurons
- Sympathetic Ganglia
- Sweat Glands
- Adrenal Glands
- Brain Cells
Neuropathic Response

Four Types of Supersensitivity described by Cannon

- **Superduration of response:**
  amplitude of response unchanged but duration prolonged

- **Hyperexcitability of stimulus:**
  lower threshold of stimulus

- **Increased Susceptibility of stimulus:**
  decreased stimulus = response of normal amplitude

- **Superreactivity of tissue**
  augmented response of tissue
Types of increased Sensitivity according to the law of denervation leading to hypersensitivity to environmental responses (molds, terpenes, bacteria, viruses, neurotransmitters, foods, toxic and nontoxic chemicals). (FHC-Dallas.)
Motor Point on skin over zone of innervation

Acetylcholine acts only at receptors within a narrow zone of innervation

Normal subcutaneous tissue
Distribution zones of motor end plates (black lines) traced by positivity of acetylcholinesterase in human lower limb muscles.

Neuropathic = Sick Nerve

In neuropathy, Acetylcholine can act at extra-junctional “hotspots” that are present throughout the muscle.

1 mmol vs. 1000 mmol

Neuropathic = Sick Nerve
Acetylcholine Sensitivity Along Muscle Membrane After Denervation

Cannon and Rosenblueth investigated complete denervation

Intact motor/efferent nerve provides not only a stimulatory but an inhibitory/stabilizing AND nourishing effect, or ‘trophic factor’

‘Trophic’ effect on end-organ so that

DE-NERVATION $\rightarrow$ A-TROPHY

NOW KNOWN: Any measure which blocks the flow of motor impulses and deprives the effector organ of excitatory input for a period of time can cause

Neuropathic DYS-FXN $\rightarrow$ DYS-TROPHY

Altered electrochemical state: SUPERSENSITIVITY
Various Degrees of Compression to Motor Neurons

CANNON:
DENERVATION > ‘A’-TROPHY

‘PARTIAL DENERVATION’, or
NEUROPATHY > ‘DYS’-TROPHY

Various conditions in motor neurons as a result of progressive compression
Neuropathic Supersensitivity > PAIN
Home Security Alarm Triggers on Sunrise and Cat ‘Burglars’
Type 1 Pain = Nociception (Extrinsic)

Type 2 Pain = Algogenic (Intrinsic)

Type 3 Pain = Super-Sensitivity (Intrinsic)
Table 1 Traditional aetiological classification of Neuropathic Pain

An estimate of the prevalence, in the USA (population 270 million) is given in brackets after each example cited

Trauma: phantom limb (50), spinal cord injury (120).
Ischaemic injury: central pain (30), painful diabetic neuropathy (600).
Infection/inflammation: post-herpetic neuralgia (500), HIV (15).
Cancer: invasion/compression of neural structures (200).
Drugs: vinca alkaloids.
Compression: sciatica (2100), trigeminal neuralgia (15).
Unknown: trigeminal neuralgia, MS (51).

Mechanisms of neuropathic pain
D. Bridges¹,², S. W. N. Thompson³ and A. S. C. Rice¹ ¹Pain Research, Department of Anaesthetics, Imperial College School of Medicine, Chelsea and Westminster Hospital Campus, London W2 1NY, UK
Most Common Cause of Nerve Injury

Spondylosis = Spinal Degeneration

- Nerve Root Vulnerable to Mechanical Trauma = Radiculo-pathy, or Radiculo-Neuropathy
- I.V.F. Narrow & Congested
- S.N.R. lacks Epineurium and Perineurium
- Tethered by Dura Mater
- Tethered by Various Ligaments

Spondylosis = Spinal Degeneration
Axial View of Disc & Spinal Nerve Root

- Autonomic Nerve Branch
- Anterior Branch
- Nerve Root & Branches
- Facet Joint Bone
- DISC
- Spinal Cord
Spinal Nerve Roots Lack Protection of Perineurium and Epineurium of Spinal/Peripheral Nerve

Bogduk, N. Clinical Anatomy of the Lumbar Spine and Sacrum, 3rd ed, p.128
Dura Mater Tethers Nerve Root

Diagram to show the effect of a central disc prolapse.

The reader must imagine that he is looking into the body along a line from the left anterior or posterior margin, emerging through the posterior disc space on the other side. The left roots are seen.

Sub arachnoid

Dura mater

Diagram to show the effect of a lateral disc prolapse.

A central disc at L4-L5 is capable of damaging all roots below L5. In fact, the most sensitive roots (L5-S2) are the most vulnerable.

A lateral disc at L4 will predominantly affect L5 root. Note the displaced root location. But this disc may also affect the S1 root. This usually occurs in the ankle jerk often being abolished with disc lesions at L4-L5.
Lateral Root & Other Ligaments

- Spinal Nerve Root
- Spinal Cord
- Lateral Ligament
Figure 18-9. Transforaminal ligaments. The various transforaminal ligaments are shown. It is possible for these structures to limit the spinal nerve to a small region of the intervertebral foramen. (A) Superior and inferior corono-transverse ligaments. (B) Superior transforaminal ligament. (C) Middle transforaminal ligament. (D) Inferior transforaminal ligament. (From Bogduk N, Twomey LT: Clinical Anatomy of the Lumbar Spine, 2nd ed. Melbourne, Churchill Livingstone, 1991, with permission.)
Figure 18-7. Intertransverse ligament. Ventral (V) and dorsal (D) leaves of the intertransverse ligaments are depicted. VR: ventral ramus of spinal nerve; MB: medial branch of dorsal ramus. (From Bogduk N, Twomey LT: Clinical Anatomy of the Lumbar Spine, 2nd ed. Melbourne, Churchill Livingstone, 1991, with permission.)
The Segmental Dorsal Ramus as a Common Cause of Chronic & Recurrent LBP.


Medial Branch of Posterior Ramus Supplies Multifidi Muscles & Passes Through Mamillo-Accessory Ligament

Dumitru, D. Electrodiagnostic Medicine, 2nd ed. Hanley & Belfus, 2002, p.719
Electrochemical ‘Highway’

- Cytoplasm contains proteins, enzymes, neurotransmitters, charged +/- ions

- Axoplasmic flow stream of cytoplasm, motor proteins, charged ions

- Electrical Properties: Resting membrane potential, action potential, DC, semicond currents

- Trophic factor: combination of axoplasmic flow and electro-chemical stimulus
  
  TROPH = Nourish
Spondylosis, or Spinal ‘Wear & Tear’ >>>>>

RADICULOPATHY & RADICULO-(NEURO)PATHY

- Nerve &/or vascular compression, angulation, torsion and traction can impede axoplasmic flow and alter electrical properties
- Spinal Nerve Roots >> Susceptible >> Peripheral nerve

10mm Hg pressure x 10-15 minutes > 50% in Compound Nerve Action Potential
1975 Sharpless

15% stretch > conduction block
1984 Rydevik

9 degrees axial rotation produces myelographic filling defect at L5 w/2x 20% physiological stretch
1983 Farfan

- Spondylosis > ‘Injury Pool’ of Sick Nerves
‘Susceptibility of Spinal Roots to Compression Block’

1975 Sharpless

10mm Hg pressure x 10-15 minutes > 50% in Compound Nerve Action Potential

CONCLUSIONS

1. Dorsal roots are far more susceptible to compression block than peripheral (sciatic) nerve. When pressure is applied for 3 minutes followed by 3-minute recovery periods, 100 mm. Hg. must be applied to sciatic nerve to achieve the same conduction block that can be produced in spinal roots by 20 mm. Hg.

2. As little as 10 mm. Hg. pressure, maintained for 15–30 minutes, reduces the compound action potentials of dorsal roots to about half of their initial values. With such small pressures, nearly complete recovery occurs in about 30 minutes.

It is difficult to appreciate the significance of the minute pressures capable of affecting root conduction. It seems doubtful that the most skillful and deft surgeon could touch a spinal root or the balloon of our compression apparatus with his gloved forefinger without producing a pressure increment of at least 5 mm. Hg. One may well consider what happens to the spinal roots when they are manipulated by the far less dextrous electrophysiologist.
>15% stretch =
- intraneural blood flow blocked
- electrical conduction block

Rydevik SPINE 1984

Fig 5. Diagram that illustrates block of axonal transport induced by compression. The rabbit vagus nerve was compressed by means of a small inflatable cuff. The cuff was applied around the nerve, which thereby was compressed at controlled pressure. Radioactively labelled amino acid (3H-leucine) was injected into the nodose ganglion of the nerve. These amino acids then are incorporated into the proteins, which are synthesized in the ganglion, and then transported down the axon at a speed of about 400 mm/24 hours. The nerve is shown schematically beneath the nodose ganglion.

ganglia. Their axons may be more than 100 cm in length. This is a remarkable distance in view of the size of the nerve cell body, which is around 100 μm. If we transfer these dimensions to a larger scale, a nerve cell body of 100 cm in diameter would have an axon with a diameter of about 10 cm and with a length in the range of 10 km.
(ONLY!!!) 9 degrees axial rotation produces myelographic filling defect at L5 w/2x normal 20% physiological stretch on nerve root

1983 Farfan
Force Absorbed

\[ K.E. = \frac{1}{2} mv^2 \]

- 20 mph = 4 x 10 mph
- 30 mph = 9 x 10 mph
- 40 mph = 16 x 10 mph
- 50 mph = 25 x 10 mph
Various Degrees of Compression to Motor Neurons

CANNON:
DENERVATION > ‘A’-TROPHY

‘PARTIAL DENERVATION’, or
NEUROPATHY > ‘DYS’-TROPHY
SPONDYLOSIS:

‘Pool of Injured Nerves’ That Accumulate Over Time

-2 meters

100%

No Symptoms

Symptoms

100% HEALTH

Time

-2 meters
Large Diameter Nerve Fibers More Susceptible to Compression > Small Diameter Fibers
# Large Myelinated Fibers

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In neuropathy, Acetylcholine can act at extra-junctional “hotspots” that are present throughout the muscle.
Muscle CONTRACTURE vs. Muscle CONTRACTION

ACh slowly depolarizes muscle membrane, and this induces electromechanical coupling, with the consequent SLOW development of TENSION WITHOUT action potentials = ‘Silent’ electrically

CONTRACT-URE is the evoked shortening of a muscle fiber in the ABSENCE of action potentials

Local Nature of Ach-Induced Contracture

Leads to Focal Taut Bands with MUSCLE TENSION
Normal Muscle & Tendon

Muscle Contracture

- Taut Bands
  Spasm, Decreased ROM
- Altered Biochemistry > Myalgic Hyperalgesia
- Referred Pain
- Tension on Tendons = Tendonosis, -opathy

Neuropathic Muscle = Shortened Muscle
The Trigger Point

Baldry, PE. Myofascial Pain and Fibromyalgia – A Clinical Guide to Diagnosis and Management. Churchill Livingstone
Identification & Quantification of Myofascial Taut Bands with Magnetic Resonance Elastography

Archives of Physical Medicine & Rehabilitation

Morphological findings of representative skeletal muscles with nontaut and taut bands. (a) Biceps femoris with a nontaut band; (b) Biceps femoris with a taut band (H&E staining, scale bar = 5 μm)

Hsieh YL, et. al. Dry needling at myofascial trigger spots of rabbit skeletal muscles modulates the biochemicals associated with pain, inflammation, and hypoxia. Evid Based Complement Alternat Med. 2012
Relationship of Tension to Muscle Length.

**The Sarcomere**

- At this length there is maximum overlap of myofilaments producing maximum number of crossbridges and maximum amount of tension.

**Whole Muscle**

- This applies to the entire muscle as well as to individual sarcomeres.

Graph: 

- Normal sarcomere length
- Tension and percent of resting length
- Muscle length and tension
- Optimum length for maximum tension
Secondary Effects of Short Muscle Syndrome

Increased traction causes "TENDONOSIS"

Increased traction in tendon sheath causes "Tenosynovosis"

Increased pressure of kneecap on the femur causes wear & tear’ or Patella-Femoral Chondromalacia
Arthralgia & Osteoarthritis

Shortened Muscle

Wolf’s Law:
- Tension
- Bone = ‘Spurs’
Paraspinal Muscle Shortening

Shortened muscles between vertebrae increase pressure on disc, nerve and facet joints
Spondylosis > Neuropathy > Muscle Shortening > Tendinosis Syndromes

- Spinal Wear & Tear ➔ Radiculo-Neuropathy
- Neuropathy ➔ Muscle Shortening
- Shortening ➔ ↑ Tension on Tendon = Tendinosis

1. Degeneration (spondylosis) in the neck causes neuropathy
2. Neuropathy causes spasm and shortening of the wrist extensors
3. Constant pull of wrist extensors on lateral epicondyle causes Tennis Elbow, Tenosynovitis
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<th>SYNDROME</th>
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<td>Pseudo’ Sciatica, Gluteal/Ischial Bursitis, Pyriformis Syndrome</td>
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<tr>
<td>Supra - Infraspinatus, Teres Major, Subscap</td>
<td>Rotator Cuff Tendonosis, Impingement</td>
</tr>
<tr>
<td>Extensor Carpi Radialis Longus and Brevis</td>
<td>Lateral Epicondylosis Tennis Elbow</td>
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<tr>
<td>Quadriceps, Adductors</td>
<td>Patella-Chondromalacia Runner’s Knee, Medial Knee Pain</td>
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<tr>
<td>Gastroc-soleus</td>
<td>Achilles Tendonosis</td>
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<tr>
<td>Neck &amp; Back Muscles</td>
<td>Cervicalgia, Lumbar Pain Facet Syndrome</td>
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<tr>
<td>Intrinsic Foot</td>
<td>Plantar Fasciitis, Heel Pain</td>
</tr>
<tr>
<td>TFL, Iliotibial Band</td>
<td>IT-Band, Knee Pain</td>
</tr>
</tbody>
</table>
Tenodopathy NOT Tendonitis


Myofascial Pain is a Type of Neuropathic Pain

‘Evidence of Neuroaxonal Degeneration in MFPS’
Chang, CW. 2008, Europ Jurnal of Pain

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Subject numbers</th>
<th>MCD (µs)</th>
<th>HNL MCD (µs)</th>
<th>Abnormal MCD (%)</th>
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<tbody>
<tr>
<td>Normal controls</td>
<td>16</td>
<td></td>
<td></td>
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<tr>
<td>Trapezius</td>
<td>8</td>
<td>32.9 ± 7.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>47.1</td>
<td>0</td>
</tr>
<tr>
<td>Levator scapulae</td>
<td>8</td>
<td>36.2 ± 6.4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>48.0</td>
<td>0</td>
</tr>
<tr>
<td>MPS patients</td>
<td>23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trapezius</td>
<td>15</td>
<td>61.5 ± 11.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>74.3</td>
<td>70.7</td>
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<tr>
<td>Levator scapulae</td>
<td>8</td>
<td>59.2 ± 10.9&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD.
HNL: highest normal limit.
For comparison of values with the same letter: <sup>a,b</sup> p < 0.01 by Student’s t-tests.
Electrophysiologic Evidence of Spinal Accessory Neuropathy in Patients With Cervical Myofascial Pain Syndrome


Fig 2. Relation between CMAP amplitude and disease duration in patients with cervical MFPS.
Radiculopathy: 3 Divisions of Nerve Root

- Motor nerve: c/o stiffness; muscle fiber contracture/taut bands, spasm, decreased joint ROM; tendinosis syndromes

- Sensory nerve: c/o parasthesias; allodynia, myalgic hyperalgesia = tender points

- Autonomic nerve: smooth muscle contracture > neurogenic or trophic edema
  - vasonstriction > cool to touch
  - sudomotor > hyperhidrosis
  - pilomotor > gooseflesh, hair loss
Neuroanatomy explains association of MFPS and Autonomically mediated symptoms
Fig. 21-2. Different parts of the autonomic nervous system. Parasympathetic fibers are shown in solid blue, sympathetic parasympathetic fibers in red, and sympathetic fibers in turquoise blue. Proximal parasympathetic fibers are shown in solid red, posterior parasympathetic fibers in internuncial red.
Autonomic Neuropathy > Neurogenic Edema: Peau d’Orange’ Effect

- Smooth muscle cells (vascular & lymphatic) Supersensitive to Ach → Contracture

  opening of cell gap

  leakage = ‘Trophic’ Edema
Jay P. Shah, MD National Institutes of Health

This technique recovered extremely small quantities (0.5L) of very small substances (molecular weight, 100kd) directly from soft tissue.

There were significant differences in the levels of pH, substance P, CGRP, bradykinin, norepinephrine, TNF, and IL-1 in those subjects with an active MTrP (symptoms, MTrP present) compared with subjects with a latent MTrP (no symptoms, MTrP present) and normal subjects (no symptoms, no MTrP).
Neuropathy → Dystrophic Muscle Histopathology

- Golgowsky and Wallraff: waxy degen., agglom. nucleoli, fatty infiltration
- Miehlke et al: Groups 1-4 Symp./Findings #3,4 ~ dystrophic nuclear changes, esp. near blood vessels, fibre degen., fat/conn. tissue
- Fassbender: swollen mitochondria, moth-eaten filaments, necrosis, dissolution of elements, inc. mucopolysaccharides = ischemia or EtOH
NEUROPATHIC- MYOFASCIAL PAIN

Non-Articular Musculoskeletal Pain Identified by Motor, Sensory & Autonomic Findings including the presence of ‘Trigger Points’, Myotomally Localized Tender & Shortened Muscle Bands (‘Taut Bands’) that can often be located by palpation and that produce local and/or referred pain, parasthesias, restricted ROM and/or Autonomic Disturbance
DIAGNOSIS of NEUROPATHIC-MYOFASCIAL PAIN SYNDROME

“Your test results were negative – get lost!”

“When all else fails, examine the patient.”

Dean Naughton
ENIGMATIC

Myotomal NOT = Dermatomal

• Referred Pain Experienced Remotely From Source & Partially

• Multiple Trigger Point Can Refer to Same Location

• Travell & Simons Myofascial Trigger Points

Gluteus minimus
Autonomic Neuropathy
Exaggerated Pilomotor Reflex
Bilateral C4-C5 Dermatomal Hair Loss
L2-L3 Dermatomal Hair Loss
Right L2-L3 Dermatomal Hair Loss
Dermatomal Hairloss Secondary to Bilateral L5 Radicuло-Neuropathy
Dermatomal Hairloss Secondary to S1 Radiculolo-Neuropathy
Muscle Contracture with Shortening > Postural deviation
MUSCLE SHORTENING  ➡  DECREASED RANGE of MOTION
Neuropathy \[\rightarrow\] Vasoconstriction: Cool to Touch Segmentally
LYMPHATIC & VASCULAR SMOOTH MUSCLE CONTRACTURE → TROPHIC EDEMA
Trophic Edema in the Lower Inner Leg
Cervical Edema + DDD = ‘Turkey Neck’
The Trigger Point

Baldry, PE. Myofascial Pain and Fibromyalgia – A Clinical Guide to Diagnosis and Management. Churchill Livingstone
Are MRI-defined fat infiltrations in the multifidus muscles associated with low back pain? (YES)
L5-S1 Degenerative Disc Narrowing Correlates w/ Level of Multifidi Dystrophy
EMG ‘Positive Sharp Waves’ or Fibrillations’ = ACUTE RADICULOPATHY

Neuropathic Myofascial Pain: Chronic, so

‘Nml EMG’

NMPS: Findings of Chronic Radiculopathy: Increased Insertional Activity, Polyphasic MUAPS but difficult to quantitate
SFEMG not practiced in community
Myofascial Trigger Point Pain is Common Epidemiologically

• Myofascial Pain in Childhood 85 cases/23 MFPS due to illness or injury Age 1-18, Majority < 10 years Rx w/ethyl chloride spray and stretch: COMPLETE RELIEF 69/85 (81%) + INCOMPLETE RELIEF 9/85 (92%) INDEFINITE/NO RELIEF 7/85 BATES and GRUNDWALDT 1958

• Asymptomatic ‘Latent’ Trigger Points – found in 54% females 45% males AGE 17-35 Median/Mean19/19.5 SOLA 1955
Myofascial Trigger Point Pain is Common

- 55% of 164 patients referred to a dental clinic for chronic head and neck pain were found to have active myofascial trigger points as the cause of their pain

- Trigger points were the primary source of pain in 74% of 96 patients with musculoskeletal pain seen by a neurologist in a community pain medical center

Prevalence of Myofascial Pain in General Internal Medicine Practice.

- 54/172 (>30%) Patients @ Primary Care: PAIN
- 16/54 (30%)=Clinical Criteria for MFPS
- @10% OF ALL PATIENTS HAVE MFPS!
- Intensity by VAS HIGH = or > ALL other PAIN

- Physicians RARELY Dx, Yet Rx Provides Substantial & Abrupt RELIEF

Myofascial Pain Common and Commonly Overlooked, Undertreated, Severe yet Rxable!!!
Myofascial Pain Findings Increase w/ Age

2006 JAGS 54:11–20
Chronic Low Back Pain in Older Adults:
Prevalence, Reliability, and Validity of Physical Examination Findings
Weiner, D. et. Al.

Incidence of Findings: Back Pain vs. No Back Pain

<table>
<thead>
<tr>
<th>Condition</th>
<th>Incidence with Back Pain (%)</th>
<th>Incidence without Back Pain (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scoliosis</td>
<td>77.5%</td>
<td>60%</td>
</tr>
<tr>
<td>Myofascial Pain Syn</td>
<td>96%</td>
<td>10%</td>
</tr>
<tr>
<td>Sacroiliac</td>
<td>84%</td>
<td>5%</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>19%</td>
<td>0%</td>
</tr>
<tr>
<td>Hip Pain</td>
<td>48%</td>
<td>0%</td>
</tr>
<tr>
<td><strong>TABLE 7-1.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Epidemiology of Trigger Points</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher in women than men</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Most common in 30- to 50-year age range</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Most commonly found in the following muscles: trapezius, levator scapulae, axial postural muscles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic pain clinics study reported incidence of 85% of patients having myofascial pain syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic shoulder girdle trigger points are found in 54% of females and 45% of males</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Overtreating Chronic Back Pain: Time to Back Off?
“I really look forward to your cheery little visits.”
STIMULATION to Modulate & Normalize Neurophysiologic Function is Both Natural: Ex: Reflexively Rubbing an Injured Area of Body AND, Commonly Used in Medicine
‘Gate Theory’ Describes Role of Large Fiber Modulation of Small Fibre Generated Pain Melzack & Wall 1965

Prolonged Relief of Pain by Brief, Intense Transcutaneous Somatic Stimulation Melzack 1975

T.E.N.S.
ELECTRO-ANALGESIA

Battery Powered Electric Nerve Stimulator 2008

TX-3 TENS and EMS

Full featured, high quality, dual channel, constant current TENS. Features 3 frequency settings: (C) continuous adjustable frequency from 2 to 160 Hz, (M) pulse width modulation, and (B) burst mode. Includes (a) 2 sets of electrodes with wires (2.5 mm jack), hard carrying case, belt clip, 9 volt battery, and instructions.

FREMS = Frequency Modulated TENS
2008 J Neurol. Mar 14
Deep brain stimulation for dystonia: outcome at long-term follow-up. Loher, TJ, et.al

CONCLUSION: DBS maintains marked long-term symptomatic and functional improvement in the majority of patients with dystonia.

Vagus nerve stimulation for epilepsy: a meta-analysis of efficacy and predictors of response

VNS is an effective and relatively safe adjunctive therapy in patients with medically refractory epilepsy not amenable to resection.
Spinal cord stimulation for ischemic heart disease and peripheral vascular disease.

Ischemic disease (ID) is now an important indication for electrical neuromodulation (NM), particularly in chronic pain conditions. NM is defined as a therapeutic modality that aims to restore functions of the nervous system or modulate neural structures involved in the dysfunction of organ systems. One of the NM methods used is chronic electrical stimulation of the spinal cord (spinal cord stimulation: SCS). SCS in ID, as applied to ischemic heart disease (IHD) and peripheral vascular disease (PVD), started in Europe in the 1970s and 1980s, respectively.

Sacral neuromodulation in lower urinary tract dysfunction

So-called idiopathic bladder overactivity still the major indication for this technique. Patients not likely to benefit from the procedure were those with complete or almost complete spinal lesions, but incomplete spinal lesions seemed to be a potential indication. This technique is now also indicated in the case of idiopathic chronic retention and chronic pelvic pain syndrome. When selection is performed, more than three-quarters of the patients showed a clinically significant response with 50% or more reduction in the frequency of incontinent episodes,
Percutaneous Tibial Nerve Stimulation: A Clinically and Cost Effective Addition to the Overactive Bladder Algorithm of Care


Percutaneous Tibial Nerve STIMULATION for the Long-Term Treatment of Overactive Bladder: 3-Year Results of the STEP Study
Peters, KM. Jrnl of Urology, June 2013

CONCLUSION: Most STEP participants with an initial positive response to 12 weekly percutaneous tibial nerve stimulation treatments safely sustained over active bladder symptom improvement to three years with an average of one treatment per month
Scribonius Largus 45 A.D.
Headache Rx: Electric Eel
ALL forms of ‘Counter–Irritation Reflex Stimulation’ operate through afferent stimulation of specialized receptors

- Massage, acupressure, MFR, Kinesiotape tactile & pressure receptors

- Exercise, traction, manual Rx, Electrical-stimulation TENS tactile, pressure, muscle spindles & Golgi tendon organs

- Diathermy, cold, ultrasound, infrared, lasers thermal receptors, photobiomodulation

- Dry Needling, Intramuscular Stimulation muscle spindle hyperstimulation, spinal reflexes, ‘current of injury’
Touch = Low Tech Afferent Stimulation

MANUAL SOFT TISSUE THERAPY: Trigger Point Massage, Pin & Stretch, Myofascial Release, Acupressure, Shiatsu Strain-Counterstrain, Rolfing
Changes in Blood Flow and Cellular Metabolism at a Myofascial Trigger Point With Trigger Point Release (Ischemic Compression):
A Proof-of-Principle Pilot Study
Albert F. Moraska, et. al.
Archives of Physical Medicine and Rehabilitation 2013;94:196-200

Responsiveness of Myofascial Trigger Points to Single and Multiple Trigger Point Release Massages:
A Randomized, Placebo Controlled Trial.
Moraska AF¹, Schmiege SJ, Mann JD, Butryn N, Krutsch JP.

Effect of ischemic compression for cervicogenic headache and elastic behavior of active trigger point in the sternocleidomastoid muscle using ultrasound imaging.
Independent Therapeutic Aides
Clinical Research on Dry Needling for MFPS

LEWITT: ‘The Needle Effect (NE) in Relief of Myofascial ’ Pain

PAIN 1979  NE: Immediate (Hyperstimulation) Analgesia Without Hypasthesia

241 Pts./312 Painful Structures = 86.8% Immediate Analgesia

288/312 sites: 92 ‘Permanent’, 58 Several Mos 63 Weeks, 32 Days, 43 NO Relief

75/244 pts. most effective c/w manipulation, traction, exercise
Clinical Research on T.P.I. vs. DN

- Prospective, randomiized, dbl-blind eval of t.p.i. therapy for LBP
  Garvey TA: SPINE 1989
  Injectate NOT critical: dry needling = ly effective

- Dbl-blind controlled study of different myofascial injection techniques
  Jaeger B: PAIN 1987
  Reduction t.p. tenderness dependent only on needle
  Reduction in referred sxs greater with solution but indep. of kind
Gunn: ‘Dry Needling of Muscle Motor Points for Chronic LBP: A Randomized Clinical Trial w/ Long Term F/U’

SPINE 1980
Runner Up Volvo Award

- 56 Men w/ C-LBP 12 – 28.6 wks. duration
- 29 Study / 27 Ctrl Pts.
- IMS avg 7.9 Rx f/u: D/C, 12 wks., 27.3 wks.
- Ctrl. 4 RTW, 14 LD, 9 DISABLED
- 18/29 RTW, 10 LD NO DISABLED
Hong CZ: Lidocaine Injection vs. Dry Needling to Myofascial T.P. The Importance of Local Twitch Response

AMER JRNL of P M & R 1994

Figure 1. Effect of trigger point injection or dry needling on subjective pain intensity. 0, no pain; 10, most severe pain.

Figure 3. Effect of trigger point injection or dry needling on range of motion of cervical spine (lateral bending).
INTRAMUSCULAR STIMULATION DRY NEEDLING RESEARCH


Cochrane Reviews

Highly regarded, rigorous reviews of the available evidence of clinical treatments.

2005: “To assess the effects of dry needling for myofascial pain in the low back region”

Thirty-five RCTs covering 2861 patients were included in this systematic review.

“Dry-needling appears to be a useful adjunct to other therapies for chronic low-back pain.”


Emerging Concepts in the Treatment of Myofascial Pain: A Review of Medications, Modalities, and Needle-based Interventions:

“DN has moderate evidence that supports its use in MPS”

2011 Annaswamy PM&R

STUDY LIMITATION: PROPER PLACEBO CONTROL
Paraspinal Stimulation Combine with Trigger Point Needling & Needle Rotation for the Treatment of Myofascial Pain: A Randomized Sham-controlled Clinical Trial
Participants: A total of 56 subjects with neck or shoulder girdle pain of more than 3 months duration and active MTrPs were recruited from a campus-wide volunteer sample. Of these, 52 completed the study (23 male and 33 female). Their mean age was 35.8 years.

Conclusions: Dry needling reduces pain and changes MTrP status. Change in trigger point status is associated with a statistically and clinically significant reduction in pain. Reduction of pain is associated with improved mood, function, and level of disability.
Mechanisms of Needle Effect?

- Local Twitch Response: Stimulation of Muscle Spindle & Spinal Reflex leading to
- Reversal of Muscle Contracture and Increased ROM
Myotatic Spinal Reflex

The Reflex Hammer Stimulates Brief Muscle Contraction, Then Release
Dry Needling Stimulates Local Twitch Response - LTR thru Spinal Reflexes

The Pin Causes Shortened Muscle Fibers To Contract Briefly, Then Relax
Chu: Does EMG (Dry Needling) Reduce Myofascial Pain Symptoms Due to Cervical Root Irritation?

One ‘Treatment’ Only

Grp 1 82/122: avg 52% decreas Pain, 14% > 75%
Grp 2 23/42: avg 39%, 0 > 75%

---

**Increased Insertional Activity**

**Twitch Response**
IMS is Like ‘Catch-and-Release’
Fly Fishing:
We are ‘Casting’ for the ‘Bites’ = Needl Grasp or
‘Local Twitch Response’
Kim HK, Kim SH, Kim MJ, Lim JA, Kang PS, Woo NS, Lee YC.
Intramuscular stimulation in chronic pain patients.
J Korean Pain Soc 2002 15:139-145
An In Vivo Microanalytical Technique for Measuring the Local Biochemical Milieu of Human Skeletal Muscle

The Milieu Level of Analytes is different in those with/without pain, those with active vs. latent or no tps, and that changes can be tracked before, during and after a LTR


Biochemicals Associated With Pain and Inflammation are Elevated in Sites Near to and Remote From Active Myofascial Trigger Points

2008 Arch PMR 89: 16-23; Shah, J., et.al.
<table>
<thead>
<tr>
<th>Needle Effect</th>
<th>Clinical Response</th>
<th>Neurophysiological Basis</th>
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<tbody>
<tr>
<td>Local Twitch Response</td>
<td>Increased ROM</td>
<td>Reversal of Muscle Contracture</td>
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<tr>
<td>Stimulation of Spinal Reflexes</td>
<td></td>
<td>? Normalization of Spindle Mechanism/Sensitivity</td>
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<tr>
<td>Hyperstimulation Analgesia</td>
<td>Decreased Pain</td>
<td>Melzack and Wall Gate Theory</td>
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<tr>
<td>Direct and Reflex Stimulation &amp; Normalization of Therapeutic Target</td>
<td>Decreased Myalgic Hyperalgesia</td>
<td>Reversal of Neuropathic Supersensitivity - Cannon and Rosenblueth’s Law of Denervation Supersensitivity</td>
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<tr>
<td>Direct and Reflex Stimulation &amp; Normalization of Therapeutic Target</td>
<td>Decreased Spontaneous Endplate Activity</td>
<td>Reversal of Neuropathic Supersensitivity</td>
</tr>
<tr>
<td>Direct &amp; Reflex Stimulation</td>
<td>Vasodilation &gt; Warming</td>
<td>? Spinal/Axon Reflexes</td>
</tr>
<tr>
<td>Minor Tissue Trauma - Bleeding</td>
<td>Inflammatory Response, release of PDGF &amp; ‘Current of Injury’</td>
<td>Reversal of Neuropathic Supersensitivity by electric stimulation - Lomo</td>
</tr>
</tbody>
</table>
Treatment Ladder of Neuropathic-Myofascial Pain

• Manual soft tissue: ‘ischemic compression’: trigger point massage, strain-counterstrain, Shiatsu, acupressure, pin & stretch, Astym
• FREQUENT FREQUENT FREQUENT FREQUENT 2x/wk (7x/week)
• Independent Therapeutic Aids: tennis ball, Theracane, Accumassage
• S-T-R-E-T-C-H: NOT strengthen
• Vapocoolant (Fluoromethane) Spray & Stretch: children
• Heat: moist heating pad/hot bath/magnesium salts
• Electrical-stim, TENS, U/S, Low Level Laser, Kinesiotape: TREAT DIRECTLY OVER TENDER MOTOR POINTS
• Osteopathic, manual PT: GENTLE, CONTROLLED, Activator
• No Impact CV for LIMBERING/RELAXATION, not ‘conditioning’
Dry Needling/IMS: Best in Mild/Moderate NMPS:
NOT a SALVAGE Treatment: PAINFUL!!!
Ergonomic & other Postural Habits: supported forearms for keyboarders, avoid sitting with legs tucked under pelvis; heel inserts for pelvic obliquity
Biofeedback for breathing & psychophysiological factors
Rx: Muscle relaxants, Anti-cholinergic/TCAs
Anti-neuropathics/gabapentin
‘Pro-neuronal’: Acetyl-L-carnitine1000mg 2-4x/day, R-lipoic acid 300 mg/day
Topical Rx Anti-Cholinergic/Neuropathic/Inflammatory CREAMS: Local, Multiple Rx, $$$$$
Treat ANXIETY, sleep disturbance, Depression
Anti-Inflammation Diet: omega-3, Vit D, turmeric, cannbanoids/CBD
Intramuscular Stimulation

Dry Needling
Learn to locate trigger points with a pointfinder.
VIDEO DEMO of Intramuscular Stimulation - IMS Dry Needling
The marijuana is covered, but there’s a co-payment for the brownies and the Grateful Dead music.