

# **Spokane Pain Conference 10/27/17**

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



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**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**

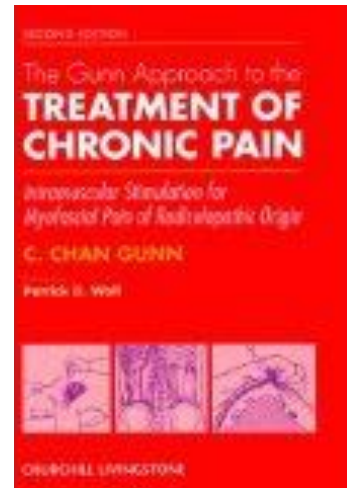
"I'm afraid it's your body, Mr. Haskins."



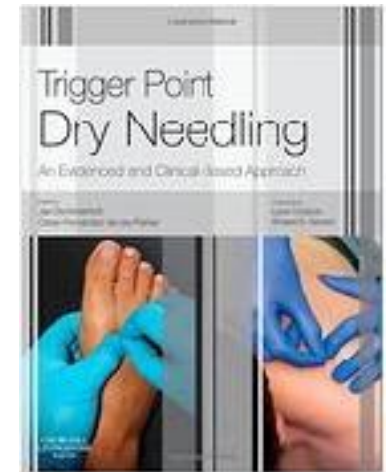
*"I'm afraid it's your body, Mr. Haskins."*



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, Infxn 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 17<sup>th</sup> Century

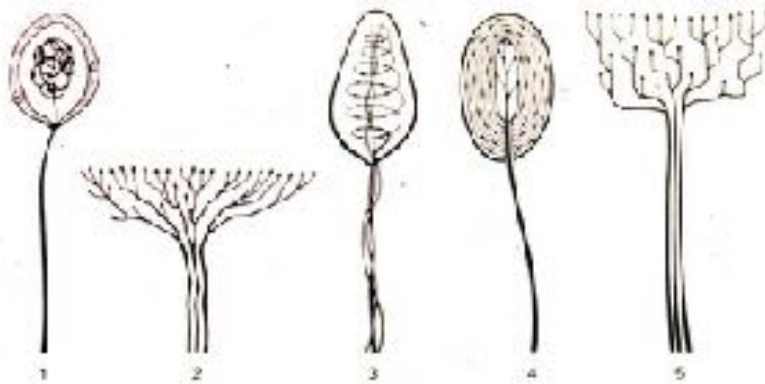


Figure 25. Descartes' (1664) concept of the pain pathway. He writes: "If for example fire (A) comes near the foot (B), the minute particles of this fire, which as you know move with great velocity, have the power to set in motion the spot of the skin of the foot which they touch, and by this means pulling upon the delicate thread (cc) which is attached to the spot of the skin, they open up at the same instant the pore (d e) 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

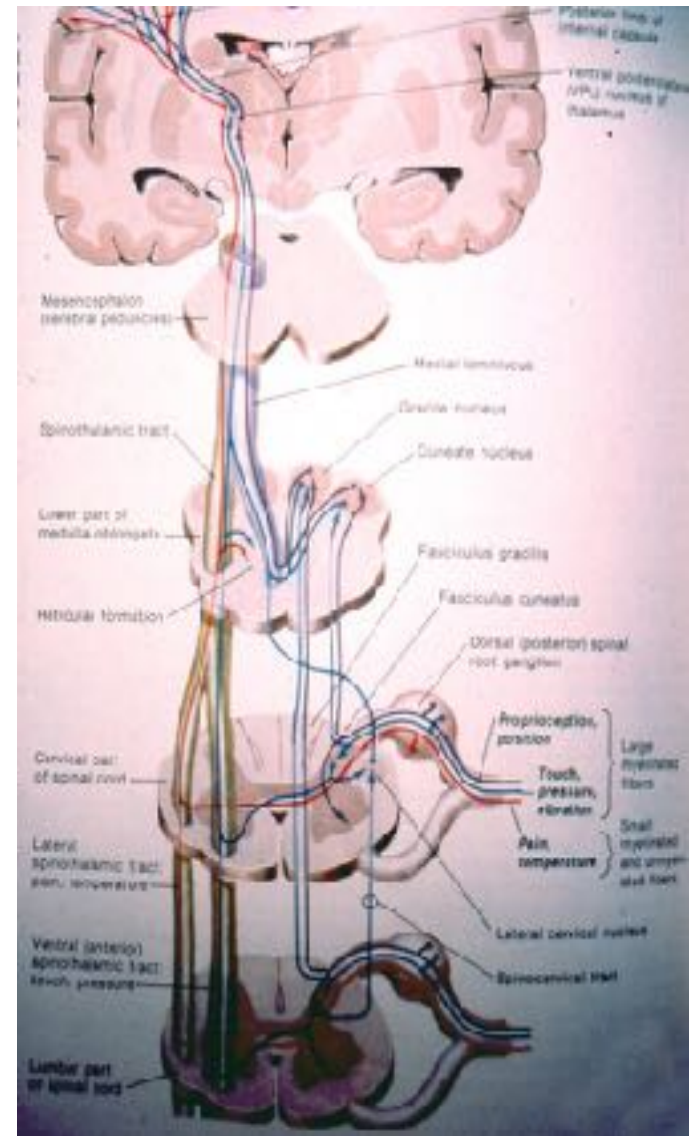
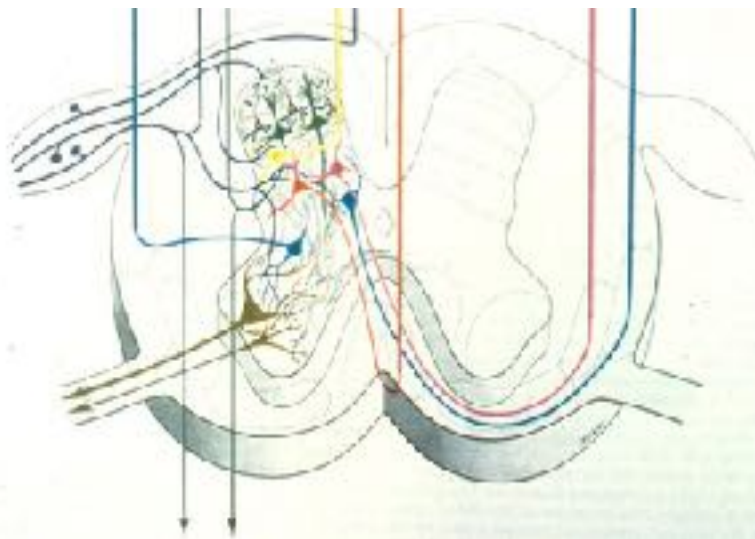
	Fiber Type		Function	Fiber Diameter (μm)	Conduction Velocity (m/s)
Myelinated	A	α	Proprioception; Somatic motor sense	12-20	70-120
		β	Touch, Pressure	5-12	30-70
		γ	Motor to muscle spindles	3-6	15-30
		δ	<b>Pain, Temperature, Touch</b>	<b>2-5</b>	<b>12-30</b>
	B		Preganglionic sympathetics	< 3	3-15
Unmyelinated	C	<i>Dorsal root</i>	Pain; Reflex responses	0.4-1.2	0.5-2
		<i>Sympathetic Fibers</i>	Postganglionic sympathetics	0.3-1.3	0.7-2.3

# Neuroanatomy A to Z

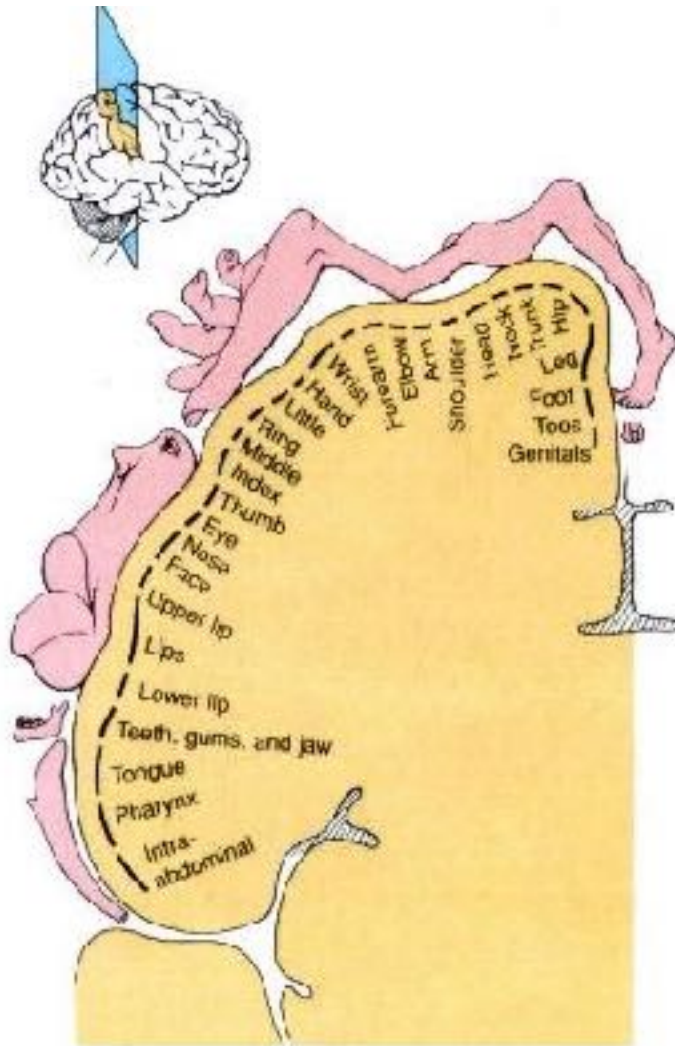


**Sense corpuscles and nerve endings that respond to:**

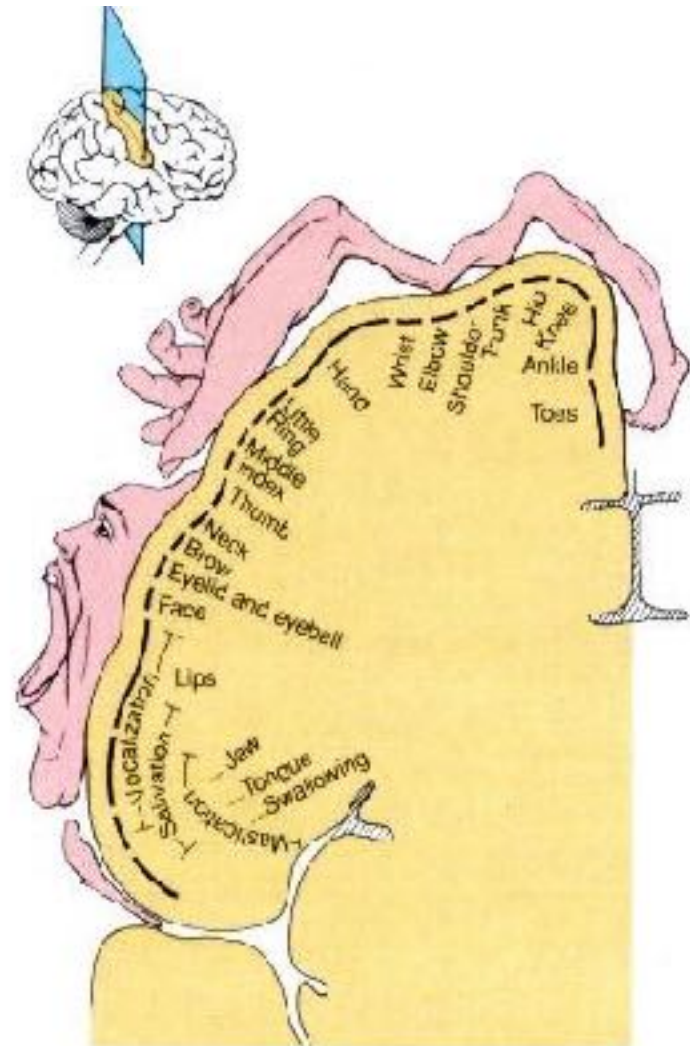
- |          |            |        |
|----------|------------|--------|
| 1 cold   | 3 touch    | 5 pain |
| 2 warmth | 4 pressure |        |



# Harry The Homunculus



(a) Somatosensory cortex in right cerebral hemisphere



(b) Motor cortex in right cerebral hemisphere

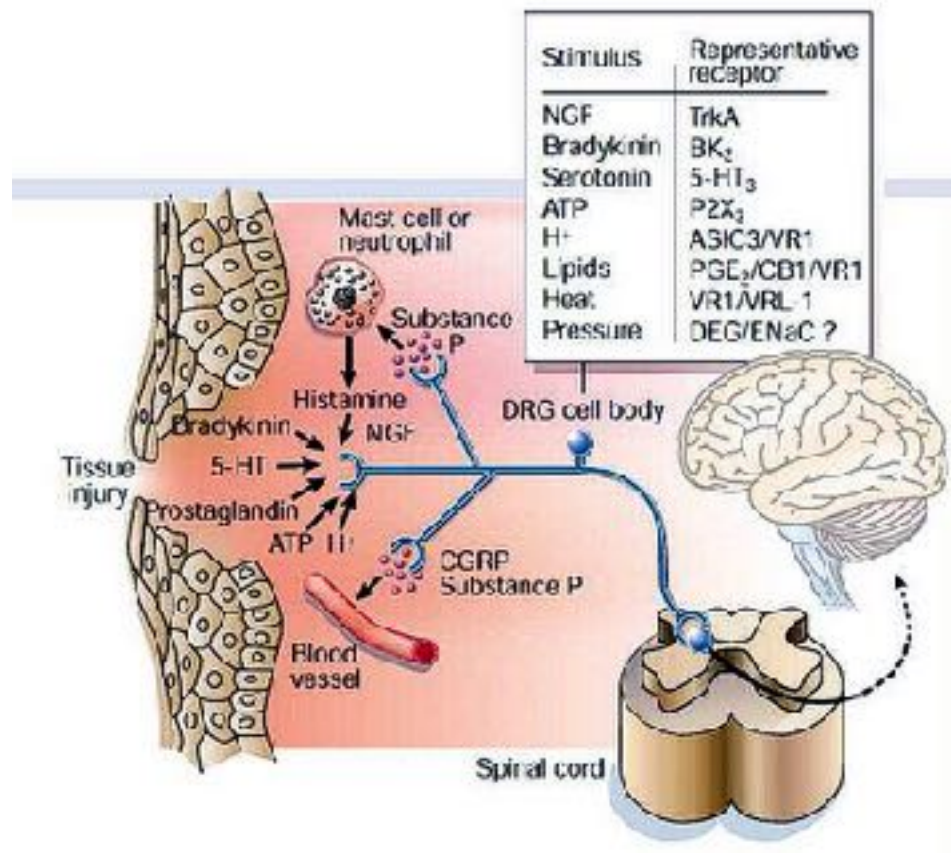
# **‘Type 1’ Pain: ‘Experimental Pain’ or ‘Monkey Business’ A-Delta Nerve Fiber**



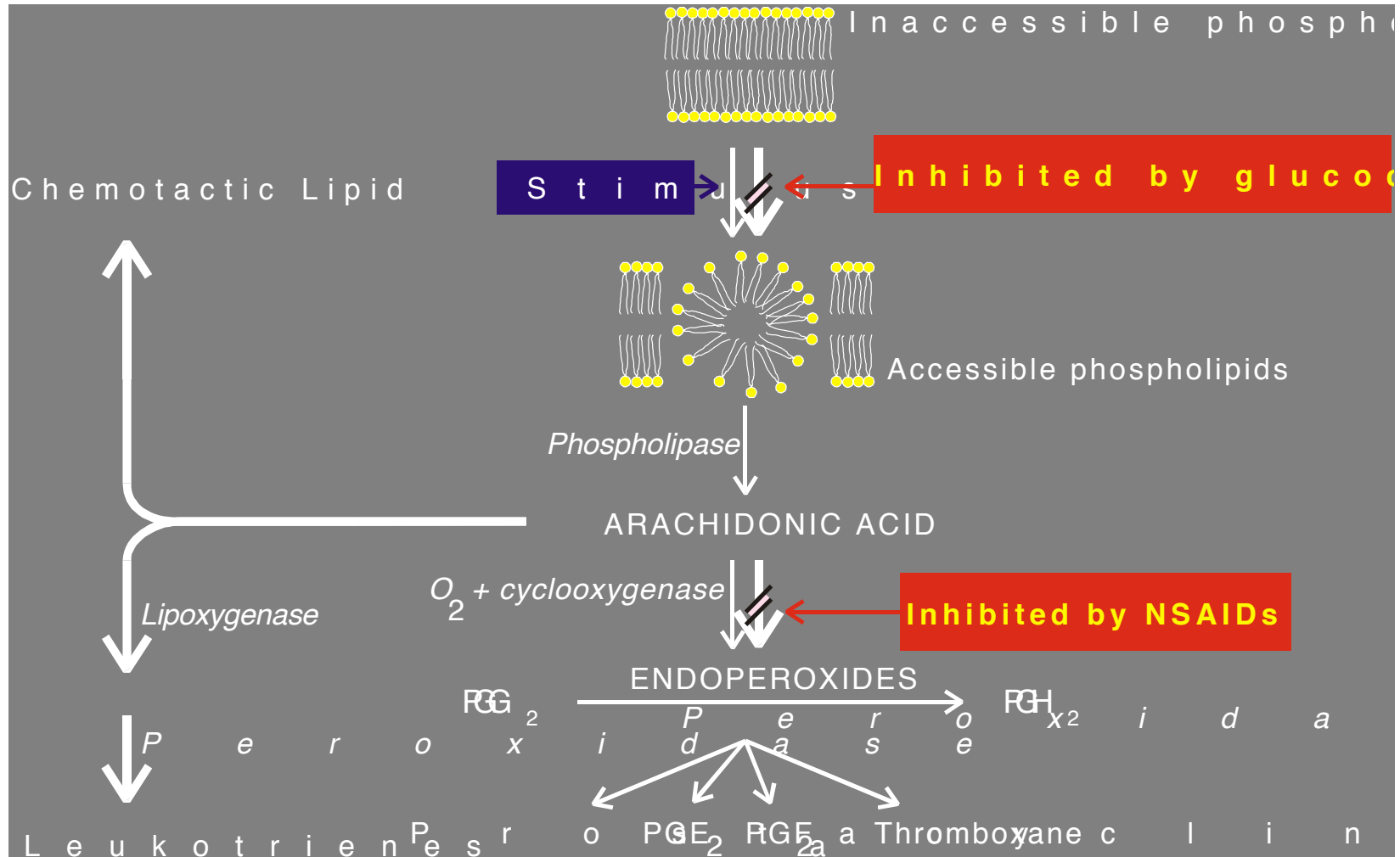
# CHARACTERISTICS of TYPES 1- 3 PAIN

	1 NOCI	2 INFLAMMATION	3 NEUROPATHIC
Localization/Locus	Cutaneous Precise Discr.	Poorly localized, vague Visceral/Referred Pattern	Poor/vague Mixed
Onset & Duration Course	Immediate On/Off	Acute Protracted	Chronic Unremitting
Quality	Sharp	Aching	ALL/MIXED
Intensity Correlation Stim/Resp	High “	Variable “	High
Behavioral Response	Fight or Flight	Concern Care, Anxiety	Depression

# **‘Type 2’ Pain: Cellular Damage Trauma or Immune Mediated ‘Chemical’ Inflammation: DOLOR RUBOR CALOR TUMOR**



# The Inflammatory 'Cascade'



# Type 2 PAIN = C Fibre

	Fiber Type	Function	Fiber Diameter ( $\mu\text{m}$ )	Conduction Velocity (m/s)
Myelinated	A $\alpha$	Proprioception; Somatic motor sense	12-20	70-120
	$\beta$	Touch, Pressure	5-12	30-70
	$\gamma$	Motor to muscle spindles	3-6	15-30
	$\delta$	Pain, Temperature, Touch	2-5	12-30
	B	Preganglionic sympathetics	< 3	3-15
Unmyelinated	<b>C Dorsal root</b>	<b>Pain; Reflex responses</b>	<b>0.4-1.2</b>	<b>0.5-2</b>
	<b>Sympathetic Fibers</b>	<b>Postganglionic sympathetics</b>	<b>0.3-1.3</b>	<b>0.7-2.3</b>

**a**

Primary afferent axons

**Aα and Aβ fibres**

Myelinated  
Large diameter  
Proprioception, light touch

Thermal threshold

None

**Aδ Fibre**

Lightly myelinated  
Medium diameter  
Nociception  
(mechanical, thermal, chemical)

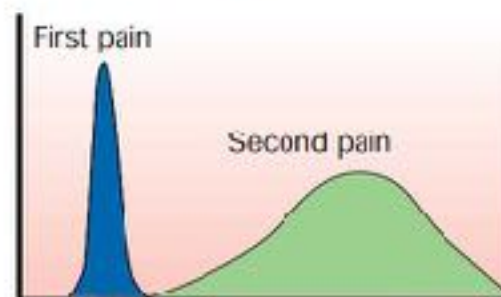
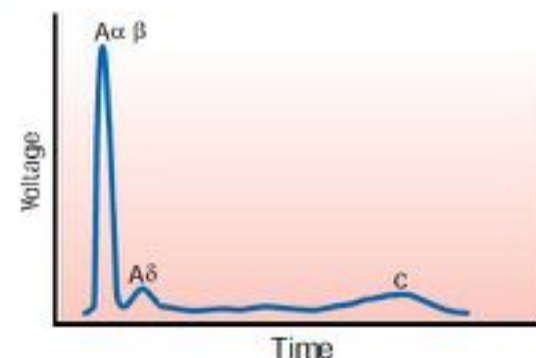
~ 53 °C Type I

~ 43 °C Type II

**C fibre**

Unmyelinated  
Small diameter  
Innocuous temperature, itch  
Nociception  
(mechanical, thermal, chemical)

~ 43 °C

**b**

**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<sup>-1</sup>, 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)**

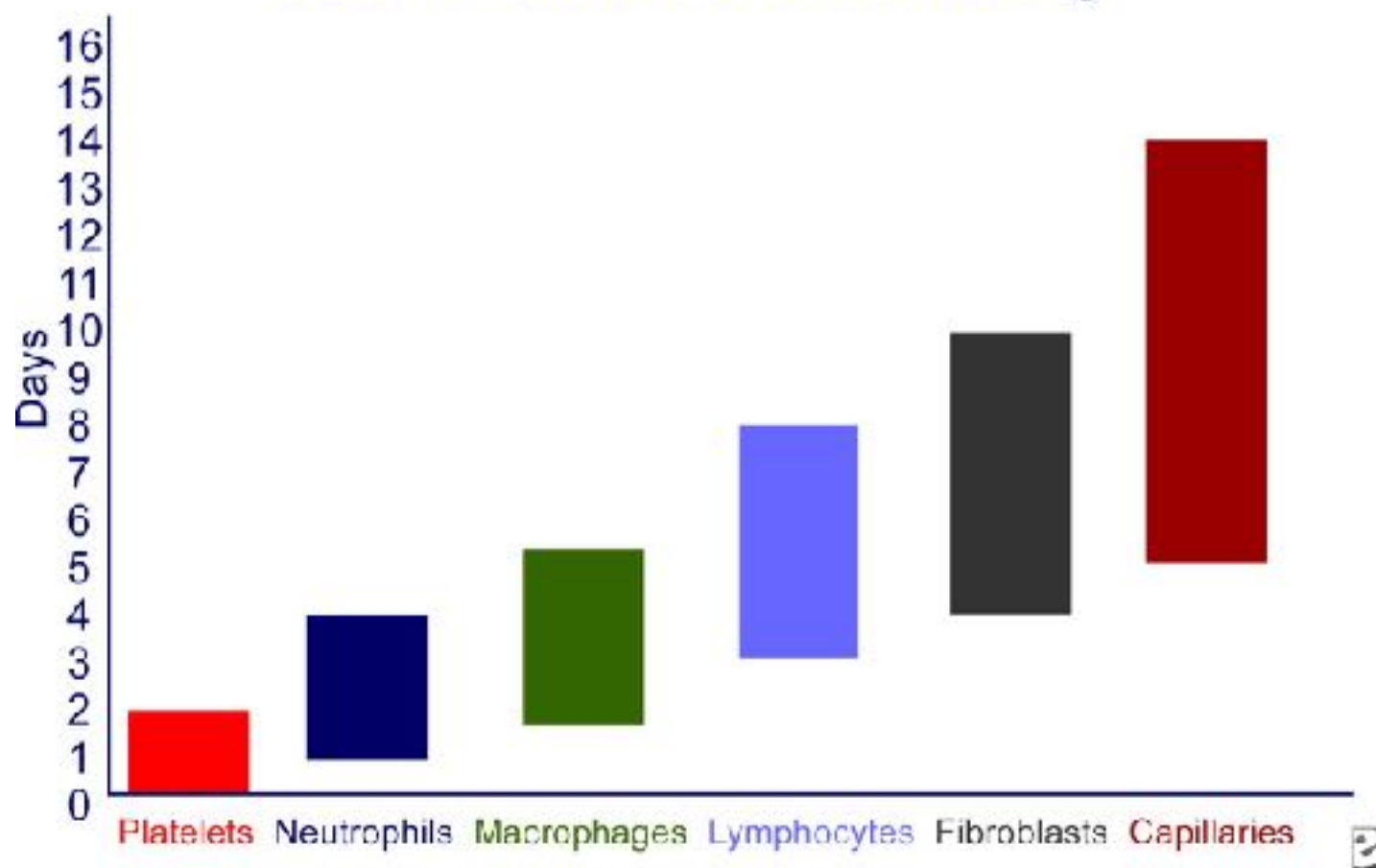
**Must Create Tissue Damage To Cause PAIN**

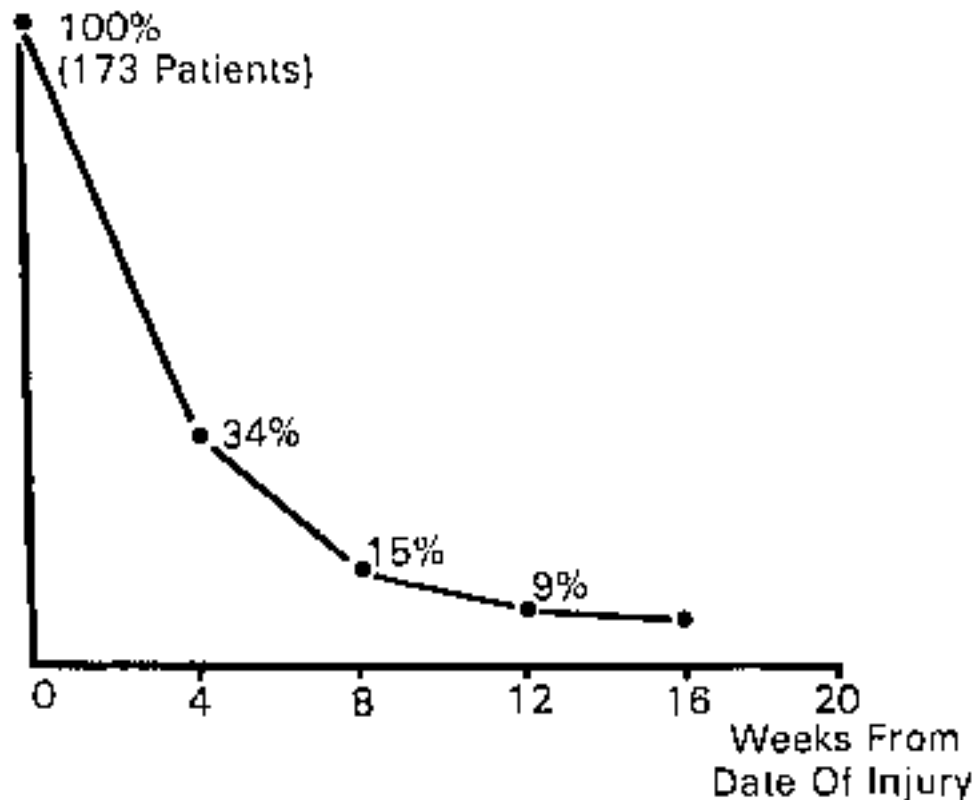


# CHARACTERISTICS of TYPES 1- 3 PAIN

	<b>1</b> <b>NOCI</b>	<b>2</b> <b>INFLAMMATION</b>	<b>3</b> <b>NEUROPATHIC</b>
<b>Localization/Locus</b>	<b>Cutaneous Precise Discr.</b>	<b>Poorly localized, vague Visceral/Referred Pattern</b>	<b>Poor/vague Mixed</b>
<b>Onset &amp; Duration Course</b>	<b>Immediate On/Off</b>	<b>Acute Protracted</b>	<b>Chronic Unremitting</b>
<b>Quality</b>	<b>Sharp</b>	<b>Aching</b>	<b>ALL/MIXED</b>
<b>Intensity Correlation Stim/Resp</b>	<b>High “</b>	<b>Variable “</b>	<b>High</b>
<b>Behavioral Response</b>	<b>Fight or Flight</b>	<b>Concern Care, Anxiety</b>	<b>Depression</b>

## Cells Involved in Wound Healing





**Fig 1.** The natural history of low-back injury. A survey of 173 patients in 1975 showed that most (85%) are recovered by the eighth week regardless of the modality of treatment. After the 12th week there remains a resistant group of less than 10% in whom progress is extremely slow.

**Gunn CC, Milbrandt WE. Dry needling of muscle motor points for chronic low back pain. A randomized clinical trial with longterm follow-up. Spine 1980;5:279-29**

# Rate of Recovery After Injury

Disability (%)

100

75

50

25

0

0

4

8

12

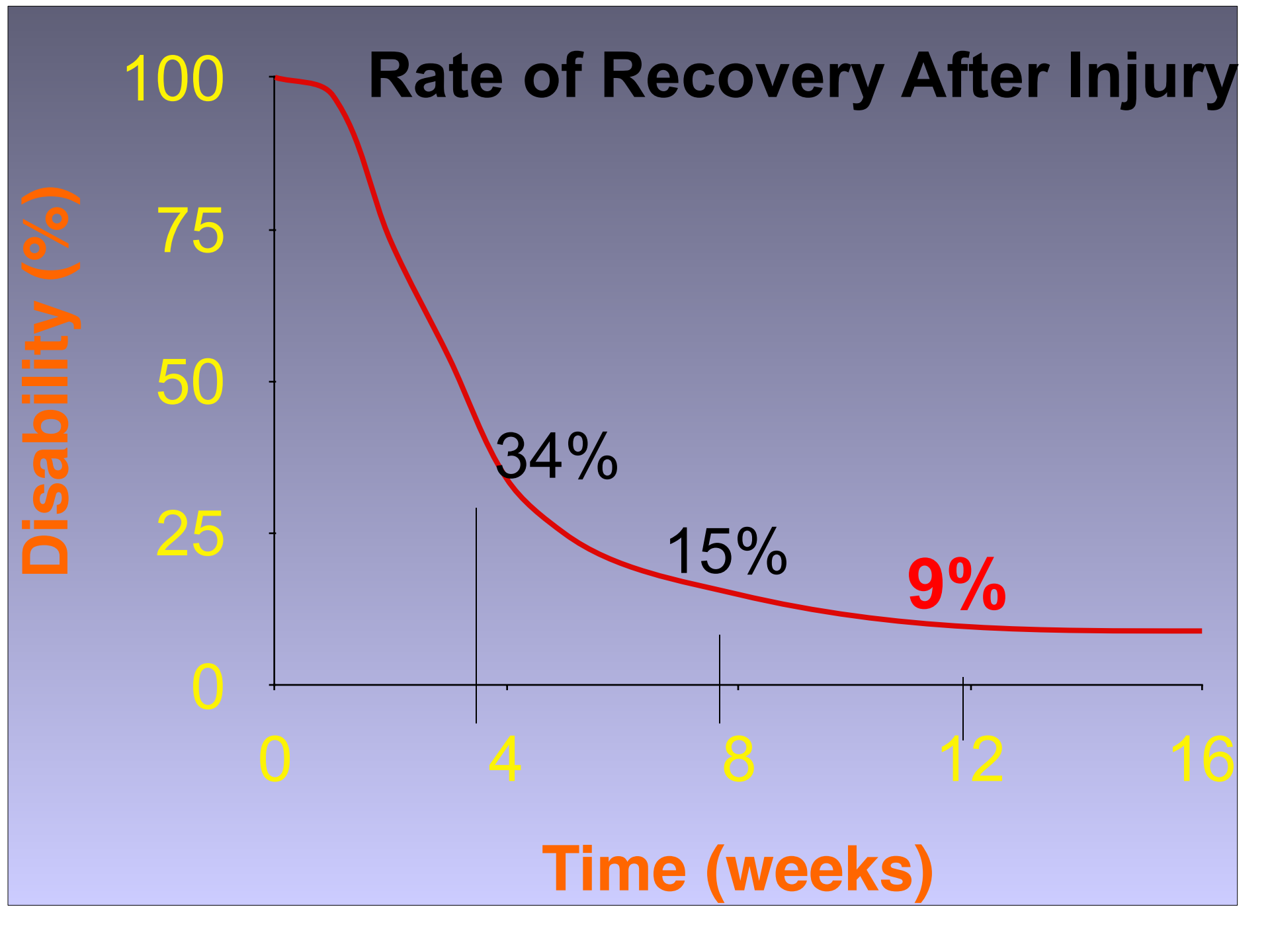
16

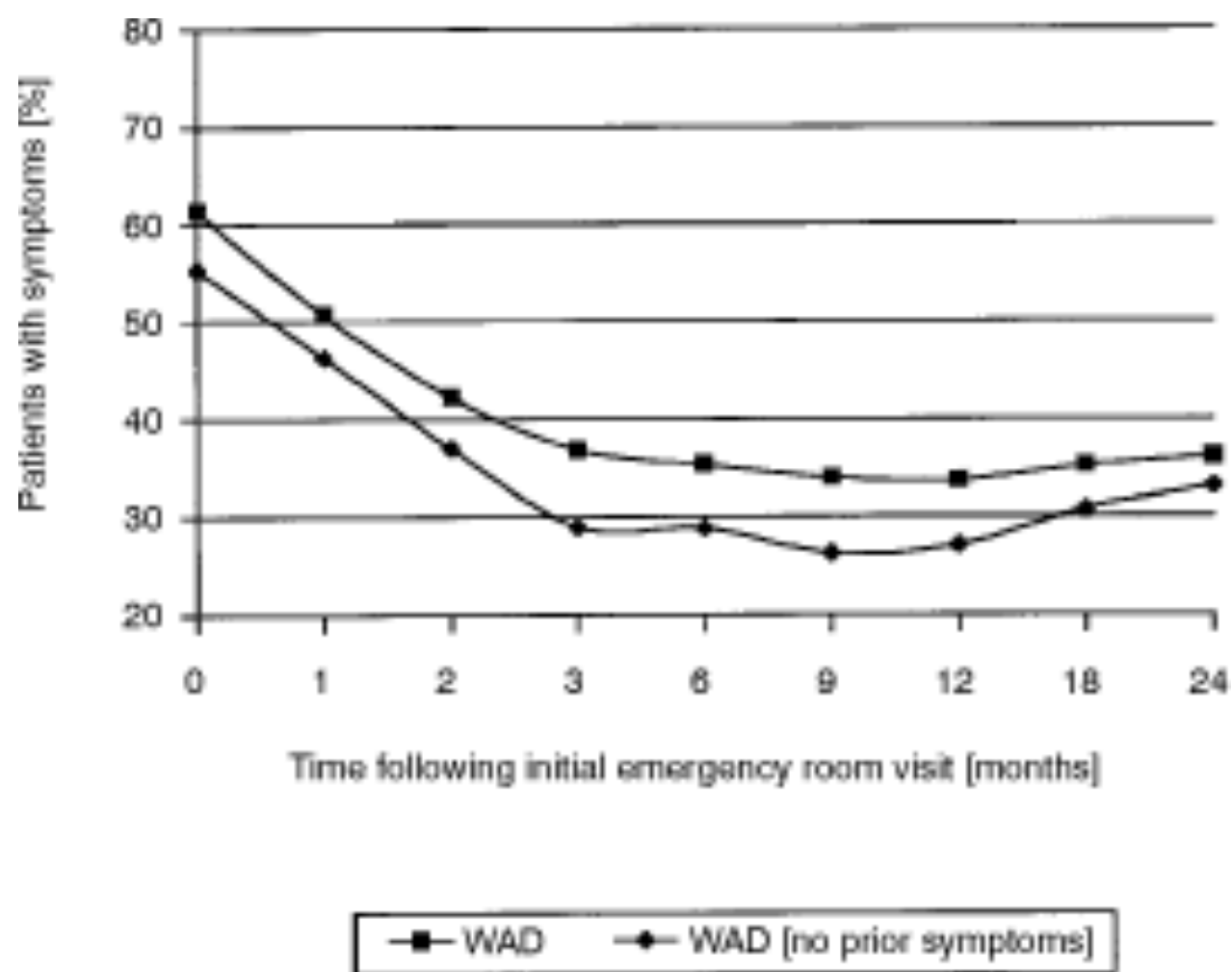
34%

15%

9%

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**

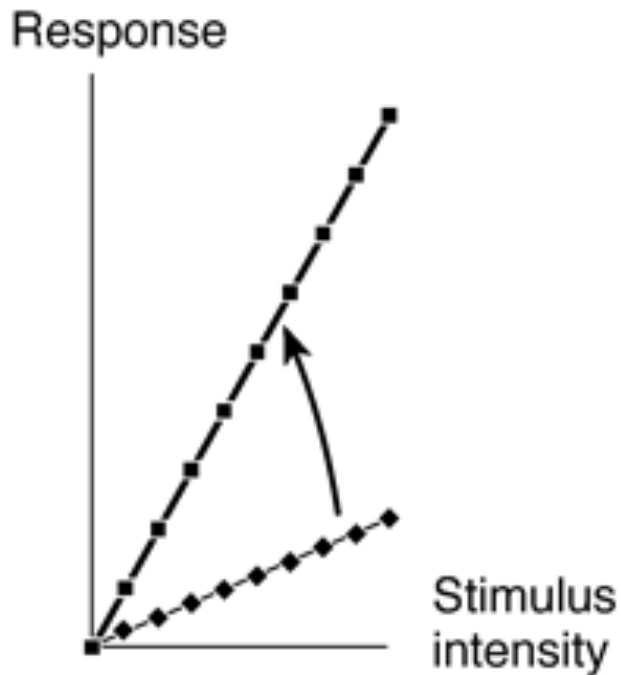
D

**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**

Peripheral effect	Central effects
<ul style="list-style-type: none"> <li>• Ectopic and spontaneous discharge</li> <li>• Ephaptic conduction</li> <li>• Alterations in ion channel expression</li> <li>• Collateral sprouting of primary afferent neurones</li> <li>• Sprouting of sympathetic neurones into the DRG</li> <li>• Nociceptor <u>sensitization</u></li> </ul>	<ul style="list-style-type: none"> <li>• Central <u>sensitization</u></li> <li>• Spinal reorganization</li> <li>• Cortical reorganization</li> <li>• Changes in inhibitory pathways</li> </ul>

**British Journal of Anesthesia**  
**Vol. 87, No. 1 12-26 2001**  
**Mechanisms of Neuropathic Pain**



(A) Hyperalgesia

– An increased response to a normally painful stimulus

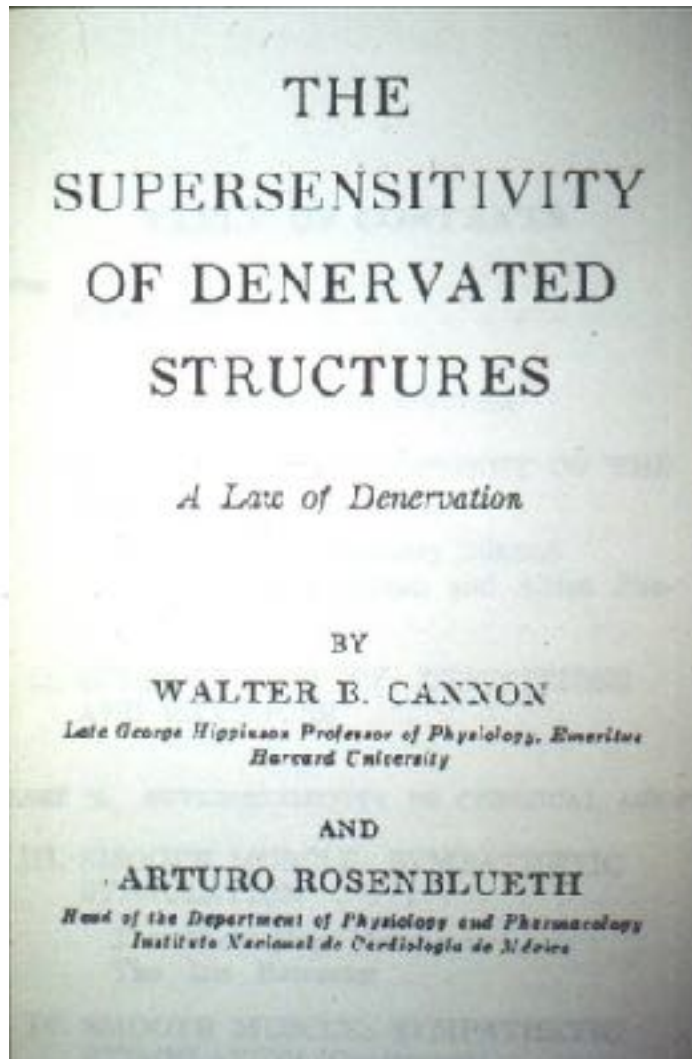


(B) Allodynia

– A painful response to a normally innocuous stimulus

**British Journal of Anaesthesia**  
**Vol. 87, No. 1 12-26 2001**  
**Mechanisms of Neuropathic Pain**

# 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.”**



**Mt. Cannon  
Glacier  
National Park  
Montana**



**Walter Cannon, Ph.D, M.D.**

**(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

D

## Four Types of Supersensitivity described by Cannon

- Superduration of response:

amplitude of response unchanged but duration prolonged

- Hyperexcitability of stimulus:

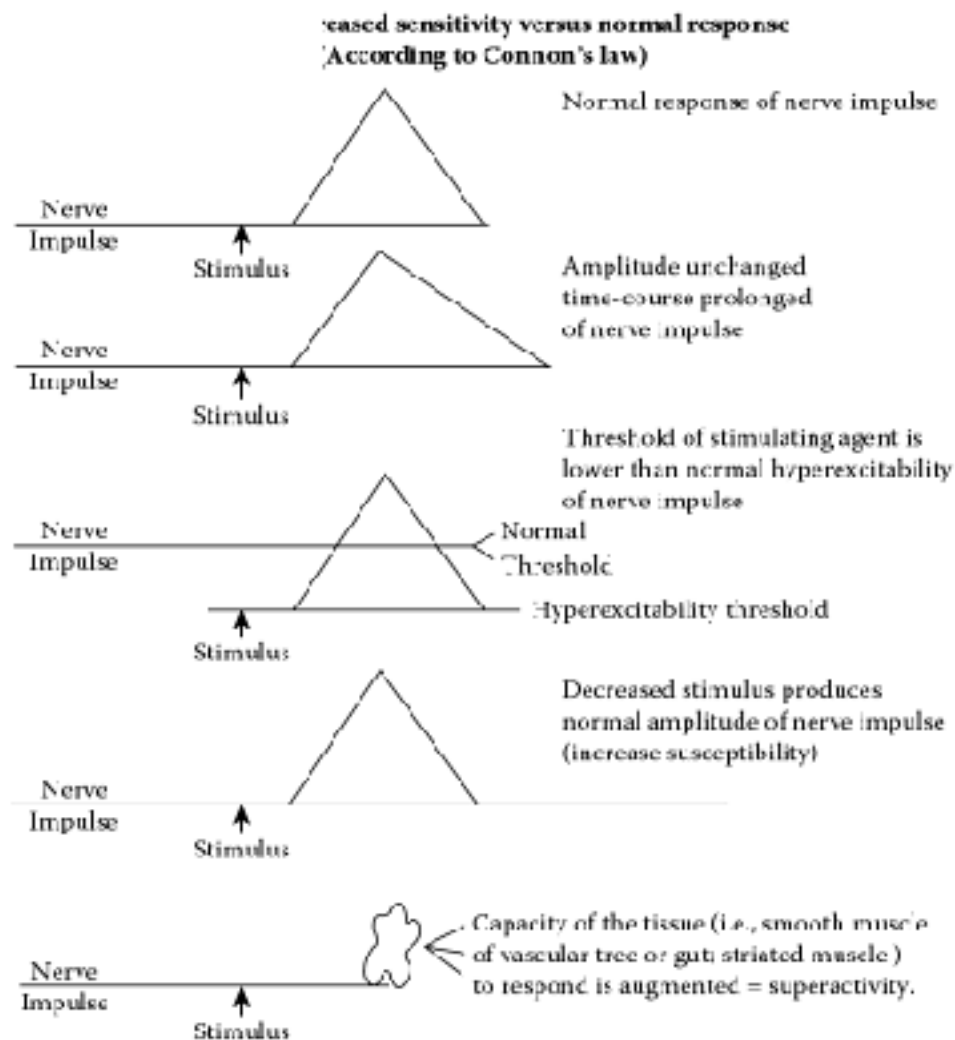
lower threshold of stimulus

- Increased Susceptibility of stimulus:

decreased stimulus = response of normal amplitude

- Superreactivity of tissue

augmented response of tissue



**FIGURE 2.12** 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). (EHC-Dallas.)

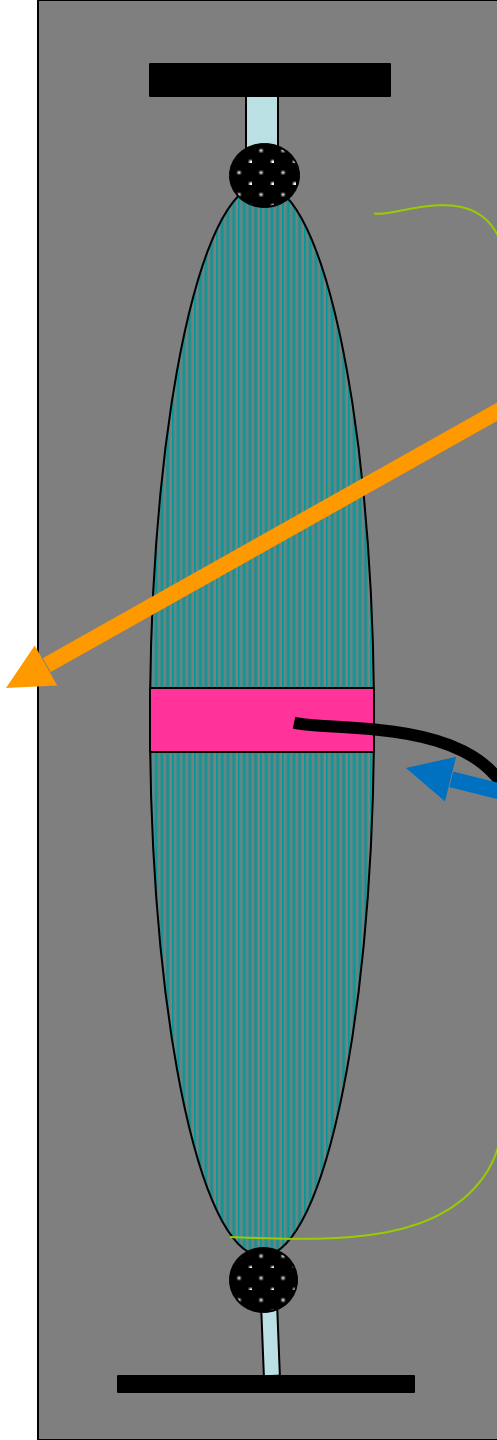
# Normal Muscle

**Motor Point on skin  
over zone of  
innervation**

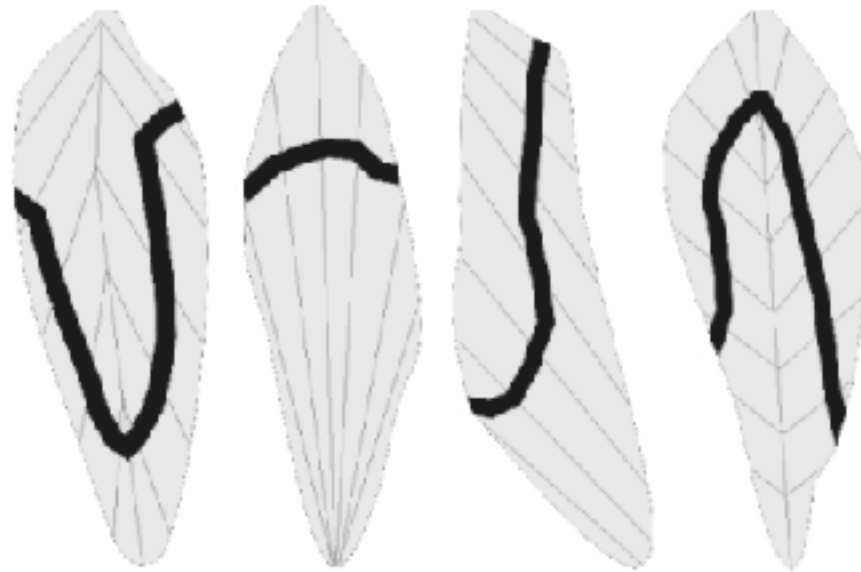
**Acetylcholine acts  
only at receptors  
within a narrow zone  
of innervation**

Nerve

**Normal subcutaneous tissue**



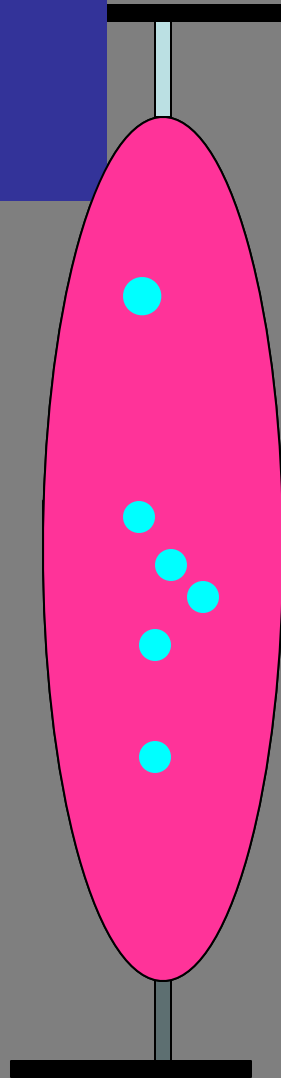
# **Distribution zones of motor end plates (black lines) traced by positivity of acetylcholinesterase in human lower limb muscles**



**Topographical localization of motor endplates in cryosections  
of whole human muscles. Muscle and Nerve 1984 May;7(4):  
287-93. Aquilonius, SM, et. al.**

# Neuropathic Muscle

**Trophic  
edema  
subcut**



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

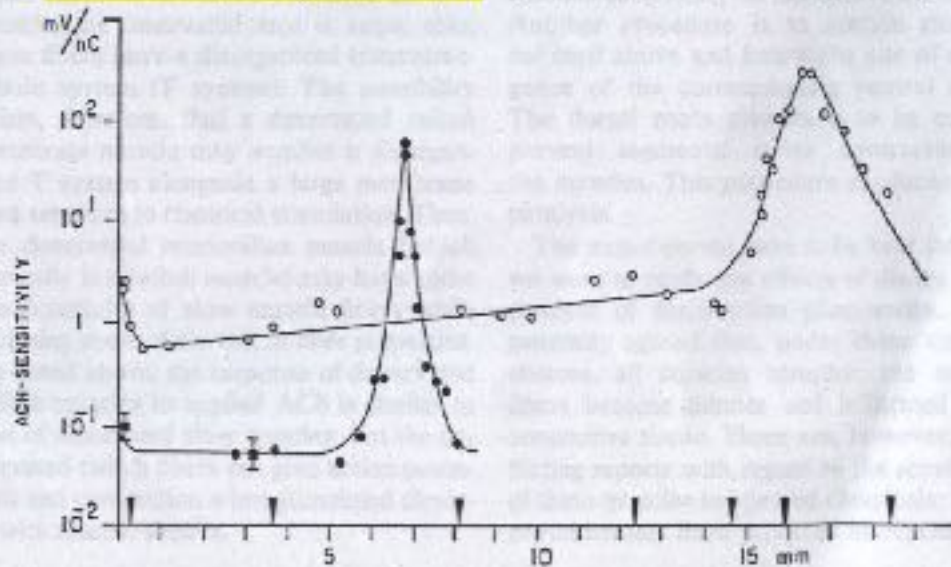
**1 mmol vs. 1000 mmol**

**Neuropathic =  
Sick Nerve**

# Acetylcholine Sensitivity Along Muscle Membrane After Denervation

Fig. 25-2.—Distribution of sensitivity to ACh applied iontophoretically along a frog muscle fiber 58 days after denervation (open circles) and along a fiber in the opposite, control muscle (dots). Distance is measured from the muscle-tendon junction. Triangles on the abscissa indicate successive positions of the intracellular recording electrode. Squares and bars denote mean sensitivity  $\pm$  S.E.M. of 10 fibers in control muscle. In normal muscle, the highest sensitivity to

ACh occurs at about 7 mm (the end-plate region). In the denervated muscle, there is almost uniform sensitivity to ACh except for an increase of the muscle-tendon junction, at about 16 mm. ACh sensitivity is expressed in threshold millivolts of depolarization per nanocoulomb of applied current ejecting ACh. (From Milred, R.: Induction of Receptors, in Manger, J. L., and DeReuck, A. V. S. [eds.]: *Enzymes and Drug Action* [Boston: Little, Brown and Company, 1962].)



Eyzaguirre C, Fidone SJ 1975 Physiology of the nervous system. Chicago, Year Book Medical Publishers. 2nd ed.

# 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

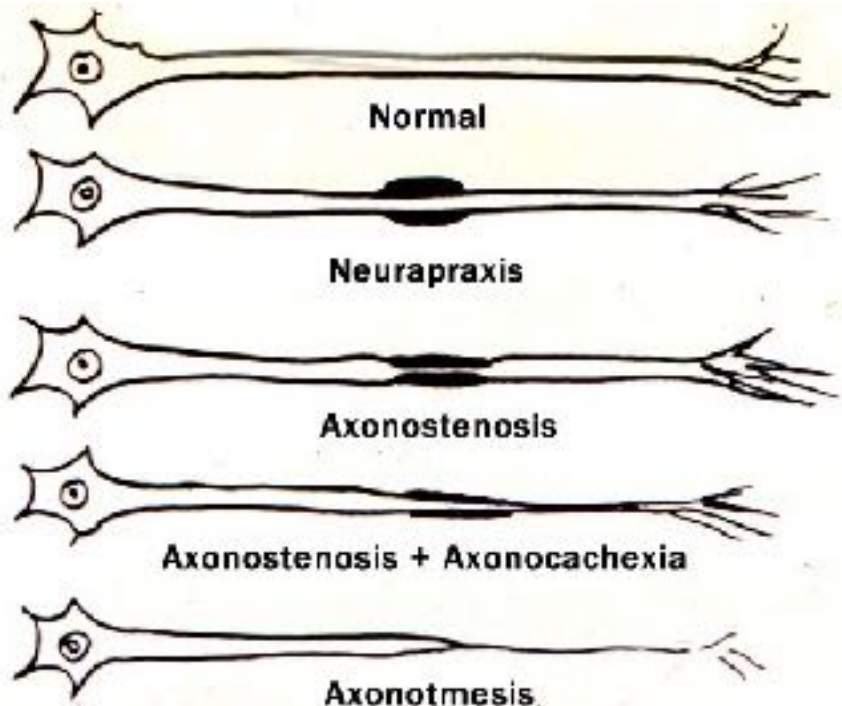
DE-NERVATION  A-TROPHY

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  DYS-TROPHY

Altered electrochemical state: SUPERSENSITIVITY

# Various Degrees of Compression to Motor Neurons



Various conditions in motor neurons  
as a result of  
progressive compression

**CANNON:  
DENERVATION > 'A'-TROPHY**

**'PARTIAL DENERVATION', or  
NEUROPATHY > 'DYS'-TROPHY**

# Neuropathic Supersensitivity > PAIN

## Home Security Alarm Triggers on Sunrise and Cat 'Burglars'



Type 1



RECEPTORS

NOXIOUS INPUT



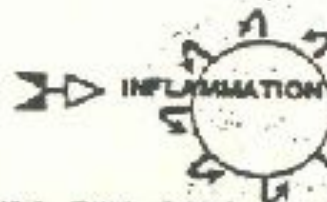
TYPE ONE PAIN = NOCICEPTION  
(EXTRINSIC)

BRAIN  
RESPONSES



FIGHT  
FIGHT  
FIGHT  
= PROTECTION

Type 2



TYPE TWO PAIN = ALGOGENIC  
(INTRINSIC)



Type 3



NORMAL INPUT



TYPE THREE 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).

British Journal of Anaesthesia, 2001, Vol. 87, No. 1 12-26 © 2001

Mechanisms of neuropathic pain

D. Bridges<sup>1,2</sup>, S. W. N. Thompson<sup>3</sup> and A. S. C. Rice<sup>1</sup> *<sup>1</sup>Pain 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**

This anatomical diagram illustrates the relationship between a lumbar vertebra, an intervertebral disc, and the associated nerve structures. The vertebra is shown in a sagittal view, with the disc positioned between it and the adjacent vertebra below. The disc is labeled 'DISC'. The nerve root and its branches are labeled 'Nerve Root & Branches'. The anterior branch is specifically labeled 'Anterior Branch'. The facet joint bone is labeled 'Facet Joint Bone'. The spinal cord is labeled 'Spinal Cord'. An autonomic nerve branch is shown originating from the nerve root and passing through the disc space, which is the focus of the study. The diagram is signed 'A. Cummings 1974' at the bottom.

## Anterior Branch

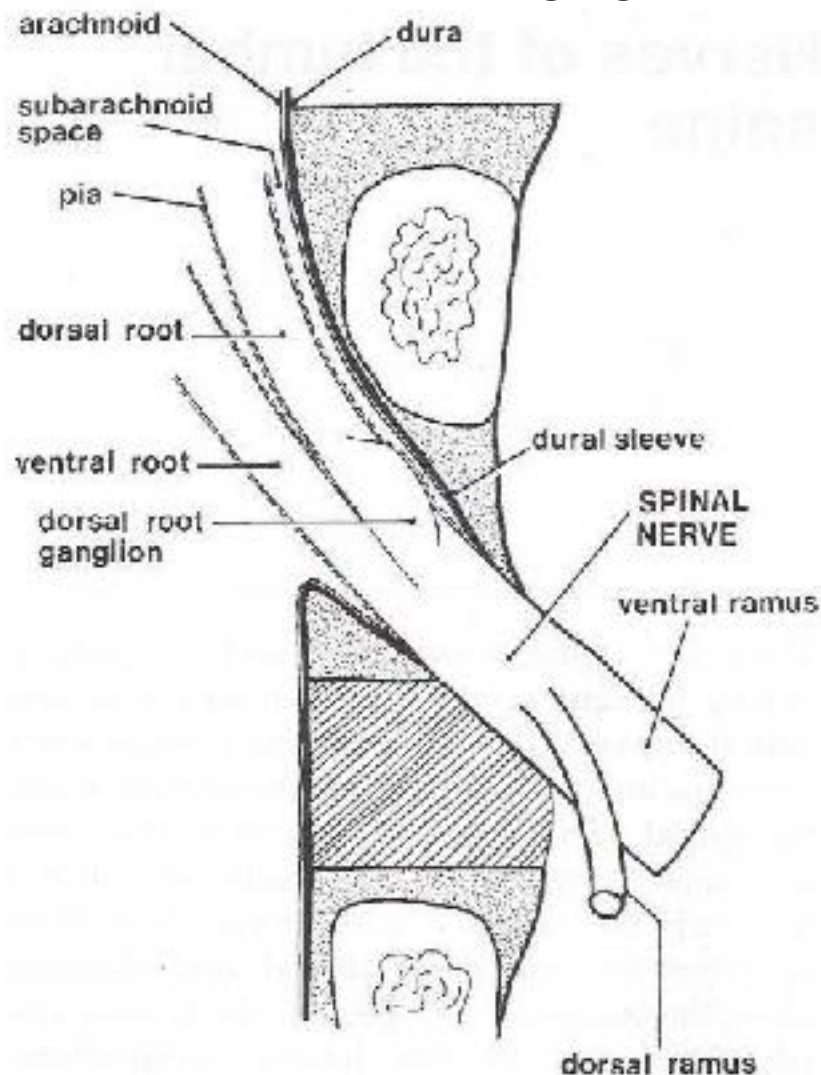
## Nerve Root & Branches

## Facet Joint Bone

# DISC

# Spinal Cord

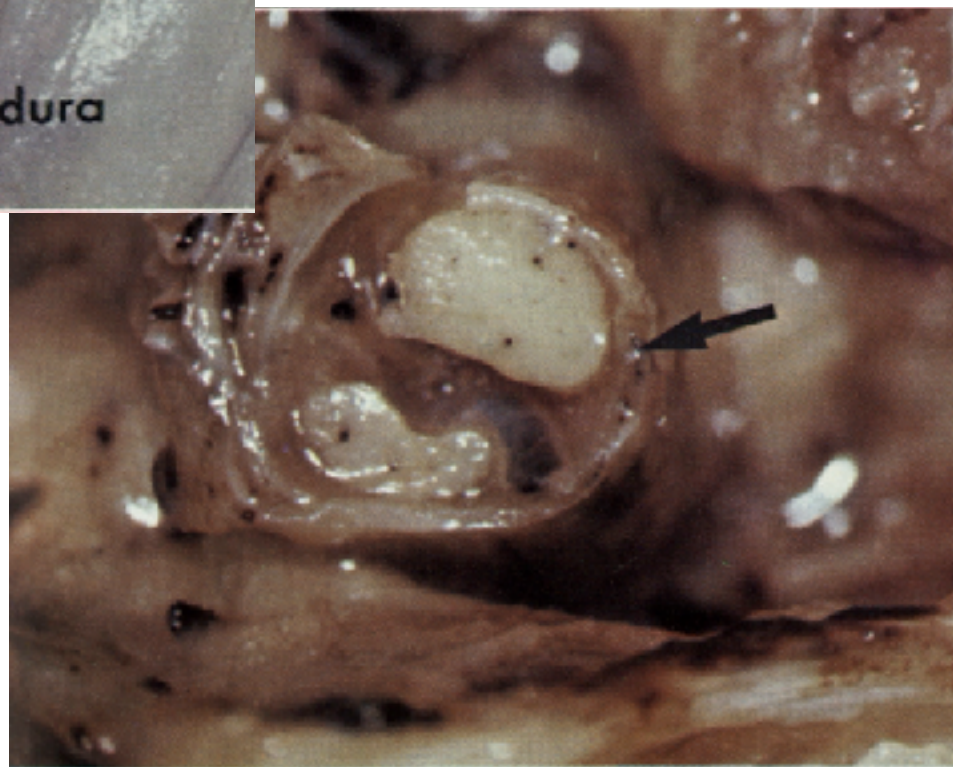
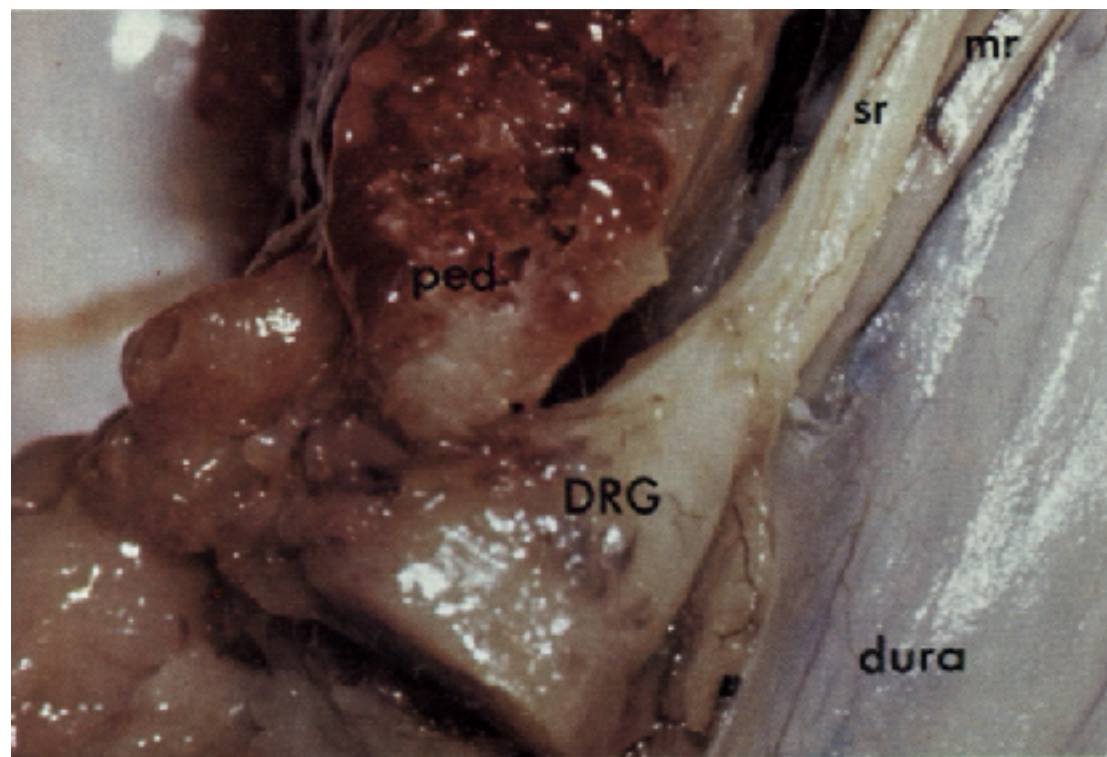
## CORONAL VIEW NEURAL FORAMINA

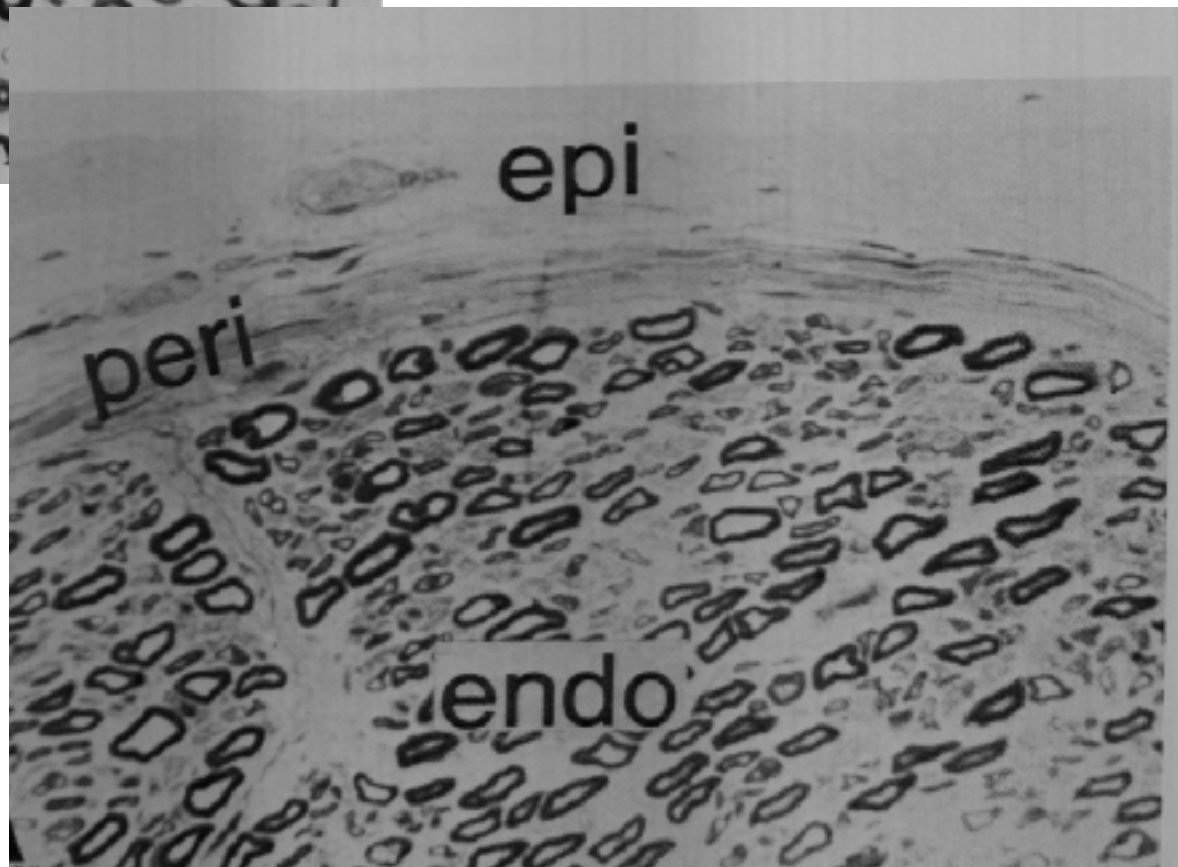
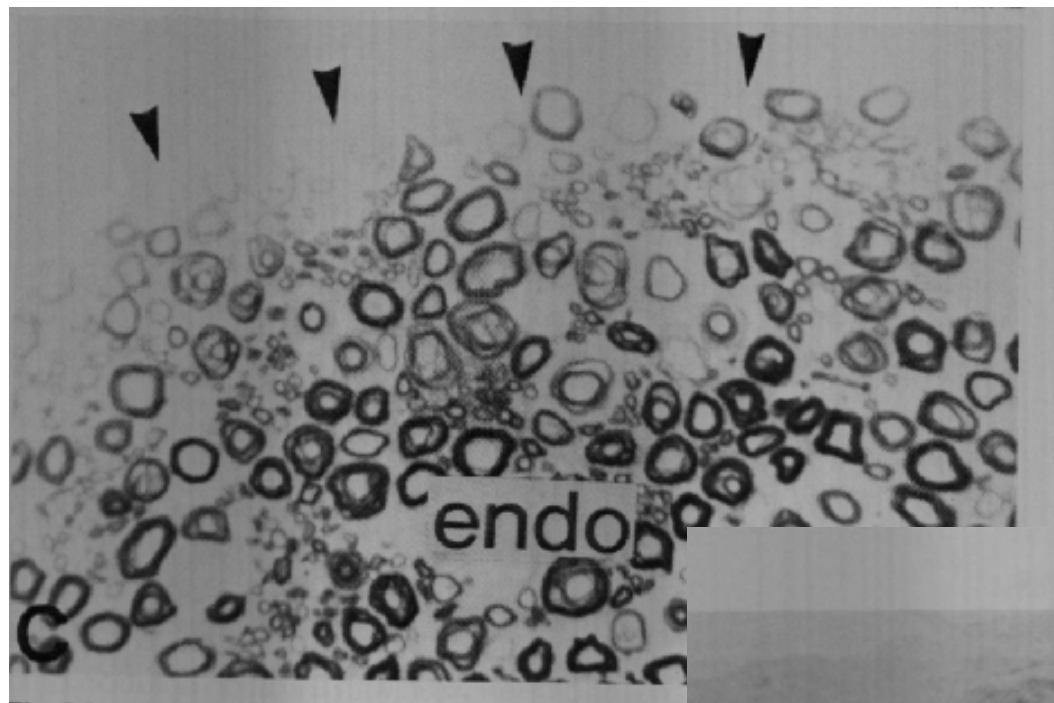


**Figure 10.1** A sketch of a lumbar spinal nerve, its roots and meningeal coverings. The nerve roots are invested by pia mater, and covered by arachnoid and dura as far as the spinal nerve. The dura of the dural sac is prolonged around the roots as their dural sleeve, which blends with the epineurium of the spinal nerve.

**Spinal Nerve Roots  
Lack Protection of  
Perineurium and  
Epineurium of Spinal/  
Peripheral Nerve**

**Bogduk, N. Clinical Anatomy of  
the Lumbar Spine and Sacrum,  
3<sup>rd</sup> ed, p.128**

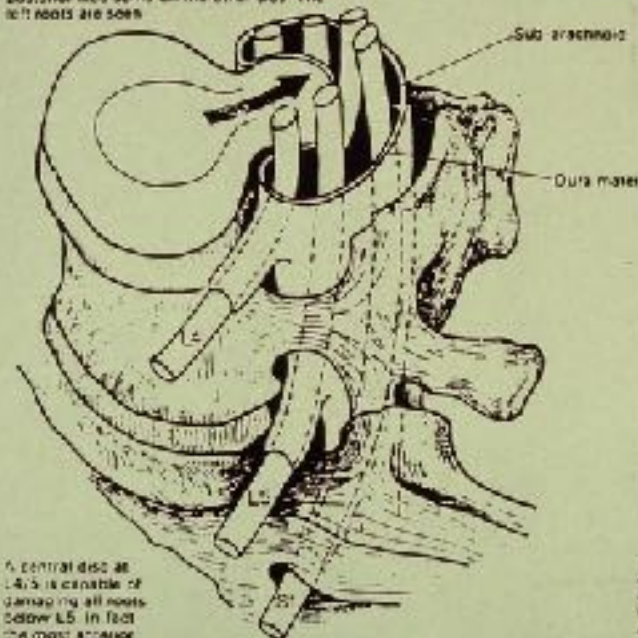




# Dura Mater Tethers Nerve Root

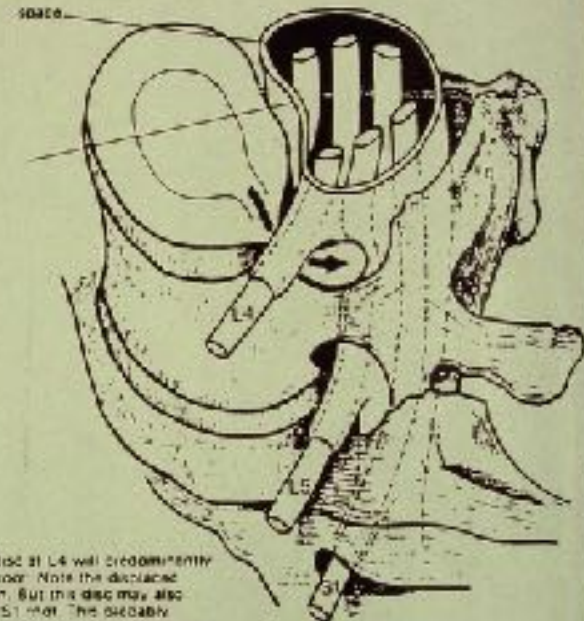
Figure 15.8: Diagram to show the effect of a Central Disc Prolapse

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



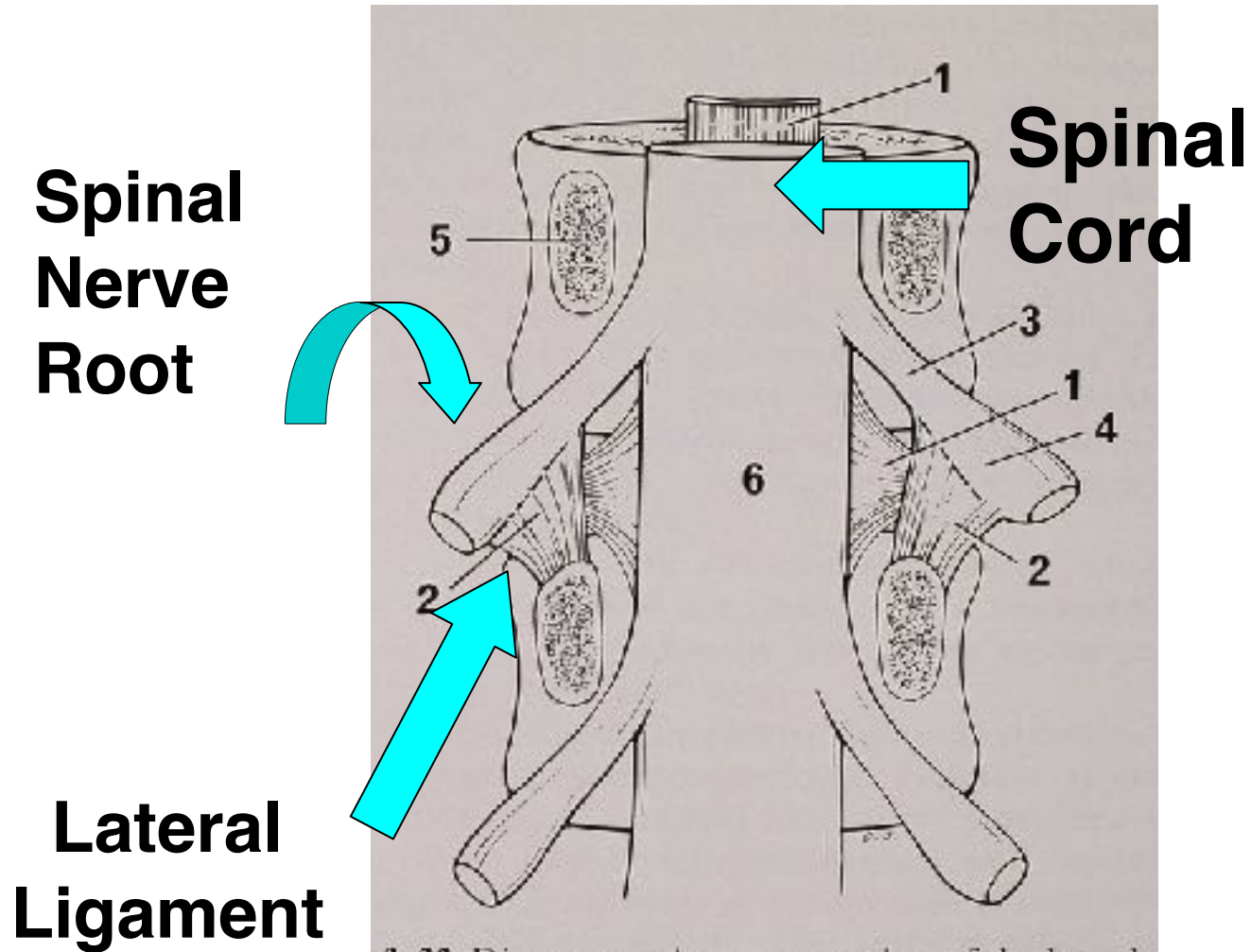
A central disc at L4/5 is capable of compressing all roots below L5. In fact the most anterior roots (L5-S2) are the most vulnerable.

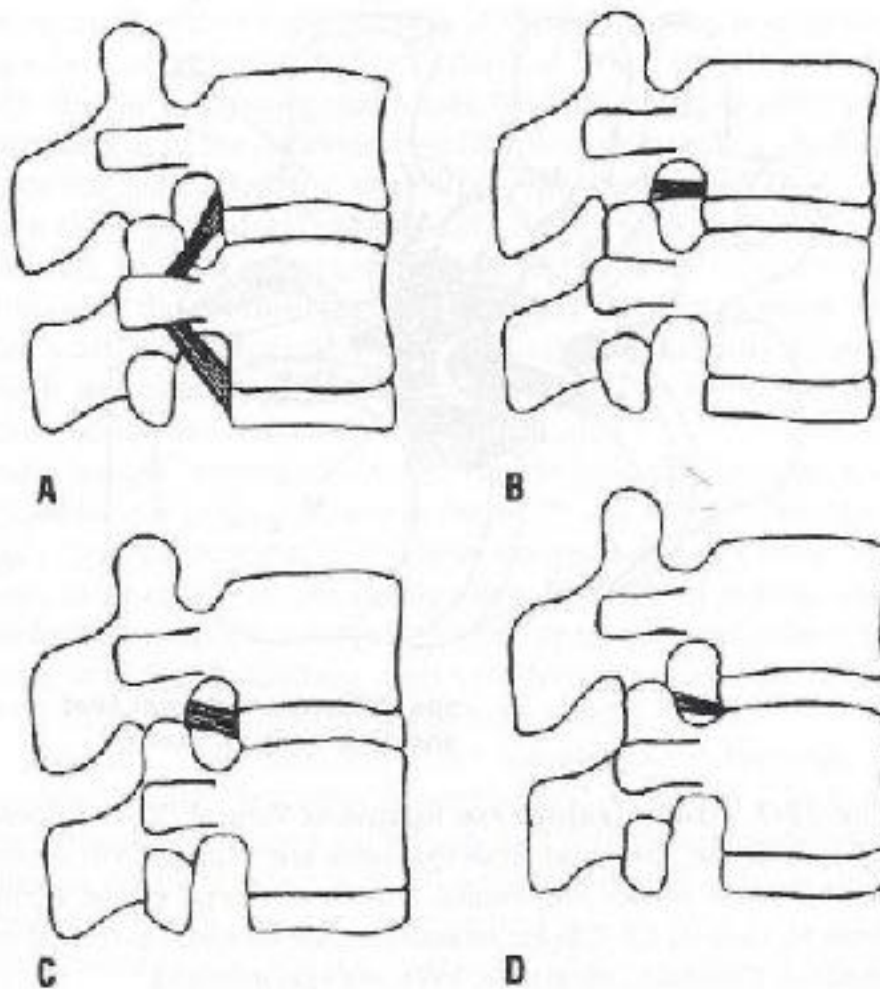
Diagram to show the effect of a Lateral Disc Prolapse



A lateral disc at L4 will predominantly affect L5 root. Note the displaced root pouch. But this disc may also affect the S1 root. This probably accounts for the ankle jerk often being abolished with disc lesions at L4/5.

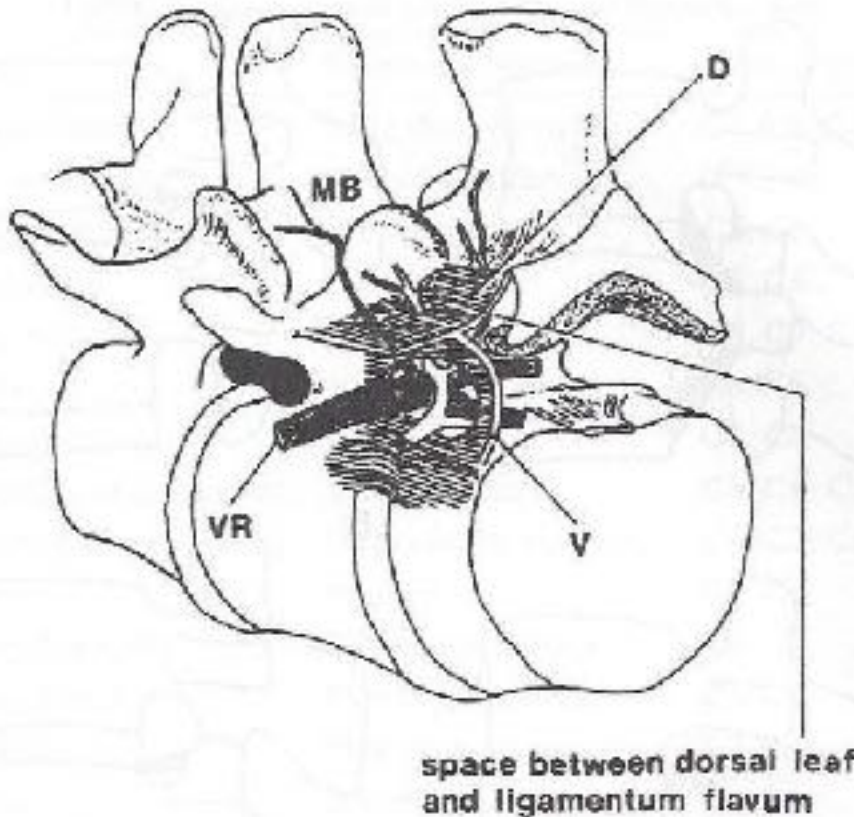
# Lateral Root & Other Ligaments





**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 corporotransverse 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.)

**Dumitru, D.  
Electrodiagnostic  
Medicine, 2nd ed.  
Hanley & Belfus, 2002,  
p.720**



**Dumitru, D.  
Electrodiagnostic  
Medicine, 2<sup>nd</sup> ed.  
Hanley & Belfus,  
2002, p.719**

**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.

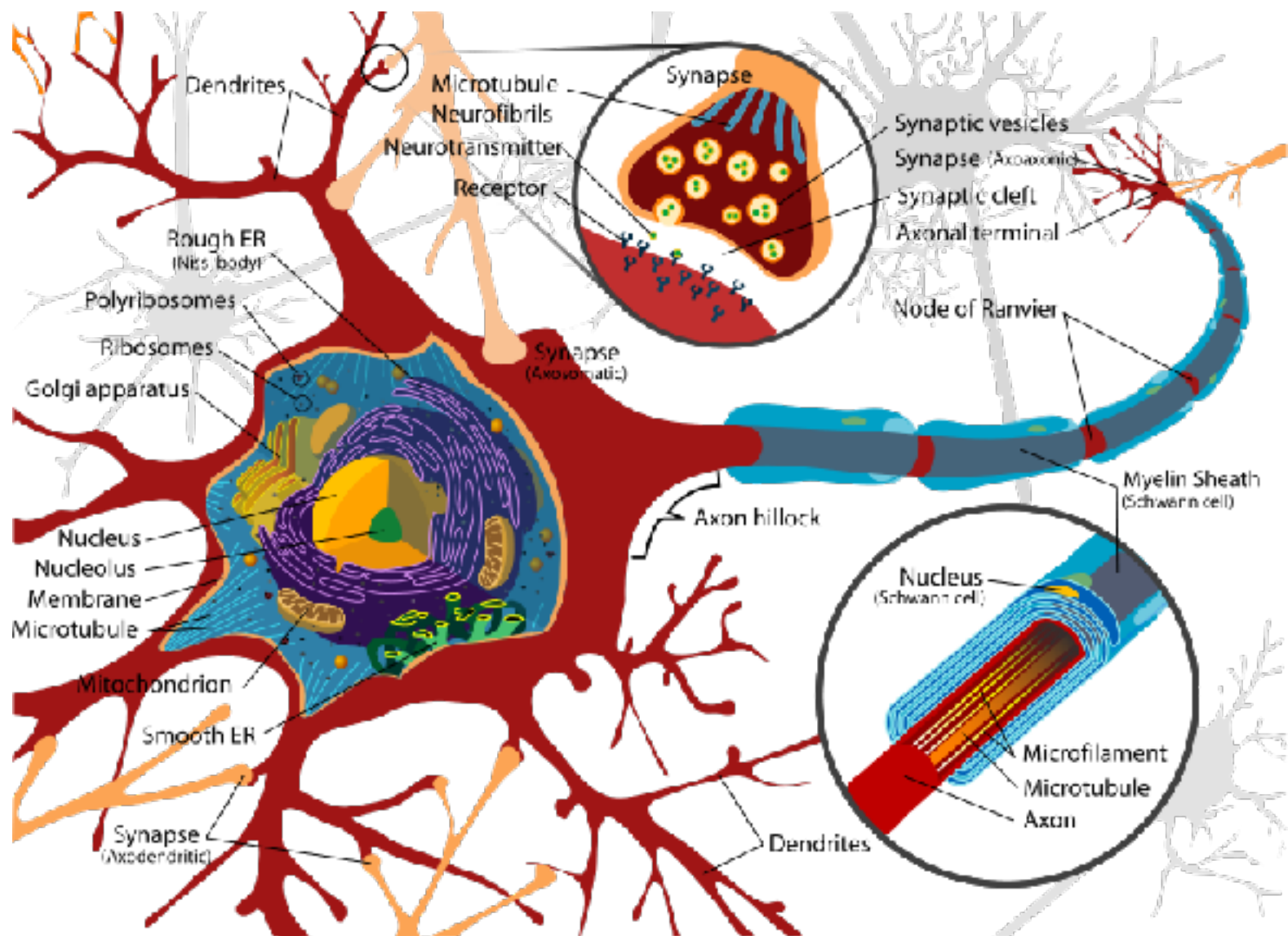
Electromyogr Clin  
Neurophysiol 1992, 32(10-11):  
507-510 Sihvonen, T.

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




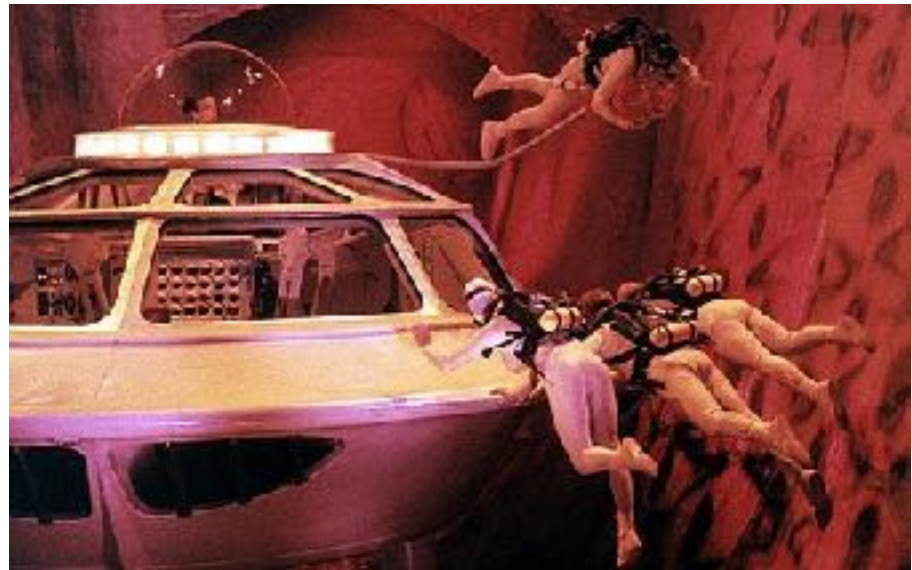
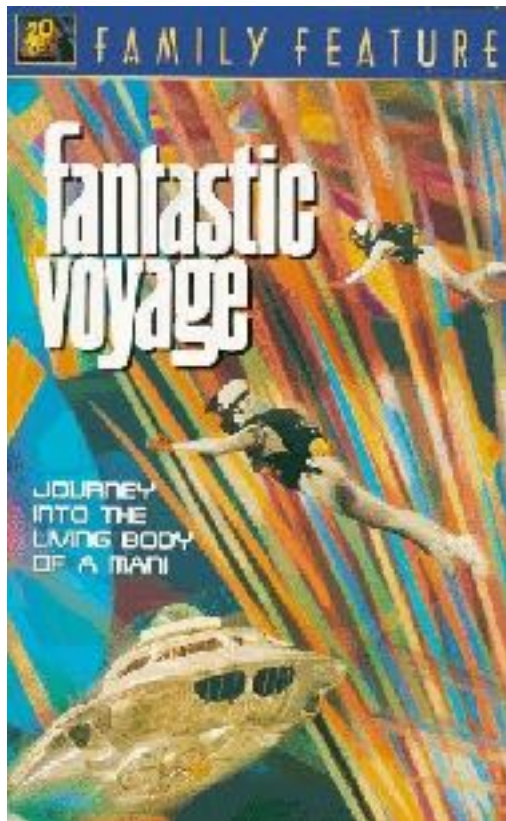
Figure 18-8. Posterior primary ramus. Medial branch of the posterior primary ramus running beneath the mamillo-accessory ligament. This is a possible site for neural entrapment, particularly when this ligamentous structure ossifies in elderly individuals. zj: zygapophyseal joint; tp: transverse process; mp: mammillary process; mal: mamillo-accessory ligament; mb: medial branch; ap: accessory process. (From Bogduk N, Wilson AS, Tynan W: The human lumbar dorsal rami. J Anat 1982; 134:383-397, with permission.)

Dumitru, D. Electrodagnostic 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



1966



# 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 >> Suceptible >> 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

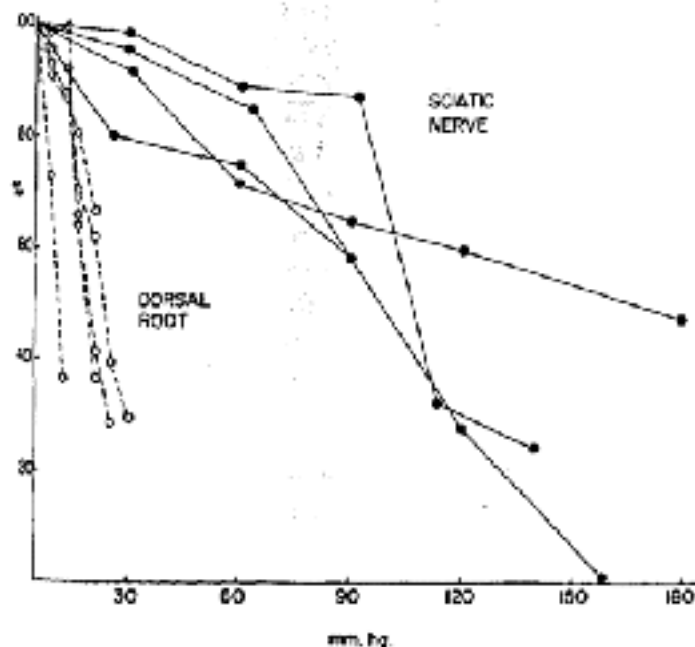
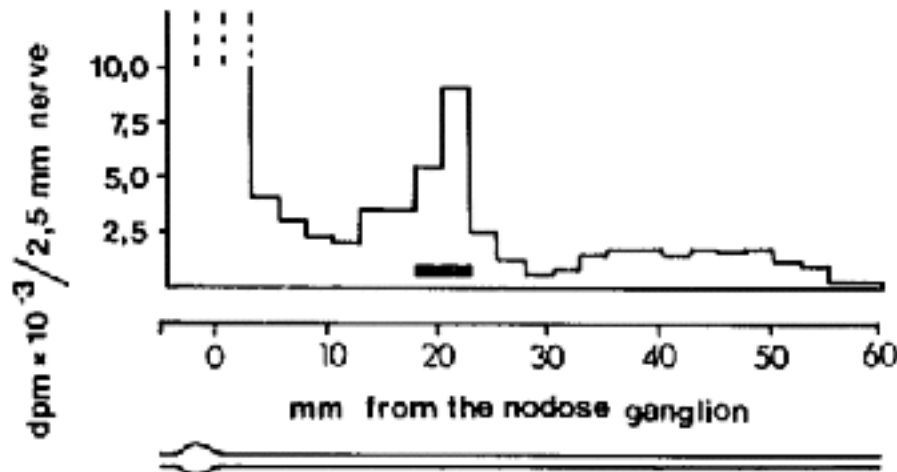


Fig. 2. Susceptibility of spinal roots to compression block. Compound action potentials integrated and expressed as Percent of initial value. Pressures applied for 3 minutes only.

## 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.



**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.<sup>39</sup> Radioactively labelled amino acid (<sup>3</sup>H-leucine) was injected into the nodose ganglion of the nerve. These aminoacids 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 histogram.

**>15% stretch =**

- **intraneural blood flow blocked**
- **electrical conduction block**

**Rydevik SPINE 1984**

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  $\mu$ m. 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.



RF8670 (RMT) © www.visualphotos.com

**(ONLY!!!) 9 degrees axial  
rotation produces  
myelographic filling defect at  
L5  
w/2x normal 20%  
physiological stretch on nerve  
root**

**1983 Farfan**



**Wall Street Journal  
Soccer 'Header' Impact Likened to Hard Punch 2/16/13**



## **Force Absorbed**

$$\mathbf{K.E. = \frac{1}{2} mv^2}$$

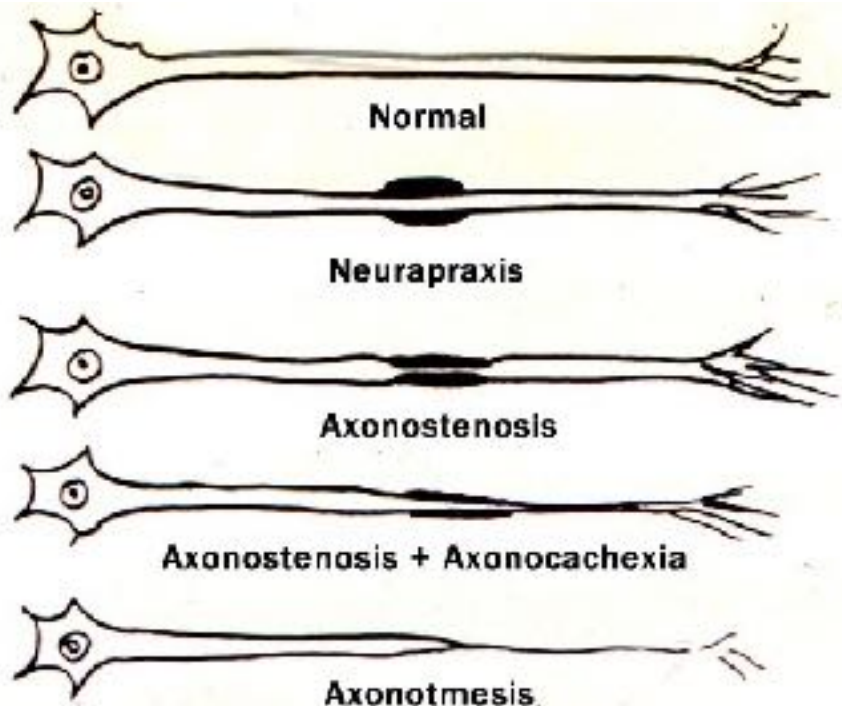
**20 mph = 4 x 10 mph**

**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



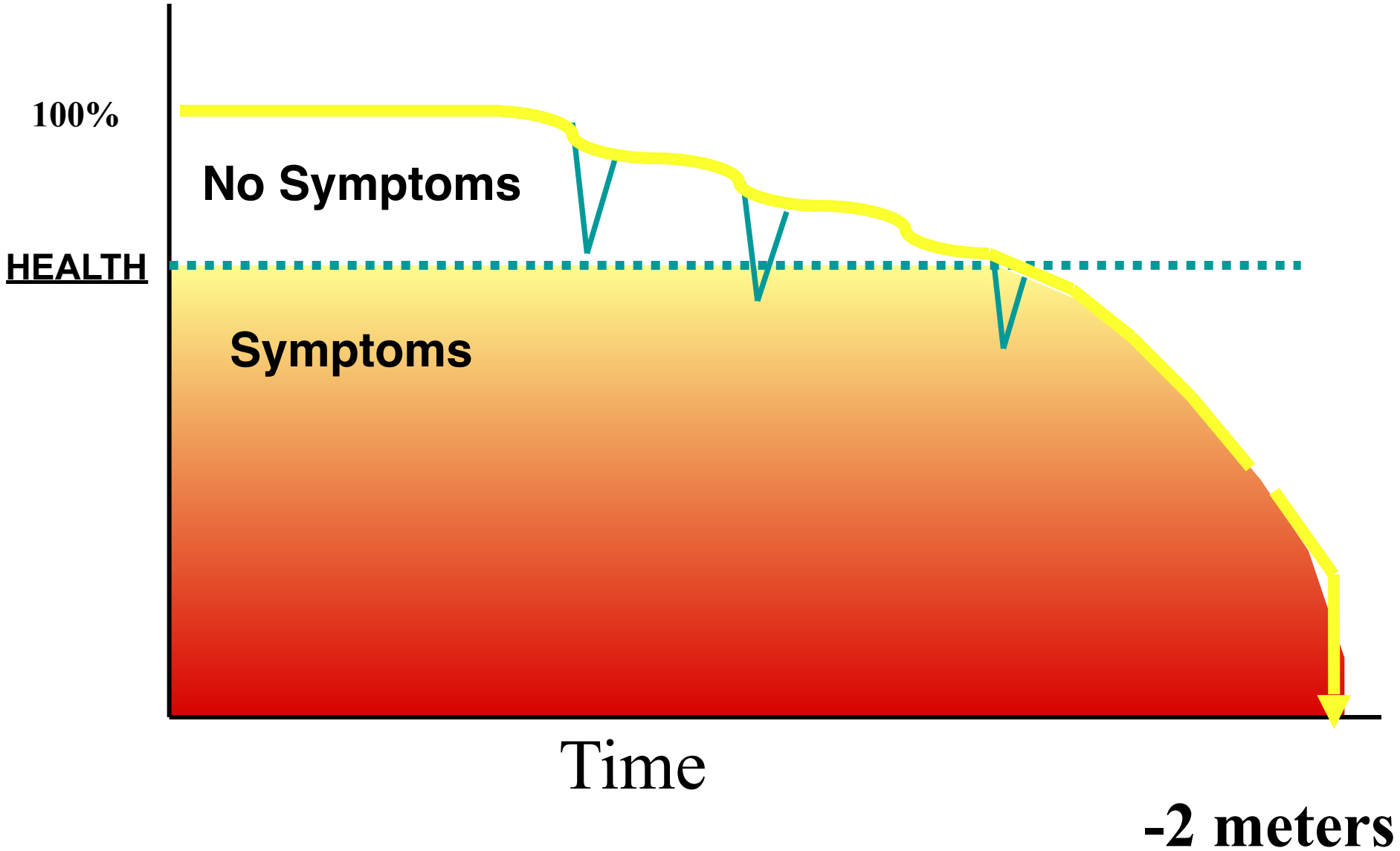
Various conditions in motor neurons  
as a result of  
progressive compression

**CANNON:**  
**DENERVATION > 'A'-TROPHY**

**'PARTIAL DENERVATION', or  
NEUROPATHY > 'DYS'-TROPHY**

# SPONDYLOSIS:

‘Pool of Injured Nerves’ That Accumulate Over Time



# Large Diameter Nerve Fibers More Susceptible Compression > Small Diameter Fibers

440

J. OCHOA, T. J. FOWLER AND R. W. GILLIATT

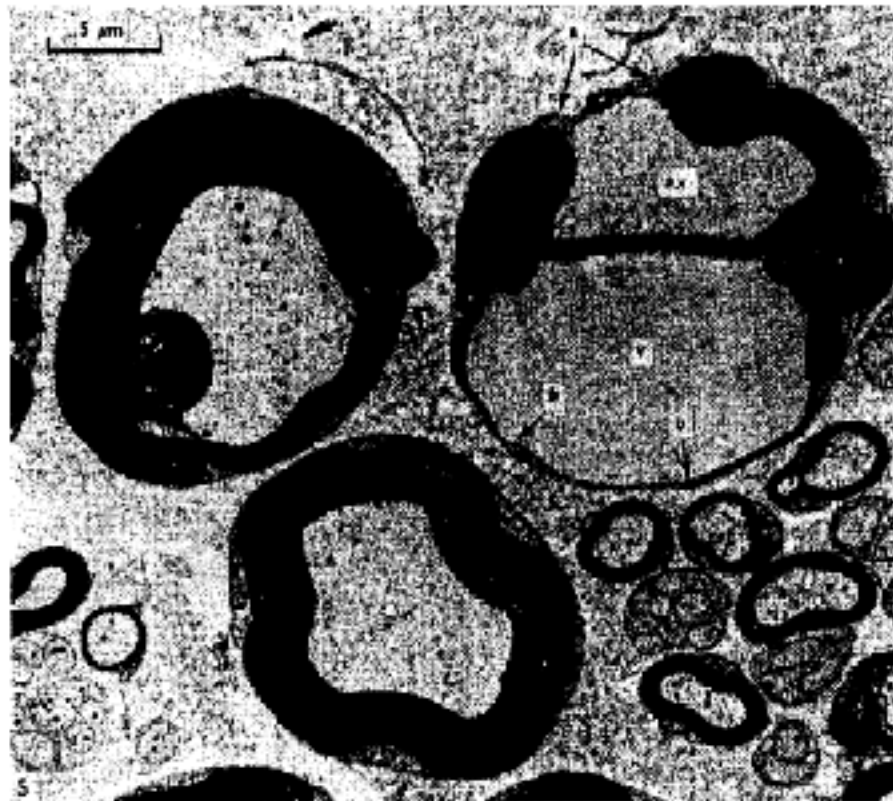


Fig. 5. Transverse section from a nerve, 18 hours after compression, to show myelin rupture and oedema. The fibre on the left shows partial rupture of myelin lamellae. The fibre on the right shows rupture of the whole thickness of the myelin sheath at *a*, the axon (*ax*) being separated from the basement membrane by a thin Schwann cell process. A collection of homogeneous material (*b*) has split the myelin sheath with rupture of the outer lamellae at *b*. Small myelinated and unmyelinated fibres appear normal.

# Large Myelinated Fibers

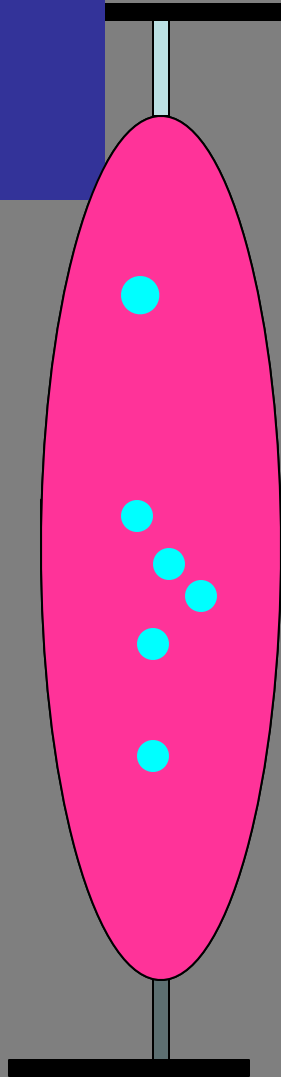
	Fiber Type		Function	Fiber Diameter ( $\mu\text{m}$ )	Conduction Velocity (m/s)
Myelinated	<b>A</b>	<b><math>\alpha</math></b>	<b>Proprioception; Somatic motor</b>	<b>12-20</b>	<b>70-120</b>
		$\beta$	Touch, Pressure	5-12	30-70
		$\gamma$	Motor to muscle spindles	3-6	15-30
		$\delta$	Pain, Temperature, Touch	2-5	12-30
	<b>B</b>		Preganglionic sympathetics	< 3	3-15
Unmyelinated	<b>C</b>	<i>Dorsal root</i>	Pain; Reflex responses	0.4-1.2	0.5-2
		<i>Sympathetic C Fibers</i>	Postganglionic sympathetics	0.3-1.3	0.7-2.3

# Cannon's Law & Neuropathic Muscle

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

**Neuropathic =  
Sick Nerve**

**Trophic  
edema  
subcut**



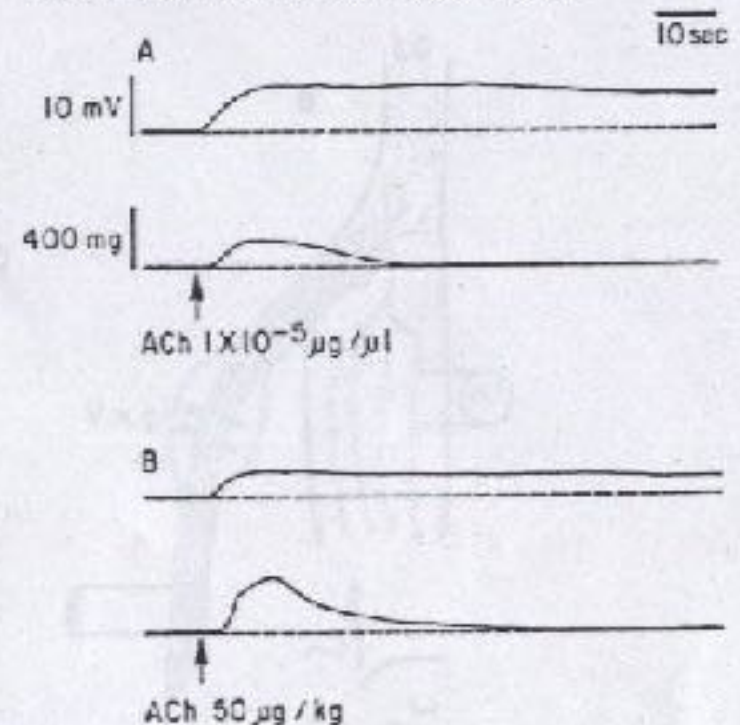
# Muscle CONTRACTURE vs. Muscle CONTRACTION

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

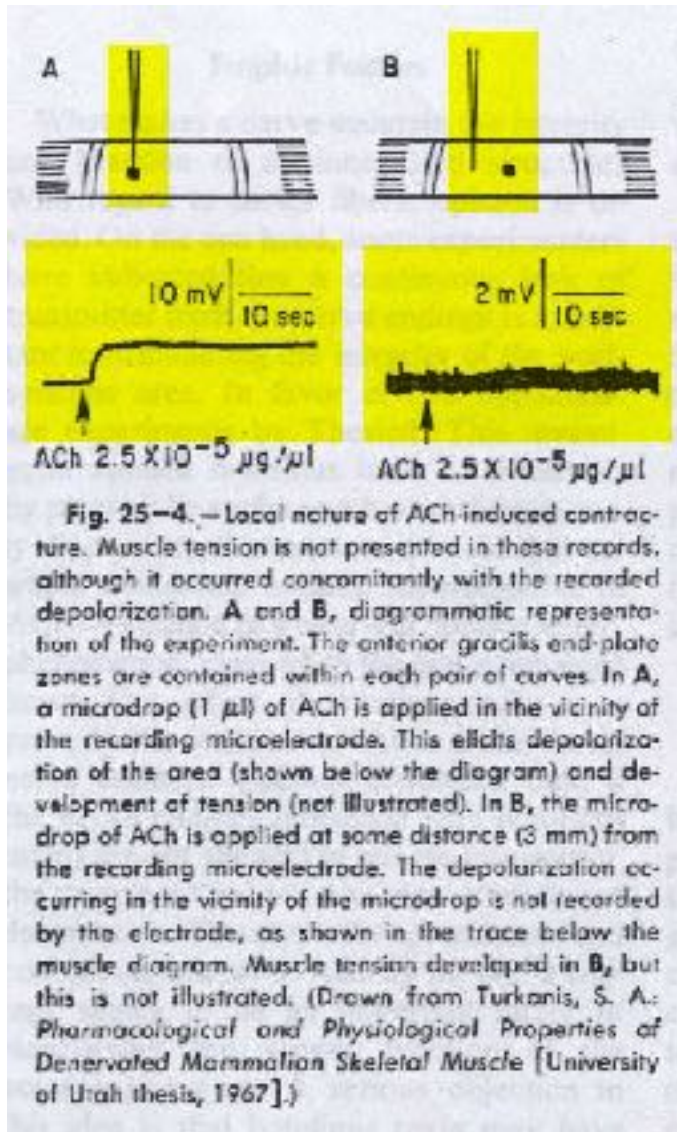
Eyzaguirre C, Fidone SJ 1975 Physiology of the nervous system. Chicago, Year Book Medical Publishers. 2nd ed.

**Fig. 25-3.** — Effects of ACh on denervated muscle; anterior gracilis of a rat denervated 24 days before the experiment. In **A** and **B** the upper traces represent depolarization recorded by a small electrode (about  $1\ \mu$ ) applied to the surface of the muscle; the lower traces represent the tension developed by the muscle. In **A**, a small drop ( $1\ \mu$ ) of ACh applied to the muscle surface in the vicinity of the recording electrode elicits muscle depolarization (upper trace) and development of tension (lower trace). In **B**, similar effects are induced by an intra-arterial injection of ACh. (Drawn from Turkonis, S. A.: *Pharmacological and Physiological Properties of Denervated Mammalian Skeletal Muscle* [University of Utah thesis, 1967].)



# Local Nature of Ach-Induced Contracture

Leads to Focal  
Taut Bands with  
**MUSCLE  
TENSION**

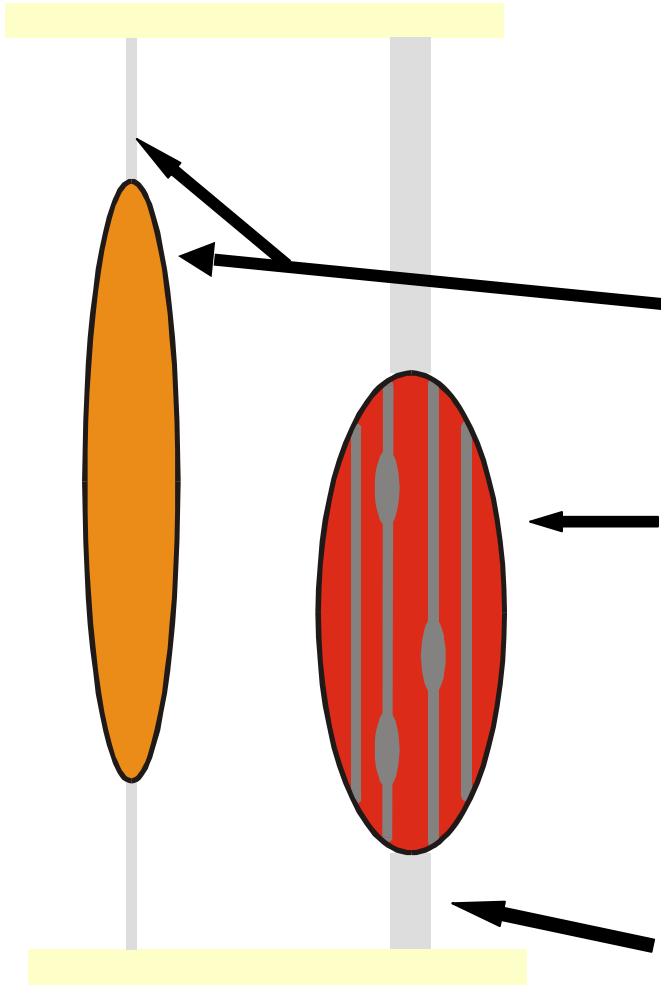


# Neuropathic Muscle = Shortened Muscle

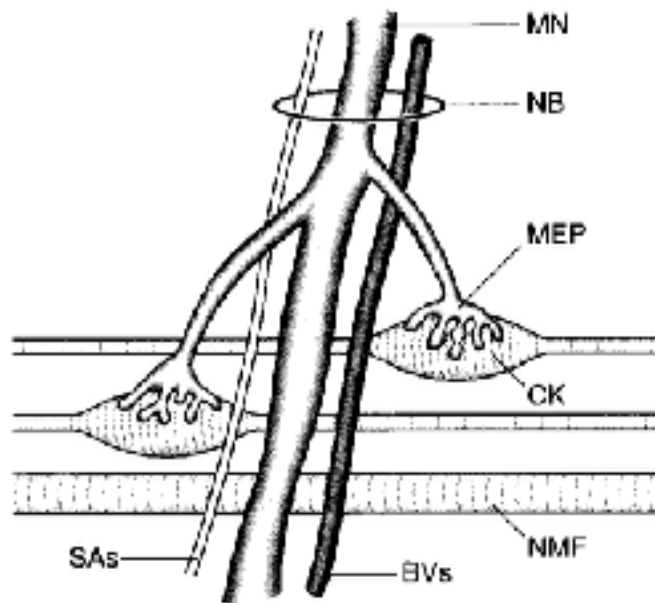
## Normal Muscle & Tendon

## Muscle Contracture

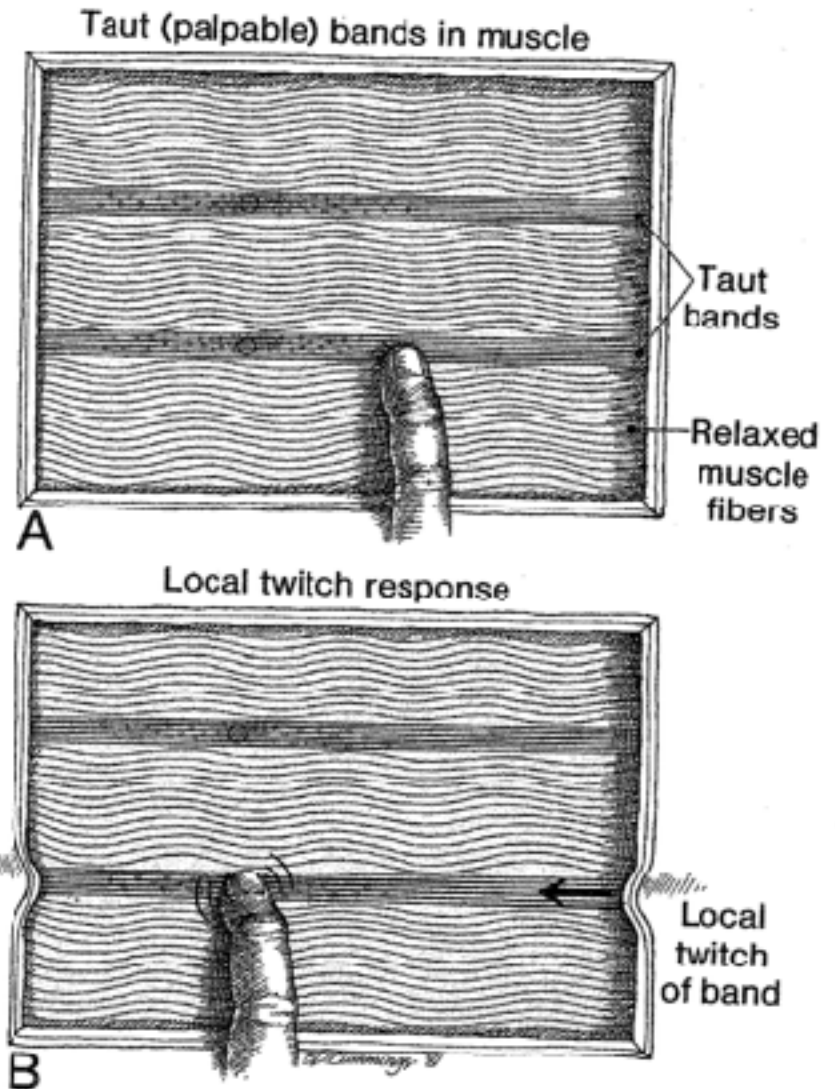
- Taut Bands  
Spasm, Decreased ROM
- Altered Biochemistry >  
Myalgic Hyperalgesia  
Referred Pain
- Tension on Tendons =  
Tendonosis, -opathy



# The Trigger Point

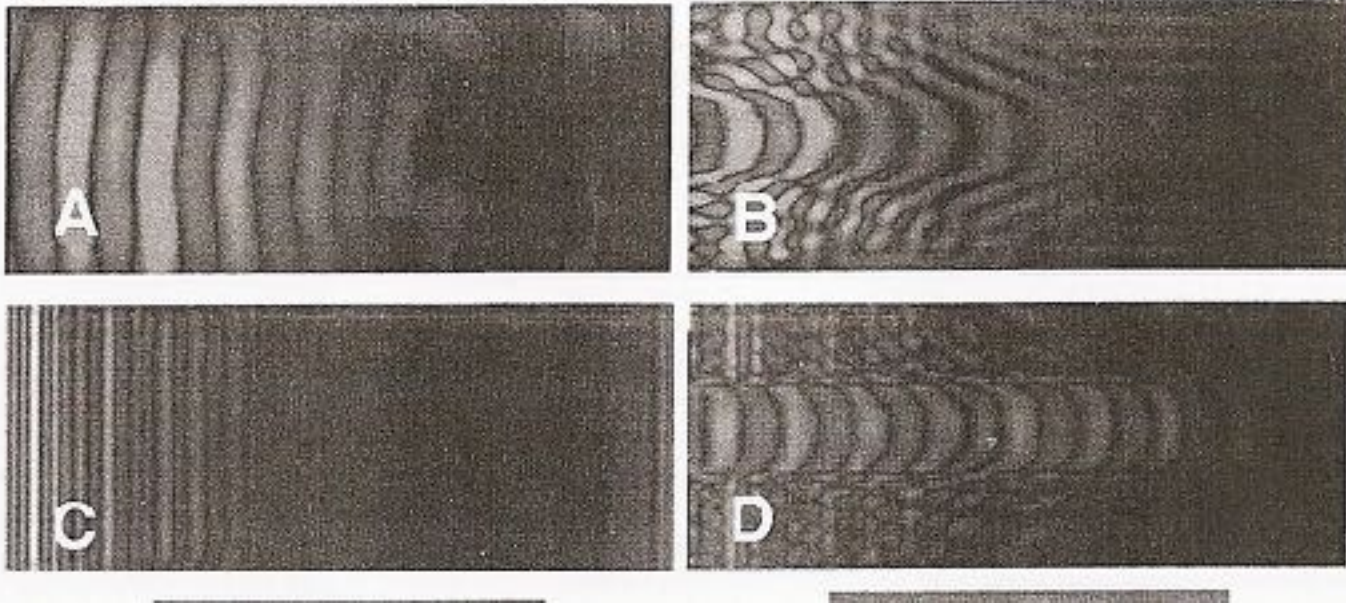


**Figure 4.2** Diagrammatic representation of part of a myofascial trigger point showing two motor endplates (MEPs) and juxta-positional contraction knots (CKs); also a neurovascular bundle (NB) containing motor nerves (MNs), nociceptive and proprioceptive sensory afferents (SAs) and blood vessels (BVs) with closely associated sympathetic fibres. Note: in normal muscle fibre (NMF) the sarcomeres are of equal length. In a muscle fibre containing a contraction knot there is shortening of the sarcomeres at that site and compensatory lengthening of them on either side.



**Baldry, PE. Myofascial Pain and Fibromyalgia – A Clinical Guide to Diagnosis and Management. Churchill Livingstone**

MYOFASCIAL TAUT BANDS, Chen

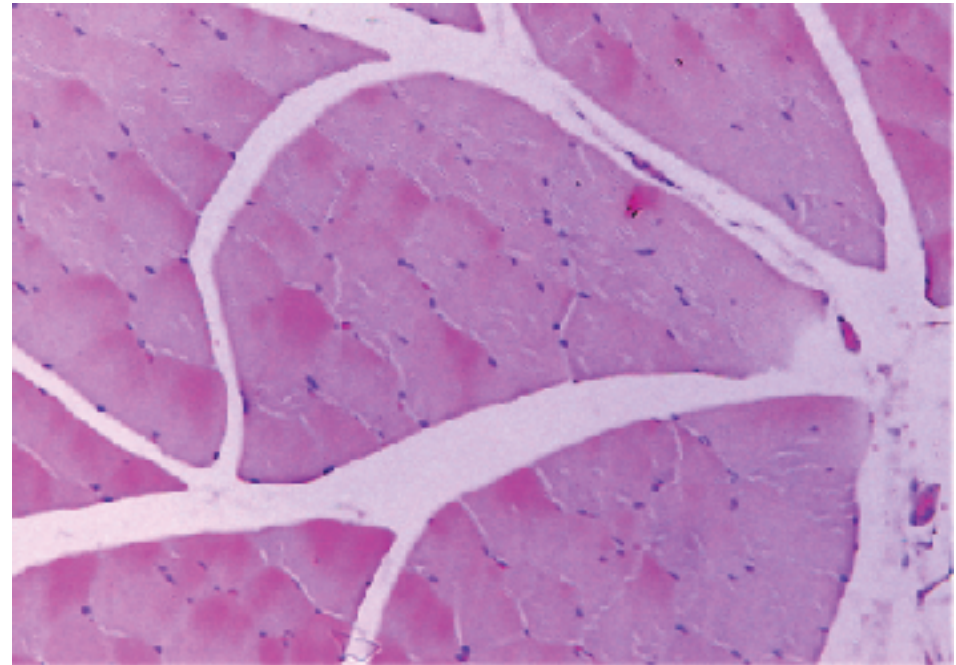
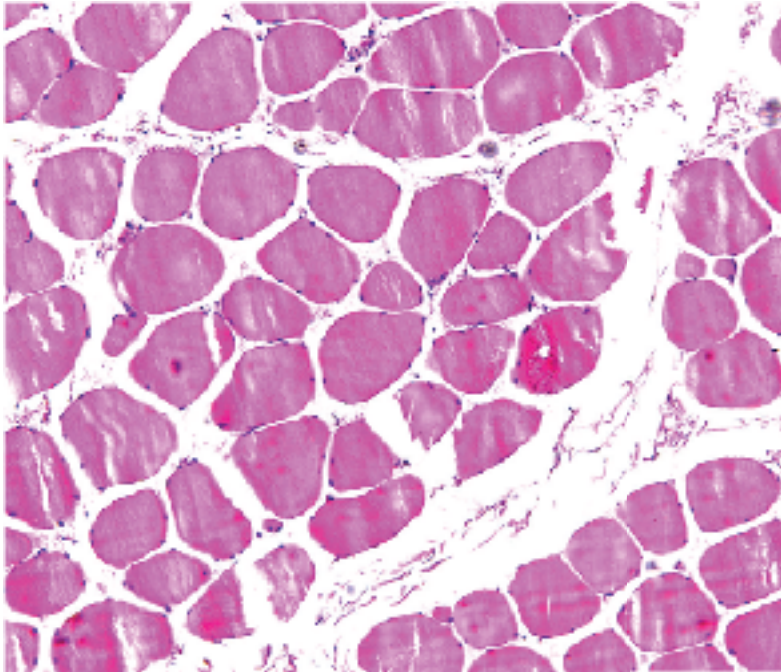


# Identification & Quantification of Myofascial Taut Bands with Magnetic Resonance Elastography

Archives of Physical Medicine & Rehabilitation

Chen, Q., et.al. Vol 88 December 2007 p.1658-1661

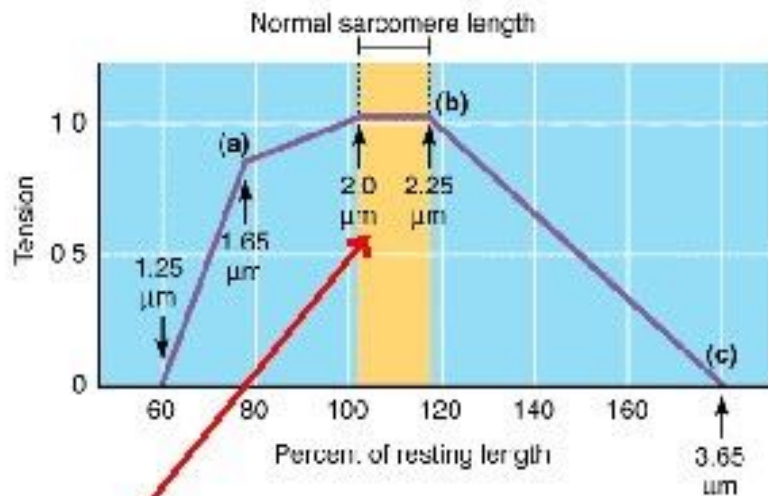
**Morphological findings of representative skeletal muscles with nontaut and taut bands. (a) Biceps femoris with a nontaut band; (b) Biceps femoris with a taut band (H&E staining, scale bar = 5  $\mu$ m)**



**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**

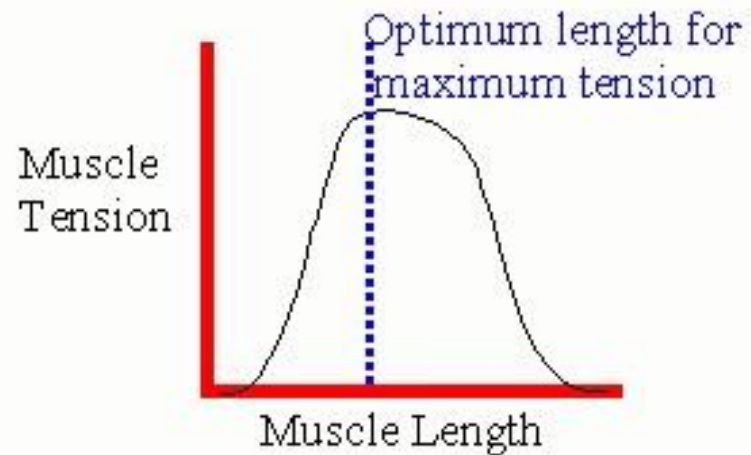
# Relationship of Tension to Muscle Length.

## The Sarcomere



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

## Whole Muscle



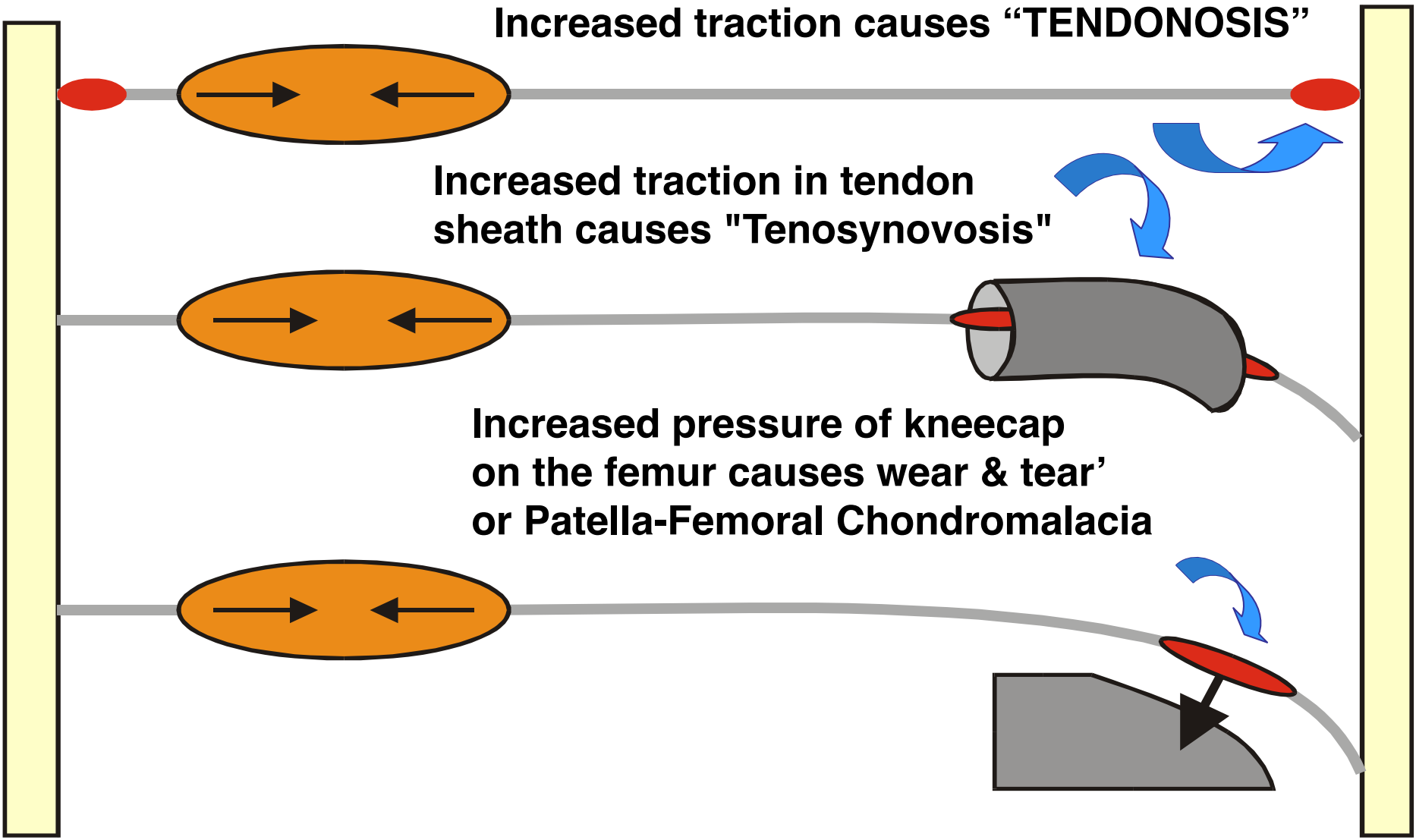
This applies to the entire muscle as well as to individual sarcomeres.

# Secondary Effects of Short Muscle Syndrome

Increased traction causes "TENDONOSIS"

Increased traction in tendon sheath causes "Tenosynovitis"

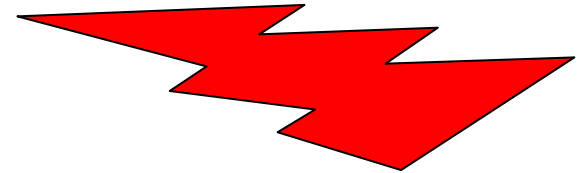
Increased pressure of kneecap on the femur causes wear & tear' or Patella-Femoral Chondromalacia



# Shortened Muscle



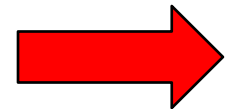
**Arthralgia &  
Osteoarthritis**



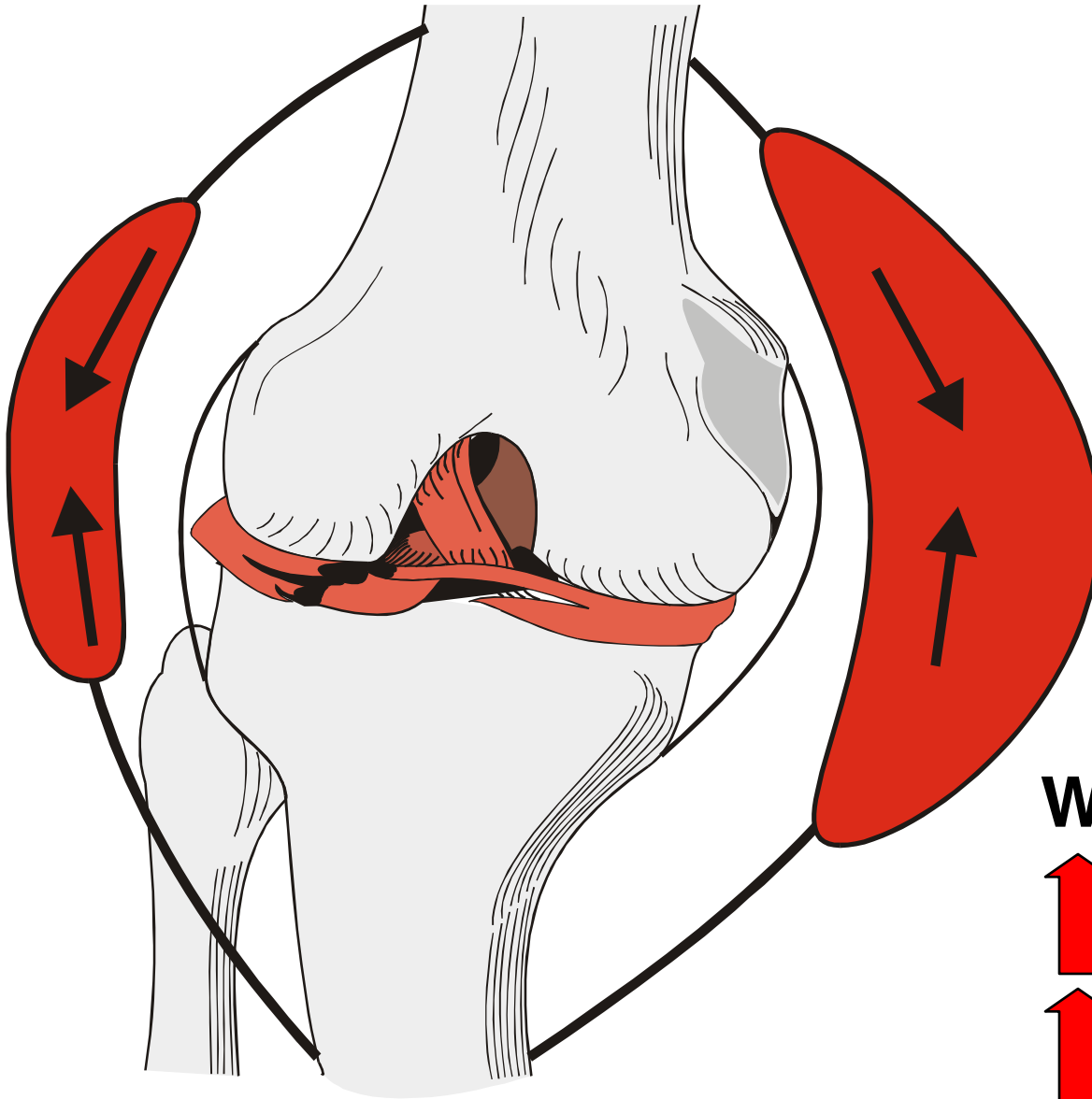
**Wolf's Law:**



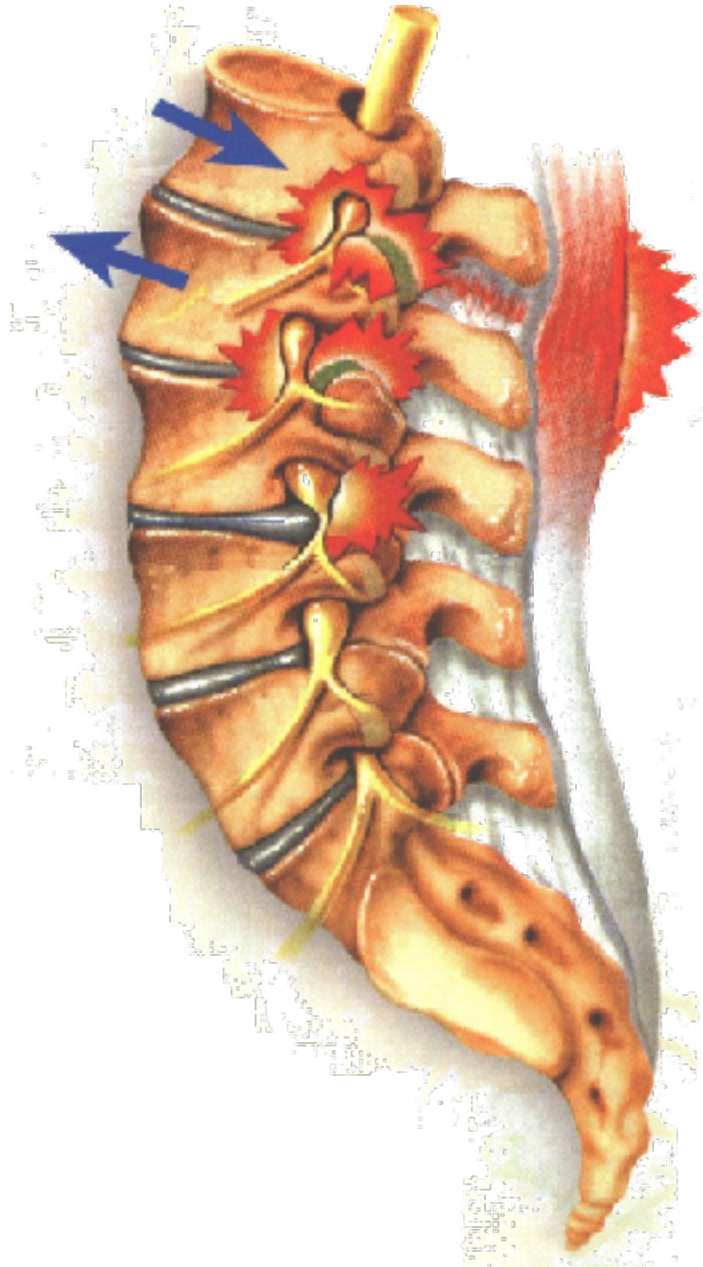
**Tension**



**Bone = 'Spurs'**



# Paraspinal Muscle Shortening



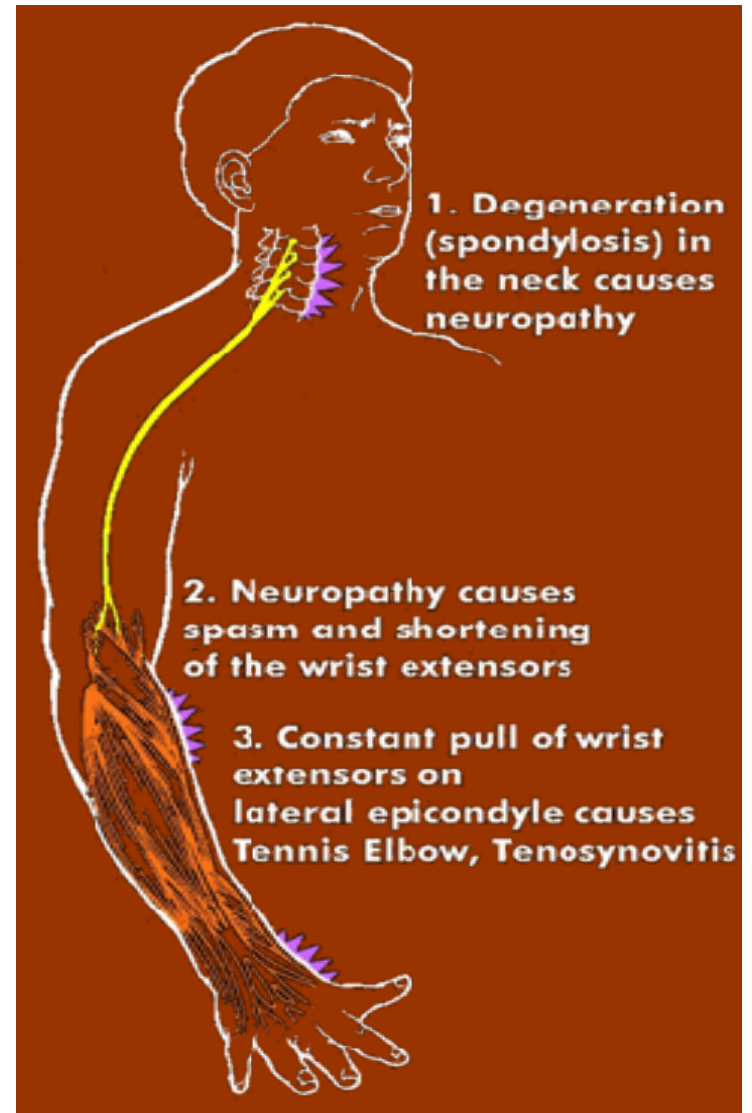
**Shortened  
muscles between  
vertebrae increase  
pressure on disc,  
nerve and facet  
joints**

# **Spondylosis > Neuropathy > Muscle Shortening > Tendinosis Syndromes**

**Spinal Wear & Tear →  
Radiculo-Neuropathy**

**Neuropathy → Muscle  
Shortening**

**Shortening → ↑ Tension  
on Tendon = Tendinosis**



# **‘SHORTENED MUSCLE’ SYNDROMES**

<b>SHORTENED MUSCLE</b>	<b>SYNDROME</b>
<b>Gluteal, Piriformis Hamstring</b>	<b>Pseudo’ Sciatica, Gluteal/Ischial Bursitis, Piriformis Syndrome</b>
<b>Supra - Infraspinatus, Teres Major, Subscap</b>	<b>Rotator Cuff Tendons, Impingement</b>
<b>Extensor Carpi Radialis Longus and Brevis</b>	<b>Lateral Epicondylitis Tennis Elbow</b>
<b>Quadriceps, Adductors</b>	<b>Patellofemoral Pain Syndrome Runner’s Knee, Medial Knee Pain</b>
<b>Gastroc-soleus</b>	<b>Achilles Tendons</b>
<b>Neck &amp; Back Muscles</b>	<b>Cervicalgia, Lumbar Pain Facet Syndrome</b>
<b>Intrinsic Foot</b>	<b>Plantar Fasciitis, Heel Pain</b>
<b>TFL, Iliotibial Band</b>	<b>IT-Band, Knee Pain</b>

# Tenodonopathy NOT Tendonitis

**Khan KM, Cook JL, Bonar F, Harcourt P, Åstrom M Histopathology of common tendonopathies. Update and implications for clinical management. Sports Med 1999 27: 393-408**

**Khan KM, Cook JL, Kannus P, Maffulli N, Bonar SF. Time to abandon the ‘tendonitis’ myth: painful overuse tendon conditions have a non-inflammatory pathology. BMJ 2002 324: 626-27**

**Fredberg U, Stengaard-Pedersen K. Chronic tendinopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. Scand J Med Sci Sports 2008 18: 3–15**

**Jonsson P, Alfredson H, Sunding K, Fahlstrom M, Cook J. New regimen for eccentric calf-muscle training in patients with chronic insertional Achilles tendinopathy: results of a pilot study. Br J Sports Med 2008 42:746–49**

**Allison GT, Purdam C. Eccentric loading for Achilles tendinopathy – strengthening or stretching? Br J Sports Med 2009 43:276-79**

# Myofascial Pain is a Type of Neuropathic Pain

## ‘Evidence of Neuroaxonal Degeneration in MFPS’

### Chang, CW. 2008, Europ Jrnal of Pain

Table 1

Values of mean consecutive difference (MCD) obtained in patients with myofascial pain syndrome (MPS) and normal controls

Muscles	Subject numbers	MCD ( $\mu$ s)	HNL MCD ( $\mu$ s)	Abnormal MCD (%)
Normal controls	16			
Trapezius	8	$32.9 \pm 7.1^a$	47.1	0
Levator scapulae	8	$36.2 \pm 6.4^b$	49.0	0
MPS patients	23			
Trapezius	15	$61.5 \pm 11.1^a$		74.3
Levator scapulae	8	$59.2 \pm 10.9^b$		70.7

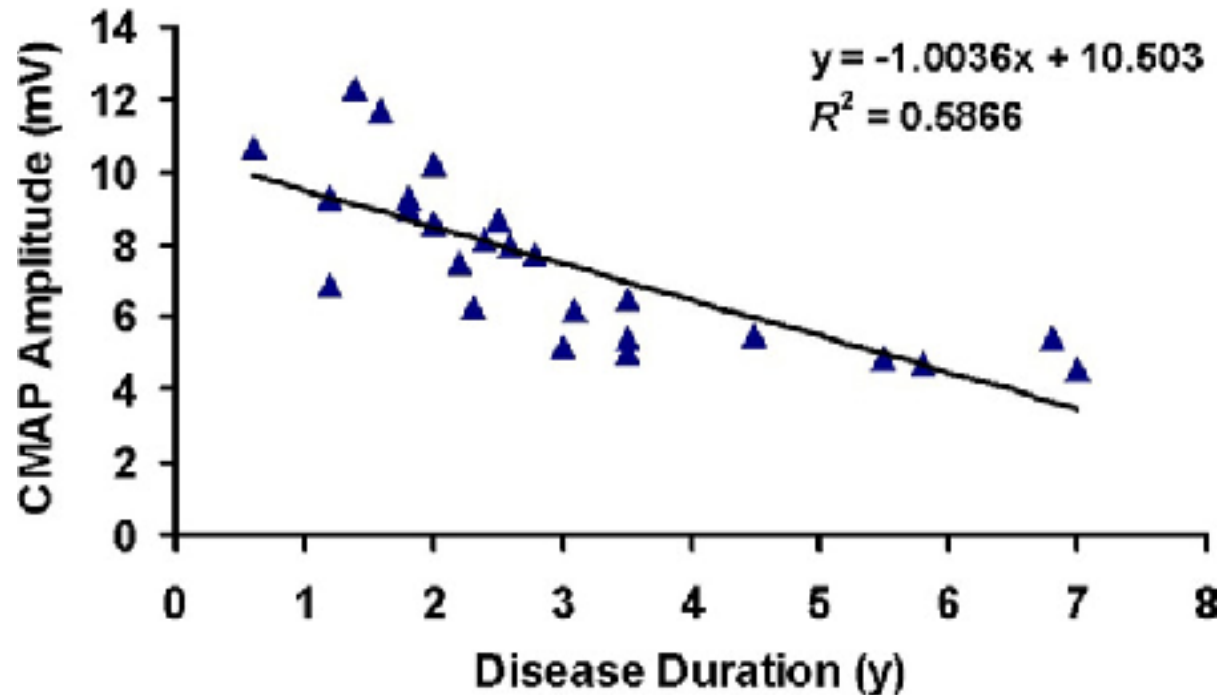
Values are mean  $\pm$  SD.

HNL: highest normal limit.

For comparison of values with the same letter:  $^{a,b}p < 0.01$  by Student's *t*-tests.

# Electrophysiologic Evidence of Spinal Accessory Neuropathy in Patients With Cervical Myofascial Pain Syndrome

Chang, et. al. Arch Phys Med Rehabil Vol 92, June 2011

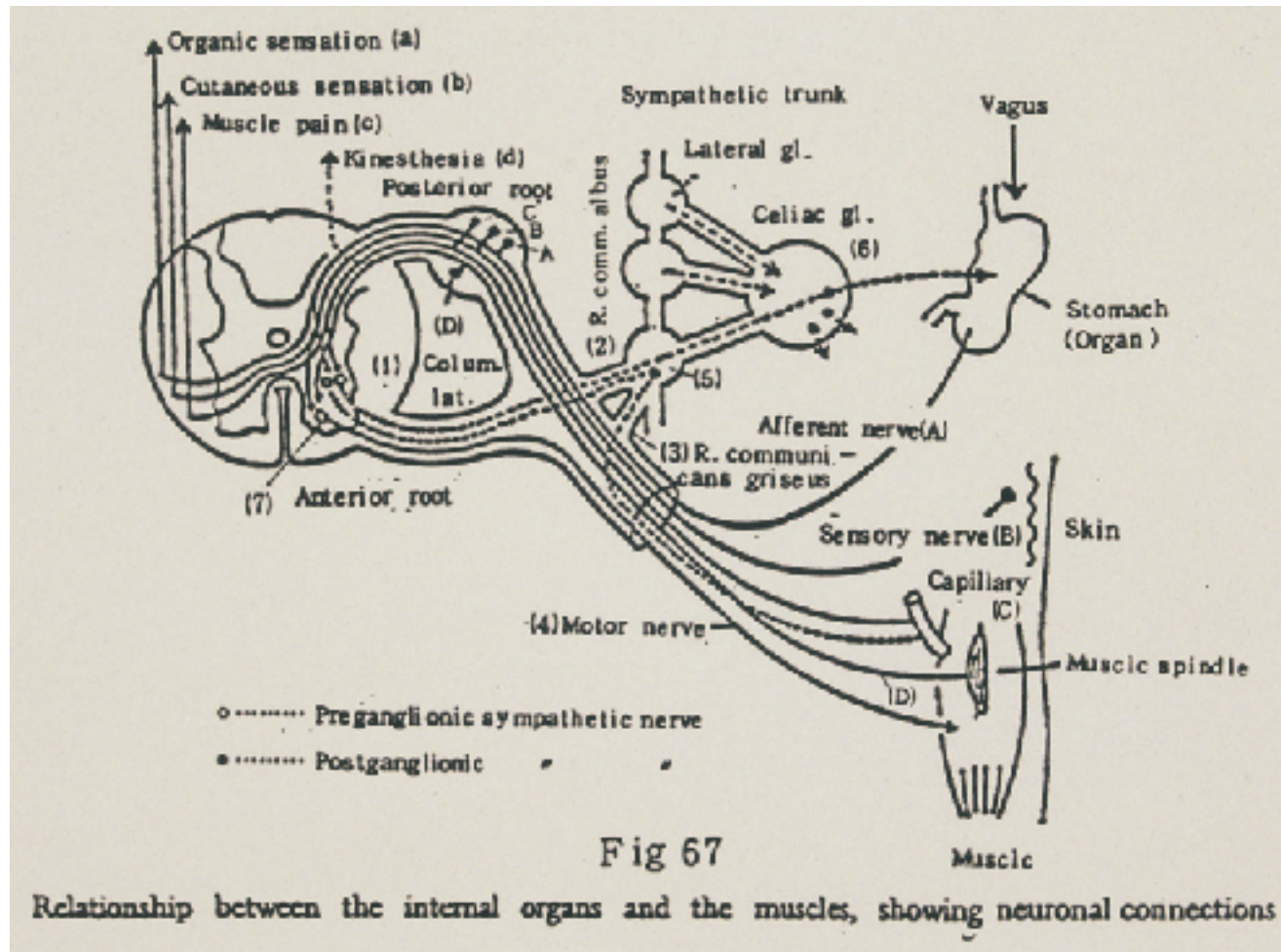


**Fig 2. Relation between CMAP amplitude and disease duration in patients with cervical MFPS.**

# **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**
- **vasoconstriction > cool to touch**  
**sudomotor > hyperhidrosis**  
**pilomotor > gooseflesh, hair loss**

# Neuroanatomy explains association of MFPS and Autonomically mediated symptoms



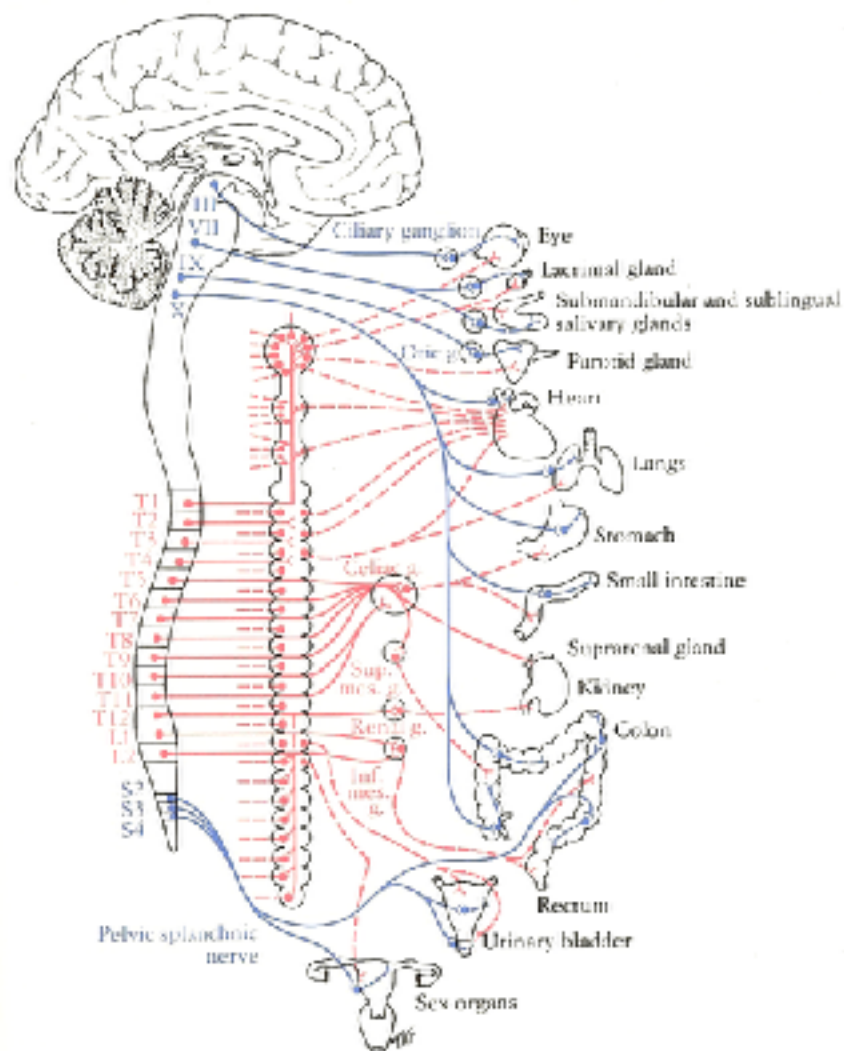
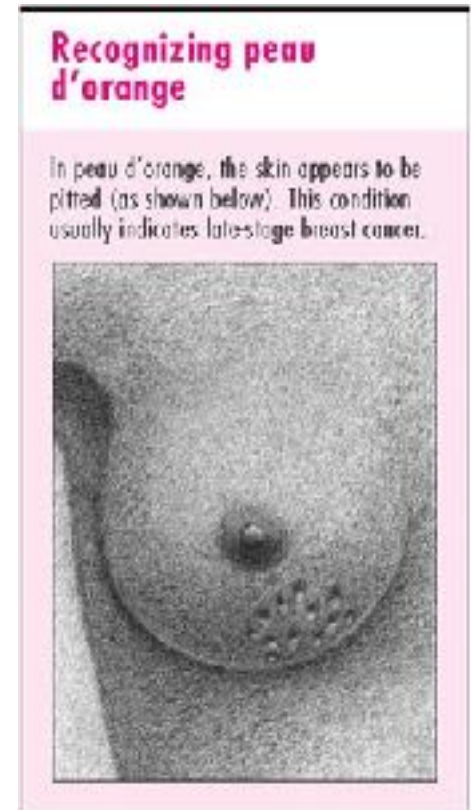
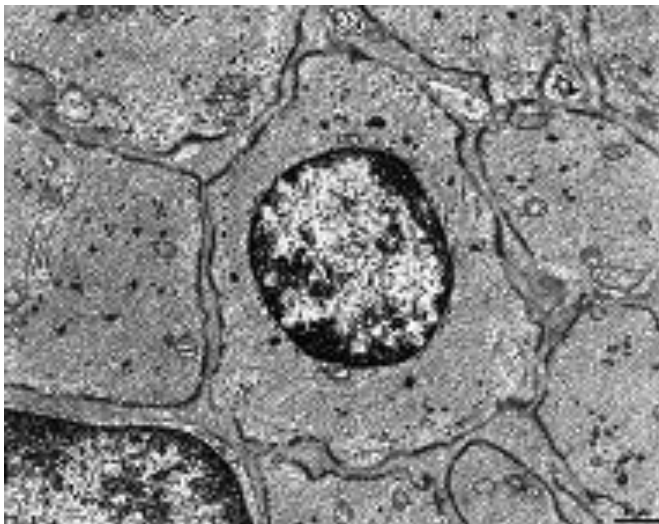


Fig. 28-2. Efferent part of autonomic nervous system. Preganglionic parasympathetic fibers are shown in solid blue, postganglionic parasympathetic fibers in interrupted blue. Preganglionic sympathetic fibers are shown in solid red, postganglionic sympathetic fibers in interrupted red.

# Autonomic Neuropathy > Neurogenic Edema: 'Peau d'Orange' Effect

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

→ ↑ opening of cell gap  
→ leakage = 'Trophic' Edema



**A Novel Microanalytical Technique for Assaying Soft  
Tissue Demonstrates Significant Quantitative  
Biochemical Differences in 3 Clinically Distinct  
Groups: Normal, Latent, and Active. 2003**  
**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. nuclei, 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!”**

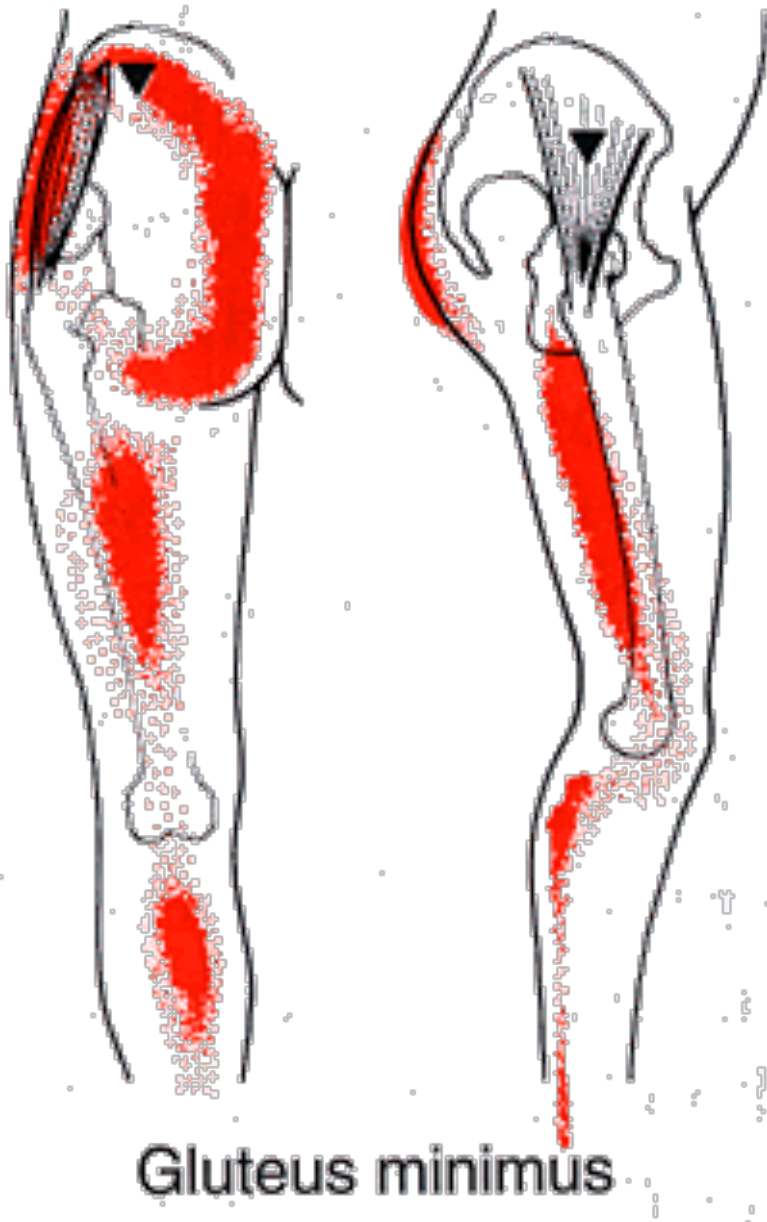
**“When all else fails,  
examine the patient.”**

**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



# **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 Radiculo-Neuropathy



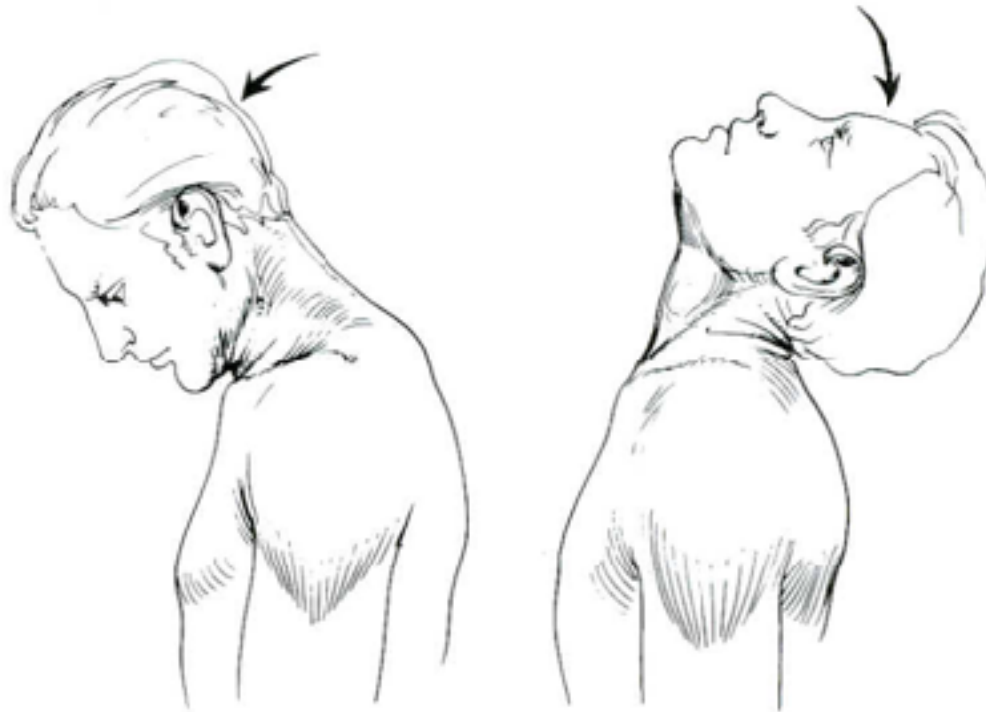
# **Dermatomal Hairloss Secondary to S1 Radiculo-Neuropathy**



# Muscle Contracture with Shortening > Postural deviation

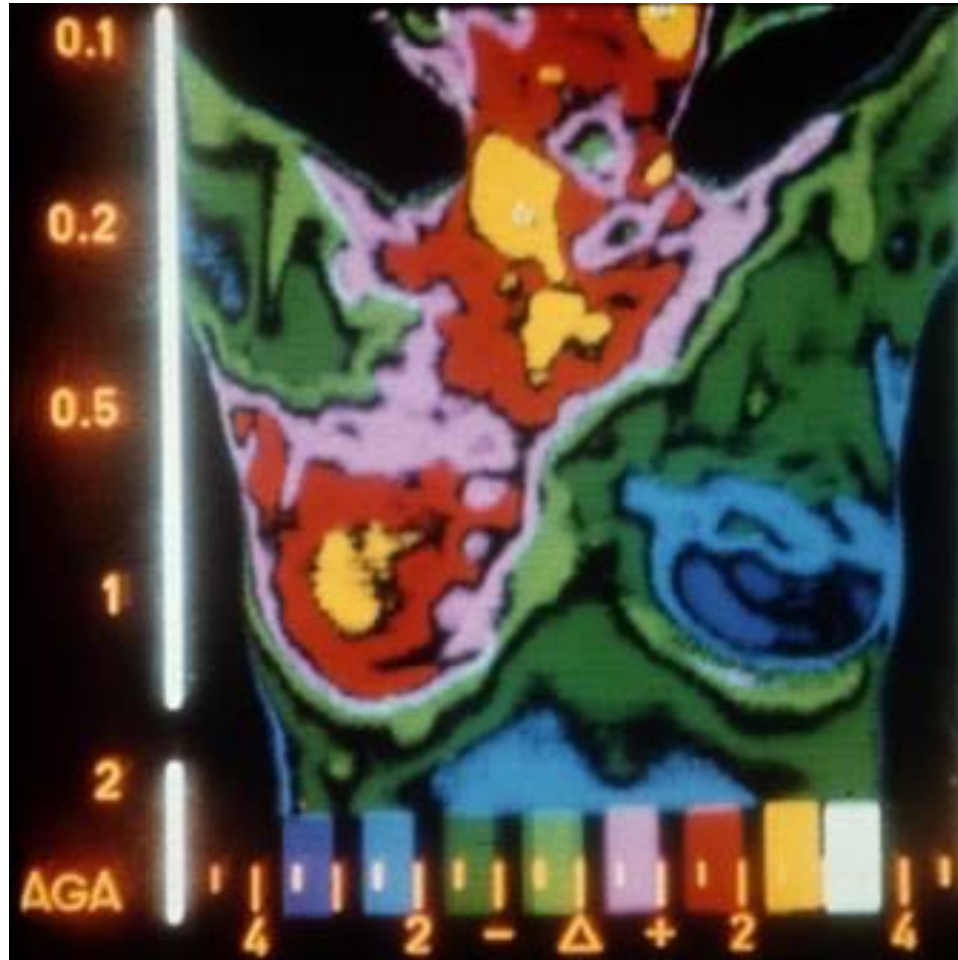


# MUSCLE SHORTENING ➡ DECREASED RANGE of MOTION



**Fig. 24. Left.** Normal range of neck flexion. **Right.** Normal range of neck extension.

**Neuropathy → Vasoconstriction:  
Cool to Touch Segmentally**



# LYMPHATIC & VASCULAR SMOOTH MUSCLE CONTRACTURE ➡ TROPHIC EDEMA





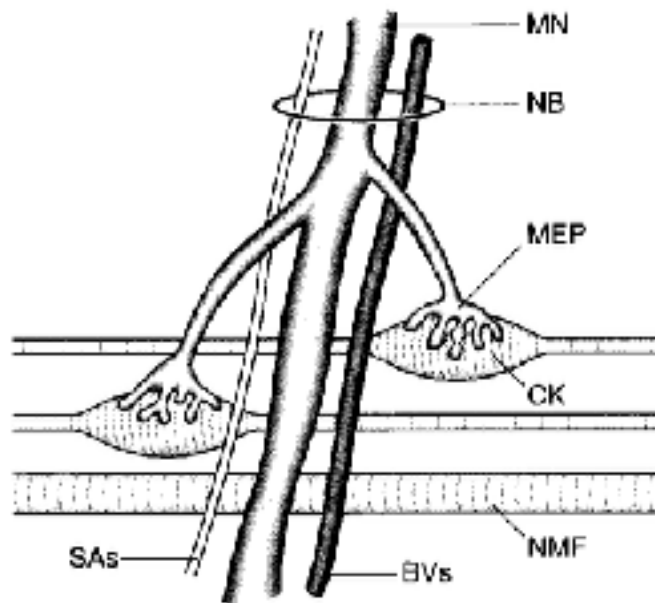
# Trophic Edema in the Lower Inner Leg



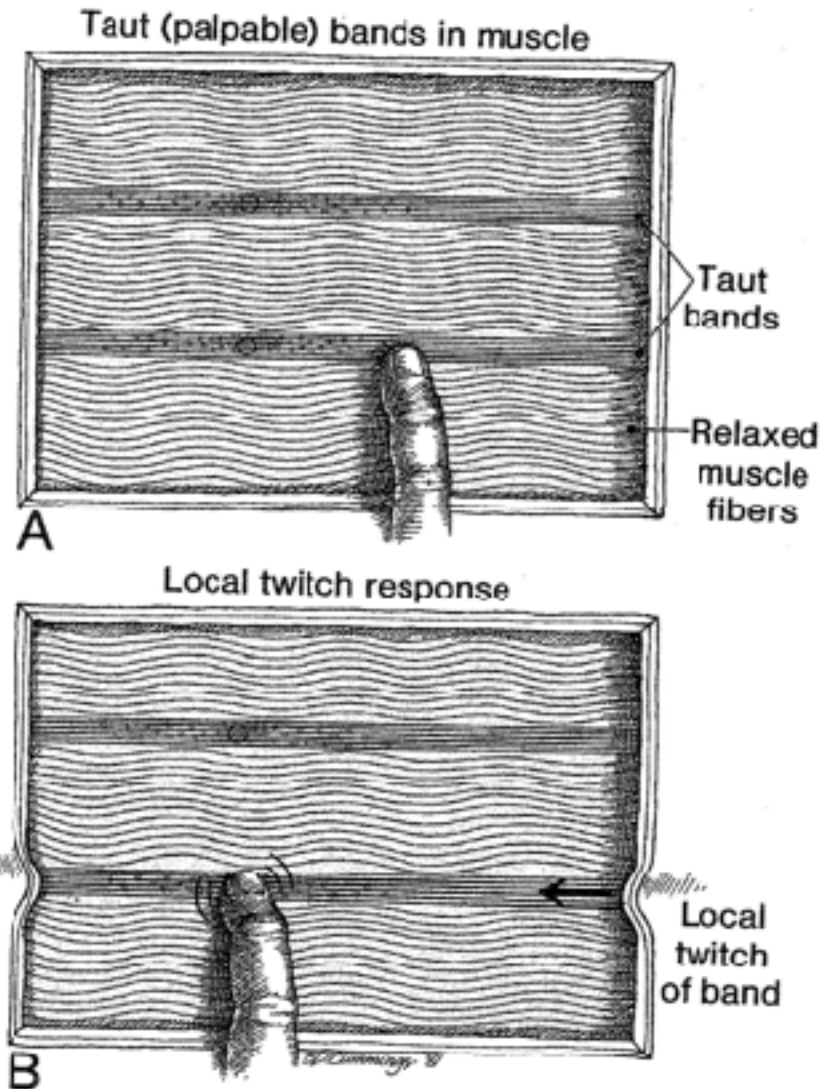
# **Cervical Edema + DDD = ‘Turkey Neck’**



# The Trigger Point

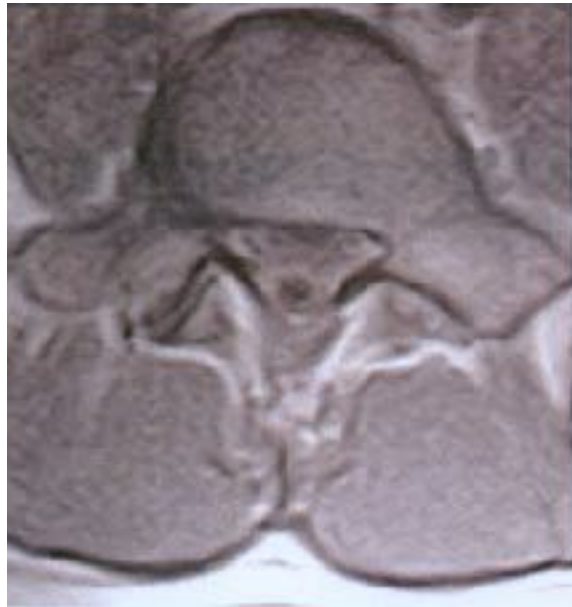


**Figure 4.2** Diagrammatic representation of part of a myofascial trigger point showing two motor endplates (MEPs) and juxta-positional contraction knots (CKs); also a neurovascular bundle (NB) containing motor nerves (MNs), nociceptive and proprioceptive sensory afferents (SAs) and blood vessels (BVs) with closely associated sympathetic fibres. Note: in normal muscle fibre (NMF) the sarcomeres are of equal length. In a muscle fibre containing a contraction knot there is shortening of the sarcomeres at that site and compensatory lengthening of them on either side.

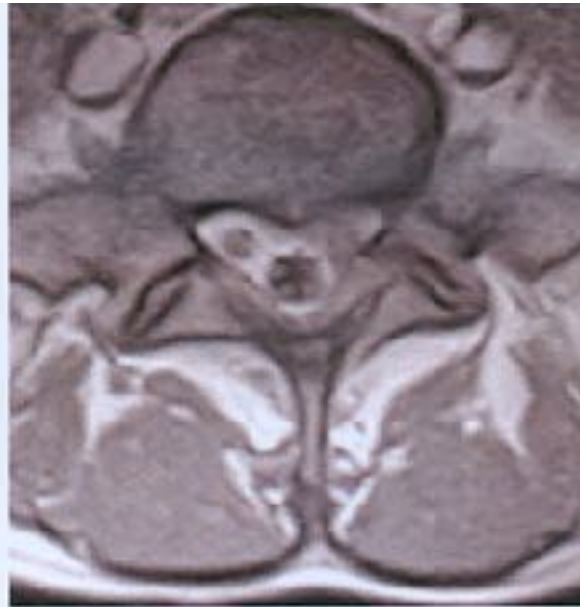


**Baldry, PE. Myofascial Pain and Fibromyalgia – A Clinical Guide to Diagnosis and Management. Churchill Livingstone**

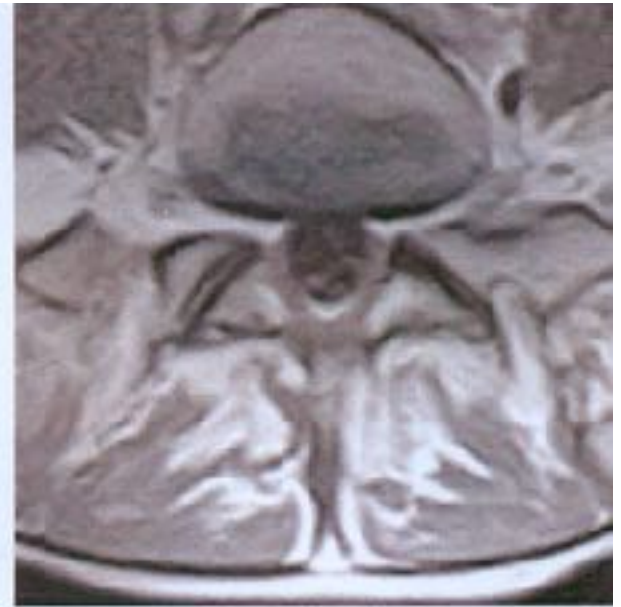
# Are MRI-defined fat infiltrations in the multifidus muscles associated with low back pain? (YES)



Grade 0 (none)



Grade 1 (slight)

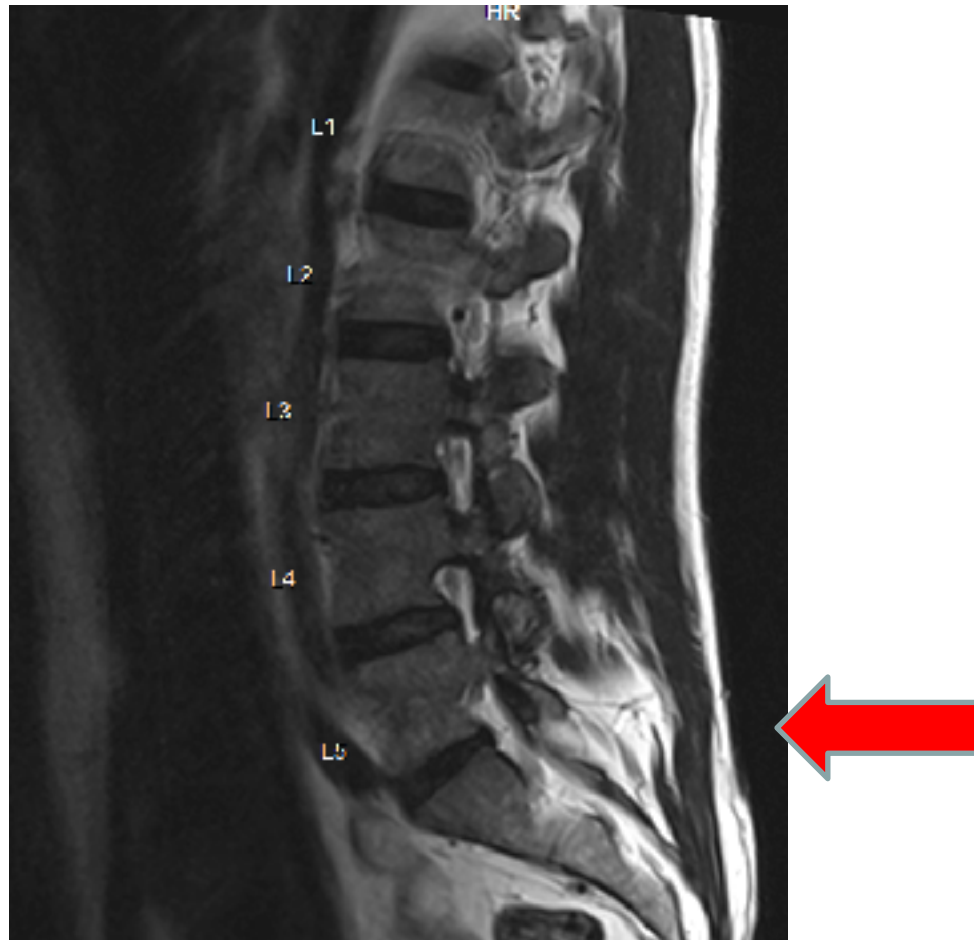


Grade 2 (severe)

**BMC Med. 2007 Jan 25;5:2. Kjaer, P., et. Al.**

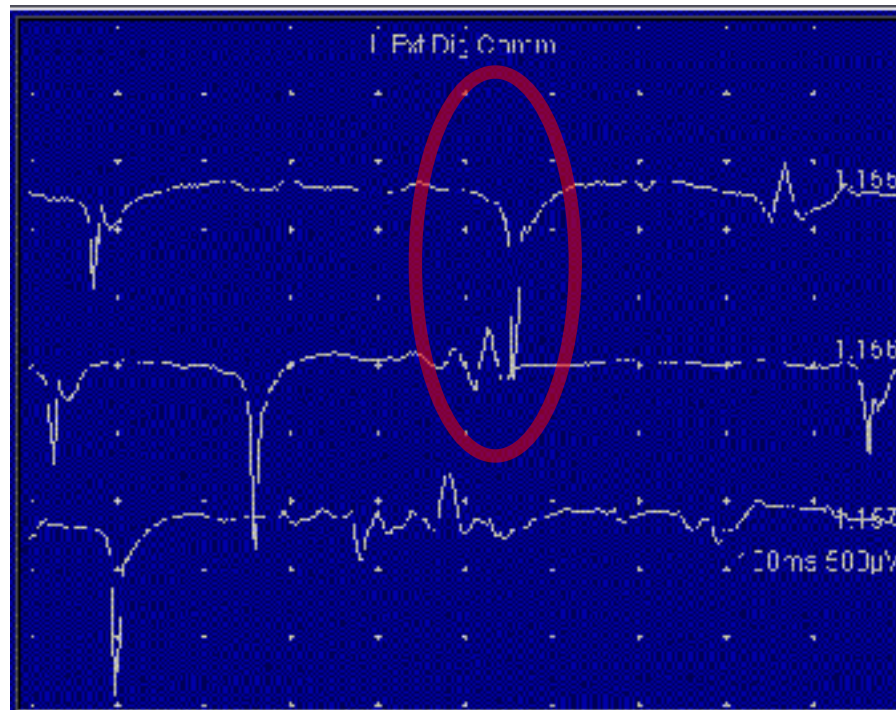
**The Back Research Center, Backcenter Funen, Part of Clinical Locomotion Science, University of Southern Denmark**

# **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/Mean 19/19.5  
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**

**Cummings, T. Needling Therapies in the Management of Myofascial Trigger Point Pain: A Systematic Review,  
Arch Phys Med Rehabil Vol 82, July 2001**

# **Prevalence of Myofascial Pain in General Internal Medicine Practice.**

Skootsky, S.A. West J Med. 1989 Aug;151(2):157-60

- **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

Scoliosis 77.5% vs. 60%

Myofascial Pain Syn 96% vs. 10

Sacroiliac 84% vs. 5%

Fibromyalgia 19% vs. 0%

Hip Pain 48% vs. 0%

## **TABLE 7-1.**

### **Epidemiology of Trigger Points**

---

Higher in women than men

Most common in 30- to 50-year age range

Most commonly found in the following muscles:  
trapezius, levator scapulae, axial postural  
muscles

Chronic pain clinics study reported incidence of  
85% of patients having myofascial pain  
syndrome

Asymptomatic shoulder girdle trigger points are  
found in 54% of females and 45% of males

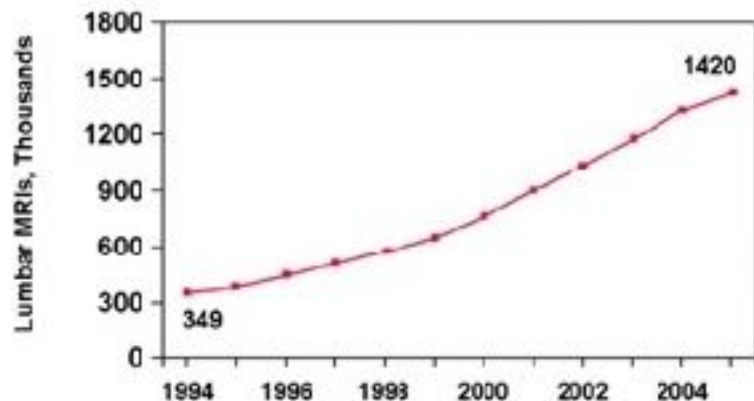
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**Rachlin ES. Myofascial Pain and Fibromyalgia –  
Trigger Point Management. Mosby, 1994**

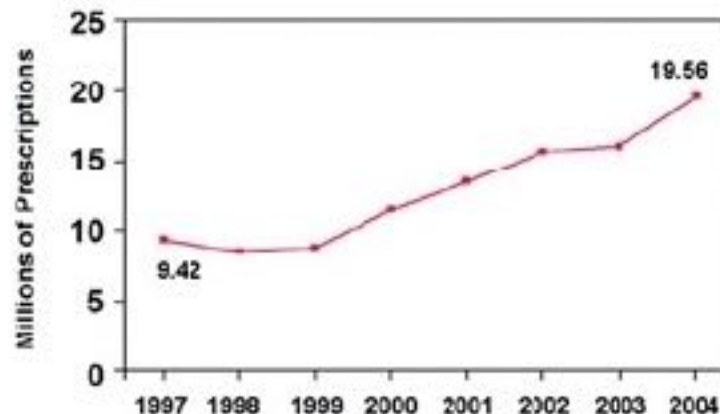
# Overtreating Chronic Back Pain: Time to Back Off?

Deyo, R. J Am Board Fam Med. 2009 Jan-Feb;22(1):62-8.

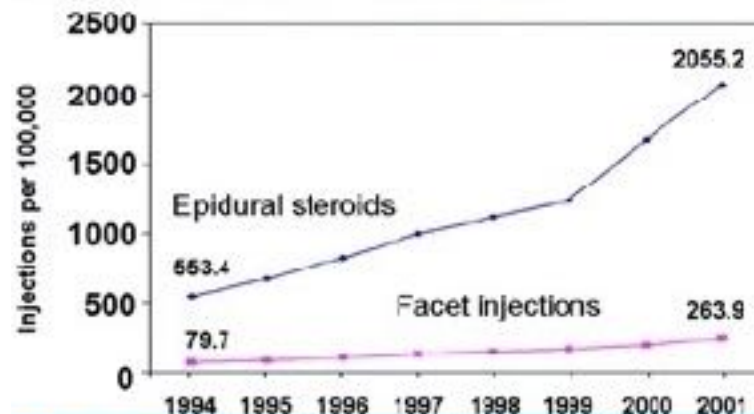
a Lumbar spine MR imaging, Medicare



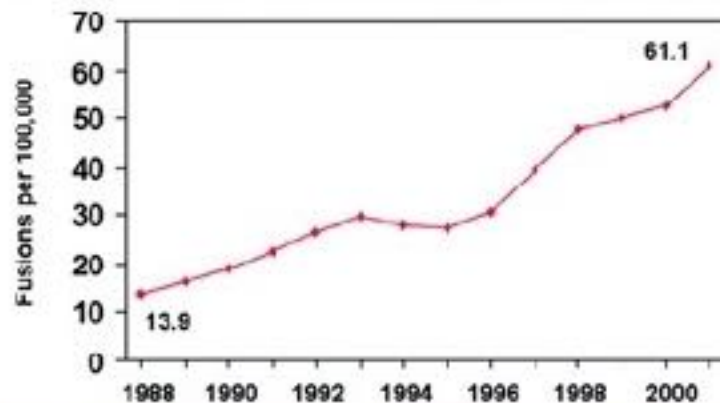
b Opioid analgesic prescriptions for spine problems



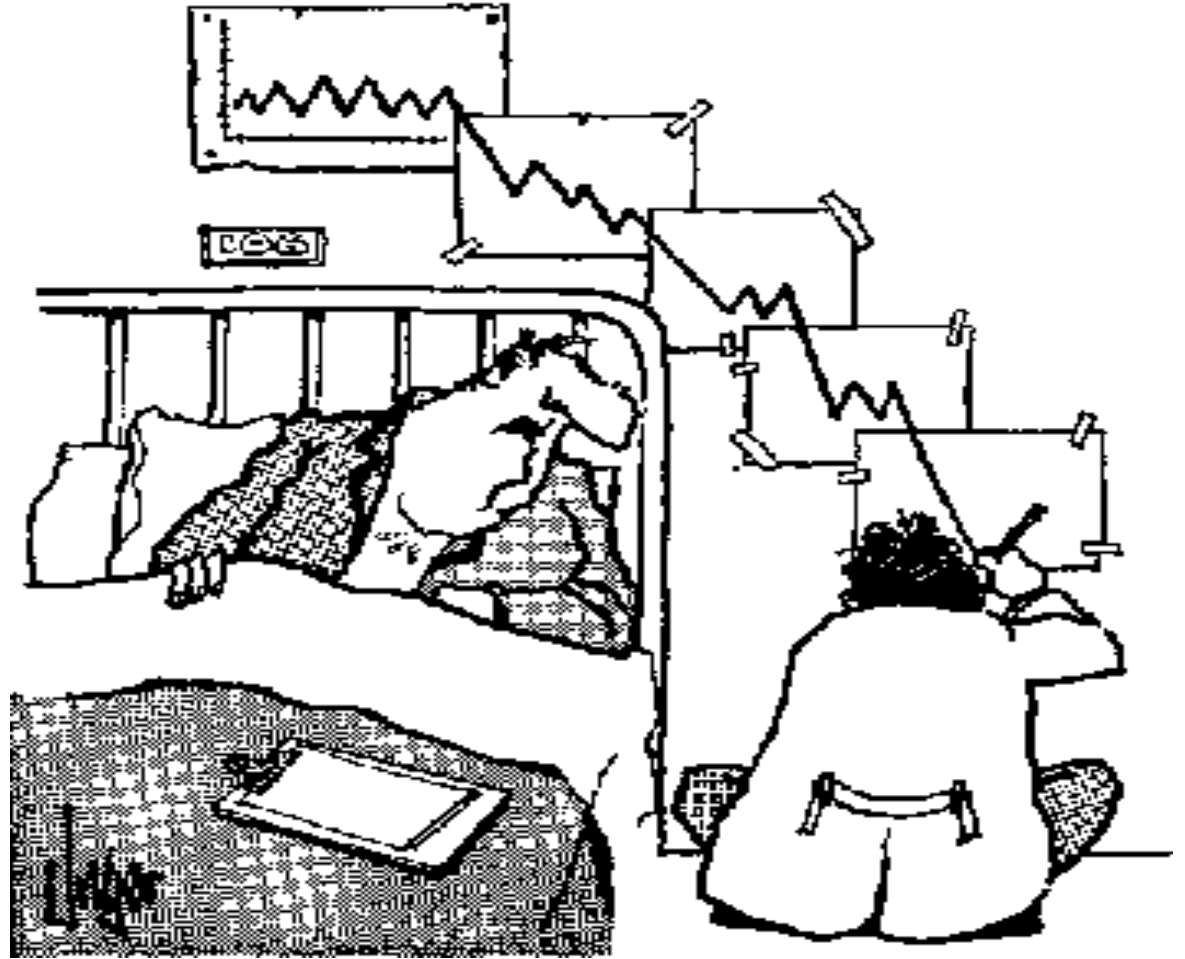
c Lumbosacral injection rates, Medicare



d Lumbar fusion rates, degenerative spine conditions



**“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**



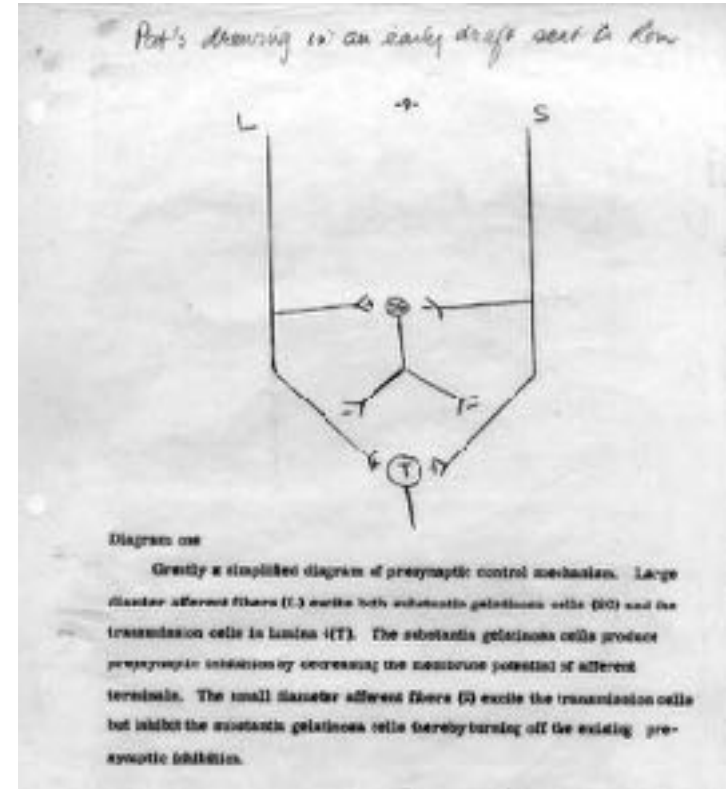
**AND, Commonly Used in Medicine**

**‘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**

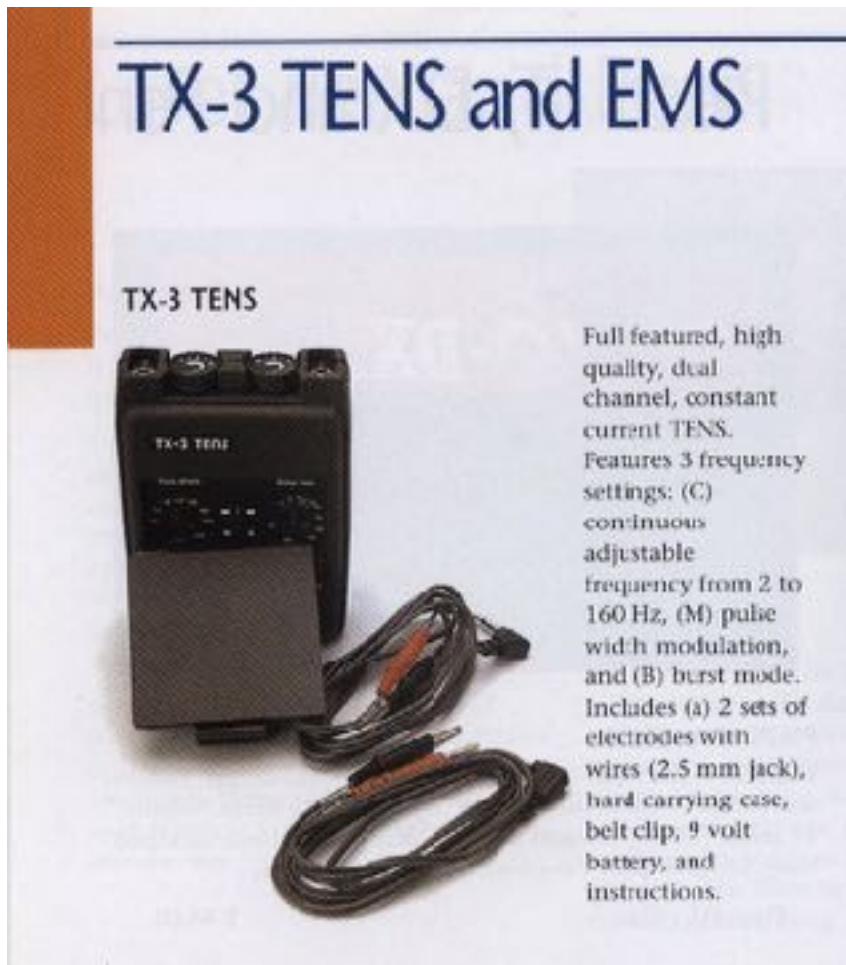


# T.E.N.S.



# ELECTRO-ANALGESIA

## Battery Powered Electric Nerve Stimulator 2008



**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**

---

**2011 J Neurosurg Dec;115(6):1248-55.**

**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**

**Curr Urol Rep (2012) 13:327–334**

---

**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

<sup>112 I.</sup>  
Capitis dolorē quemuis ueterem &  
intolerabilem protinus tollit, & impera  
petuum remediāt torpedo uiua nigra,  
imposita eo loco q̄ in dolore est, donec  
desinat dolor, & obstupeſcat ea pars:  
quod cum primū ſenſerit, remoueatūr  
remedium, ne ſenſus auferatur eius par  
tis. Plures aut̄ parādæ ſunt eius generis  
torpedines: quia nōnunq̄ uix ad duas,  
trēsue reſponder curatio, id eſt, corpor,  
quod ſignū eſt remediationis.

Scribonius Largus Designatus  
"Scripta mea Latina medicalia, codex inlar"  
editio prima, cap. XI. De compositione medicamentum liber.  
Ed. J.M.Berthold, Argentor, 1786.

# **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 AF<sup>1</sup>, Schmiede SJ, Mann JD, Butryn N, Krutsch JP.](#)  
[Am J Phys Med Rehabil. 2017 Sep;96\(9\):639-645.](#)

**Effect of ischemic compression for cervicogenic headache and elastic behavior of active trigger point in the sternocleidomastoid muscle using ultrasound imaging.**

Jafari M<sup>1</sup>, Bahrpeyma F<sup>2</sup>, Togha M<sup>3</sup>. [J Bodyw Mov Ther. 2017 Oct;21\(4\):933-939](#)

# 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, randomized, 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 sx's 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

Vol. 73, No. 4, July/August 1994

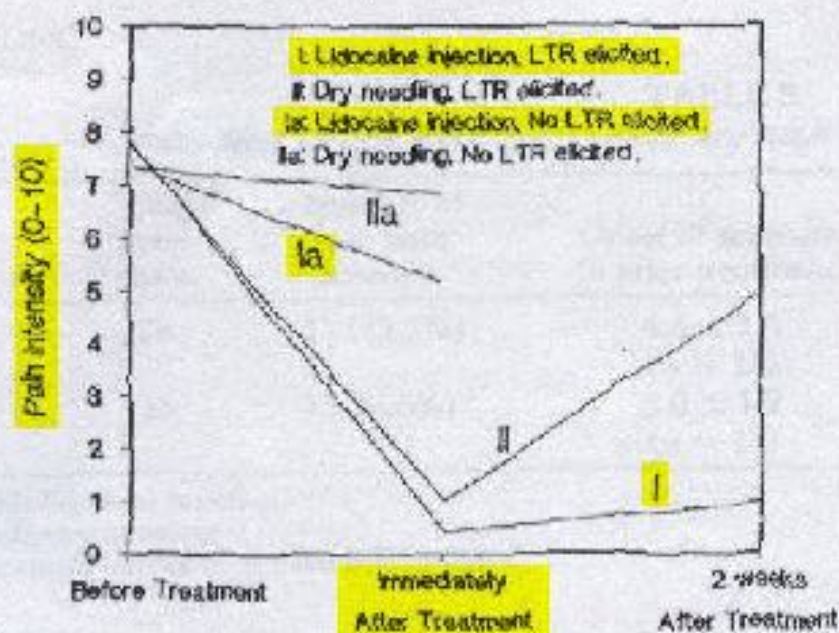


Figure 1. Effect of trigger point injection or dry needling on subjective pain intensity: 0, no pain; 10, most severe pain.

TRIGGER POINT INJECTION

26

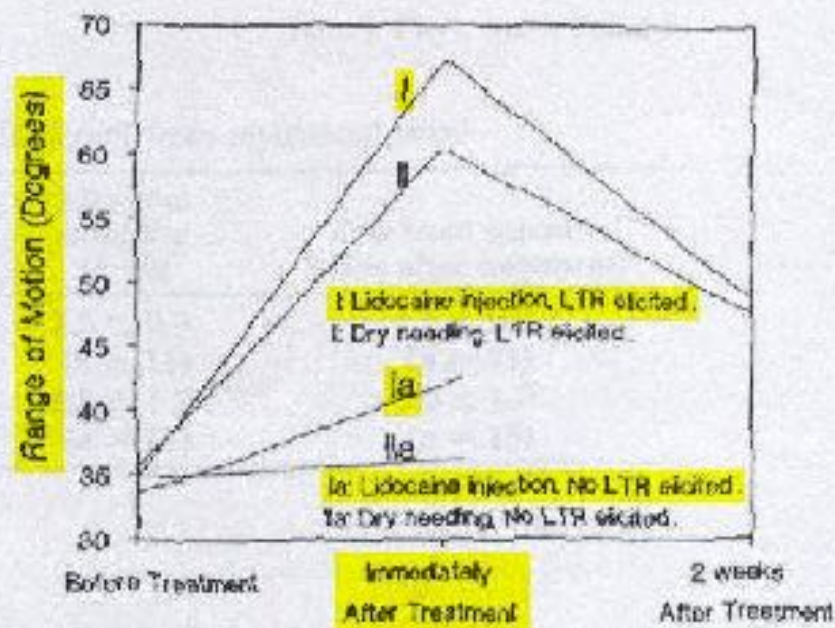


Figure 3. Effect of trigger point injection or dry needling on range of motion of cervical spine (lateral bending).

# **INTRAMUSCULAR STIMULATION DRY NEEDLING** **RESEARCH**

- **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 (In Korean with English introduction)**
- **Kim JK, Lim KJ, Kim C, Kim HS. Intramuscular stimulation therapy in failed back surgery syndrome patients. J Korean Pain Soc 2003 16:60-67 (In Korean with English introduction)**
- **Ga H, Koh HJ, Choi JH, Kim CH. Intramuscular and nerve root stimulation vs lidocaine injection of trigger points in myofascial pain syndrome. J Rehabil Med 2007 39: 374-378**
- **Ga H, Choi JH, Park CH, Yoon HJ. Dry needling of trigger points with and without paraspinal needling in myofascial pain syndromes in elderly patients. J Altern Complement Med 2007 13: 617-624**
- **Lim SM, Seo KH, Cho B, Ahn K, Park YH. A systematic review of the effectiveness and safety of intramuscular stimulation therapy. J Korean Med Assoc 2011 54(10): 1070-1080**

# 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.”**

Furlan AD, et.al. Acupuncture and dry-needling for low back pain. Cochrane Database of Systematic Reviews 2005, Issue 1. Art. No.: CD001351

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

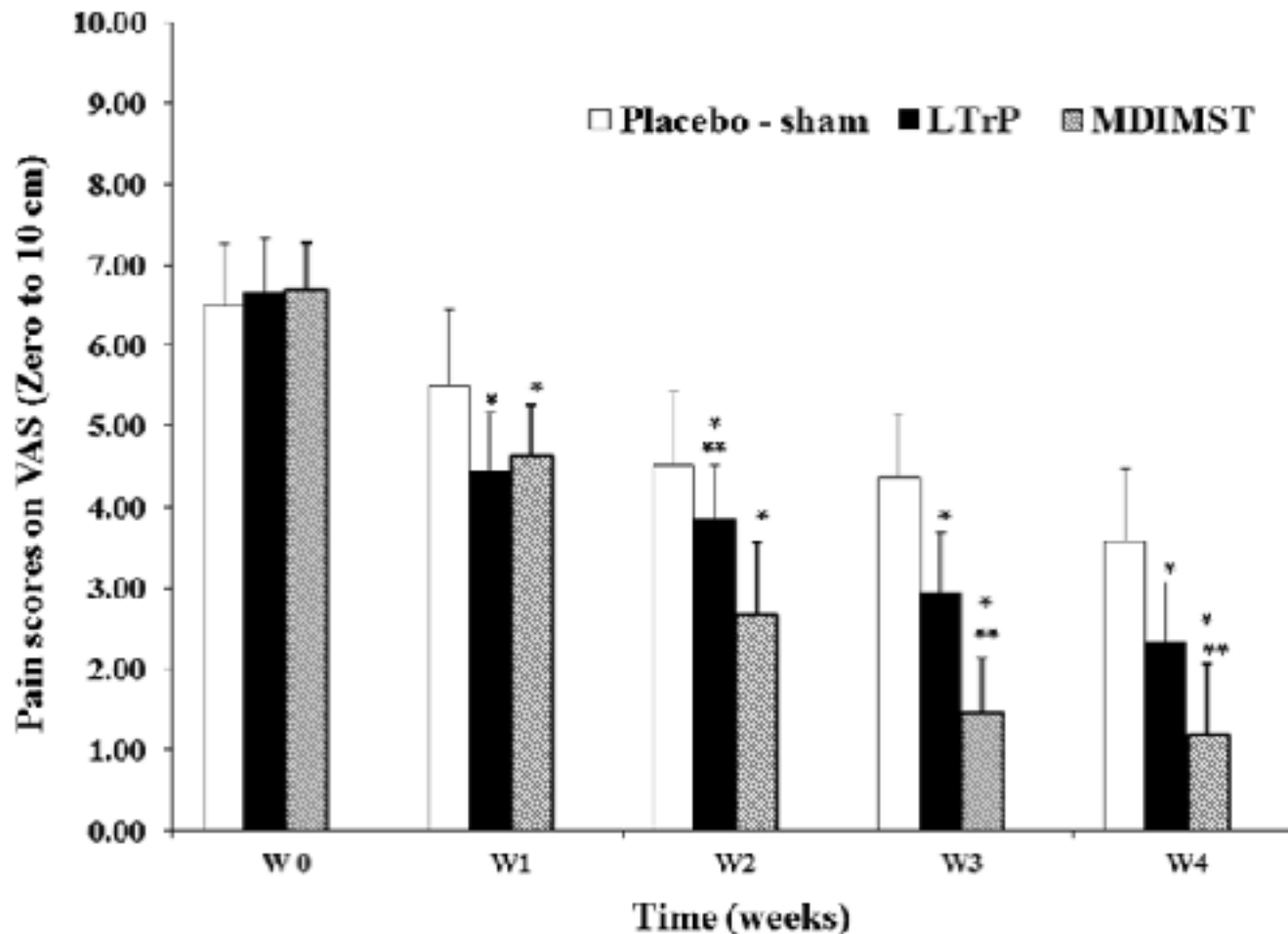
**“DN has moderate evidence that supports its use in MPS”**

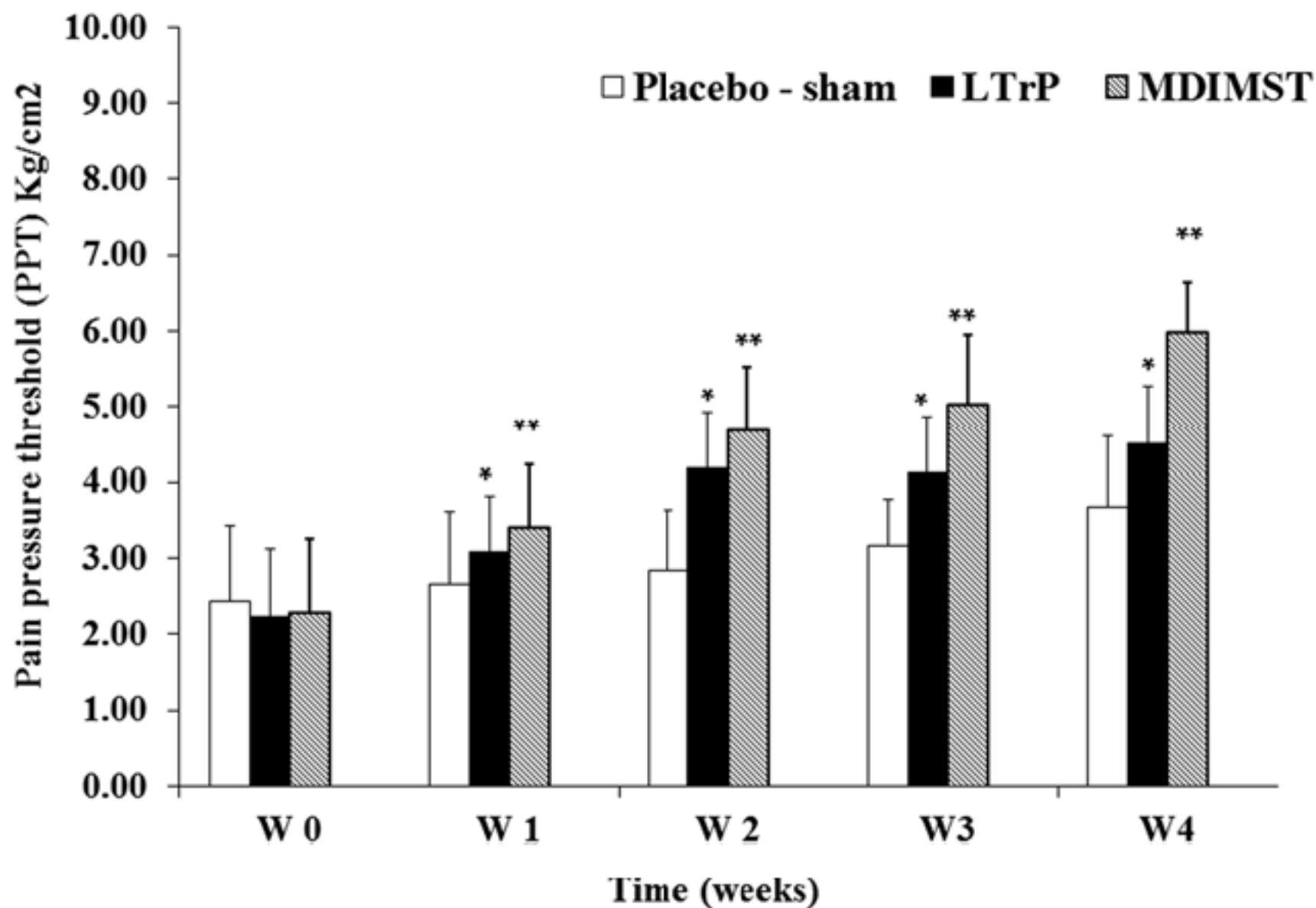
**2011 Annaswamy PM&R**

**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

Couto, C., et. al. Clinical Journal of Pain Vol 30(3) 2014





PMR July 2015 Volume 7, Issue 7, 711–718

## **Original Research CME**

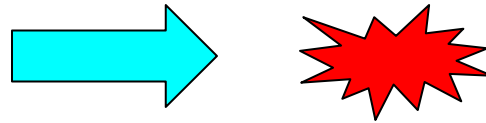
# **Dry Needling Alters Trigger Points in the Upper Trapezius Muscle and Reduces Pain in Subjects With Chronic Myofascial Pain**

*Lynn H. Gerber, MD, Jay Shah, MD, et. al.*

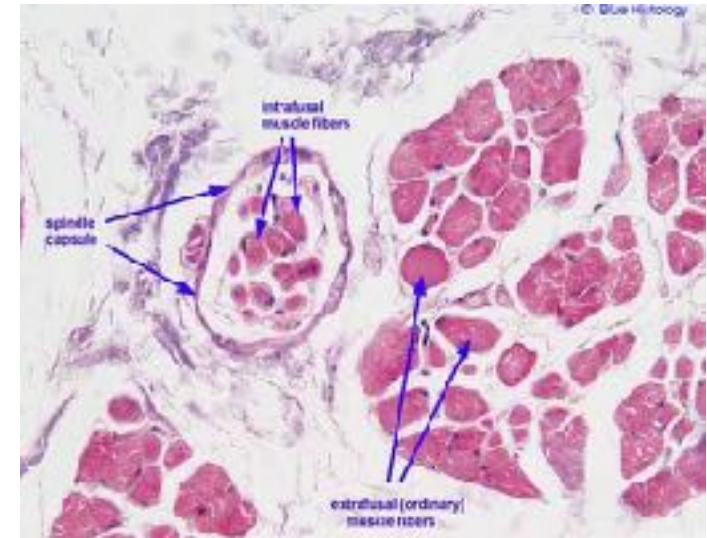
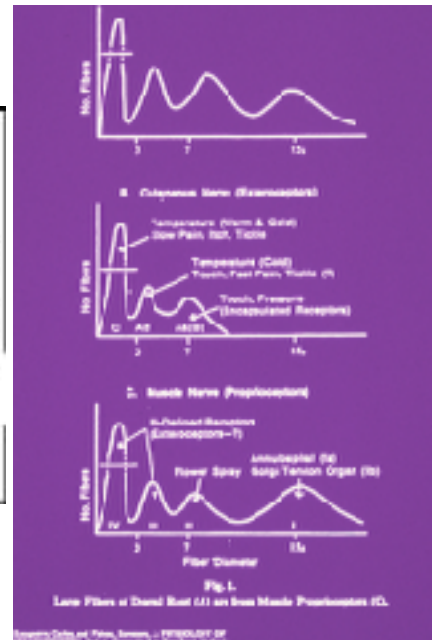
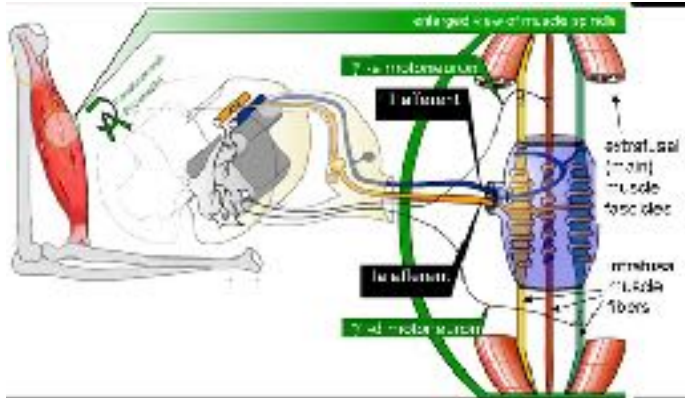
**Participants:** A total of 56 subjects with neck or shoulder girdle pain of more than 3 months duration and active MTrPs were recruited from a campus-wide volunteer sample. Of these, 52 completed the study (23 male and 33 female). Their mean age was 35.8 years.

**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**

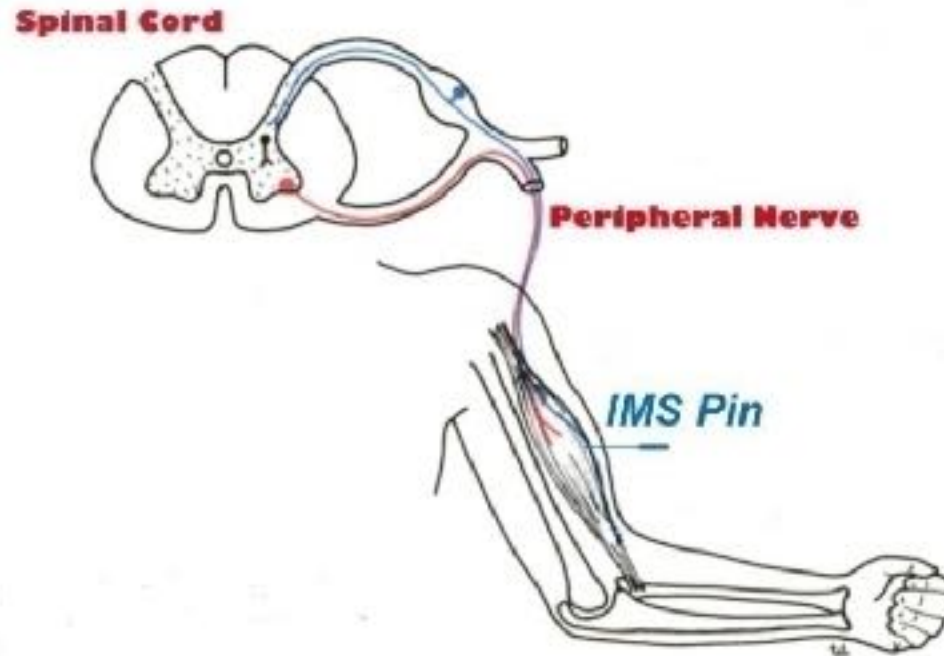


# Myotatic Spinal Reflex



**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?

Electromyogr. clin. Neurophysiol. 1997

One 'Treatment' Only

Grp 1 82/122: avg 52% decr Pain, 14% > 75%

Grp 2 23/42: avg 39%, 0 > 75%

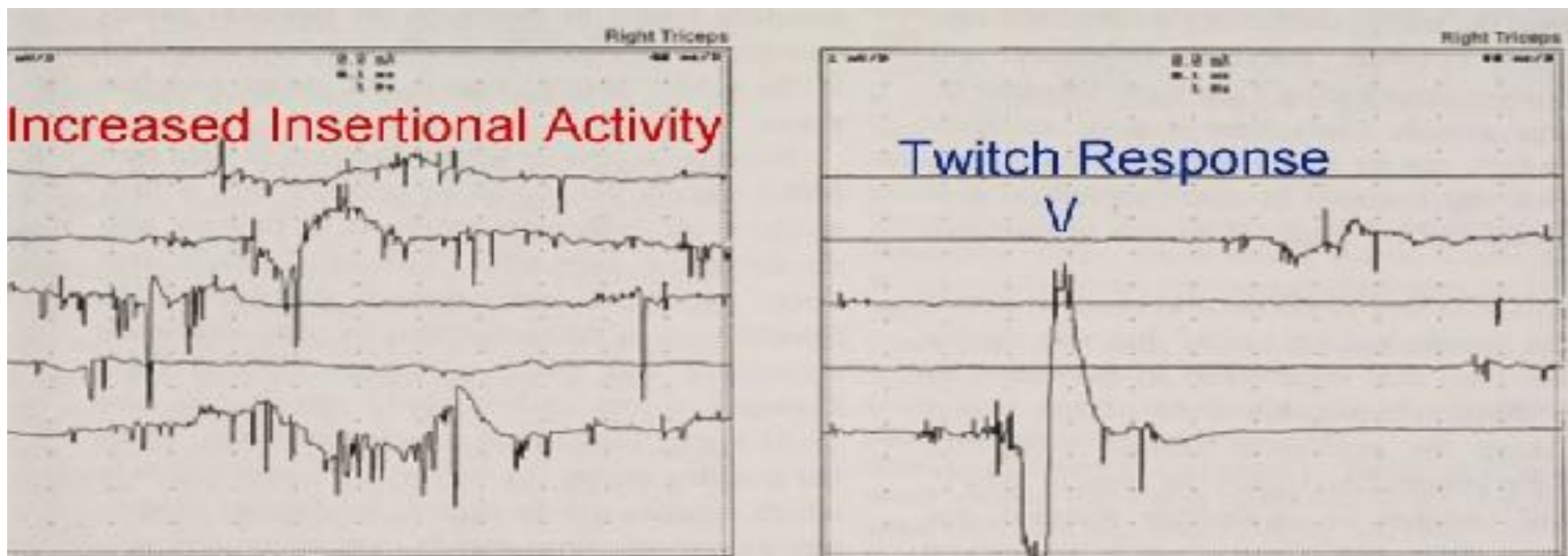


Fig. 4. - Insertional activity in a triceps muscle at a tender point within a myofascial band showing (A) increased insertional activity consisting of grouped endplate spikes (negative or negative-positive form) and positive spikes, and Trace (B) showing a grouped insertional activity culminating into a large twitch response. Sensitivity in both traces was set at 1 mV/division Sweep speed was set at 10 ms/division for Trace A and 80 ms/division for Trace B.

[Note: Tracings recorded with Keypoint Electromyograph, DANTEC Corp., Allendale, NJ, USA.]

## Before Twitch Response

muscle fibers

• Pain Causing  
Chemicals

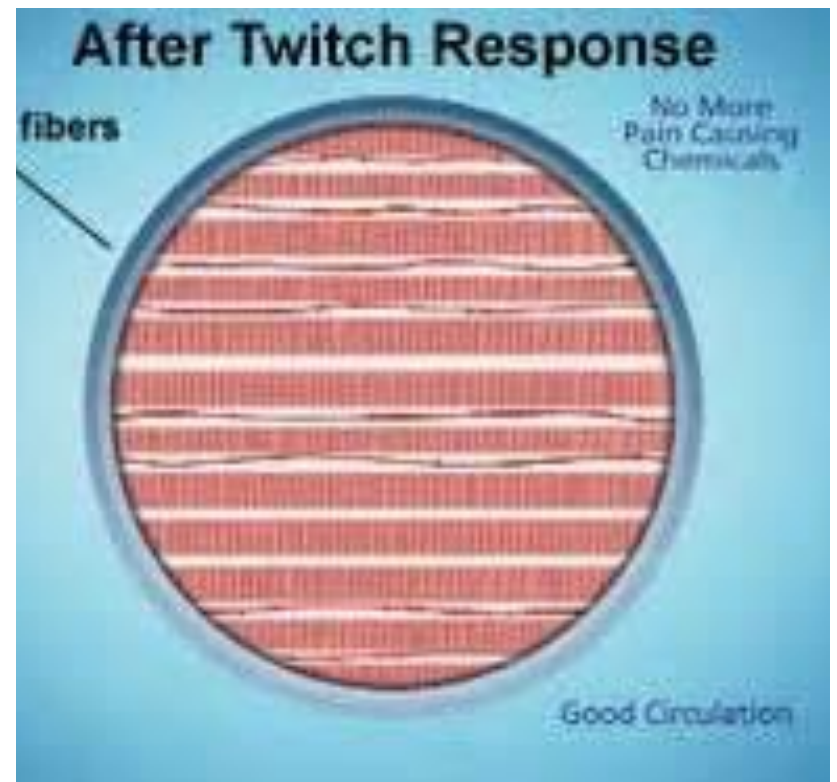
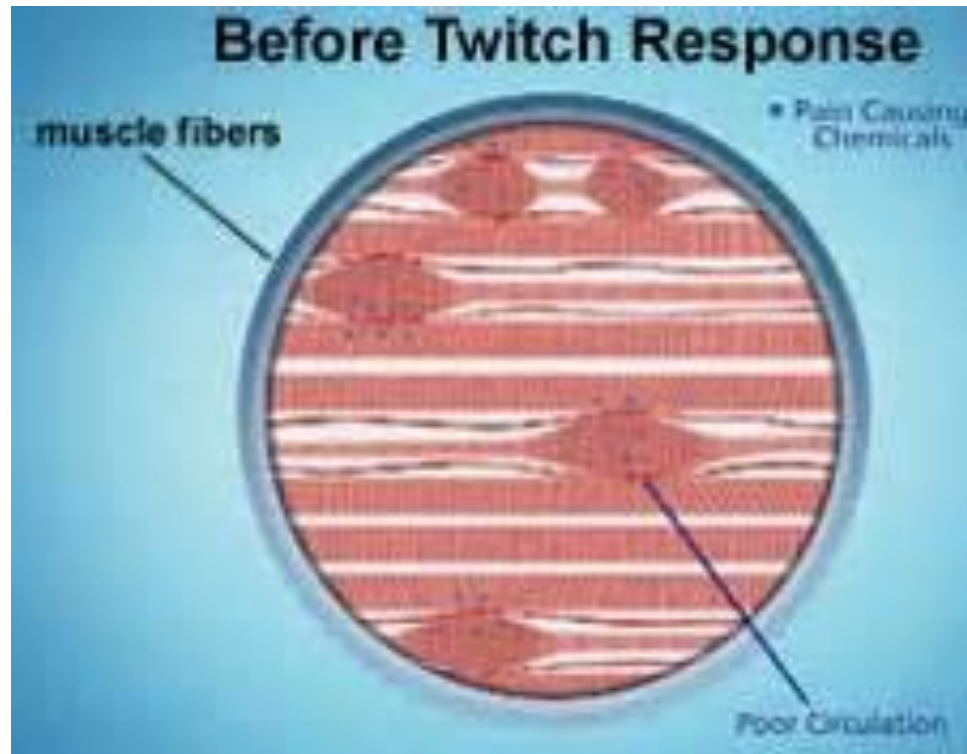
Poor Circulation

## After Twitch Response

fibers

No More  
Pain Causing  
Chemicals

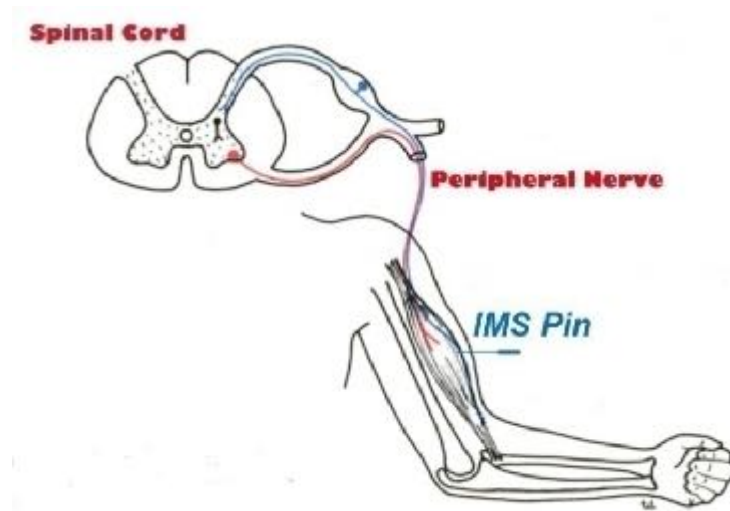
Good Circulation

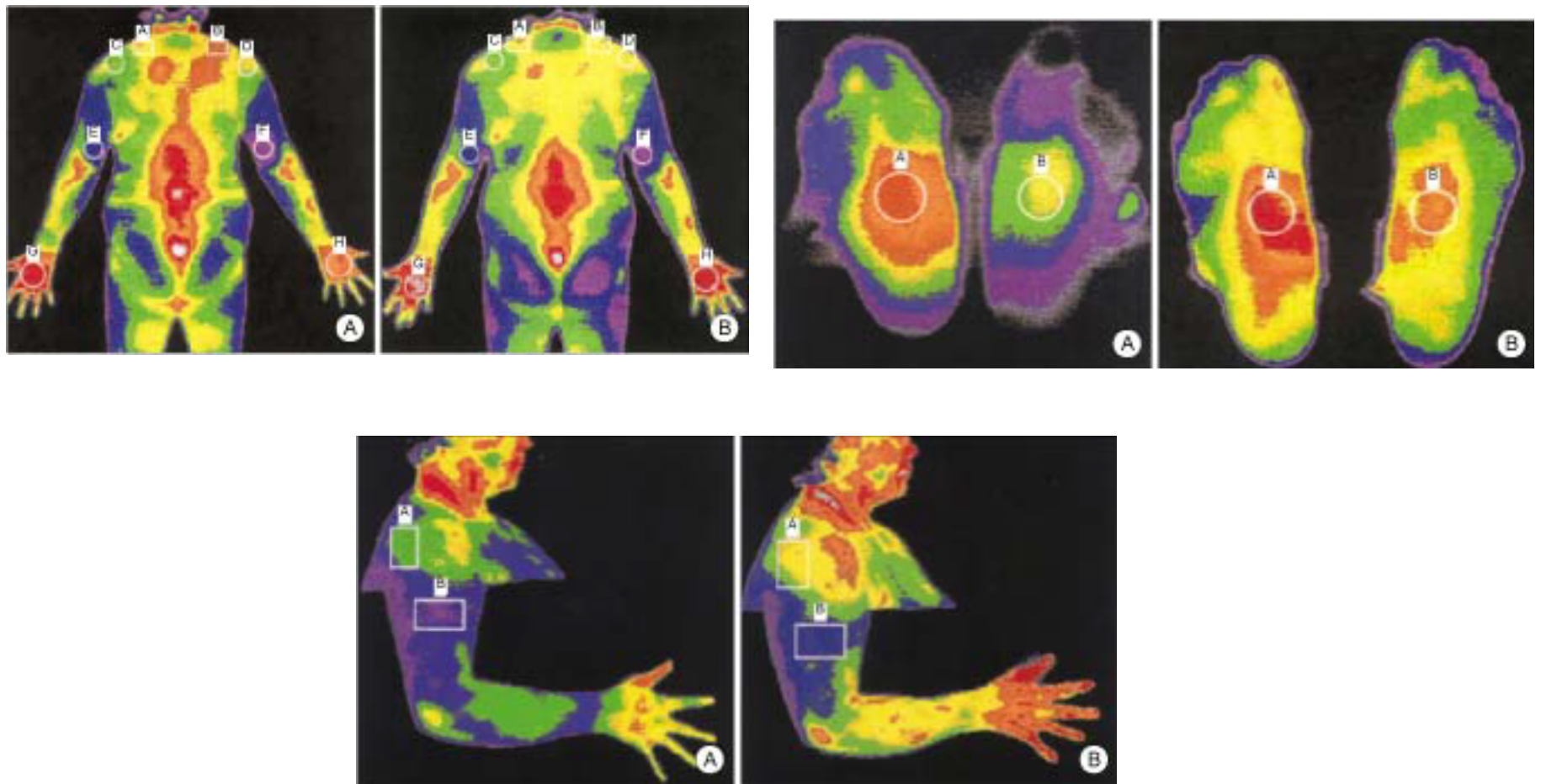




**IMS is Like 'Catch-and-Release'  
Fly Fishing:**

**We are 'Casting' for the 'Bites' =  
Needle 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**

**2005 Jrnl Appl Physiol 99: 1977-84. Shah, J.**

**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.**

Needle Effect	Clinical Response	Neurophysiological Basis
Local Twitch Response  Stimulation of Spinal Reflexes	Increased ROM	Reversal of Muscle Contracture  ? Normalization of Spindle Mechanism/Sensitivity
Hyperstimulation Analgesia	Decreased Pain	Melzack and Wall Gate Theory
Direct and Reflex Stimulation & Normalization of Therapeutic Target	Decreased Myalgic Hyperalgesia	Reversal of Neuropathic Supersensitivity - Cannon and Rosenblueth's Law of Denervation Supersensitivity
Direct and Reflex Stimulation & Normalization of Therapeutic Target	Decreased Spontaneous Endplate Activity	Reversal of Neuropathic Supersensitivity  Cannon's Law
Direct & Reflex Stimulation	Vasodilation > Warming  Normalization of Local Biochemical Milieu	?Spinal/Axon Reflexes
Minor Tissue Trauma - Bleeding	Inflammatory Response, release of PDGF & 'Current of Injury'	Reversal of Neuropathic Supersensitivity by electric stimulation - Lomo

# Treatment Ladder of Neuropathic-Myofascial Pain

- Manual soft tissue: 'ischemic compression': trigger point massage, strain-counterstrain, Shiatsu, acupressure, pin & stretch, Astym
- 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'

# **Treatment Ladder of Neuropathic-Myofascial Pain cont'd**

- **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-carnitine 1000mg 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**

# Thank You!

