Nociceptive and neuropathic pain management

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Seminar objectives

- Better understand acute, chronic and neuropathic pain from the laser therapy perspective
- Identify treatment targets unique and significant to pain management
- Have knowledge and confidence to manage pain presentations next week with laser therapy

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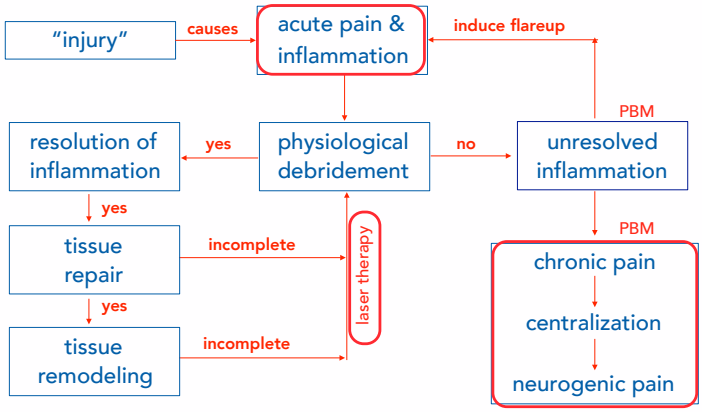
Why are we using laser?



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Key: acute inflammation

- An essential part of wound healing
- Acceleration is preferable to inhibition
- Growth factors are secreted
- Growth factors are mitogenic and angiogenic

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What we'll cover

Laser therapy

Energy Density

Dosage

Power Density

Average Power

Peak Power

Average Dose

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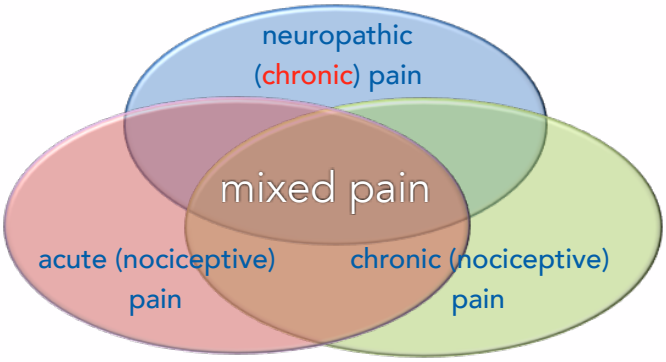
What we'll cover

Nociceptive versus neuropathic pain



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What we'll cover

Pain management: treatment players

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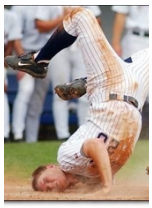
What we'll cover

Treatment guidelines & strategies



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What we'll cover

- Super brief review of laser therapy
- Nociceptive and neuropathic pain
- Pain management: the "treatment players"
- Treatment guidelines and strategies

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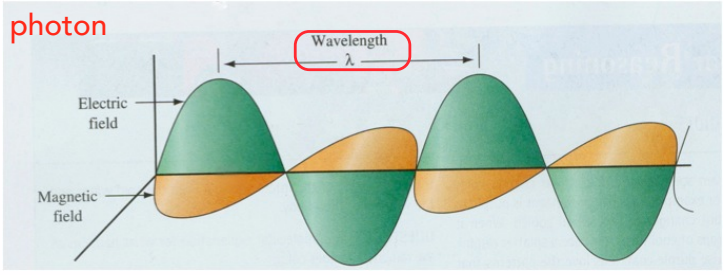
Photons are electromagnetic waves



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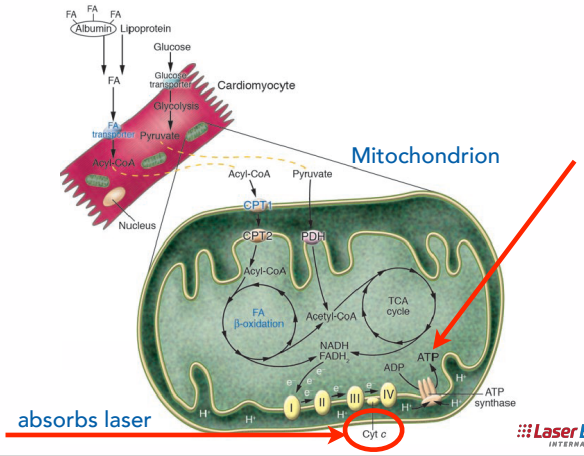
Laser stimulates ATP production



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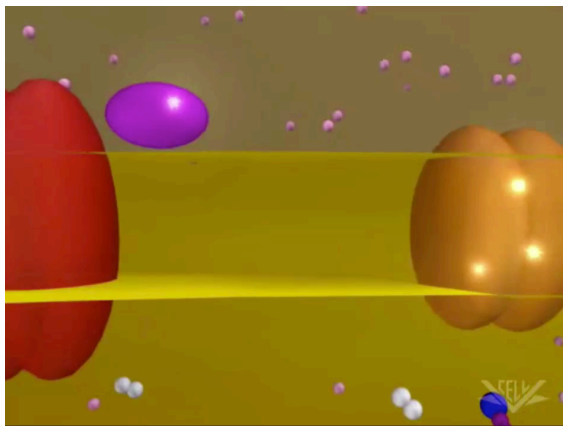
Mitochondria need electrons: no electrons → cell death!

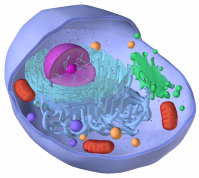


Main purpose of laser therapy?

recharge the mitochondria!

laser must reach target tissues in the correct amount!

photonic energy



cell

pos. & neg. factors

patient factors

laser factors

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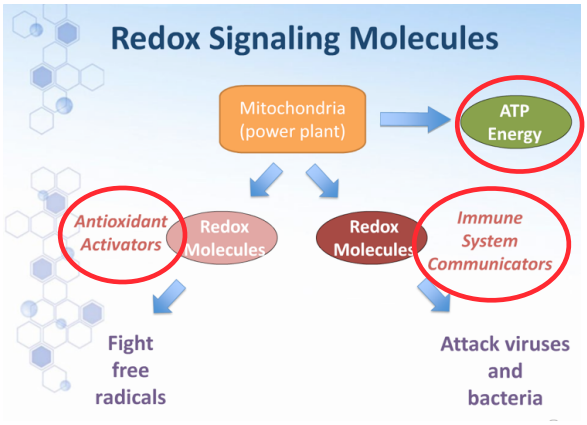
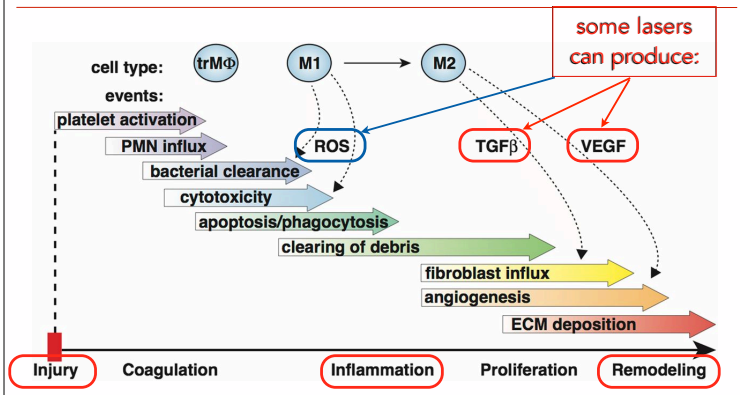
Why recharge the mitochondria?



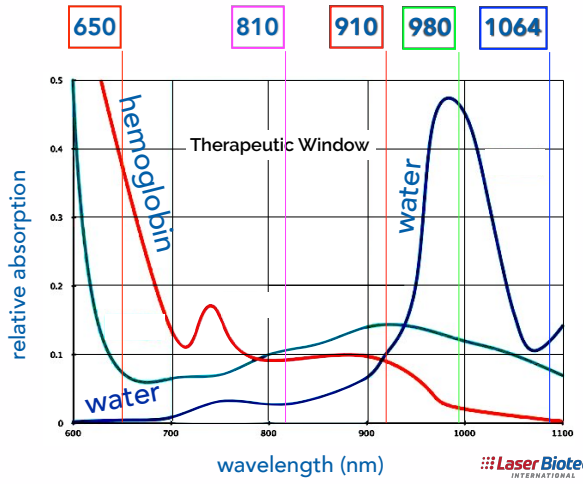
Mitochondria functions

- ATP Synthesis (aerobic)
- ATP Consumption (anaerobic or aerobic with uncoupling)
- Redox Poise Homeostasis
- Platelet Aggregation and Activation
- Neutrophil Chemotaxis
- Late Neutrophil Oxidative Burst
- Macrophage Activation
- T-Cell Activation
- Sperm cell motility/fertilization
- Angiogenesis
- Lymphedema
- Nitric Oxide Synthesis
- Apoptosis/Caspase Activation
- Prostaglandin Inactivation
- Cholesterol Synthesis
- Cortisol Synthesis
- Mineralocorticoid Synthesis
- Sex Steroid Synthesis
- Vitamin D Metabolism
- Cytoskeleton Architecture/ Mechanotransduction
- Calcium Storage and Release
- Iron Storage and Metabolism
- DNA and RNA--De Novo Pyrimidine Synthesis (DHO-QO)
- Lipids--Fatty Acid Oxidation
- Proteins--Amino Acid Metabolism
- Sugars--Carbohydrate Metabolism (Krebs Cycle)
- Urea Cycle and NH_3 Metabolism
- Peripheral Benzodiazapine Receptor

Injury to inflammation to tissue repair



Photon absorption depends on wavelength



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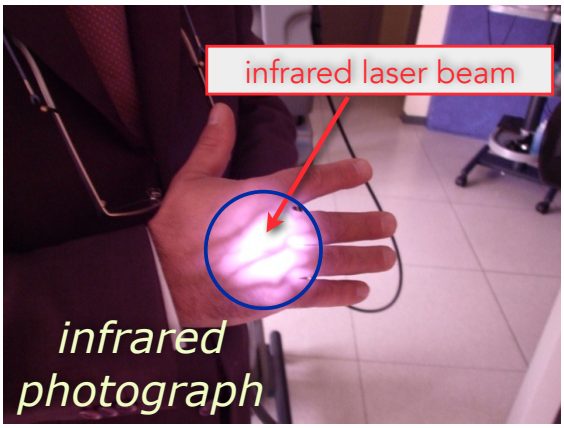
What are the dark lines on my hand?



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Water and blood "steal" laser energy

- Caution: Target tissues with high water content require lower power density (e.g., bursa)
- Treating through vascular layers would require _____ dose (e.g., targeting piriformis)
- Water and/or blood layers reduce laser energy in transit to the target tissue

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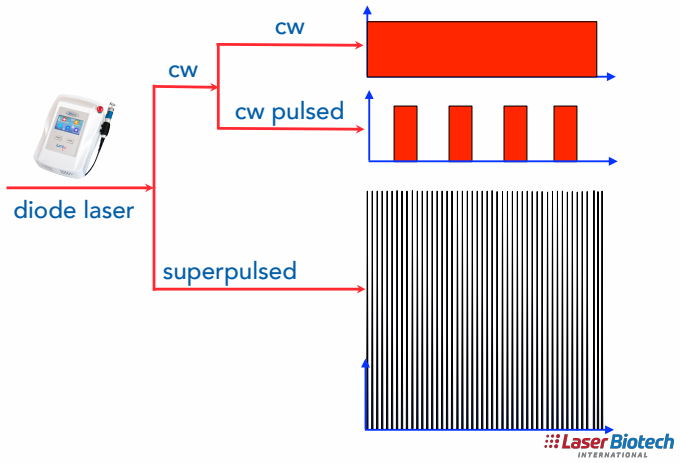
Best biostimulation wavelength

635 - 670 nm	best superficial laser (CW red), e.g., skin conditions
800 - 830 nm	excellent penetrating CW laser; best for mostly vascular tissues
904 - 910 nm	excellent all tissue laser (SP); lowest thermal and highest peak power
915 - 980 nm	excellent thermal effects (CW); large superficial to mid depth treatment
1064 nm	excellent penetrating CW laser; best for mostly avascular tissues

Two types of therapeutic lasers



Clinical pearl

- Acute conditions: 20 kHz pulse rate
  - priority - shorten inflammatory cycle
- Chronic conditions: 40 kHz pulse rate
  - priority - tissue repair and remodel (enhanced gene expression)

World Association for Laser Therapy

www.WALTZA.co.za

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WALT on tissue penetration

Factors affecting the laser depth of tissue penetration:

- The laser's wavelength
- Whether the laser is superpulsed
- The power output
- The treatment technique used
- The technical design of the apparatus

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Laser terminology

- Average power (AP) and energy
- Power density ("intensity")
- Energy density (dose)
- Dosage principles
- Average dose per tissue type

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Laser and x-ray photons

- **mAs**: dictates the quantity of x-ray photons emitted in one second
- **mW**: dictates the quantity of laser photons emitted in one second (power)

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Dose: most important parameter

- Power = photons per second
- Power density = "intensity of photons"
- Power density = average power (W)
  ÷ size of beam or "spot size" (cm^2)
- Power density = W/cm^2
- Energy density = power density X time (sec)
  = J / cm^2 (dose)

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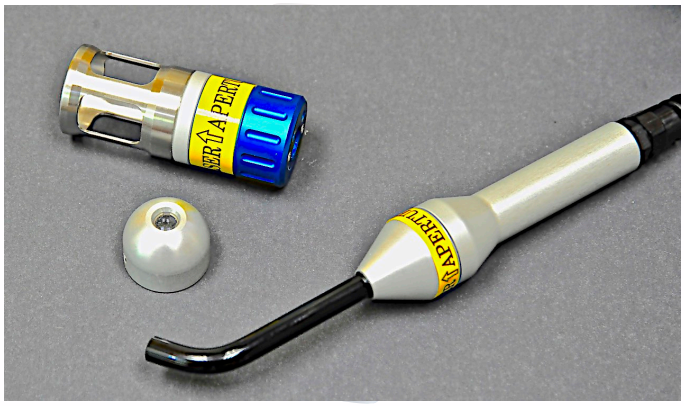
Adjustable power density (Lumix)



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Average dose per tissue type

- Muscles: **7** J/cm^2
- Nerve, skin, bursa: **5** J/cm^2
- Tendon, ligament, fascia, bone: **10** J/cm^2

must compensate for tissue depth!

use 50% rule

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What we'll cover

- Super brief review of laser therapy
- Nociceptive and neuropathic pain
- Pain management: the "treatment players"
- Treatment guidelines and strategies

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Acute vs. chronic

- Acute Inflammation: GOOD
- Chronic Inflammation: BAD
- It is better to:
  - Re-initiate the acute phase of inflammation (e.g., Graston, dry needling, prolotherapy)
  - Then treat it with lasers

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Treating Chronic Conditions

- "Injuries" of more than 8 weeks
- Steady improvement usually occurs gradually during the first 2-3 treatments
- Most patients feel some form of immediate pain relief 2 hours post-treatment, but it may not be sustainable
- Treatment effects are cumulative

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Chronic and neurogenic pain

- Chronic pain: lasting 6 weeks or more
- Neurogenic pain: pain due to dysfunction of the peripheral or central nervous system in the absence of nociceptor stimulation by trauma or disease
- Neurogenic pain (syndromes): about 25% of the chronic pain conditions are neurogenic in etiology

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Nociceptive versus neuropathic pain

Nociceptive Pain

- An appropriate physiologic response to a painful stimulus
- Can be acute or chronic

Neuropathic Pain

- An inappropriate response caused by primary lesion or dysfunction in the CNS or PNF
- Mostly chronic

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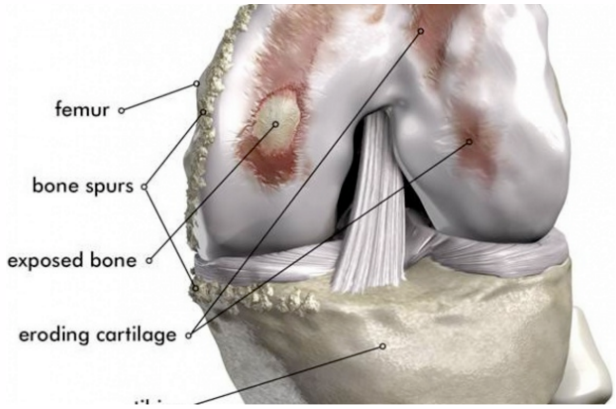
Example of nociceptive pain



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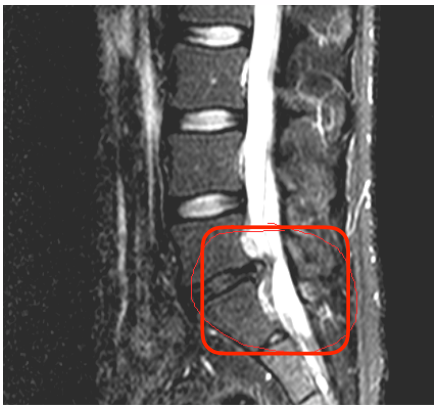
Example of neuropathic pain



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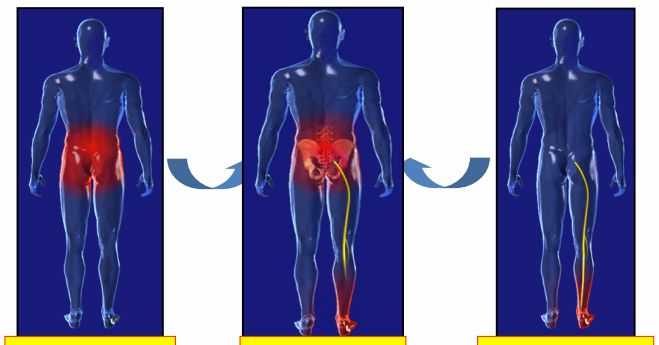
Pain patterns



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Neuropathic pain presentations

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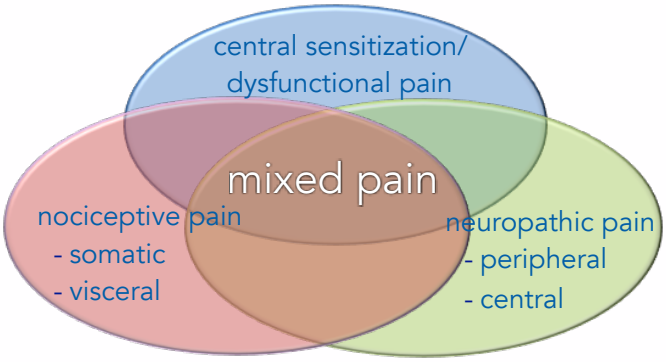
Classifying pathophysiological pain



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Simulated demonstration

Chronic knee pain post-surgery:

- Local tissues
- DRG's: knee
- Sensory cortex: knee
- Medulla

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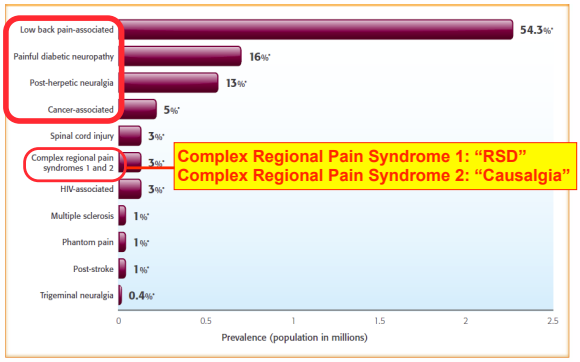
Neuropathic pain prevalence

73%



*Percentages based on total prevalence of neuropathic pain in US population of 270,000,000 individuals.

LBP alone accounts for 54%!

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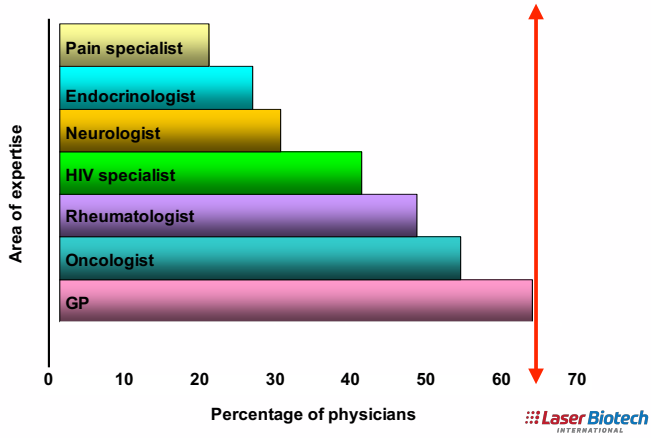
Neuropathic pain: diagnostic failure rates by MD type



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Neuropathic pain: treatment failure

- Diagnosis failure rates are alarmingly high, e.g, 64%
- Conventional approach is to chemically or mechanically:
  - Inhibit
  - Ablate
- Laser therapy approach (PBM) is to:
  - Biomodulate

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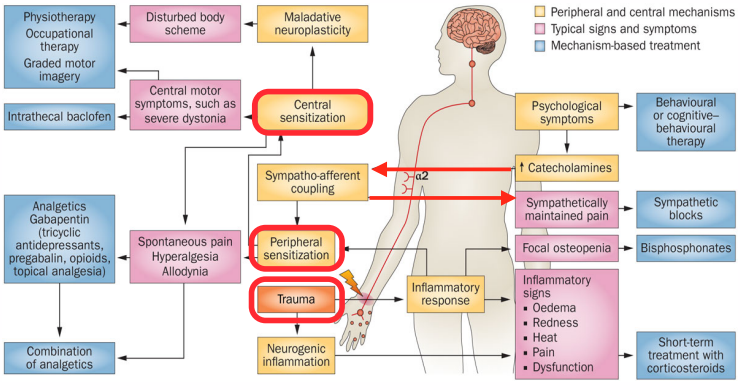
Conventional approach to pain



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Pain: Key terms

- Hyperalgesia: an increased response to a stimulus that is normally painful
- Neuralgia: pain in the distribution of a nerve or nerves
- Neuropathy: a disturbance of function or pathological change in a nerve

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Key terms (cont'd)

- Neuropathic pain: pain initiated or caused by a primary lesion or dysfunction in the CNS or PNS (NSAIDs don't work)
- Nociceptive pain: an appropriate physiologic response to a painful stimulus

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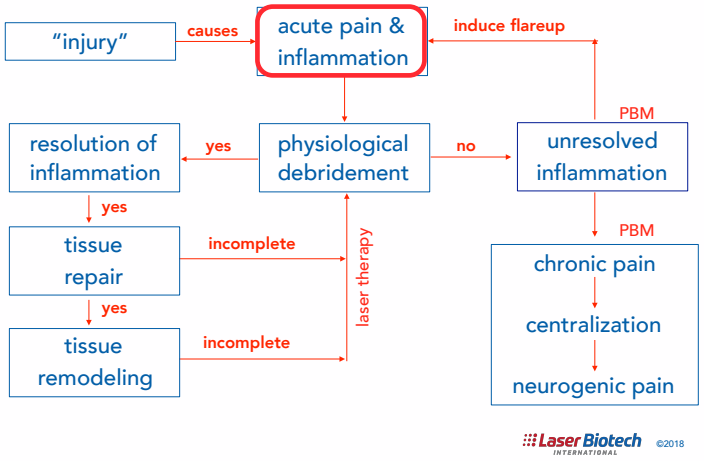
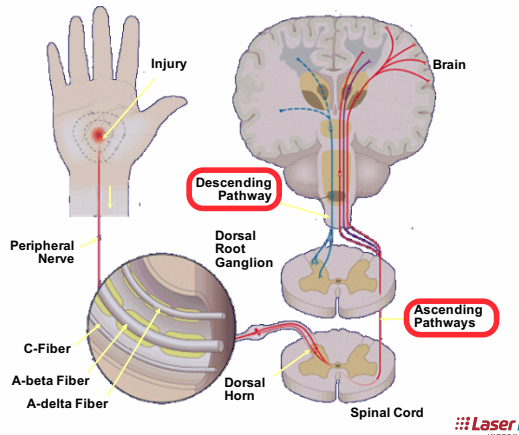
Laser approach to nerve "injury"



Pain pathways are bidirectional



Chronic pain due to lost inhibition?

7574 • The Journal of Neuroscience, April 24, 2013 • 33(17):7574–7582

Neurobiology of Disease

Chronic Pain: Lost Inhibition?

Luke A. Henderson,[1] Chris C. Peck,[2,5] Esben T. Petersen,[3] Caroline D. Rae,[4] Andrew M. Youssef,[1] Jenna M. Reeves,[1] Sophie L. Wilcox,[1] Rahena Akhter,[2] Greg M. Murray,[2] and Sylvia M. Gustin[1]
[1]Department of Anatomy and Histology and [2]Faculty of Dentistry, University of Sydney, Sydney 2006, New South Wales, Australia, [3]Departments of Radiology and Radiotherapy, University Medical Center Utrecht, 3584 CX Utrecht, The Netherlands, [4]Neuroscience Research Australia, Randwick 2031, New South Wales, Australia, and [5]School of Dentistry and Health Sciences, Charles Sturt University, Orange, New South Wales 2800, Australia

too many pain signals ascending or not enough modulating signals descending?

Craniofacial pain is rather unique

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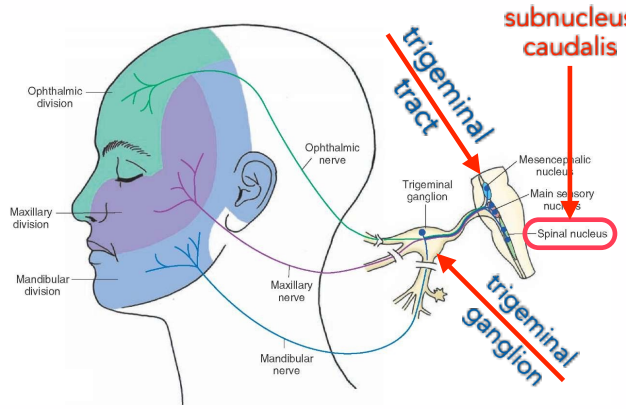
The mighty trigeminal nerve (CN V)



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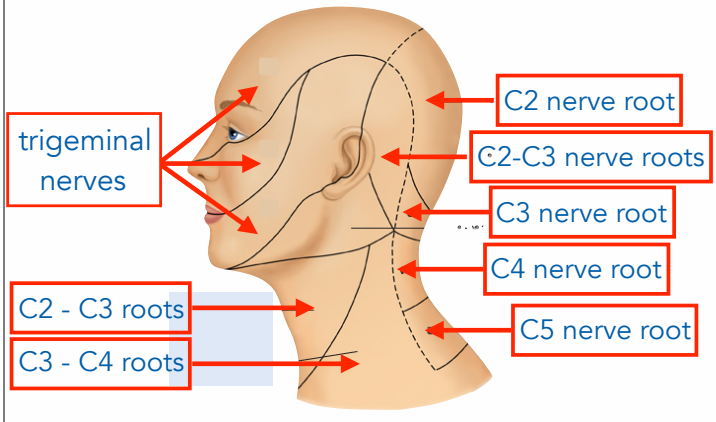
The craniocervical map



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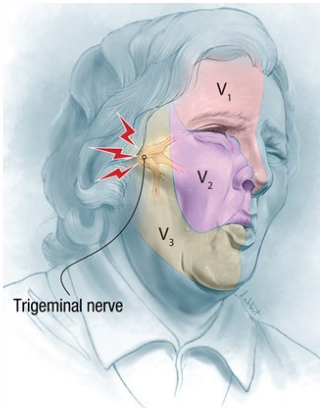
Trigeminal "neuralgia" presentations

Trigeminal neuropathic pain (TNP)



Swedish Neuroscience Institute

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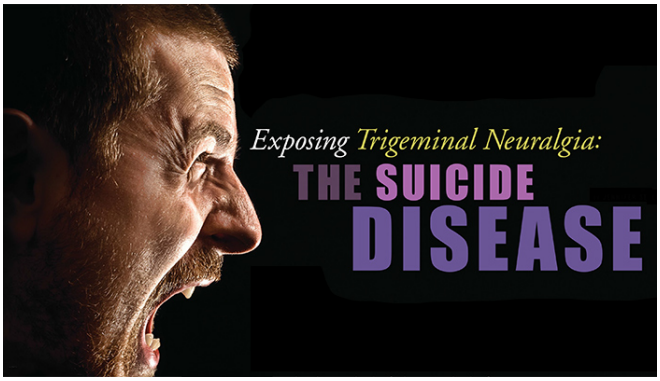
TNP, the suicide disease



Pain Pathways Magazine

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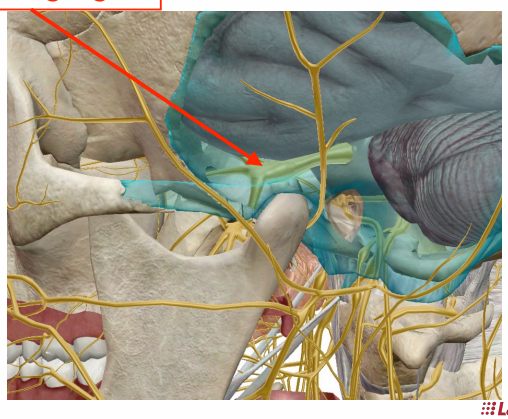
Line of sight to trigeminal ganglion

Trigeminal ganglion



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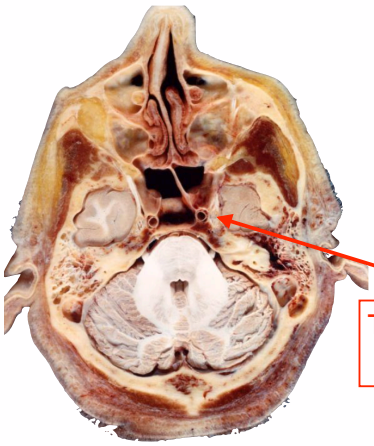
But, how deep is it?



Trigeminal ganglion: about 5 cm depth

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Treating the trigeminal ganglion

J. Tregaskes, DMD, MS

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Tracing trigeminal V3

J. Tregaskes, DMD, MS

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Treating TN trigger zones

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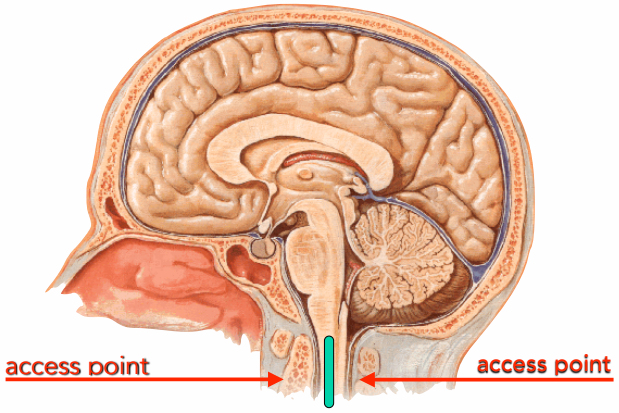
Targeting the subnucleus caudalis



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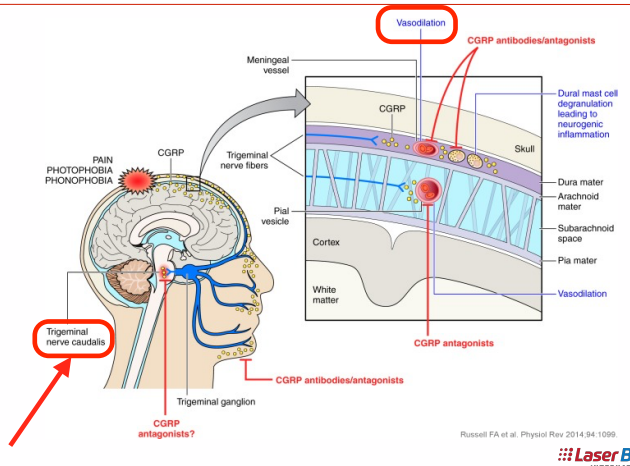
CGRP are vasodilators → migraines



Russell FA et al. Physiol Rev 2014;94:1099.

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Simulated demonstration

- Trigger points right side of face
- Right mandibular pain
- Diagnosed as neuropathic pain

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What we'll cover

- Super brief review of laser therapy
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- Treatment guidelines

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Neuropathic pain: "treatment players"

- Local tissue, precipitating lesion
- DRGs
- Sympathetic chain
- Thalamus and medulla
- Sensory cortex

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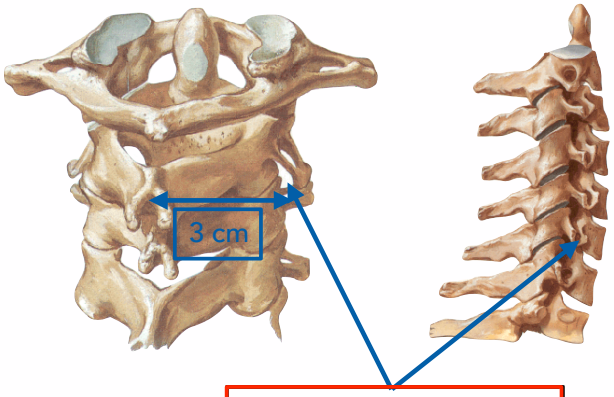
DRG/VRG live near the IVF



cervical spine IVF

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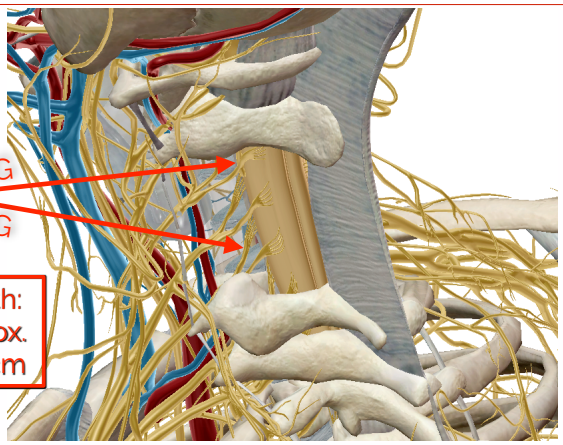
Cervical VRG / DRG



depth: approx. 2-8 cm

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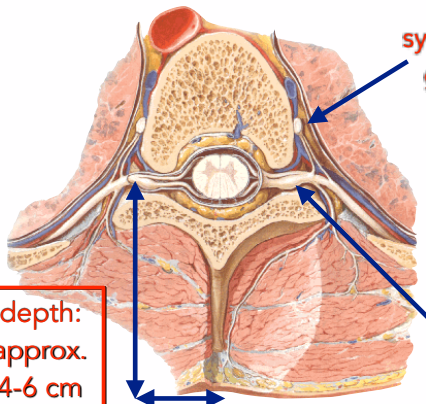
Thoracic access to DRG/VRG



sympathetic ganglion

dorsal & ventral roots

depth: approx. 4-6 cm

2 cm

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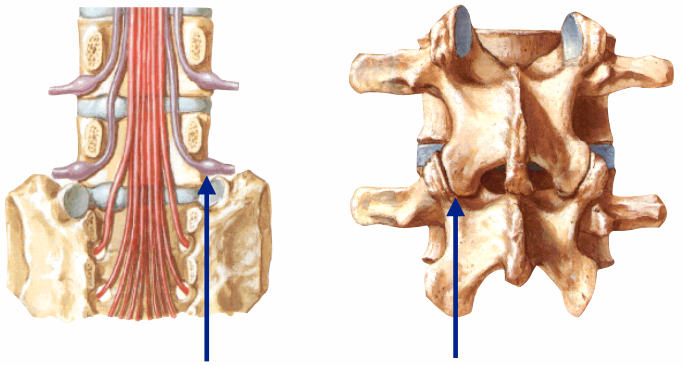
Facet joint & DRG/VRG are close



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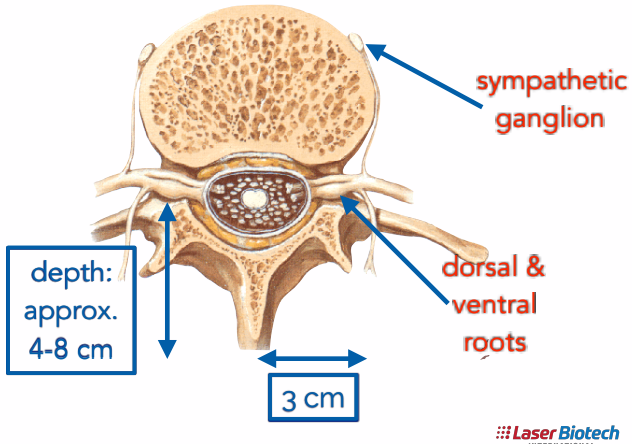
Lumbar access to nerve roots



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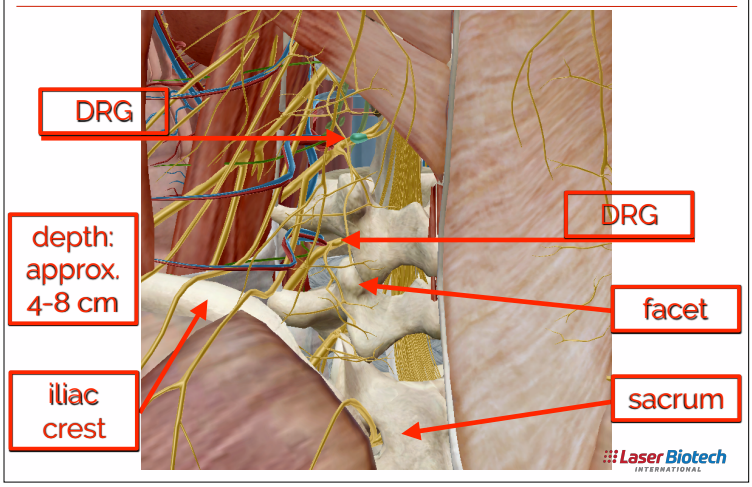
Lumbar access to DRG



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Peripheral nervous system origin of phantom limb pain

Apostol Vaso, Haim-Moshe Adahan, Artan Gjika, Skerdi Zahaj, Tefik Zhurda, Gentian Vyshka, Marshall Devor

Received 31 December 2013; received in revised form 6 April 2014; accepted 14 April 2014. published online 23 April 2014.

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

Abstract | Full Text | PDF | Images | References

Summary

Intraforaminal injection of lidocaine eliminated phantom limb pain in amputees, suggesting that its primary driver is hyperexcitable afferent neurons rather than maladaptive cortical plasticity.

Abstract

Nearly all amputees continue to feel their missing limb as if it still existed, and many experience chronic phantom limb pain (PLP). What is the origin of these sensations? There is currently a broad consensus among investigators that PLP is a top-down phenomenon, triggered by loss of sensory input and caused by maladaptive cortical plasticity. We tested the alternative hypothesis that PLP is primarily a bottom-up process, due not to the loss of input but rather to exaggerated input, generated ectopically in axotomized primary afferent neurons in the dorsal root ganglia (DRGs) that used to innervate the limb. In 31 amputees, the local anesthetic lidocaine was applied intrathecally and/or to the DRG surface (intraforaminal epidural block). This rapidly and reversibly extinguished PLP and also nonpainful phantom limb sensation (npPLS). Control injections were ineffective. For intraforaminal block, the effect was topographically appropriate. The suppression of PLP and npPLS could also be demonstrated using dilute lidocaine concentrations that are sufficient to suppress DRG ectopia but not to block the propagation of impulses generated further distally in the nerve. PLP is driven primarily by activity generated within the DRG. We recommend the DRG as a target for treatment of PLP and perhaps also other types of regional neuropathic pain.

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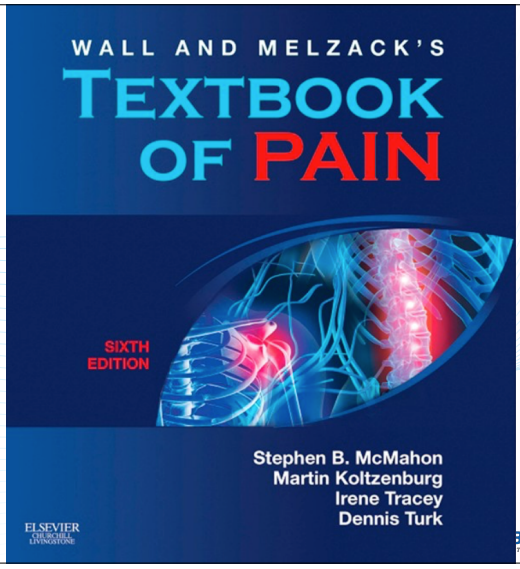
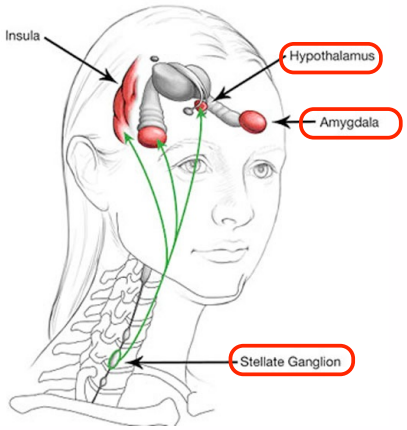


Summary

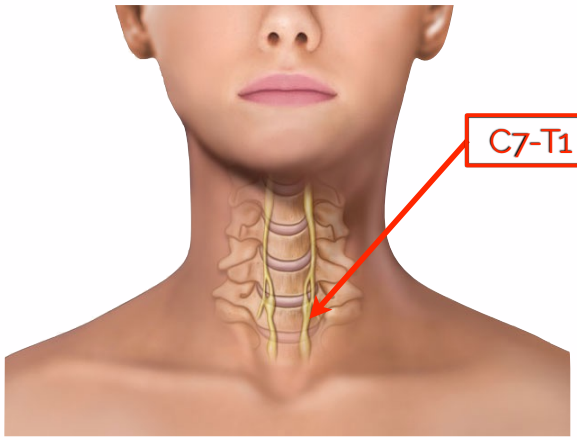
Clinically relevant forms of pain cannot be fully understood without appreciating the various forms of plasticity that develop in the spinal dorsal horn after injury or with disease. All major components of the spinal cord nociceptive network are subject to potential short- and long-term plasticity. This applies in particular to the synaptic contacts between nociceptive nerve fibers and spinal dorsal horn neurons. Synaptic strength is not static but may be enhanced or depressed for long periods. For example, inflammation and neuropathy may induce long-term potentiation at the first synaptic relay between nociceptive nerve fibers and neurons in the superficial spinal dorsal horn. Long-term potentiation is a cellular model for pain amplification. Nociceptive nerve fibers, spinal interneurons, and projection neurons communicate with each other and with spinal microglia and astrocytes. Transfer of information in spinal nociceptive circuits is under powerful segmental and supraspinal control, both inhibitory and facilitatory. Proper modulation is essential for normal nociception but may change quickly and significantly under conditions of peripheral inflammation or nerve injury. Long-term potentiation and disinhibition are two forms of neural plasticity that contribute to hyperalgesia. Disinhibition in addition causes breakdown of somatotopic and modality borders, which leads to spreading pain and allodynia. The list of relevant substances released into the spinal dorsal horn by neurons and glial cells in the course of neuropathy, trauma, or inflammation is growing rapidly, as is the ensemble of participating receptors and signaling pathways. We are now beginning to understand that depending on the primary cause of pain, the spinal nociceptive network may enter distinct modes of operation. This then leads to different forms of pain amplification, pain generation, and pain referral. The various forms of spinal dorsal horn plasticity are currently unfolding as summarized in this chapter. In-depth understanding of plasticity in spinal nociceptive pathways is a key requirement for targeting chronic pain states.

For example, inflammation and neuropathy may induce long-term potentiation at the first synaptic relay between nociceptive nerve fibers and neurons in the superficial spinal dorsal horn. Long-term potentiation is a cellular model for pain amplification.

Stellate ganglion projections



Stellate ganglion access



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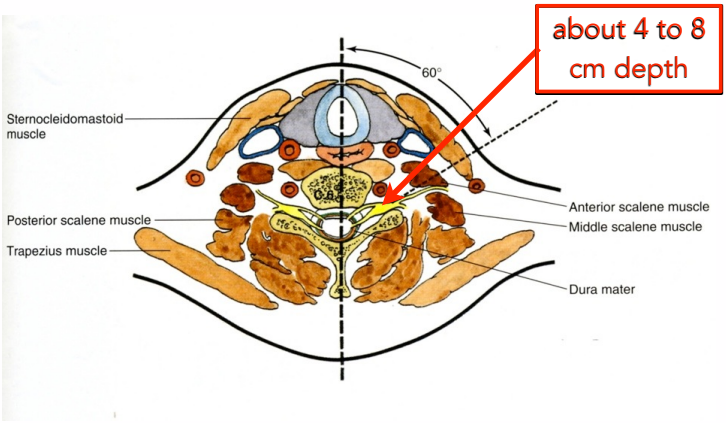
Stellate ganglion depth



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Neuropathic pain: more "treatment players"

- Thalamus and medulla
- Sensory cortex

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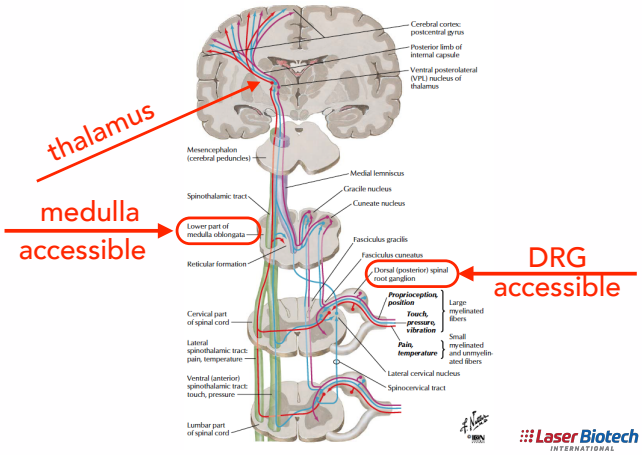
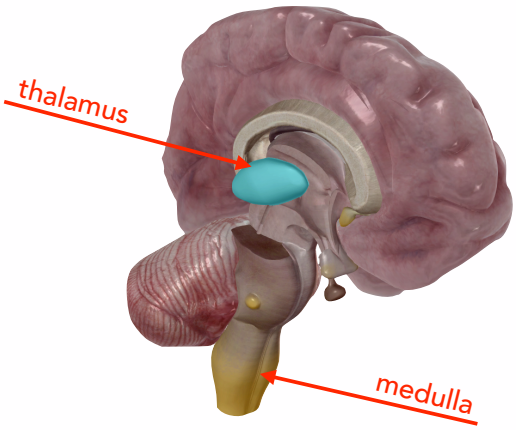
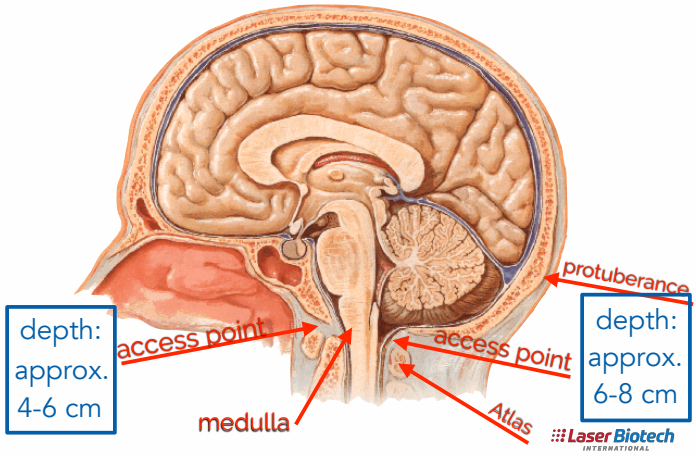
Role of DRG's and thalamus



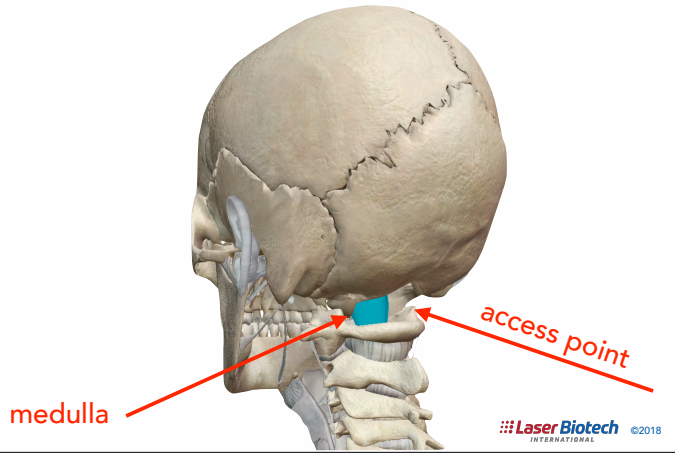
Thalamus & medulla oblongata



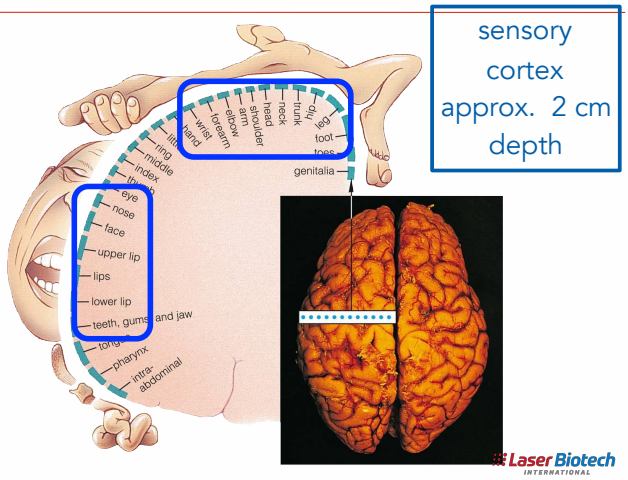
Targeting the medulla



Medulla and access point

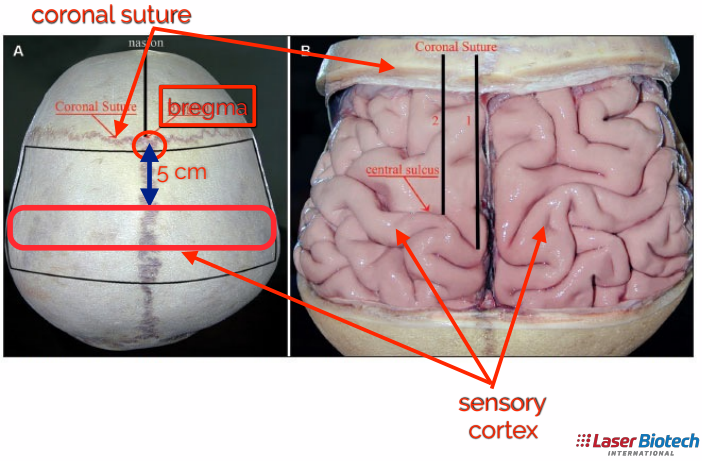


Sensory cortex: sagittal map

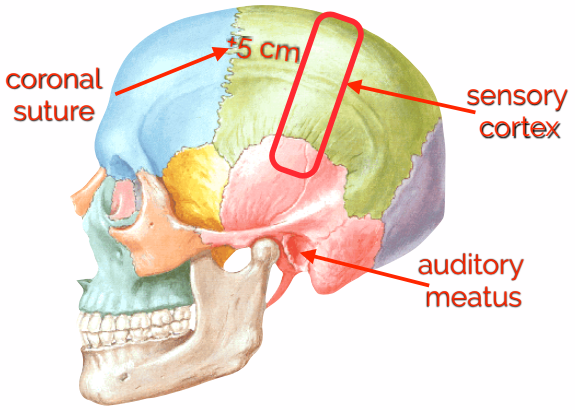


sensory cortex approx. 2 cm depth

Locating the sensory cortex



Sensory cortex landmarks



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Neuropathic and centralized pain

Physiological processes:

- Sensitization
- Centralization
- Sprouting
- Normal and dysfunctional reflexes

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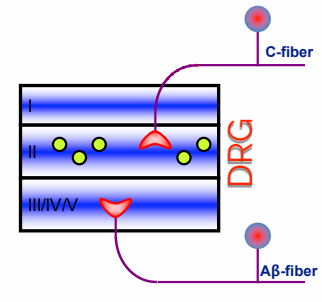
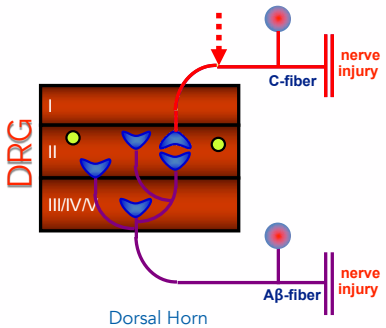
DRG remodeled: sensitization



Dorsal Horn
- Normal termination pattern



Dorsal Horn
- Sensitization / wind-up
- C-fiber terminal atrophy
- A-fiber sprouting

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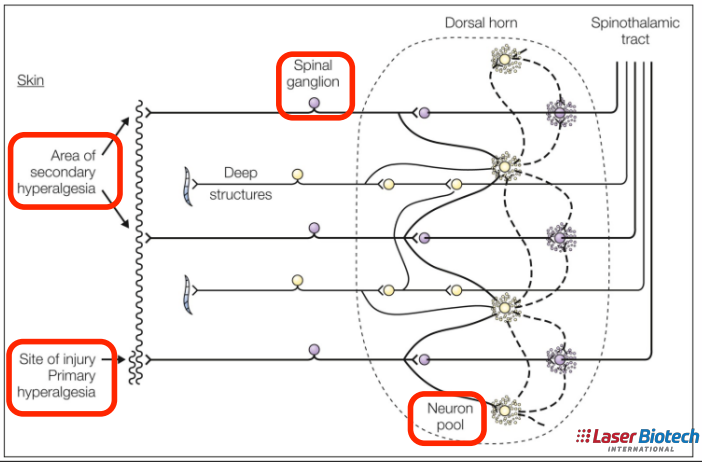
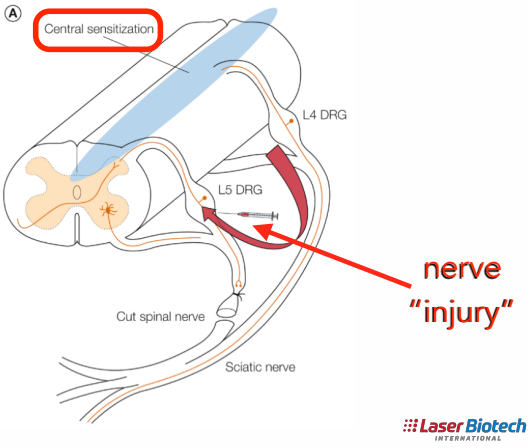
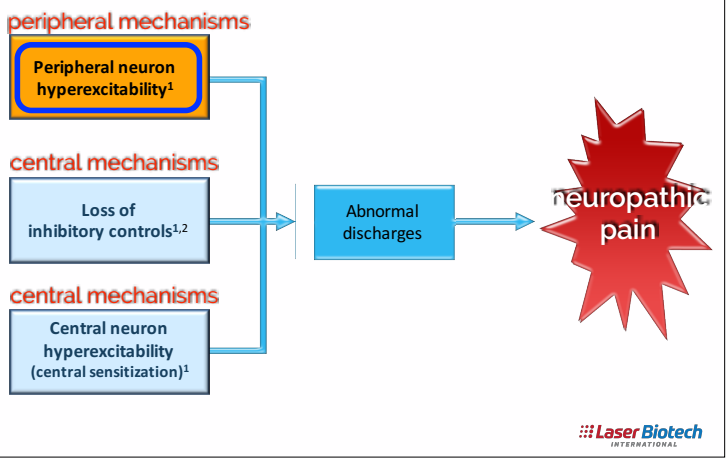
Dorsal horn spill over



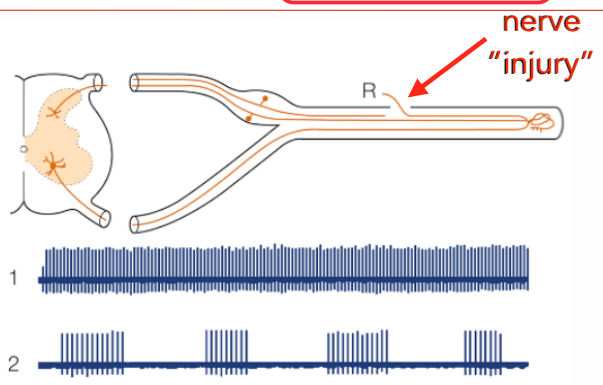
Neuropathic pain centralization



Neuropathic pain pathophysiology



Neuronal lesion causes spontaneous ectopic firing



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Effects at local tissue and CNS

- ↑ threshold activation after injury
- ↑ response to noxious stimuli
- ↑ spontaneous activity

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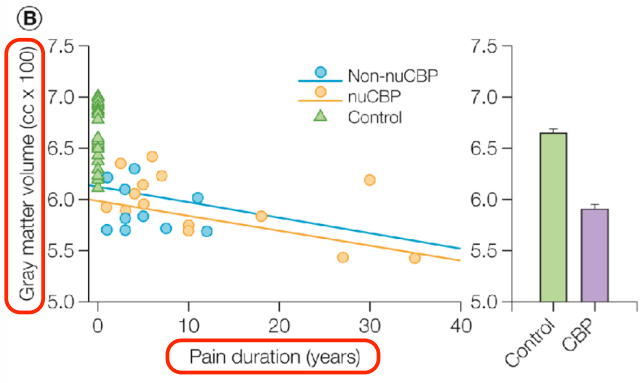
Chronic back pain effects on brain



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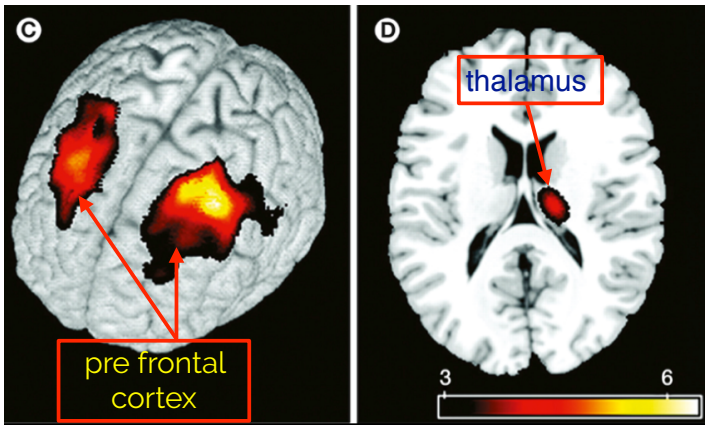
Chronic pain = shrinking brain!



What we'll cover

- Super brief review of laser therapy
- Nociceptive and neuropathic pain
- Pain management: the "treatment players"
- Treatment guidelines

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Laser treatment: 5 components

1. Local tissue
2. Nerve supply plus DRG ← chronic issues
3. Blood supply
4. Lymphatic flow (if relevant)
5. Meridian pathways (if trained)

Principle: time proximity

- A therapeutic laser produces the best clinical effect when used close to the onset of tissue injury
- Effects manifest first on acute conditions or the acute component of chronic conditions (flare-up)

Firm skin contact

Lumbar spine - push into tissue

Dr. Mathesie, DC

Using a clear barrier

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Treating joint problems: motion

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Fracture treatment

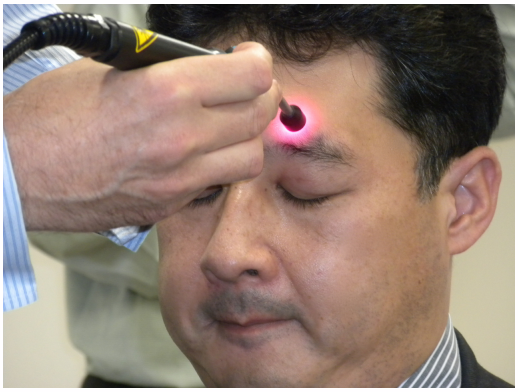
04/25/10

04/30/10

05/10/10

Mike Mathesie, DC
Lumix 2 - 45W

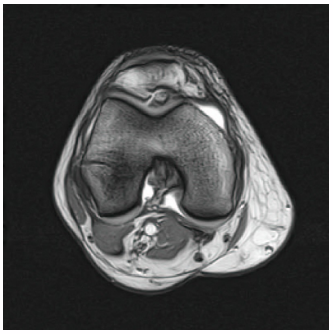
Treating sinusitis

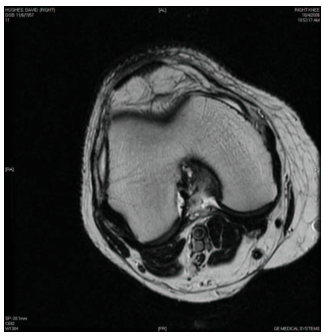


Tinnitus and sinusitis treatment

Lumix 2 - 250W
Dr. T. Lahue, DC

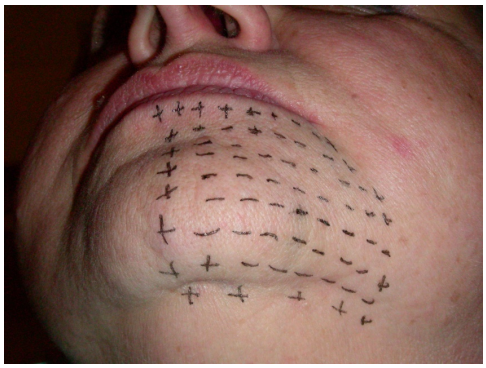
Meniscal inflammation





Andrzej Zielke, MD
Lumix 2 - 250W

Paresthesia



Paresthesia

Anatomical considerations: shoulder

Treatment done by assistants

Maria Kasper, DPM
Lumix 2 - 45W

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Correct diagnosis is essential

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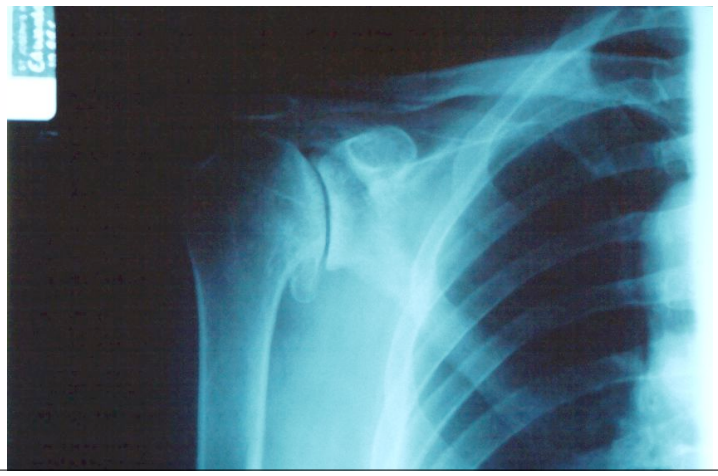
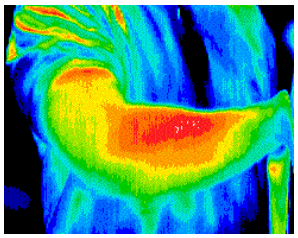
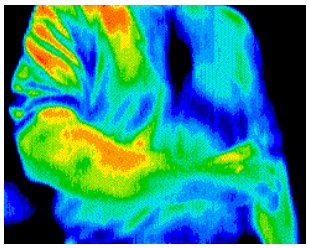
Shoulder pain: radiographic image



Elbow pain: IR thermogram

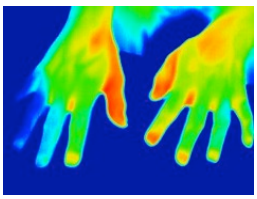


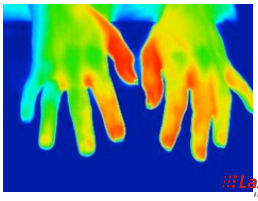


Neuropathic pain: case study

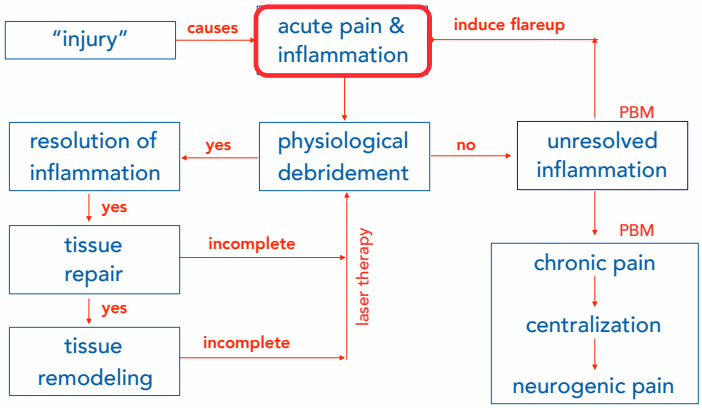
Andrzej Zielke, MD using Lumix 2 - 250W

47 years old white female with long lasting progressive RSD after multiple surgical reconstruction of right wrist and forearm. She was treated for years with stelate ganglion blocks that produced pain relief up to 24-48 hours and no long term improvement. For the last year she has been receiving laser therapy in subclavian approach once to twice per month only, due to limited resources and medical insurance denying more often treatment. The patient noticed significant pain relief lasting about a week up to 10 days associated with less pain long term and improvement in function and overall level of activity. Typically, the patient experiences almost complete pain relief within the first 3 minutes of treatment that is associated with normalization of temperature in the affected hand.
The thermographs below were taken immediately before and after laser treatment. Please notice normalization of temperature in right hand that coincided with typical pain relief.





Revisit the laser therapy landscape



Treatment guidelines

- Acute pain relief with pulse rates (PR) of 20 kHz
- Reduction of inflammation and tissue healing: PR ≥ 30 kHz and AP ≥ 0.5 W
- Induce acute flare up in treated tissue: use high pulse rates (> 40 kHz) and AP ≥ 1.0 W
- Treat the dorsal root ganglia corresponding to the target tissues
- Treat the medulla in the upper cervical
- Treat the trigeminal ganglion and subnucleus caudalis in neuropathic facial pain

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Treatment guidelines

- If you can't reach it, you can't treat it!™
- Protocols are guidelines, not recipes
- Keep it simple: 3 tissue types - target dose per tissue type
- Keep it simple: 50% rule for estimating treatment times for the target depth
- Acute inflammation is the open window - find it or activate it

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Treatment guidelines

- Acute injury and pain relief
  point and shoot
- Tissue repair and pain relief
  stoke the fires to initiate healing
- Tissue remodeling and chronic pain
  shoot the roots as well as the branch
- Neurogenic pain and pain syndromes
  pesky points and surprising segments

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Treatment guidelines

- Skin contact/proximity required - move as necessary for thermal factor
- Average power determines power density and treatment time
- Peak power and wavelength drive tissue penetration
- Power density X treatment time = dose

Treatment guidelines

- Acute treatment most profound response, with time proximity
- Chronic treatment - make it acute again!
- Specialized therapeutic effects:
  - 20 kHz or less - normalizes
  - 30 kHz or more - supports, stimulates regeneration
  - 40-60 kHz - re-initiates acute inflammatory cycle

How Lumix lasers address pain management

Lumix 2, 3 & 4

- 4 wavelengths from: 650, 810, 910, 980 or 1064 nm
- Pulse rate 1 kHz to 100 kHz
- Average powers 0.5 W to 35 W
- Pulse powers 45 W to 250 W

Lumix 2, 3 & 4

Simulated demonstration

Patient with:

- spinal fusion at L4/L5 and L5/S1
- right knee replacement surgery
- LBP with radiation
- burning pain in right foot

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Recommended resources

- Laser Biotech webinars and seminars
  - website: www.LaserBiotech.com
- North American Association for Laser Therapy
  - website: www.NAALT.org
- World Association for Laser Therapy
  - website: www.WALTA.co.za

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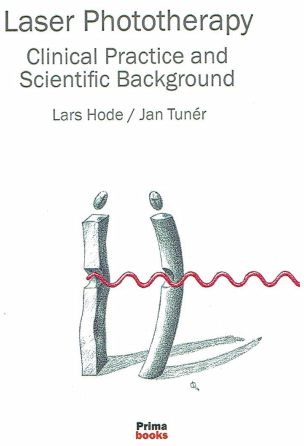
Recommended resources



Clinical science and plenty of protocols

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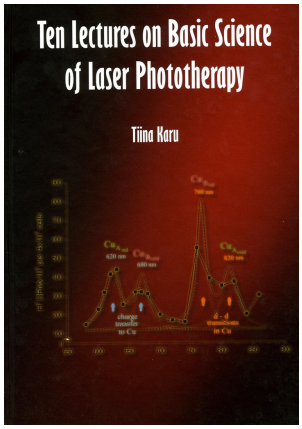
Dr. Karu: best laser researcher



NM and Tiina Karu, PhD

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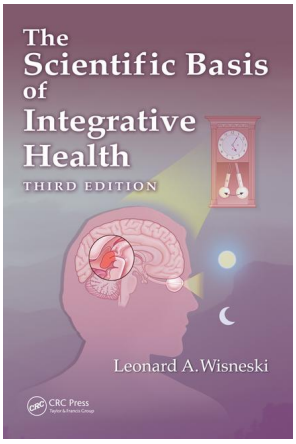
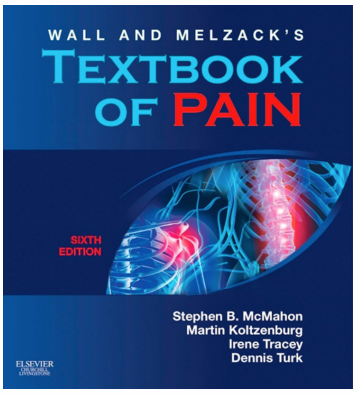
Recommended resources





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Recommended resources

- in Japanese
- soon to be translated into English

Craniofacial Pain:
protocols of laser treatments

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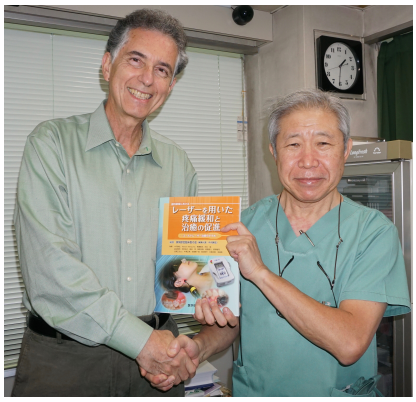
Recommended resources



NM and Dr. Katsuhiro Nakazawa, DDS, PhD

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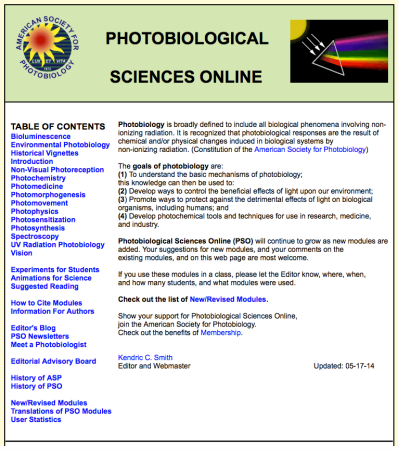
www.Photobiology.info



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(2) Develop ways to control the beneficial effects of light upon our environment;
(3) Promote ways to protect against the detrimental effects of light on biological organisms, including humans; and
(4) Develop photochemical tools and techniques for use in research, medicine, and industry.

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Editor and Webmaster

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Founder of Photobiology in the US

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Laser photobiostimulation

"discovered" as therapy by Endre Mester, MD (Hungary)

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