

# Stanford Chronic Pain Management Resident Rotation Syllabus

**Version 2009-10**

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<http://paincenter.stanford.edu>

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## **Pain Management Faculty**

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- Raymond Gaeta, M.D., Associate Professor, Medical Director, Pain Management Center
- Ravi Prasad, Ph.D., Clinical Assistant Professor, Assistant Director, Division of Pain Management
- Wendye Robbins, M.D., Clinical Assistant Professor
- Ian Carroll, M.D., Clinical Instructor
- Kale Wedemeyer, M.D., Clinical Instructor
- Vanila Singh, M.D., Clinical Assistant Professor
- Josh Kirz, Ph.D., Clinical Assistant Professor
- Tim Dawson, M.D., Clinical Assistant Professor
- Alpana Gowda, M.D., Clinical Instructor
- Meredith Barad, M.D., Clinical Instructor
- Mike Brook, M.D., Clinical Assistant Professor
- Michael Leong, M.D., Clinical Assistant Professor
- Einar Ottestad, M.D., Clinical Instructor
- Stephen Coleman, M.D., Clinical Assistant Professor
- Jiang-Ti Kong, M.D., Clinical Instructor

### **VOLUNTEER CLINICAL FACULTY**

- William Brose, M.D.
- Steven Feinberg, M.D.
- John Massey, M.D.
- Annu Navani, M.D.
- Peter Abaci, M.D.
- Rebecca Posner, M.D.

## **Goals & Objectives**

*Sean Mackey, MD, PhD*

The Pain Management Center clinic rotation is intended to provide opportunities for new patient evaluation and a continuity of care. The resident who is then supervised by one of the attending physicians evaluates patients with assorted chronic painful conditions. Clinic is held Monday through Friday during which exposure to the full range of chronic painful conditions is achieved. More complex cases are reviewed every Friday in an interdisciplinary case conference.

### **PATIENT CARE**

Goal: To gain an understanding of the domains of gathering history and physical information, making differential diagnoses, assimilation of clinical data from multidisciplinary team members for the purpose of creating an integrated treatment plan, and implementing the treatment plan.

Objectives: Upon completing this rotation, residents should understand

- How to elicit a directed neurological history and perform a detailed neurological exam
- Basic neuroimaging with an ability to identify significant findings
- How to perform a comprehensive musculoskeletal examination with emphasis on both structure and function as it applies to diagnosing acute and chronic pain problems
- How to assess for psychiatric and psychological comorbidities (e.g., chemical dependency issues, somatoform disorders, mood disturbances, personality disorders, etc.)
- The role of psychological and psychiatric treatment
- How to create and implement complex treatment plans across a variety of disciplines
- The common obstacles that often interfere with successful implementation of treatment plans and how to overcome them

## MEDICAL KNOWLEDGE

Goal: To acquire a comprehensive understanding of the essential basic and applied medical and social sciences as they relate to management of pain conditions in an outpatient continuity of care clinic.

Objectives: Upon completing this rotation, residents should understand

- The anatomy, physiology, and pharmacology of pain transmission and modulation
- The role of opioid and non-opioid medications for treatment of pain conditions
- The role of surgical and interventional treatment modalities for various pain conditions
- The role of functional, vocational, and psychological treatment modalities for various pain conditions
- The use of various strategies and techniques as a means of reducing the impact of pain on a patient's life while concurrently improving functional capacity and quality of life

## PRACTICE-BASED LEARNING & IMPROVEMENT

Goal: To be able to perform self-assessments; retrieve, understand, and apply scientific evidence related to the practice of pain medicine; and make meaningful contributions to the education of others.

Objectives: Upon completing this rotation, residents should be able to

- Identify their personal strengths and weaknesses and how this affects their practice

- Incorporate feedback from performance evaluations into daily practice
- Utilize hospital and university information technology systems to optimize learning
- Actively contribute to the education of patients, families, students, and other health professionals

## INTERPERSONAL & COMMUNICATION SKILLS

Goal: To be able to demonstrate strong interpersonal and communication (verbal and written) skills that result in effective exchange of information with patients, families, and other health professionals.

Objectives: Upon completing this rotation, residents should be able to

- Provide concise yet comprehensive communication with other health professionals
- Communicate effectively with patients, family members, and the general public across broad socio-economic and cultural domains
- Work collaboratively with other physicians and health professionals from other disciplines
- Maintain appropriate, timely, and accurate medical records

## PROFESSIONALISM

Goal: To strictly adhere to ethical principles in all aspects of practice.

Objectives: Upon completing this rotation, residents should be able to

- Demonstrate compassion and respect for patients, families, and other health care professionals
- Have a pervasive sense of personal integrity
- Demonstrate sensitivity to individuals of diverse socio-economic and cultural backgrounds

## SYSTEMS-BASED PRACTICE

Goal: To understand how the practice of pain medicine is a part of a larger context of health care organization and to be able to utilize resources from the larger system to optimize patient care.

Objectives: Upon completing this rotation, residents should be able to

- Be proficient with cost analysis and cost containment as it relates to patient care
- Consistently employ quality assurance and improvement techniques in professional work
- Work collaboratively with others to improve health care procedures and systems to positively impact patient care

## PROCEDURAL ROTATION

This aspect of the chronic pain rotation provides experience in the interventional management of pain including neural blockade, joint injection, and interventional spine therapies. Attention to the preoperative assessment for indications, alternative therapies, side effects and expected outcome are stressed.

### *PATIENT CARE*

Goal: To master the skills requisite to perform interventional pain management procedures.

Objectives: Upon completing this rotation, residents should understand

- How to conduct a thorough preoperative evaluation
- The appropriateness of various interventions among varying pain populations
- The common complications that can occur with interventional procedures and how to address them

### *MEDICAL KNOWLEDGE*

Goal: To acquire a comprehensive understanding of the essential basic and applied medical and social sciences as they relate to interventional pain management procedures.

Objectives: Upon completing this rotation, residents should understand

- The anatomy, physiology, and pharmacology of pain transmission and modulation
- The safe use of fluoroscopy for the delivery of interventional blockade
- The surgical concepts around the implantation of spinal cord stimulators and intrathecal medication delivery systems

### *PRACTICE-BASED LEARNING & IMPROVEMENT*

Goal: To be able to perform self-assessments; retrieve, understand, and apply scientific evidence related to the practice of pain medicine; and make meaningful contributions to the education of others.

Objectives: Upon completing this rotation, residents should be able to

- Identify their personal strengths and weaknesses and how this affects their practice
- Incorporate feedback from performance evaluations into daily practice
- Utilize hospital and university information technology systems to optimize learning
- Actively contribute to the education of patients, families, students, and other health professionals

### *INTERPERSONAL & COMMUNICATION SKILLS*

Goal: To be able to demonstrate strong interpersonal and communication (verbal and written) skills that result in effective exchange of information with patients, families, and other health professionals.

Objectives: Upon completing this rotation, residents should be able to

- Provide appropriate communication with OR nursing staff to ensure the highest quality of care for the patient
- Communicate effectively with patients, family members, and the general public across broad socio-economic and cultural domains
- Work collaboratively with other physicians and health professionals from other disciplines
- Maintain appropriate, timely, and accurate medical records

### *PROFESSIONALISM*

Goal: To strictly adhere to ethical principles in all aspects of practice.

Objectives: Upon completing this rotation, residents should be able to

- Demonstrate compassion and respect for patients, families, and other health care professionals
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### *SYSTEMS-BASED PRACTICE*

Goal: To understand how the practice of pain medicine is a part of a larger context of health care organization and to be able to utilize resources from the larger system to optimize patient care.

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## **Pathophysiology**

### **Cancer-related Pain**

*Theresa Mallick-Searle, NP*

#### NEUROPATHY

Chemotherapy associated neuropathy arises due to different mechanisms, including disruption of tubulin function by chemotherapeutic agents w/release of cytokines, resulting degeneration of sensory neurons and sensitization of primary nociceptive afferents.

Radiotherapy can cause tissue fibrosis with nerve compression and microvascular obstruction of the nerve.

Nervous tissue compression or lesion contributes to central sensitization.

### **METASTATIC CANCER-INDUCED BONE PAIN**

Injury or infiltration of sensory neurons that innervate the bone marrow cause pain.

Alterations in normal bone turnover occur, with loss of mechanisms that normally regulate the balance between osteoclast and osteoblast activity.

With advanced disease, the bone loses mechanical strength and is subject to osteolysis, pathological fracture, and microfractures.

Bony metastases can cause painful muscle spasm.

### **DIRECTLY TUMOR-RELATED PAIN**

Stretching of hollow visera, distortion of the capsule of solid organs, inflammation of the mucosa, and ischemia or necrosis activate visceral nociceptors, resulting in visceral pain.

Tumor infiltration in nerve plexuses and damage to nerve tissue can cause neuropathic pain.

Cancer cells can cause invasion of mechanically sensitive tissues (e.g visceral pain) or entrapment and injury of nerves (e.g. neuropathic pain).

### **CHEMICAL**

Tumors themselves secrete inflammatory and prohyperalgesic mediators.

Local and systemic inflammatory response, with production of pro-inflammatory cytokines, which facilitate pain transmission.

Tumors contain immune system cells that release factors including endothelin, prostaglandins, and TNF- $\alpha$ , which excite or sensitize peripheral nociceptive primary afferents.

## **Chronic Pain States**

### **ACUTE AND CHRONIC NECK AND LOW BACK PAIN**

*Alpana Gowda, MD*

Chou R, et al. Diagnosis and Treatment of Low Back Pain: A Joint Clinical Practice Guideline from the American College of Physicians and the American Pain Society. *Annals of Internal Medicine*. 2007;147:478-491. [PDF](#)

### **NEUROPATHIC PAIN STATES**

#### ***COMPLEX REGIONAL PAIN SYNDROMES, TYPES 1 & 2***

Sean Mackey, MD, PhD

Mackey S, Feinberg S. Pharmacologic Therapies for Complex Regional Pain Syndrome. *Curr Pain Headache Reports*. 2007;11:38-43 [PDF](#)

Harden R, Bruehl S. Diagnosis of Complex Regional Pain Syndrome. *Clin J Pain*. 2006;22:415-419. [PDF](#)

### *POSTHERPETIC NEURALGIA*

Wendye Robbins, MD

Basbaum A, Julius D, Robbins W. Vanilloids. In: Bountra C, Munglani R, Schmidt W, eds. *Pain: Current Understanding, Emerging Therapies, and Novel Approaches to Drug Discovery*. New York, NY: Marcel Dekker; 2003:531-538. [PDF](#)

### *PHANTOM LIMB PAIN*

Meredith Barad, MD

#### **Incidence**

60-80% of patients experience phantom limb pain almost immediately after the loss of a limb.

#### **Onset:**

75% of phantom pain patient experience onset within the first week while 25% of in the weeks, and rarely in the years, following.

#### **Duration:**

Usually fades in days to weeks, but in 30% of patients can persist for years.

#### **Quality:**

Episodic pain with multiple daily events lasting seconds to minutes. The pain has been reported as stabbing, shooting, boring, burning, like the limb is stuck in a block of ice. This pain is not to be confused with stump pain which is often due to a scar neuroma and present in 10-25% of amputees.

#### **Phantom Sensations:**

NOT PAIN. This is often described as the feeling that the limb is still present, usually more vivid in the most distal part of the extremity which may reflect the larger cortical representation of the more distal part of limb. The most common sensation is telescoping. In 50% of cases involving the upper limb, the arm will get progressively shorter until the patient is left with the hand alone dangling from the stump. These limbs can telescope sometimes at will. Paresthesias have also been reported.

#### **Risk Factors:**

pre-amputation pain, phantom sensations.

Less frequent in children and adolescent amputees and patients with congenital limb defects. The phantom limb is not affected by the reason for surgery, the limb or the location on the limb of the amputation, sex, age, marital or socioeconomic status of the patient.

#### **Position:**

The phantom usually occupies the habitual position – such as elbow partially flexed and forearm pronated. The phantom can occupy an unusual even painful position, that can sometimes be reset by movement of the stump.

**Treatment:**

There is a paucity of well-designed trials, but basically the same principles used for central neuropathic pain apply.

**Medications:**

TCAs, Gabapentin, other AEDs, Opiates (consider Methadone), NMDA antagonists.

**Procedures:**

SCS, DBS of the ventral caudal nucleus of the thalamus and MCS have all been shown to be effective in small numbers of patients. Non-pharmacologic therapy: Mirror treatment, desensitization

**Peri-operative management:**

Epidural (these patients have been shown to have considerably less pain at 6 months) Nikolajsen et al. 1997. Consider preoperative gabapentin.

**References:**

F. Cervero, T.S. Jensen, Editors. *Handbook of Clinical Neurology*, Vol. 81 (3rd series). Chapter 45. *Pain*. 2006

Nikolajsen L, Ilkjaer S, Christensen JH, Kroner K, Jensen TS. Randomised trial of epidural bupivacaine and morphine in prevention of stump and phantom pain in lower-limb amputation. *Lancet* 350: 1353–1357.

Katayama Y et al. Motor cortex stimulation for phantom limb pain: comprehensive therapy with spinal cord and thalamic stimulation. *Stereotact Funct Neurosurg*. 2001;77(1-4):159-62.

**PERIPHERAL NEUROPATHIES (E.G., DIABETIC NEUROPATHY)**

*Ian Carroll, MD, MS*

Hocking G, Cousins M. Ketamine in Chronic Pain Management: An Evidence-Based Review. *Anesth Analg*. 2003;97:1730-9. [PDF](#)

**SOMATIC PAIN CONDITIONS****FACET ARTHROPATHY**

*Jiang-Ti Kong, MD*

**Anatomy and innervations**

The basic anatomical unit of the spine is consisted of three joints: the intervertebral disc, and the paired zygapophysial (or facet) joints (Fig 1a). The three joints function together to support and stabilize the spine and limit motion in all planes.[1] The upper lumbar facets (L2-3, L3-4) are in a more sagittal plane than coronal; thus protecting the spine from excessive axial rotation and lateral bending. The lower lumbar facets (L4-5, L5-S1) are just the opposite, with a more coronal orientation.

As people age, the overall orientation of the lumbar facets becomes more sagittal, the consequence of which is less protection from the shearing force caused by flexion and extension.[2] The orientation of

the individual facet may also differ at the same spinal level, referred to as tropism. Tropism is positively associated with disc degeneration and herniation.[3] Furthermore, as the discs begin to degenerate and shrink in height, more loads are transmitted to the facet joints, eventually leading to their degeneration as well.

The zygapophysial joints are true synovial joints, consisted of the superior and the inferior articulating process (SAP and IAP), from the spinal level below and above; and the joint capsule.[4] The facet joints are innervated by medial branch nerves, which are branches of the dorsal primary rami of the spinal nerves (Fig 1b). Each medial branch sends out a proximal ascending and a distal descending branch. The former innervates the caudal aspect of the facet, while the latter innervates the superior and medial aspects of the facet at a level below. Hence, each facet joint is innervated by two medial branches, one from the same level, and one from a level above.

### **Clinical Presentation and Diagnosis**

Lumbar facet pain often refers to the low back, buttock, and thigh. Less commonly it may involve the groin and the flank (Fig 2), along with diffuse crampy leg pain and stiffness. Patients with facet arthropathy often display the following signs: 1) Paraspinal tenderness; 2) pain on facet joint loading maneuvers (axial rotation, lateral bending, flexion /extension); 3) Absence of signs of nerve root irritation; 4) Non-radiating back, +/- hip, +/- buttock pain on straight leg raising.[4]

The diagnosis of lumbar facet arthropathy is not intuitive. Even though many signs and symptoms are ascribed to facet arthropathy, multiple large, randomized, well-controlled trials showed that NO historic or physical examination findings can reliably diagnose facet arthropathy.[5] Furthermore, the evidence in the literature does not support the use of any radiographic imaging (x-ray, CT, or MRI) to diagnose facet pain.[6] In fact, the agreed method to diagnose lumbar facet arthropathy is response to facet blocks: intra-articular or medial branch blocks (MBB). Although both blocks are frequently used, the evidence supporting the use of MBB is much stronger as judged by quality and number of clinical trials.[1]

Medial branch blocks are performed using fluoroscopy, with the x-ray beam 10-40 degrees ipsilateral oblique.[7] The target is usually taken at the junction between the transverse process and the SAP (Fig 3). Half a milliliter to 1mL of local anesthetic plus 10-20mg of methylprednisolone is injected to each medial branch via a 22G or 25g needle. The pain relief from MBB comes in two phases: early phase which happens within ½ hour due to the local anesthetic, and a late phase which takes place within one week of the injection. The duration of the relief is often limited, roughly about one month. Given the high false positive rate of MBB (25-40%), two consecutive positive responses to MBB are required before proceeding to radiofrequency ablation (RFA) of the medial branches.

### **Treatment**

A multimodal approach to the treatment of facet arthropathy is recommended.[1] It involves physical therapy, medications, and psychological interventions. Tailored exercise programs combined with yoga have been shown to reduce pain and prevent relapses in patients with chronic low back pain. In terms of medications, NSAIDS, and the judicious use of antidepressants and muscle relaxants are advocated. Untreated psychiatric disorders including PTSD, depression, anxiety and substance abuse adversely

affect the outcomes of patients with chronic low back pain. It is therefore critical to address these disorders while treating pain.

Evidence from literature supports radiofrequency ablation (**RFA**) of medial branches (MB) as the **interventional treatment of choice** for facet arthropathy.[1] The fluoroscopic technique for RFA is identical to that of MBB.[8] However, rather than injecting medications, it uses radiofrequency waves to denervate the medial branch. Before the ablation, the location of the needle is verified by sensory stimulation at 50Hz. If the probe is sufficiently close to the medial branch, the patient should feel the stimulation at less than 1V (some uses 0.5V). Next, motor stimulation at 2Hz is performed up to 2-2.5V, at which point only the multifidus (paraspinal) muscles are supposed to contract, and NOT the muscles in the girdles and /or limbs.

Two types of RFA are performed: continuous and pulsed. The continuous technique was the original technique used for RFA since the 1970's. It works by heating tissues to 60-100°C, which causes near instantaneous coagulation necrosis of the medial branch, essentially a heat neurectomy. In contrast, the pulse RFA delivers bursts of radiofrequency waves at 42°C for 20ms with 480ms silence time in between, without causing neuronal death. It is thought that the pulsed RF works by using a shifting electromagnetic field around the medial branch to modify pain processing mechanisms at the spinal and supraspinal level. Direct head-to-head comparison of continuous vs. pulsed RF showed that both methods work to reduce pain. However, there is significant difference in the duration of analgesia: the continuous RF results in up to one year of pain reduction, while the pulsed RF ≤ 4 months. The recommendation is thus continuous RF for lumbar medial branch ablation, and reserve pulsed RFA for neuropathic type of conditions mediated by peripheral nerves.[1]

Cautions and risks: radiofrequency ablation procedures are in general safe. The most common side effects from RFA is reactive flaring of pain right after the ablation procedure, which can be treated by injecting steroid via the needle immediately after ablation.[4] The administration of steroid itself carries certain risks, including suppression of the hypothalamic-pituitary-adrenal axis up to 4 weeks, impaired insulin sensitivity up to 1 week, as well as increased risk for infection. If appropriate sensory and motor tests are performed, the risk of injuring other nerves is minimal.

*Cervical facet syndrome:* The pathophysiology, presentation, diagnosis and treatment of cervical facet arthropathy largely mirror that of the lumbar facet disease, with the following exceptions: [4]

1. *Presentation:* The cervical facets refer pain to the occiput, neck, shoulder and upper arm (Fig 4). Typical signs of cervical facet disease include decreased range of motion in cervical spine, local tenderness over spinal process and affected facets, and pain upon extension and ipsilateral bending (i.e. facet loading maneuver). Of particular mention is the third occipital nerve, the medial branch of C3, which innervates the C2-3 facet. C2-3 arthropathy often contributes to the development of various headaches that involve the occiput.
2. *Diagnosis:* cervical facet joint injections are NOT recommended due to technical difficulties, the increased risk of injuring the vertebral arteries and exiting nerves, as well as the lack of positive clinical trials. Cervical MBB is the recommended technique for diagnosis and short term treatment of facet disease. It may be done via either an anterior-posterior or lateral approach (Fig 5a and 5b).

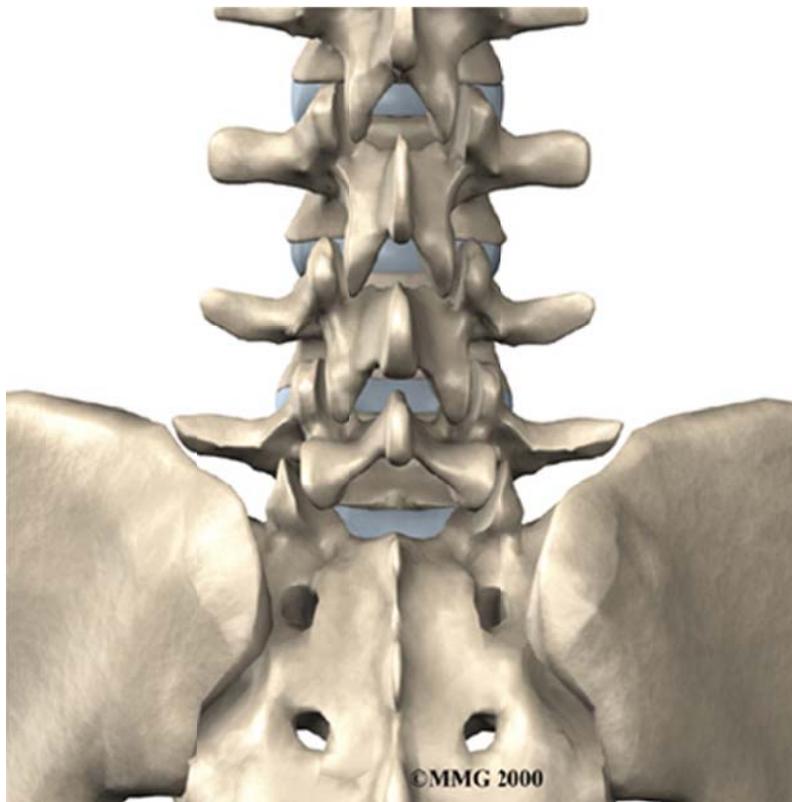
3. *Treatment*: radiofrequency ablation is again the interventional treatment of choice. The efficacy of the traditional continuous RFA was shown to last between 9 and 12 months by two well controlled, randomized, prospective trials.

## Conclusion

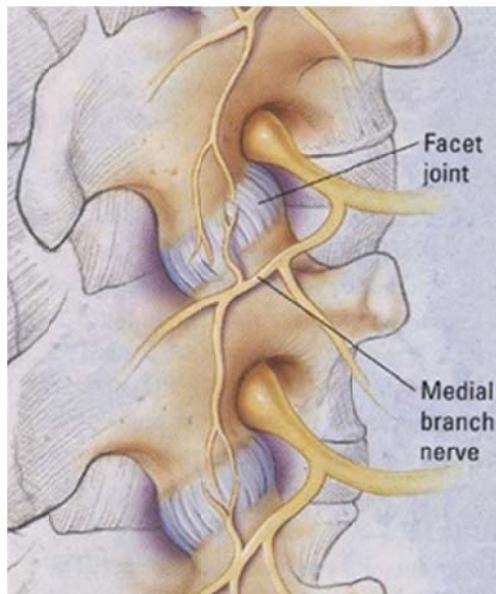
Facet arthropathy is a common etiology that leads to axial neck and low back pain. It is diagnosed via medial branch blocks. The definitive interventional treatment is continuous RFA of the medial branches. It is also important to approach patients with facet disease via a multimodal treatment approach in addition to the invasive procedures to prevent relapses.

1. Cohen, S.P. and S.N. Raja, *Pathogenesis, diagnosis, and treatment of lumbar zygapophysial (facet) joint pain*. *Anesthesiology*, 2007. **106**(3): p. 591-614.
2. Boden, S.D., et al., *Orientation of the lumbar facet joints: association with degenerative disc disease*. *J Bone Joint Surg Am*, 1996. **78**(3): p. 403-11.
3. Vanharanta, H., et al., *The relationship of facet tropism to degenerative disc disease*. *Spine (Phila Pa 1976)*, 1993. **18**(8): p. 1000-5.
4. Clemans, R.R., Benzon, H.T., *Facet Syndrome: Facet Joint Injections and Facet Nerve Blocks*, in *Essentials of Pain Medicine and Regional Anesthesia*, S. Benzon, Molloy, Liu, Fishman, Editor. 2005. p. 341-347.
5. Revel, M., et al., *Capacity of the clinical picture to characterize low back pain relieved by facet joint anesthesia. Proposed criteria to identify patients with painful facet joints*. *Spine (Phila Pa 1976)*, 1998. **23**(18): p. 1972-6; discussion 1977.
6. Cohen, S.P., et al., *Clinical predictors of success and failure for lumbar facet radiofrequency denervation*. *Clin J Pain*, 2007. **23**(1): p. 45-52.
7. Czervionke, L.F., Fenton, D.S., *Facet Joint Injection and Medial Branch Block*, in *Image-Guided Spine Intervention*, a.Z. Fenton, Editor. 2003, Saunders: Philadelphia, PA.
8. Fenton, D.S., Czervionke, L.F., *Facet Denervation*, in *Image-Guided Spine Intervention*, a.Z. Fenton, Editor. 2003, Saunders: Philadelphia, PA.

**Figure 1a.** Anatomy of Lumbar Facets

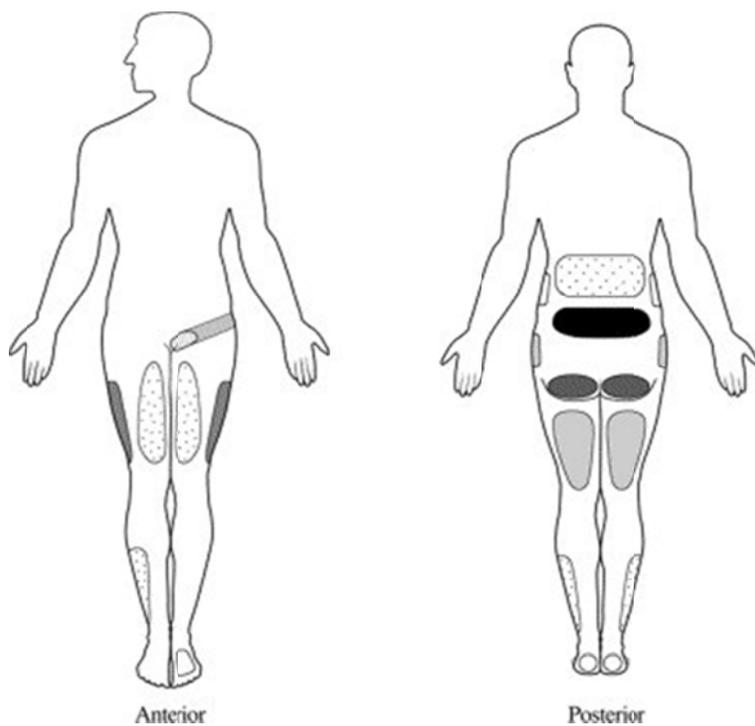


**Figure 1b.** Anatomy of Spinal Medial Branches

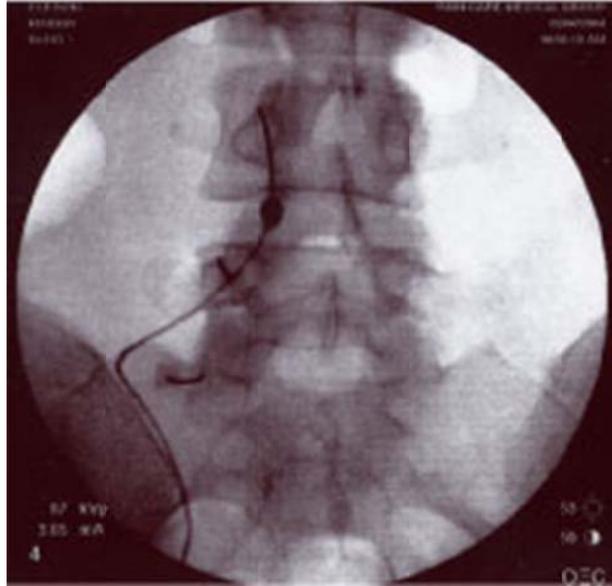


**Figure 2:** Referral Patterns of Lumbar Facet Arthropathy. In descending order, the most common referral patterns extend from the **darkest (low back) to the lightest regions (flank and foot).**

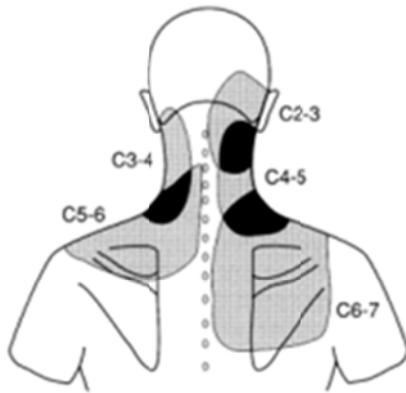
Adapted from Cohen and Raja 2007.



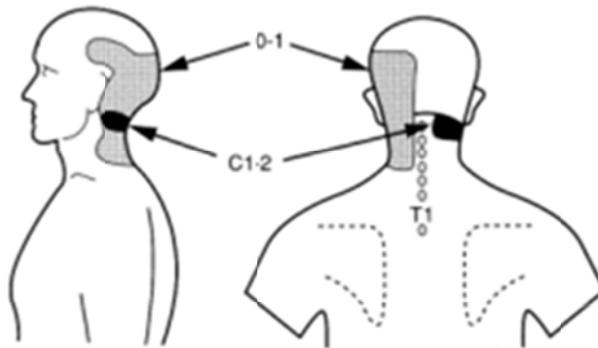
**Figure 3.** Radiography for Lumbar Medial Branch Blocks at Left L4, L5, ala. Notice the Target is at the junction between the superior articulating process (SAP), and transverse process (TP). This picture is taken after aligning the endplates at L4-5, and oblique the beam toward the left about 10-20 degrees.



**Figure 4.** Referral Patterns for Cervical Facet Arthropathy, Adapted from [www.weblockpain.com](http://www.weblockpain.com)

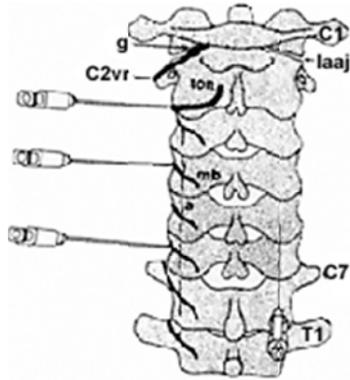


**Pain Referral Patterns from Cervical C2-3 through C6-7 Facet Joint Injections.** Shaded areas indicate areas of pain experienced by asymptomatic volunteers after injection of facet joints C2-3 through C6-7. (From Dwyer AB, Aprill C, Bogduk N. Cervical zygapophyseal joint pain patterns. I: A study in normal volunteers. *Spine* 1990; 15:453-457.)



**Pain Referral Patterns from the Atlanto-occipital (O-C1) and Lateral Atlantoaxial (C1-2) Joints.** The larger, lightly shaded area is the location of pain induced by injection of the atlanto-occipital (O-C1) joints of asymptomatic volunteers, and the more darkly shaded areas are from similar injections of the lateral atlantoaxial joints (C1-2). (From Dreyfuss P, Michaelsen M, Fletcher D. Atlanto-occipital and lateral atlanto-axial joint pain patterns. *Spine* 1994; 19:1125-1131.)

**Figure 5a.** Cervical MBB Approached by Anterior-Posterior Orientation of the Fluoroscope



**Figure 5b.** Cervical MBB Approached via Lateral Orientation of the Fluoroscope



### **Definition and Diagnosis:**

Myofascial pain (MP) is local and referred pain that arises from myofascial trigger points. Trigger points are defined as localized sensitive areas in muscles that contain palpable, taut bands of muscles.[1] Palpation of trigger points often reproduces the patient's pain which often radiates and refers. Myofascial pain differs from fibromyalgia in location, pathogenesis and treatment (see chapter on Fibromyalgia).

MP is quite common and appears in up to 30% of patients who presents to orthopedic or general medical clinics. It tends to affect women slightly more often than man.

### **Differential Diagnosis:**

MP is a relatively common and benign disorder, which can be confused with other more serious local and systemic diseases.[1] It is therefore important to rule out these other disorders before making the diagnosis of myofascial pain. Local disorders that may mimic MP include arthritis, bursitis, fasciitis, tendinopathy, neuropathy, and referred visceral pain. Systemic disorders include endocrine disorders (hypothyroidism, Cushing's disease and hyperparathyroidism); infections (hepatitis C, lyme disease etc); and inflammatory disorders (RA, SLE, PMR, Sjogren's and myositis).

### **Pathogenesis:**

Myofascial pain often results from acute injury, or, more commonly repetitive strain from abnormal posture and non-ergonomic body mechanics. These injuries can be acute or chronic. In some cases no obvious causes are found.

Although the details are not entirely clear, a generally accepted concept is that pathologic increase in acetylcholine release by motor endplates leads to prolonged muscle contraction, strain, and ischemia.[2] Ischemia in turn results in release of local vasoactive mediators which then further increase acetylcholine release, creating a positive feedback loop for myofascial pain. This model also explains why botulinum toxin (Botox), which irreversibly blocks the release of acetylcholine, is helpful in treating MP (see below).

### **Treatments:**

1. **Non-invasive physical modalities:** The cornerstone of all treatment approaches to myofascial pain is slow and sustained muscle stretching exercises.[1] Paradoxically, the contracted, painful muscle also tends to be weakened, hence requiring a strengthening program as well. The literature supports herapeutic exercises for the neck, low back and knee pain. Other modalities, such as TENS and relaxation therapy, are also useful if indicated
2. **Injection therapies:** Both trigger point injections and dry needling appear to be helpful for MP, although data suggests injection therapy results in more improvement than dry needling.[3] The medication used for injection, i.e. saline, lidocaine, bupivacaine, or botox did not seem to matter per several previous trials. However, recent data have shown that Botox may be more efficacious than the others.[4, 5]

3. **Medications:** Few randomized controlled trials exist that evaluate the efficacy of pharmacotherapy for MP, partly because this order is more commonly treated by physical modalities and injections. Based on trials in patients with low back pain, arthritis and tension headaches, NSAIDs, tricyclic antidepressants (if insomnia and depression), and tizanidine may be helpful as adjuvants in treating MP.[1] There is some data supporting tramadol, which is not the author's choice due to potential for addiction and interaction with other antidepressants (may cause seizure and serotonin syndrome).

### **Outcome summary:**

Myofascial pain deserves the attention of the physician because treatment outcome is often not ideal. For example, Cassisi et al found that patients with myofascial low back pain do worse than those with disc herniation.[6] They tend to harbor inaccurate beliefs about their pain condition and pessimism regarding their treatment. Therefore, a multidisciplinary program is often necessary to treat patients with MP, addressing emotional, ergonomic, rehabilitational and pharmacologic aspects of their disease.

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## *FIBROMYALGIA*

*Jiang-Ti Kong, MD*

### **I. Diagnosis and Epidemiology:**

- a. Diagnosis: The diagnosis of fibromyalgia (FM) is clinical and includes the follow two elements as required by the College of American Rheumatology:[1]
  - i. Chronic widespread pain (CWP) of at least 3 months' duration, present above and below the diaphragm, on both sides of the body, plus axial pain.

- ii. Tenderness in at least 11/18 characteristic locations (Fig 1) when 4 kg/cm<sup>2</sup> is applied (roughly the pressure that results in the blanching of the nail bed).
  - iii. It differs from myofascial pain, which is local (Table 1)
- b. Epidemiology: The prevalence of fibromyalgia is estimated to be between 0.5 to 5% of the population. There does not seem to be a race, national, or ethnic bias. However, it tends to occur in women between the ages of 20 and 50 years. The female to male ratio is 10:1.[1]

## II. Associated Symptoms:

In addition to the CWP and tenderness, fibromyalgia is often associated with several symptoms including fatigue, sleep disturbance, mood disorders, and discognition.[2]

Several large patient survey studies from the US and Germany (all published in 2008) ranked fatigue at the top of the list of patient-perceived major manifestations of fibromyalgia (Table 2), second only to pain and stiffness. Non-restorative sleep (NRS) is the second major complaint of FM patients. It was initially thought secondary to alpha intrusion into the delta rhythm of sleep, which prevents the progression into the restorative stages 3 and 4 of Non-REM sleep (aka slow wave sleep). Mood disorders, particularly depression, anxiety, and PTSD are also present in up to 74% of patients with FM. Lastly, cognitive dysfunction (referred to as “fibro-fog” by some) is common in FM patients. It manifests in areas including working memory, concentration, motivation and other executive tasks. Some estimated this overall cognitive decline to be equivalent to 20 years of aging.

Other symptoms, such as subjective joint swelling, allodynia/hyperalagia, paresthesia without obvious EMG/NCS findings, are also common in FM patients.[2]

## III. Coexisting pain disorders:

Patients with FM also tend to have other chronic pain syndromes such as: irritable bowel syndrome, interstitial cystitis, TMJ, and migraines which also involve central sensitization as part of their etiology (see below). Other associated syndromes include, but not limited to: restless leg syndrome, chronic fatigue syndrome and Raynaud phenomenon.[1]

## IV. Pathophysiology:

Currently, we do not have sufficient knowledge to identify the exact mechanisms leading to fibromyalgia. However, mounting evidences have suggested abnormalities in pain processing, particularly the **amplification of ascending** nociceptive signals, and the **reduction in descending** pain inhibition are involved in the development of FM.[3, 4]

- a. Ascending pathway: simply speaking, the ascending nociceptive pathway include primary afferent neuron whose cell body resides in the DRG, spinal secondary neurons, thalamic tertiary neurons, and finally cortical and subcortical neurons (Fig 2). In FM, abnormally high levels of pro-nociceptive mediators are found at the primary and spinal secondary neuron synapses,

including substance P, NGF, glutamate and prostaglandin. Their presence leads to hyperexcitability of the primary afferent neurons and abnormal transformation of spinal secondary neurons to wide dynamic range neurons. These changes are then lead to the development of hyperalgesia and allodynia, respectively.

- b. **Descending Pathway:** it refers to the neurocircuit connecting cortical structures (somatosensory and frontal cortex) to hypothalamus, the periaqueductal gray and other midbrain/pons structures (e.g. raphe magnus), which then arrives at and modifies the inputs from the synaptic junction between the spinal secondary and primary afferent neurons. Experimental evidences have shown the concentrations of biogenic amines (5HT, DA, NE) in the CSF are decreased in FM patients. These biogenic amines are considered major neurotransmitters responsible for descending pain inhibition.
- c. **Additional mechanisms:** neuro-endocrine abnormalities, including growth hormone deficiency (present in 1/3 of FM patients), hypothyroidism, and decreased level of cortisol were also thought related to the pathogenesis of FM via a central process.[1] However, most of these deficiencies are mild and multiple small clinical trials with hormonal replacement did not demonstrate sustainable benefit.[5] The current recommendation is therefore: no hormonal treatment unless clinically significant deficiency is present.

## V. **Treatment:**

The treatment of fibromyalgia needs to be comprehensive, addressing the myofascial, CNS, emotional and physical aspect of this complex disease. **The three cornerstones FM thus include: medications, exercise, and psychological interventions.**[1]

### 1. **Medications.**

- a. **The FDA approved drugs:** Three drugs are currently approved by the FDA for fibromyalgia: **pregabalin (lyrica), duloxetine (cymbalta), and milnacipran (savella).**[6] Pregabalin primarily works on the inappropriately activated ascending nociceptive pathway while duloxetine and milnacipran increase the concentration of biogenic amines to enhance the descending inhibition. For details on the mechanism, indications, dosage and major side effects of these medications, please refer to Table 3a.
- b. **Other medications (Table 3b):**
  - i. A single controlled trial showed efficacy of **gabapentin** (neurontin) in FM related pain, function and sleep disturbance. The median effective dose 1800mg per day.[6] Gabapentin is structurally similar to pregabalin; but pregabalin does not require a protein transporter to be absorbed, is analgesic at a lower dose, and easier to titrate.
  - ii. **Tricyclic antidepressants** (amitriptyline, nortriptyline, and desipramine) inhibits the reuptake of serotonin and norepinephrine at synapses, also helps with restorative sleep. However, their use is often limited by anticholinergic side effects.

- iii. Cyclobenzaprine, a tricyclic muscle relaxant, and sodium oxybate, a GABA precursor, were also shown to be moderately helpful.
- iv. NSAIDS and opioids were found NOT to be helpful for FM. Tramadol, however, was shown to be a moderately beneficial adjunct.

**c. Typical algorithms:**

- i. First drug selection: is based on the patient's symptoms, as well as cost. Often a second generation tri-cyclic such as desipramine or nortriptyline is selected if the patient has significant depression. Another choice would be gabapentin if sleep is a problem. Alternatively, lyrica, cymbalta or savella may be used if financially feasible.
- ii. Dose escalation: most of the above drugs are not therapeutic at the initial dose and needs to be escalated. The patient should remain at the therapeutic target dose for at least 4 weeks before deciding the drug ineffective.
- iii. Drug combinations: if the first drug does not work, one may choose to wean it off and try a second one; or adding a second to the first agent. Recent data showed that the combination of nortriptyline plus gabapentin is more effect than either alone for neuropathic pain. This may be applicable to FM.
- iv. Novel agents: **Naltrexone** 4.5mg po qhs may be used by itself or in combination with another drug. It may decrease neuro-inflammation at the glial cell level and is efficacious in certain FM patients.[7]

2. **Exercise Interventions:** Exercise has been proven to be beneficial to patients with fibromyalgia in multiple randomized controlled trials.[8] However, due to their altered physiology in pain processing and abnormal response to endorphins, FM patients often have increase pain and thus poor tolerance with standardized exercise regimens. Their **exercise program thus needs to be tailored**. Below are recommendations from a recent review article:[8]

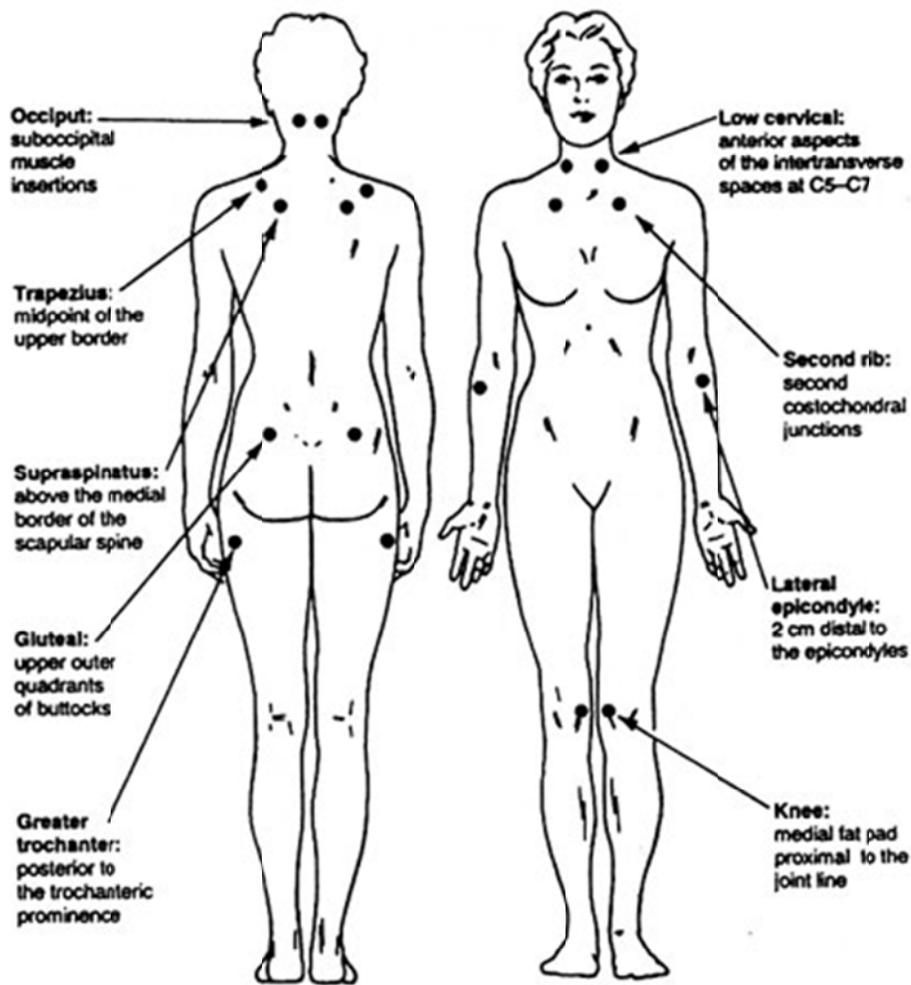
- a. Goals: low-intensity non-repetitive, low-impact, individualized programs are preferred.
- b. Examples: warm water-based gentle aerobics, mixed-type exercises; walking, stationary bikes, posture and flexibility (i.e. yoga, Qigong).
- c. Recommended progression: 1) breath, posture, and relaxation training; 2) flexibility; 3) strength and balance; 4) aerobics (aquatics, walk, bike etc)
- d. Addressing comorbidities: treat sleep and mood disorders; diet counseling for obesity, evaluate balance and kinesthetic training for those with autonomic instability.
- e. Avoid: heavy weight, excessive repetition, eccentric muscle works. Realize the average 40-year-old patient who has FM has the physical stamina of and 80-year-old without FM.

3. **Psychologic Interventions:**[9]

- a. Interventions with definitive evidence for efficacy:
  - i. **Patient education:** involves explaining the neuropathophysiology of fibromyalgia; the biopsychosocial model of chronic pain; and de-stigmatization of FM.
  - ii. **CBT:** cognitive therapy aims at modifying maladaptive thoughts to change affect and behavior; while behavior therapy uses the operant behavior technique to reward adaptive behavior (e.g. pacing, graded exercise) and punish maladaptive behavior (e.g. secondary gain, catastrophizing). **Relaxation techniques** are often utilized in CBT.
  - iii. **Biofeedback:** multiple randomized controlled trials support the use of heart-rate-biofeedback; while more data is needed to support EMG- and EEG- biofeedback.
- b. Interventions with equivocal evidence for efficacy: meditation alone, and Qigong. Hydrotherapy (i.e. warm spa) was found to be helpful only short term.
- c. Interventions with minimal efficacy: acupuncture, massage.

## VI. **Conclusion:**

Fibromyalgia is a clinically diagnosed disease with chronic wide-spread pain involving bilateral muscles and joints, both above and below the diaphragm, both peripheral and axial. Affected individuals often have additional symptoms including fatigue, non-restorative sleep, stiffness, mood and cognitive dysfunction. Disregulated pain perception and inhibition are indicated in its pathogenesis. Its treatment demands a multidisciplinary approach, including medications, tailored physical therapy, and psychological interventions.

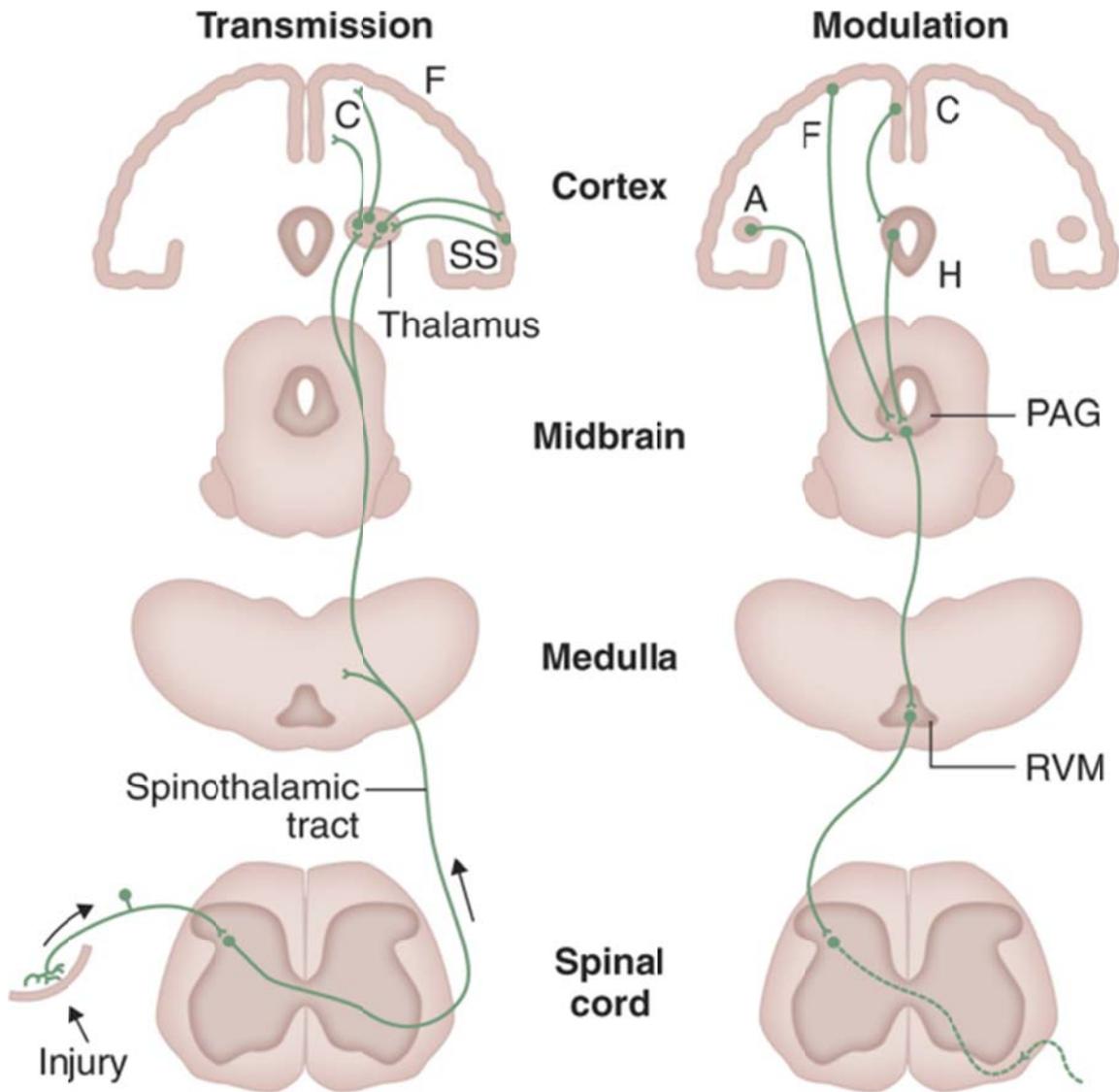


**Fibromyalgia Tender Points**

General locations of the 18 tender points that make up the criteria for identifying fibromyalgia.

**Figure 1:** Fibromyalgia Tender Points

Adapted from Web Image from Google



**Figure 2:** Simplified Model of

Ascending (transmission) and Descending (modulation) Pathways of Pain

*C: cingulate cortex, SS: somatosensory cortex, F: frontal cortex. Right, pain modulation via the dorsolateral funiculus. A: amygdala, H: hypothalamus, PAG: periaqueductal gray, RVA: rostroventromedial medulla.*

*Adapted from Cecil Medicine on MD Consult*

**Table 1: Myofascial Pain vs Fibromyalgia**

	<b>Myofascial Pain</b>	<b>Fibromyalgia</b>
Location	Local	Widespread
Trigger point	+	-
Tender point	-	+
Severe fatigue	Uncommon	Common
Primary Treatment	Stretching, PT, Trigger Point Injection	Multidisciplinary: Drugs, psych, tailored PT

1. Trigger point: taut point in muscle, the pressure upon which produces radiating pain
2. Tender point: sensitive points, where pressure produces localized pain

**Table 2:** Adapted from Bennet 2009 Review Article

A comparison of the major patient-perceived manifestations of fibromyalgia

<b>OMERACT 7 Patient Delphi</b>	<b>NFA Survey</b>	<b>DFV Survey</b>
Pain or physical discomfort	Morning stiffness	Pain
Joint pain or aching	Fatigue	Fatigue
Fatigue or lack of energy	Nonrestorative sleep	Nonrestorative sleep
Poor sleep	Pain	Morning stiffness
Fibro-fog	Forgetfulness	Poor concentration
Stiffness	Poor concentration	Lack of energy
Disorganized thinking	Difficulty falling asleep	Low productivity
Difficulty with moving	Muscle spasms	Forgetfulness
Having to push yourself to accomplish things	Anxiety	Irritability
Problems with setting goals and completing tasks	Depression	Weather sensitivity
Tenderness to touch	Headaches	Feeling hands are swollen
Depression	Anger	Dizziness
Limitations in normal daily activities	Restless legs	Headaches
Poor memory	Abdominal pain	Visual disturbances

**OMERACT:** Rigorous evaluations performed by the Outcome Measures in Rheumatology Clinical Trials (OMERACT) involving patient group interviews (Delphi groups).

**NFA:** Internet survey conducted by National fibromyalgia Association (NFA) on 2569 people diagnosed with fibromyalgia

**DFV:** Mail-in questionnaire survey conducted by the German Fibromyalgia Association (DFV) on 3996 patients.

**Table 3: Common Medications Used to Treat Fibromyalgia**

**Table 3a: FDA approved Medications**

Drug Name	Duloxetine*	Milnacipran*	Pregabalin*
Trade Name	Cymbalta	Savella	Lyrica
Mechanism	SSNRI	SSNRI	Ca++ Ch. Bck
Also Treats:	depression	depression	sleep
Side Effects	N/V, SA	N/V, SA	WT gain, dizziness
Starting Dose (once a day)	20-30mg	12.5mg	50-100mg
Dosing Frequency	QD or BID	BID	BID or TID
Target Regimen	60mg QD	50mg BID	450mg / day

**Table 3b: Other Medications**

Drug Name	Gabapentin	Tricyclic Antidepressants	Naltrexone
Trade Name	Neurontin	Elavil, pamelor, norpramine	
Mechanism	Ca <sup>++</sup> Ch. Bkck	Inhibits 5HT, DA, NE reuptake	Glial cell modulator
Also Treats:	sleep	depression, sleep	cannot use w opioid
Side Effects	WT gain, dizziness	anticholinergic; lethal OD	None
Starting Dose (once a day)	100-300mg	10-25mg	4.5mg
Dosing Frequency	TID or QID	QD or BID	QHS
Target Regimen	1800mg / day	50-150mg /day	4.5mg QHS

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## **Treatment**

### **Cancer-Related Pain**

#### **CANCER BONE PAIN**

*Stephanie Jones, MD*

*Michael Leong, MD*

#### **3 Key Points:**

1. What are 3 of the 5 main areas of the body where cancer bone pain occurs?
2. What common pathologic fracture is treated by orthopedic surgery?
3. What are 3 oral or intravenous analgesic classes used to treat cancer bone pain?

#### **Etiology:**

- One of the most common causes of cancer-related pain

- Third most common metastatic site after lung and liver
- Bone fractures, hypercalcemia, and associated neurologic deficits affect quality of life

### **Tumor types:**

- Primary bone tumors include: multiple myeloma, osteosarcoma, Ewing's sarcoma
- Metastatic bone tumors include: breast, prostate, lung, thyroid, and kidney

### **Pathophysiology:**

- Bone remodeling is modulated by osteoclasts (resorption) and osteoblasts (building)
- Skeletal imbalance and uncoupled resorption results in destruction of bone architecture and subsequent weakness to connective tissue
- Osteoclast may be activated by tumor cells
- Prostaglandins may stimulate osteoclastic cells
- Different tumors have different presentations
- Lytic lesions are associated with Multiple Myeloma
- Pathologic fractures are associated with lung and renal carcinoma

Hypercalcemia associated with osteolytic metastases but extent of metastatic bone disease does not correlate with hypercalcemia

### **Additional Pain mechanisms:**

- Many nerves are found in the periosteum
- Stretching of periosteum, mechanical stress of weakened bone and nerve entrapment
- Osteoclastic bone destruction may activate pain receptors

### **Clinical presentation:**

- Osteolytic bone metastases present with bone pain, pathological fractures, hypercalcemia, or rarely swelling / neurologic symptoms
- Most common presenting symptom is pain 50%
- 5 most involved areas (vertebrae, pelvis, ribs, femur, and skull)
- Spinal instability is due to bone loss of one or more vertebral bodies tending to collapse resulting in kyphosis

### **Radiological Studies:**

radiography, scintigraphy, CT scan and MRI

### **Therapies:**

- Radiotherapy: local bone pain; tumor shrinkage and inhibition of chemical pain mediators
- Single fraction of local or hemibody irradiation within 24 hours
- Rapid response suggests effect on chemical mediators of the inflammatory response
- Usually low dose (4 to 8 Grays) in 1 treatment or over course of one week
- Chemotherapy / Hormonal Therapy
- Concept is to deprive the tumor cells of growth stimulus induced by hormones, particularly in breast, prostate, endometrial cancers

Chemotherapy regimen is disease specific and beyond the scope of this handbook

Patients can experience pain relief without showing objective tumor response

### **Orthopedic / Physiatriic Approaches**

10-30% with bone metastases develop long bone fractures require orthopedic treatment – femur for stability and to promote ambulation

Conservative treatment of bone fractures in axial skeleton is successful since bones have a better blood supply

Vertebroplasty (injection of methylmethacrylate) and Kyphoplasty (injection of methylmethacrylate into a balloon supported cavity)

### **Analgesics:**

- Refer to WHO guidelines for first steps ([see WHO section below](#))
- NSAIDs treat the stimulation of free nervous ending of periosteal tissues; no conclusive evidence that one is more effective than another
- Choline magnesium trisalicylate does not inhibit prostaglandin synthesis and has an analgesic effect on malignant bone pain
- Celecoxib as a COX-2 inhibitor does not affect platelet aggregation but may have a class effect for cardiovascular risk (although not clear with published data)

Corticosteroids are potent anti-inflammatories and decrease nociceptive input; duration of pain relief from systemic steroids is generally short

Calcitonin inhibits sodium and calcium reorption by renal tubule and reduces osteoclastic bone resorption but short duration of action and poor efficacy for rapid development of tachyphylaxis; with EBM review effective for CRPS!

### **Bisphosphonates:**

Mechanism of inhibitory effect on bone resorption is unknown as well as pain relief

- Clodronate – oral or intravenous
  - 1600 to 3200 mg / day or 300 to 600 mg / day
  - for i.v. 600 mg single dose or 300 mg qd for 5 days
  - slow infusion rate due to renal toxicity
- Pamidronate
  - In Breast CA 30 to 50% reduction of pain
  - 60 mg i.v. every 2 weeks
  - monthly infusions of 90 mg infused over 2 hours provided protection of skeletal complications and decrease of bone pain
  - Treatment well tolerated
  - Side-effects: transient low grade fever, nausea, myalgia, bone pain, mild infusion-site reactions
- Zoledronic Acid
  - Phase 3 studies in breast CA and multiple myeloma
  - i.v. 4 mg infused over 15 minutes every 3-4 weeks for 12 months
  - also effected in primary malignancies per unblended studies
- Ibandronate
  - i.v. 6 mg and oral for breast CA
  - Acute phase reactions, gastrointestinal toxicity, renal toxicity, osteonecrosis of the jaw (oral ulcerations expose underlying bone) – requires dental exam
- Radioisotopes:

- Strontium-99 systemically administered to deliver radiation to the body; similar chemical structure to calcium
- Uptake is greatest in areas of marked osteoblastic activity, such as prostate
- Invasive: per interventional and sympathetic blocks sections

## High-dose Opioids

Intraspinal analgesia: opioids vs Ziconotide

## Surgical Ablative

Percutaneous cervical cordotomy: interruption of ascending spinothalamic tract for unilateral bone pain below C5 dermatome

Pituitary ablation: widely disseminated pain of bony metastatic origin and when primary tumor is hormonally responsive

## Psychological Support:

High incidence of depression and anxiety for patient and family

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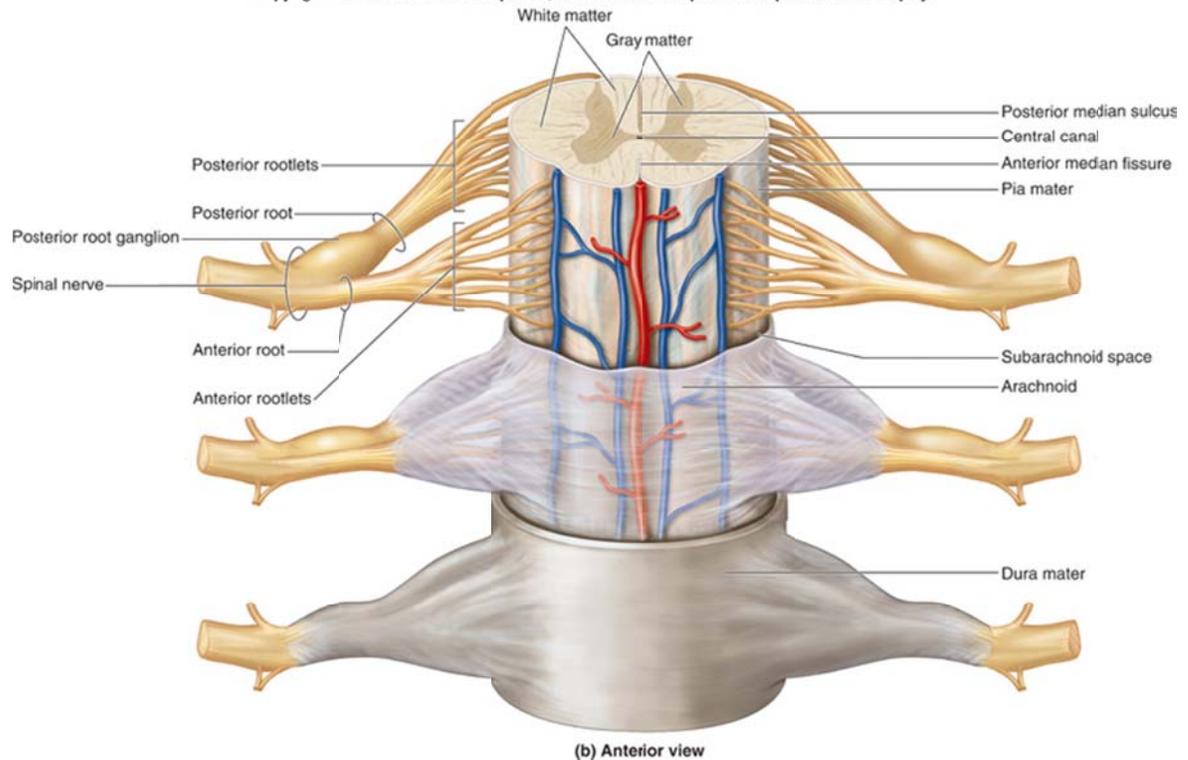
## SPINAL ANESTHESIA

*O. Jameson Stokes, MD*

### Anatomy

Vertebral column and ligaments: The skin and subcutaneous tissues, the supraspinous and interspinous ligaments, and ligamentum flavum are all essential components<sup>1</sup>.

Meninges and spinal cord: The meninges form three connective tissue membranes that cover and protect the spinal cord. the dura mater is the outermost layer and consists of fibroelastic fibers. The inner layer of the dura is closely attached to the middle meningeal layer, the arachnoid mater. The arachnoid mater is composed of overlapping layers of epithelial cells connected by tight junctions, which allows this layer to function as the principal physiologic barrier to substances traversing in and out of the cerebrospinal fluid (CSF)<sup>2</sup>. The pia mater is the innermost meningeal layer and is composed of a thin layer of highly vascular connective tissue adherent to the spinal cord. In contrast to the arachnoid mater, the pia mater is fenestrated, which allows the spinal cord to communicate freely within the CSF. In most adults, the caudal tip of the spinal cord ends between L1 and L2.



### Physiology of CSF:

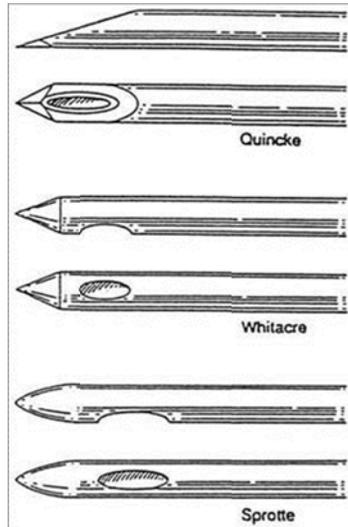
Cerebrospinal Fluid Volume: Local anesthetic solutions injected will become diluted in the volume of the CSF before reaching their site of action within the central nervous system. Thus, individual variations in lumbosacral CSF volumes can have a significant effect on the extent and duration of spinal anesthesia. Recent studies utilizing fast spin-echo MRI demonstrate a wide variability between individuals in the volume of lumbosacral CSF, with a mean volume of  $50 \pm 20$  mL, but a range of 28 to 81 mL<sup>3</sup>.

### Cerebrospinal fluid density and baricity:

Solutions that have the same density as CSF have a baricity of 1.0000 and are classified isobaric. Solutions that are denser than CSF are classified as hyperbaric, whereas solutions that are less dense than CSF are termed hypobaric. CSF density is poorly predictive of peak block height, suggesting that CSF density is only one of a number of factors influencing the extent of spinal anesthesia.

### Technical aspects of spinal anesthesia:

Needle design: Cutting or non-cutting spinal needles are the two main categories. Cutting (Quincke) needles cut through dural fibers, while non-cutting (pencil point like Sprotte, Whitacre, Gertie-Marx) needles spread the fibers. In an invitro investigation non-cutting needles demonstrated 2 to 3 times less CSF leakage compared to cutting needles of corresponding size<sup>4</sup>.



**Factors influencing block height:**

Baricity: The baricity of local anesthetic solution relative to the patient position is the most important determinant. It is possible, by choosing the appropriate baricity and patient position, to "direct" the local anesthetic solution to the dermatomal segments that require anesthesia. Because of the variability in mean CSF density in patients, a local anesthetic solution must have a density of less than 0.9990 to function reliably as a hypobaric spinal anesthetic. In contrast, solutions with a density of greater than 1.0015 can be expected to function reliably as hyperbaric spinal anesthetics<sup>5</sup>.

Age, height, weight, and anatomic configuration of the spine have not been shown to be clinically important or a reliable predictor of block height<sup>6</sup>.

**Factors influencing duration of spinal anesthesia:**

Local anesthetic: The primary factor in determining block duration is the choice of local anesthetic agent. Increasing the dose clearly increases duration of spinal anesthesia for all the commonly used spinal local anesthetics. For a given dose, higher peak blocks tend to regress faster. Thus, isobaric local anesthetic solutions usually produce blocks of longer duration than hyperbaric blocks using the same dose. It is thought that the lower cephalad spread results in a relatively higher local anesthetic concentration in the CSF and the spinal nerve roots, which requires more time for the local anesthetic to decrease below the minimally effective concentration<sup>7</sup>.

**Physiologic effects of spinal anesthesia:**

Cardiovascular physiology

- Hypotension: due to decrease in cardiac output and systemic vascular resistance
- Treatment: correction of decreased venous return, SVR, and CO.
- Bradycardia: blockage of sympathetic cardioaccelerator fibers at T1 to T5 levels
- Treatment: administration of ephedrine, atropine, or epinephrine.

Thermoregulatory physiology

- Perioperative hypothermia
- Due to redistribution of core heat to periphery caused by vasodilatation from sympathetic blockade and decreased threshold for vasoconstriction and shivering
- Treatment: Active warming, warmed fluids, covering exposed skin, and limiting block height

## Pulmonary physiology

Spinal blockade to even midthoracic levels has been shown to have minimal effect on inspiratory muscle function. In contrast, expiratory muscle function has been shown to decrease in proportion to the height of spinal blockade<sup>8</sup>.

## Central nervous system physiology

Spinal anesthesia has been shown to have sedative effects in the absence of intravenous sedation. The proposed mechanism for this independent sedative effect is a decrease in reticular activating system activity due to interruption of ascending afferent sensory input to the brain. Clinically, the degree of sedation correlates with the level of peak block height<sup>9</sup>.

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## NEUROLYSIS

*Stephanie Jones, MD*

### Introduction

- Definition – intentional injury of a nerve by chemical, thermal, cryogenic, or surgical means (in order to relieve pain)
- Used primarily for refractory cancer pain (less often for nonmalignant pain secondary to risks)
- Best used in conjunction with other techniques/medications for optimal outcomes (rarely effective as the sole method of pain management)

- Chemical neurolysis – most commonly performed with 50 - 100% ethyl alcohol or phenol; alcohol is extremely noxious, therefore local anesthetic is strongly suggested prior to administration; phenol is painless and analgesic as well as neurolytic
- Mechanisms
  - alcohol leads to the extraction of cholesterol and phospholipid from the neural membrane and the precipitation of lipoproteins and mucoproteins
  - phenol 6-10% in water produces protein coagulation and necrosis of the neural structures
- Radiofrequency thermocoagulation – may lower the risk of undesired deficits encountered with chemical ablation by better localization of the resulting lesion (untoward spread associated with injection techniques using chemical neurolytics; chemical neurolysis preferred for interventions that depend on disrupting a more diffuse neural network, ie. Celiac plexus, superior hypogastric plexus)
- Cryoanalgesia – less commonly employed, less durable analgesia

### **Risks**

- Postablative dysesthesias, neuritis, neurologic deficits, damage to nontargeted neural and non-neural tissues, failure, non-permanence
- Unfortunately, neurolytic substances damage tissue indiscriminately; application may lead to excessive, persistent neurologic injury; potential for undesired effects can be somewhat assessed with prognostic local anesthetic blocks
- Aberrant spread with resultant tissue injury – vest avoided by verifying needle placement with imaging techniques (fluoro, CT, US), serial aspiration prior to injection, alert to paresthesias, appreciation of tissue compliance, electrical stimulation, and/or test doses with local anesthetic
- Neurolytic agents predominantly affect neuronal axons, not the cell bodies; subsequently, pain relief is temporary due to axonal regeneration and neural plasticity; pain relief therefore averages 3 – 6 months in patients with stable disease

### **Patient selection**

- Most often malignant pain
- Sometimes applicable to non-cancer pain in the presence of another advanced, irreversible, or progressive illness
- Decisions should be carefully individualized
- Life expectancy – effects of neuroablation average 3 – 6 months, with duration of relief influenced by incomplete neurolysis, new pain due to disease progression, and/or postdenervation neuropathic pain; therefore, optimal time to intervene is probably within 6 – 12 months of predicted death
- When death is imminent – treatments associated with extensive preparation, discomfort, or recuperation should be avoided (ie. Intrathecal drug delivery system implantation); on the other hand, treatments with higher risks may be more liberally applied (ie. Intrathecal neurolysis)
- Contraindications – absolute contraindications- inadequate informed consent; relative contraindications – local infection, bleeding diathesis, spinal cord compression, tumor spread at the injection site or when local tumor growth would prevent access to the neural target and make treatment effects unpredictable

### **Types of pain amenable to neurolysis**

- Neurolysis most appropriate and effective for discrete, well-localized pain in a single area that the patient can reliably identify (when attempting to provide more extensive coverage, treatment more likely to fail and increased risk of undesired neurologic deficits)
- Visceral pain – although visceral pain is often vague and broadly based, it is often amenable to sympathetic neurolysis (ie. Celiac plexus, superior hypogastric plexus)
- Patients with vague pain (ie. “hurts everywhere” “can’t describe it”) or those whose complaints are inconsistent or change over time, are poor candidates for neurolysis
- Somatic or visceral pain appears to respond more favorably than neuropathic pain

### **Specific neurolytic procedures**

- Peripheral nerves
  - Usually avoided secondary to higher incidence of neuritis (more common with alcohol than phenol)
  - Risks of weakness due to mixed nerves (sensorimotor)
  - Risk of failure due to overlapping sensory distribution
  - Intercostal neurolysis – risk of pneumothorax; neighboring nerves need to be blocked for overlapping innervation; risk of neuritis
  - Cranial nerves – alcohol, phenol, or radiofrequency ablation of the trigeminal nerve or its branches, and occasionally 9<sup>th</sup> and 10<sup>th</sup> cranial nerves, are performed for refractory orofacial pain
- Sympathetic nerves
  - Blockade to treat refractory visceral pain, sympathetically maintained pain, hyperhidrosis, and peripheral ischemia – successful treatment associated with low incidence of new pain and no numbness or motor weakness
  - Celiac plexus/splanchnic nerve neurolysis – neurolysis usually reserved for abdominal malignancy (ie. Pancreatic cancer, gastric cancer) due to risks of neurolysis
  - Superior hypogastric plexus neurolysis – malignant visceral pelvic pain
  - Most lower-extremity malignant pain is somatically mediated, creating little role for lumbar sympatholysis; upper extremity cancer pain rarely sympathetically-mediated and stellate ganglion neurolysis associated with brachial plexus injury (so usually avoided); lumbar sympatholysis has been shown to be effective in the treatment of ischemic lower extremity pain

### **Neuraxial neurolysis**

- Less common in contemporary practice
- Intrathecal or epidural application of neurolytics only considered in advanced, irreversible disease (usually cancer) secondary to the significance of the possible adverse outcomes
- Anatomy - selectively interrupt sensory transmission while sparing motor function in the affected area, possible due to the division between sensory and motor fibers of the spinal cord (the dorsal root carries sensory fibers and the ventral root carries motor and sympathetic fibers)
- Predictable, segmental sensory loss occurs by proper patient positioning, correct selection of the targeted level of injection, and choosing the appropriate neurolytic agent for intrathecal procedures based on baricity (alcohol is hypobaric and phenol is hyperbaric compared to the cerebrospinal fluid)

- Intrathecal injection of absolute alcohol or phenol and glycerine, with respective hypobaricity and hyperbaricity, confer control over drug spread
- Especially useful for malignant chest wall pain, since dorsal targeted fibers are distant from those innervating the extremities and sphincters (lowers incidence of paralysis, incontinence)
- Studies suggest better outcomes for somatic pain than visceral pain

Unfortunately, there is a lack of controlled studies comparing different treatment techniques for a variety of clinical syndromes; this has been a barrier to the development of evidence-based algorithms when approaching treatment options in different pain syndromes; therefore, the decision to utilize neurolysis must be made after careful, individualized evaluation of each patient presentation, and fully informed consent is essential due to the significance of the adverse possible outcomes (ie. Paralysis, postablation neuritis)

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## WORLD HEALTH ORGANIZATION (WHO) 3-STEP ANALGESIC LADDER (1984)

*Theresa Mallick-Searle, NP*

If pain occurs, there should be prompt oral administration of drugs in the following order: non-opiates; then, as necessary, mild opiates; then stronger opiates such as morphine, until patient is “free” of pain. To calm fears and anxiety, additional drugs, “adjuvants”, should be used.

To maintain freedom from pain, drugs should be given “by the clock”, that is q3-6hr, rather than “on demand”.

This 3-step approach of administering the right drug in the right dose at the right time is inexpensive and 80-90% effective (WHO); 69-100% effective (Jadad, JAMA 1995).

The Principles of the WHO Analgesic Ladder include:

- Using analgesics in adequate doses.
- Titration for the individual patient.
- Administration on a strictly regular basis to prevent pain not pro re nata (PRN), as pain occurs.
- Developing a management plan for breakthrough pain.
- Using oral route whenever possible.
- Review and reassess analgesic requirements
- Keep it simple.

### Mild Pain

Non-opiate (Tylenol, NSAIDS)

+/- Adjuvants (anticonvulsants, antidepressants, corticosteroids, disphosphonates (bisphosphonate), anxiolytics)

### **Mild to Moderate Pain**

Opiate/weaker (long & short acting formulations; po, IM, IV)

+/- Non-opiate

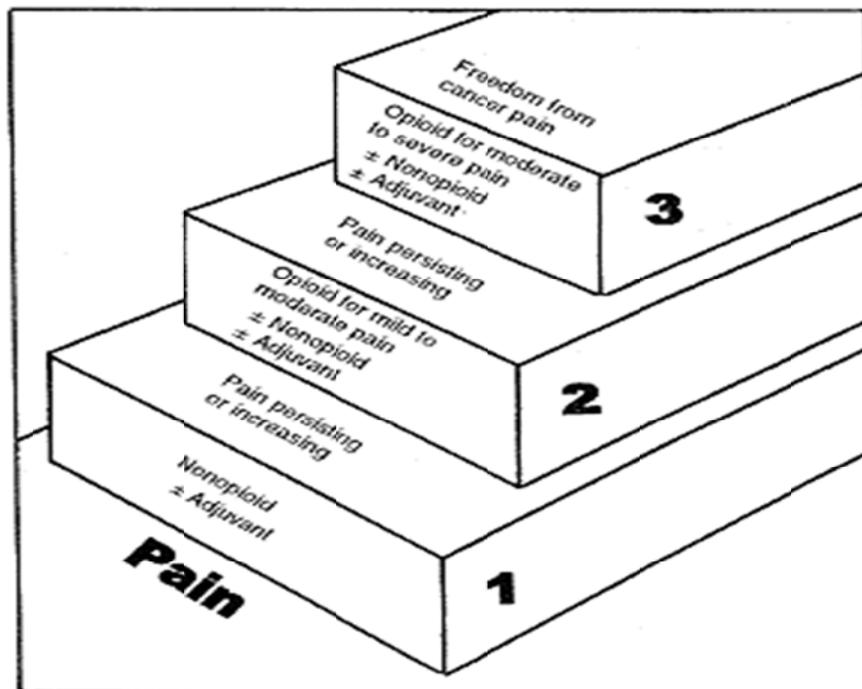
+/- Adjuvants

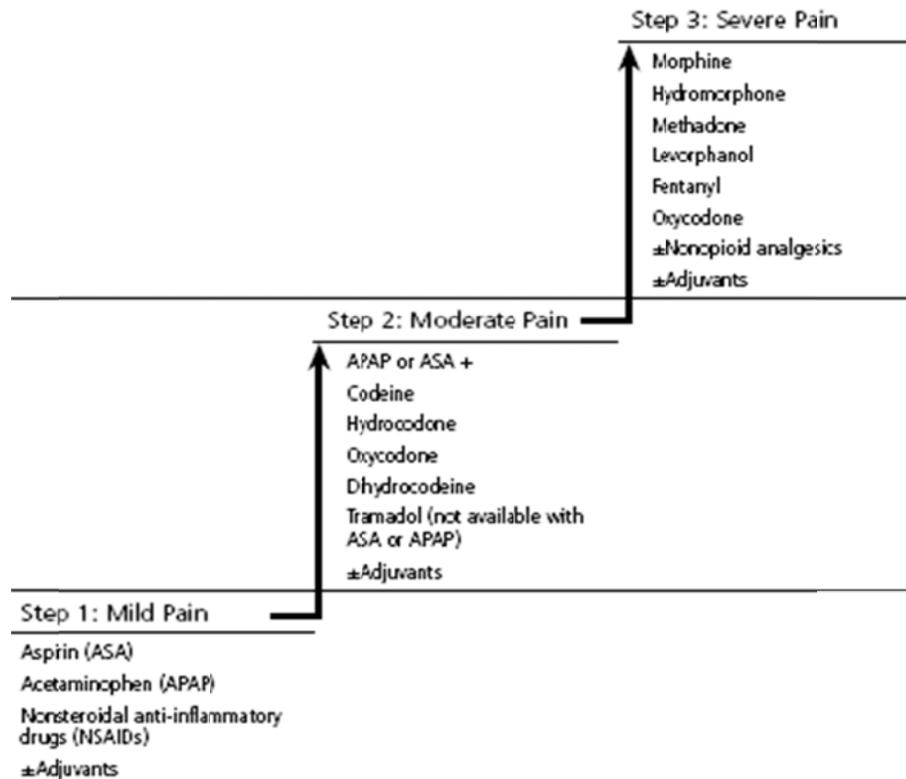
### **Moderate to Severe Pain**

Opiate/stronger

+/- Non-opiate

+/- Adjuvants





## Questions

1. Identify four causes of cancer-related pain.
2. Describe the WHO analgesic ladder.
3. According to the WHO analgesic ladder, when would it be appropriate to use an opiate as an analgesic agent, alone or in combination?
4. What are adjuvants?

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# **Chronic Pain (Non-Cancer Related)**

## OPIOID ANALGESICS – INCL. TOLERANCE & ADDICTION

Ballantyne J, Mao J. Opioid Therapy for Chronic Pain. *NEJM*. 2003;349:1943-53. [PDF](#)

## ANTICONVULSANTS

*Stephen Coleman, MD*

Backonja, M. Anticonvulsants (Antineuropathics) for Neuropathic Pain Syndromes. *Clin J Pain*. 2000;16:S67-S72. [PDF](#)

## MUSCLE RELAXANTS FOR BACK SPASMS

*Michael Leong, MD*

Acute musculoskeletal spasm of the back is a common disorder that causes localized pain, stiffness, reduced mobility, impaired activities of daily living, and sleep disturbances (Ralph et al., 2008).

Low back pain is the fifth most common reason to seek a physician (Chou et al., 2007; Chou & Huffman, 2007).

Health care costs attributable to low back pain in the U.S. were estimated at \$26.3 billion in 1998 (Chou et al., 2007) and have certainly increased in the past decade.

- Most episodes of acute low back pain are nonspecific (Cherkin et al., 1998).
  - Nonspecific back pain is defined as mechanical back pain, facet joint pain, osteoarthritis, muscle sprains, and muscle spasms (Bratton, 1999).
  - Acute low back pain is likely caused by reflex spasms in the paraspinal muscles (Indahl et al., 1995).
  - Typical pharmacologic treatments for acute low back pain are NSAIDS, acetaminophen, and muscle relaxants.
  - Baclofen has demonstrated efficacy in treating muscle spasm associated with acute, painful musculoskeletal conditions (Dapas 1985).

Muscle relaxants can be classified in general to having central versus spinal effects in reducing spasticity. Hence, Stanford's Pain Center usually advocates using predominantly Baclofen or tizanidine modulating spinal polysynaptic reflexes rather than other more centrally acting agents. The following are a quick and noncomprehensive guide to some of the more common medications.

- Baclofen (Lioresal)
  - Treatment of spastic movement from spinal cord injury, multiple sclerosis, dystonias, or trauma.
  - Mech of Action: modulates Gaba B receptors
  - Dosage: oral start at 5 to 10 mg TID; max recommended oral dose is 20 mg QID per package insert but titrated to higher doses for SCI patients
  - Intrathecal: please refer to intrathecal section but for quick guide dosages between 2 to 20 mcg/hour are not uncommon for SCI
  - Side-effects: vomiting, weakness, drowsiness, visual hallucination and seizures at high dosages, and withdrawal symptoms esp. intrathecal

- Tizanidine (Zanaflex)
  - Treatment of multiple sclerosis, spinal cord injury, spastic diplegia.
  - Mech of Action: alpha-2 agonist similar to clonidine with much weaker blood pressure effects
  - Dosage: 2 to 4 mg tid starting at 2 mg night for spasms and sedation
  - Side-effects: sedation, 5% incidence of liver function test elevation, hallucination, withdrawal effect if high doses due to rebound hypertension, tachycardia

### Central Agents:

- Carisoprodol (Soma)
  - Treatment of muscle spasms
  - Mech of Action: unknown central action; metabolized to Meprobamate – high abuse potential
  - Dosage: 250 to 350 mg tid and qhs x 2 to 3 weeks
  - Side-effects: sedation at low doses, sedative / hypnotic effects at high doses, abuse potential
- Cyclobenzaprine (Flexeril, Flexmid, Amrix)
  - Treatment of pain and muscle spasms
  - Mech of Action: unknown central action but chemical structure is similar to 1<sup>st</sup> generation TCA's: amitriptyline, imipramine
  - Dosage: 5 to 10 mg tid; 15 to 30 mg qd ER
  - Side-effects: sedation, depression, headaches, severe dizziness, blurred vision
- Metaxalone (Skelaxin)
  - Treatment of strains, sprains, muscle spasms
  - Mech of Action: unknown central action, general CNS depressant
  - Dosage: 400 to 800 mg tid to qid
  - Side-effects: nausea, vomiting, sedation, and CNS effects (headache, irritability)
- Methocarbamol (Robaxin)
  - Treatment of skeletal muscle spasms
  - Mech of Action: unknown central action, carbamate of guaifenesin
  - Dosage: 500 mg 1000 mg qid titrate to a maximum of 8000 mg per day; also available IV/ IM
  - Side-effects: nausea, vomiting, sedation, and CNS effects (headache, irritability)

Other central agents include: chlorzoxazone (Parafon Forte), ophenadrine (Norflex) – anticholinergic to treat Parkinson's disease, dantrolene, and benzodiazepines, such as diazepam (Valium).

Moreover, for spinal cord injury patients, other analgesics may be helpful such as antidepressants - TCA's and antiepileptics – gabapentin and pregabalin (Lyrica) for pain and spasms.

### Further Reading:

Chou R, et al. Comparative Efficacy and Safety of Skeletal Muscle Relaxants for Spasticity and Musculoskeletal Conditions. *J Pain Symptom Management*. 2004;28(2):140-175. [PDF](#)

Van Tulder M, et al. Muscle Relaxants for Nonspecific Low Back Pain. *Spine*. 2003;28(17):1978-1992. [PDF](#)

## EPIDURAL ANALGESIA

O. Jameson Stokes, MD, MS

Epidural anesthesia has been used since shortly after the start of the 20th century<sup>1</sup>. The advent of the Tuohy needle and epidural catheter in the 1940s led to an interest in caudal and lumbar epidural anesthesia for labor anesthesia. Currently, this is a very popular technique for operative anesthesia with the ability to extend epidural analgesia into the postoperative period.

### **Anatomy**

Anterior boundaries: posterior longitudinal ligament

Posterior boundaries: ligamentum flavum and periosteum of the laminae

Lateral boundaries: intervertebral foramina containing neural elements

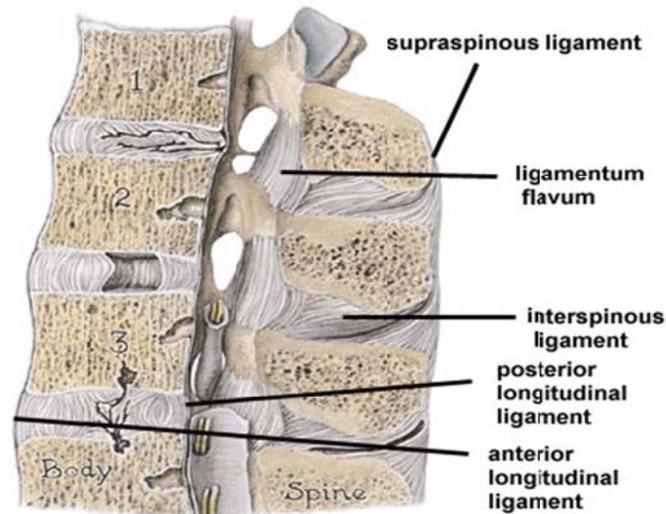
Superior boundaries: foramen magnum where spinal dura attaches to cranial dura

Caudal boundaries: sacral hiatus and closed by sacrococcygeal ligament

### **Layers to pass through prior to entering the epidural space<sup>2</sup>:**

Skin → Subcutaneous tissues → Supraspinous ligament → Interspinous ligament → Ligamentum flavum → Epidural space

The depth from skin to epidural space in adults is dependent upon the amount of adipose tissue present. The most frequently encountered distance was 5 cm, but the distance ranged from 2.5 to 9 cm in a study by Gutierrez<sup>3</sup>.



### **Selection of interspace and approach:**

The guiding principle behind the choice of the interspace is to place the epidural catheter closest to the dermatome in the middle of the surgical incision. Following this principle will result in maximal concentration of local anesthetic or opioid delivered at the dermatomes where anesthesia, analgesia, or motor block are desired.

**Question. If the patient is undergoing gastrectomy with a T6-T11 surgical incision, where would you place the epidural catheter?**

**Answer. Ideally the catheter should be placed between T8 and T10.**

There are two standard approaches to the epidural space: midline and paramedian.

The midline approach. This is the easiest, because the needle is inserted perpendicular to the skin and parallel to the spinous processes at that spinal level.

The paramedian approach. The target area when the needle is angled 10° to 15° from midline is larger than the target area for the midline approach. This approach is useful in elderly patients who may have osteoporotic changes narrowing the interspace or degenerative interspinous ligaments, which could produce a false loss of resistance to injection<sup>4</sup>. Also, in the thoracic region from T5-T10, the angulation of spinous processes require a very acute angle of insertion when the midline approach is attempted therefore requiring adaptation toward the paramedian approach.

**Question. What is the largest interspace in the body?**

**Answer. The largest interspace in the body is the L5/S1 interspace. The paramedian approach to this interspace is called the "Taylor approach".**

### **My advice on placing the epidural:**

Perform a complete H&P with review of pertinent lab and radiographic studies.

Consent the patient with an explanation of all possible complications.

Let the nurses know that you are placing an epidural. He/she should be near the bedside and actively involved in monitoring the patient during the procedure. This provides more help with on-the-fly positioning and can help you in case of complications (syncope, hypotension, arrhythmia, etc.)

Apply pulse oximetry and blood pressure cuff.

Raise the bed height

Choose an appropriate interspace and approach technique prior to prepping the patient.

Betadine requires drying, so Chlorohexidine is my first choice for skin prep.

The paramedian approach is favored in the thoracic region. The midline approach is favored in the lumbar region.

Loss of resistance technique can be accomplished with air or saline. I prefer air because the infiltration of saline into the tissues via a "false loss of resistance" will result in the distortion of field anatomy and decrease your ability to quickly determine malpositioning of the needle.

Never withdraw the catheter from the needle once it has passed the tip. This could shear the catheter tip, leaving it into the epidural space.

**Question. What would you do if a catheter tip sheared off when placing an epidural catheter?**

**Answer. No direct harm usually results from this since the materials used are non-irritating and "tissue-implantable". The patient should be informed of the foreign body present, but surgical removal is usually not required.**

Without fluoroscopic guidance, the epidural catheter cannot reliably be directed once it leaves the tip of the catheter.

Advance the catheter about 5 cm into the epidural space. Lack of control in catheter placement may result in knotting or extension past the intended area of epidural analgesia. Catheters placed 3 cm or less into the epidural space are likely to be fall out. Monitor your depth!

A "test dose" will consist of 3 mL of 2% lidocaine with 5-10 micrograms of epinephrine. If the catheter rests intravascularly, an increase in heart rate of 15-20 beats per minute should be seen on the monitors. If the tip rests intrathecaly, the test dose will result in a dense sensory and motor block, but is inadequate in dosage to induce a high spinal.

A "negative" test dose does not eliminate the possibility of intravascular or intrathecal placement. Elderly patients or those taking beta blocking medications may not display a significant heart rate increase from intravascular injection. "Negative" aspiration of the epidural catheter does not rule out intravascular or intrathecal placement. It may take 10 minutes or more for the full manifestations of an intrathecal test dose to be seen. Profound hypotension and bradycardia may be early signs. Constant vigilance is required whenever epidural analgesia is used.

## **Epidural Complications<sup>5</sup>:**

### Headache

May be the result of dural puncture (1-2% incidence), usually self-limiting  
Treatment: Analgesia, Bed rest, Hydration, Caffeine, Blood patch

### Backache

At insertion site, usually transient  
Treatment: Analgesics and reassurance, monitor for fever or neuro deficits

### Sympathetic blockade

Hypotension, respiratory distress, bradycardia, unconsciousness, block higher than T4, numbness tingling in fingers or arms, Horner's syndrome  
Treatment: Hydration, vasopressors, resuscitation, stop infusion

### Nerve damage

Rare and usually transient  
Treatment: Investigation and neurology consultation

### Epidural Abscess

Insidious and slow over hours to days, back pain and tenderness, fever and leukocytosis are usual, mental status changes, weakness over hours and days

Diagnosis: MRI with gadolinium

Treatment: Surgical decompression, medical treatment for mild or early cases

## Epidural Hematoma

Acute and abrupt, from minutes to hours, back pain and tenderness, weakness progresses rapidly

Diagnosis: MRI with gadolinium

Treatment: Surgical decompression

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5 Geibler RM, Scherer RV, Peters J. Incidence of neurologic complications related to thoracic epidural catheterization. Anesthesiology. 1997; 86:55-63.

Image. [members.cox.net/injections/esi\\_principles.htm](http://members.cox.net/injections/esi_principles.htm)

## PERIPHERAL NERVE BLOCKS FOR CHRONIC PAIN

*Einar Ottestad, MD*

### 1. Trigeminal Ganglion

- Indication: Trigeminal neuralgia, diagnostic and ablation
- Anatomy: in Meckel's cave. Medial is cavernous sinus, superior is temporal lobe, posterior is brainstem
- Function: Sensory - oral mucosa, gingiva, tooth pulp, anterior 2/3 tongue, V1/V2/V3 cutaneous head/face
- Motor - muscles of mastication (V3)
- Autonomic - ciliary, sphenopalatine, otic, submaxillary ganglia
- Procedure: Fluoroguided. Entry 2 cm lateral to commisure labialis directed towards pupil. Target is foramen ovale as seen on fluoro. Stim.

### 2. Maxillary nerve V2

- Indication: Maxillofacial surgery, upper jaw pain
- Anatomy: V2. From gasserian ganglion it travels along cavernous sinus through foramen rotundum. It extends to the superior aspect of the pterygopalatine fossa along inferior portion of the orbit and exits infraorbital foramen.
- Function: Pure sensory
  1. Intracranial group to medial cranial fossa dura mater

- 2. Pterygopalatine group to temporal and lateral zygomatic area, and sphenopalatine branches to mucosa
- 3. Infraorbital canal group to incisors/canines/premolars, maxillary antrum, nasal cavity
- 4. Infraorbital face group to skin
- Procedure: ID mandibular notch. Insert needle at inferior/posterior aspect of notch and advance until needle finds lateral pterygoid plate. Redirect anterior and superior into pterygopalatine fossa until stim.

### 3. Mandibular nerve V3

- Indication: Mandible pain, tongue pain
- Anatomy: V3. Outside foramen ovale nerve traverses anterior and inferior deep in infratemporal fossa anterior to middle meningeal artery, lateral to otic ganglion, medial to external pterygoid/masseter/ramus. Gives off Nervus spinosus to dura. Motor fibers to muscles of mastication. Sensory to buccal membranes, auriculotemporal, auricular, external meatal, articular, parotid, superficial temporal branches, lingual, inferior alveolar, mental nerves.
- Function: Motor - muscles of mastication
- Sensory - jaw, temple, anterior 2/3 tongue, medial and superior of ear
- Procedure: Needle in mandibular notch, advance through infratemporal fossa, lateral pterygoid is endpoint. Walk needle backward off plate until stim to jaw/tongue/ear.

### 4. Glossopharyngeal nerve

- Indication: glossopharyngeal neuralgia, head and neck cancer pain.
- Anatomy: CN IX. Exits jugular foramen near vagus, accessory nerve, jugular vein. Lies in groove between jugular vein and carotid artery. Posterior to styloid process.
- Function: Sensory - posterior third of tongue sensation and taste, tonsils, mouth and pharynx, middle ear
- Motor - stylopharyngeus muscle, elevates larynx
- Autonomics - from carotid sinus nerve (carotid sinus and body) blood pressure/respiration/pulse. Parasympathetics to otic ganglion and parotid gland.
- Procedure: Fluoroscopy. Needle advanced perpendicular to skin to contact styloid process, then walked posteriorly. Negative aspiration then inject. Can stim.

### 5. Greater and lesser occipital nerves

- Indication: Occipital neuralgia, cancer pain, cervicogenic headache
- Anatomy: Greater occipital comes from dorsal primary ramus of C2 and some C3. Ascends in posterior neck over dorsal surface of rectus capitis, then pierces semispinalis, runs deep to trapezius, becomes superficial below superior nuchal line. Lesser occipital nerve comes from ventral primary rami of C2 and C3. Passes superiorly along posterior border of SCM.
- Function: GON - medial portion of scalp as far as vertex
- LON - lateral portion of posterior scalp and cranial surface of pinna of ear.
- Procedure: GON - Patient is seated and flexed forward. Note occipital protuberance, mastoid, and occipital artery. Artery usually 1/3 from protuberance to mastoid. Encounter bone and withdraw a few mm. Inject medial and lateral to artery. Can stim.
- LON - Infiltrate posterior border of SCM at midpoint. Alternate technique is to medial to insertion of SCM at mastoid process. Aim needle cephalad and medial until skull contact, withdraw few mms, inject.

### 6. Intercostal nerve

- Indications: Chest wall pain from trauma, fractures, zoster, surgery, cancer, chest tubes

- Anatomy: Arises from anterior division of thoracic paravertebral nerve. Can overlap from dermatome above and below.
- 4 branches.
  - unmyelinated postganglionic fibers of gray rami communicantes which interfaces with sympathetic chain
  - Posterior cutaneous branch to paraspinal muscle and skin
  - lateral cutaneous division which arises in anterior axillary line to chest and abdominal wall
  - anterior cutaneous branch to midline chest and abdominal wall
- Function: Intercostal muscles and cutaneous.
- Procedure: Prone, seated, or lateral decubitus. Posterior axillary line. Insert needle to rib, then walk inferior 2 more mm into intercostal groove. Inject. Can use fluoro or ultrasound to confirm position.

## 7. Suprascapular nerve

- Indications: Shoulder surgery or pain. Suprascapular nerve entrapment.
- Anatomy: Origin is C5 and C6 nerve roots, occasionally C4. Passes underneath coricoclavicular ligament to suprascapular notch along with suprascapular artery and vein.
- Function: Sensory - 70% of shoulder joint
- Motor - supraspinatus (shoulder abduction) and infraspinatus (external rotation)
- Procedure: Palpate scapular spine all the way to acromion. ID a point on scapular spine 3-4 cm medial to acromion, and insert needle 1 cm superior in the direction of the scapula. Walk superomedial or superolateral into notch. Stim, ultrasound, or fluoro can be helpful.

## 8. Ilioinguinal and iliohypogastric nerves

- Indications: Surgical anesthesia. Neuralgias.
- Anatomy: Both originate T12/L1, travel around inside ilium. At the ASIS they pierce the transversus abdominis, and a bit more medially they pierce the internal oblique to lie between internal and external oblique. The ilioinguinal dives into superficial inguinal ring and travels to groin. Often overlap between these nerves and genitofemoral.
- Function: Ilioinguinal is sensory to upper medial thigh and genitals. Iliohypogastric is sensory to pubis.
- Procedure: Palpate ASIS. Draw line to umbilicus. 2 cm towards umbilicus on this line insert blunt needle. First pop is external to internal oblique. Give some local anesthetic in case the nerves have already become more superficial. Then insert to second pop which is between internal oblique and transversus abdominis. Give more local anesthetic. Can also stimulate in both layers to ID nerves or use ultrasound to see fascial planes.

## 9. Genitofemoral nerve

- Indications: Groin pain
- Anatomy: Originates L1/L2, travels through psoas major muscle, descends posterior to ureter, divides above inguinal ligament. Femoral branch travels lateral to femoral artery to femoral triangle. Genital branch travels through inguinal canal/deep inguinal ring to spermatic cord/round ligament to scrotum/mons and labia majora.
- Function: Sensory - genital area, overlap with ilioinguinal
- Motor: Cremaster
- Procedure: Femoral branch - SQ infiltration lateral to femoral artery.
- Genital branch - palpate pubic tubercle. Insert needle inferior to inguinal ligament and immediately lateral to pubic tubercle.

## 10. Lateral femoral cutaneous nerve

- Indications: Meralgia paresthetica. Surgical procedures involving lateral thigh.
- Anatomy: Originates L2/L3. leaves psoas muscle to course medial to ASIS, under inguinal ligament and beneath fascia lata then to lateral thigh
- Function: Sensory to lateral thigh from above knee to greater trochanter.
- Procedure: Palpate ASIS. 1 cm medial and 1 cm inferior, insert needle through first pop. Inject. Stim or ultrasound can be used.

## 11. Pudental Nerve

- Indications: Genital, perineal, rectal pain.
- Anatomy: Origin at S2-S4. Passes inferiorly between piriformis and coccygeal muscles, leaves pelvis with pudental vessels through greater sciatic foramen, passes around ischial spine, re-enters pelvis in lesser sciatic foramen. Travels in Alcock's canal near ichium then divides into inferior rectal nerve, perineal nerve, dorsal nerve of penis/clitoris.
- Function: Sensory - perianal, posterior 2/3rd scrotum/labia, dorsum penis/clitoris
- Motor - anal sphincter, urogenital triangle muscles
- Procedure - Several choices.
  1. Lithotomy position. Palpate ischial spine in vagina or rectum. Palpate ichial tuberosity. Insert needle 1-2 cm medial and posterior to ischial tuberosity and advance towards finger on ischial spine. inject.
  2. Lithotomy. Insert stimuplex 1-2 cm medial and posterior to ichial tuberosity and direct perpendicular to skin. Elicit stim and inject.
  3. Prone. Fluoro to ID ischial spine. Insert stimuplex needle through buttock to ischial spine. Walk medially without going deeper than spine to obtain stim. Inject.
  4. Prone. Ultrasound to ID pudental artery and ischial spine. Insert needle medial to artery and inject.

### References:

Raj P et al. Interventional pain management: image-guided procedures. Saunders Elsevier 2008.

Peng P, et al. Ultrasound-guided interventional procedures for patients with chronic pelvic pain - a description of techniques and review of literature. Pain physician 2008; 11:215-224.

### Questions:

1. What are the complications of trigeminal nerve blocks?
2. What are some interventions if diagnostic blocks provide short term pain relief?

## SYMPATHETIC NERVE BLOCKS

*Stephanie Jones, MD*

Sympathetic nervous system implicated in numerous pain syndromes:

Complex Regional Pain Syndrome – sympathetic blocks shown to have about 60% efficacy in the treatment of CRPS, effects somewhat better if applied early, and worse if delayed > 6 months

Lower extremity advanced ischemic disease caused by peripheral arteriosclerotic disease (other than from diabetic or small vessel disease) – responds well to lumbar sympathectomy, symptomatic improvement in 70% of cases

Herpes Zoster/Post-herpetic neuralgia - No evidence exists from controlled prospective studies to support the use of sympathetic blocks in the prevention of post-herpetic neuralgia during acute HZ, but may be beneficial in pain relief during the acute phase of HZ

### **Sphenopalatine Ganglion Blockade**

Anatomy – sphenopalatine ganglion located in the pterygopalatine fossa (sphenopalatine fossa), posterior to the middle nasal conchae and anterior to the pterygoid canal; close to the maxillary nerve; sympathetic nerve passes through this ganglion to supply the sensory, vasomotor, and secretory fibers to the sphenopalatine, lacrimal, and nasal glands, and also to some of the sympathetic fibers along the cranial blood vessels

Indications – atypical facial pain, headaches

Technique –

1) simplest approach – advance two soft, cotton-tipped applicators soaked with viscous lidocaine through the nares, along the middle turbinate posteriorly; a second applicator is then applied superior and posterior to the first one and both are left in position for 30 minutes (some case studies support self-administered sphenopalatine block by patients with refractory facial pain/headaches)

2) fluoroscopic guidance – needle inserted between mandibular rami, under fluoroscopy, and under the zygoma, aiming at sphenopalatine fossa (paresthesia to maxillary nerve may occur); on AP view the needle tip should lie just adjacent to the lateral nasal cavity wall; contrast dye or radiofrequency stimulation can confirm position (stimulation should produce tingling in the nasal area and cavity); can then apply 1 ml of local for diagnostic blockade, neurolytic for neurolysis, or RFA can be performed

Complications – mechanical – trauma to maxillary nerve, intravascular injection, epistaxis, pain at site of injection, pharmacologic – intravascular injection, damage to maxillary nerve with neurolytic agent, seizure from IV injection

### **Cervicothoracic/Stellate Ganglion Block**

Anatomy – sympathetic flow to the head, neck, and upper extremities is derived from the upper 5 – 7 thoracic spinal segments; cell bodies located in the gray matter of the dorsolateral spinal cord; they exit with the anterior primary rami as white rami communicante; fiber ascends along the anterior lateral surface of the spinal column, to the three cervical sympathetic ganglia (superior, middle, and inferior cervical sympathetic ganglia); in 80% of individuals, the inferior cervical and the first thoracic sympathetic ganglia fuse to form the “stellate ganglion” (or cervicothoracic ganglion); cervicothoracic sympathetic ganglia supply the head, neck, and most of the upper extremity sympathetic flow; stellate ganglion resides anterolateral to the body of C7 at the neck of the 1st rib and is bordered medially by the longus coli muscle, laterally by the anterior scalene muscle, anteriorly by the subclavian artery, posteriorly by the prevertebral fascia, and inferiorly by the dome of the lung. \*in 20% of people, nerves from the 2nd and 3<sup>rd</sup> thoracic sympathetic ganglia bypass the stellate ganglion and directly join the brachial plexus, called “Kuntz fibers,” explaining why stellate ganglion alone sometimes does not provide complete sympathetic block to the upper extremity

Indications – head and neck (if sympathetically-maintained), upper extremity and chest wall CRPS and other SMP (sympathetically-maintained pain), vascular insufficiency/vascular disorders of the upper extremities, head, and neck, including some vascular types of headaches (possibly migraines, cluster headaches)

Technique – classically use either C6 or C7 vertebra as landmark; after identification, either by fluoroscopy or palpation (C6 is lateral to cricoid), the C6 transverse process (Chassaignac’s process) is

used as a landmark; at the junction of the body and transverse process of either C6 or C7, the periosteum is contacted using a 27-g short, beveled needle; carotid artery displaced laterally; once periosteum contacted, the needle withdrawn a few millimeters; after negative aspiration of CSF or blood, 1 ml dye can be injected for fluoroscopic approach, often approach is blind; 0.5 ml of local anesthetic is injected as a test dose – wait 2-3 minutes to monitor patient for signs of toxicity or spinal anesthesia, then the rest of the dose, 2 – 10 ml of local is injected, with incremental aspiration (carefully!); sympathetic blockade confirmed by Horner's (ptosis, anhydrosis, miosis) – although may get Horner's and still not complete upper extremity sympathetic blockade; Malmqvist et al. developed criteria comprising four elements of change believed necessary to confirm complete sympathetic block to the arm - (a) Horner's syndrome, (b) skin temperature rise to 34°C or higher, (c) greater than 50% increase in skin blood flow, and (d) complete abolition of skin resistance response. (Some studies reveal inadequacy of complete sympathetic blockade with the C6 approach)\*

Complications – mechanical – pain, hematoma, pneumothorax, pneumomediastinum, esophageal injury, brachial plexus injury, vasovagal response; pharmacologic – horner's (expected), spinal analgesia, brachial plexus and phrenic nerve block with difficulty breathing, recurrent laryngeal block with hoarseness (avoid bilateral stellates!), seizure secondary to intravascular injection

Contraindications – contralateral phrenic palsy, blood dyscrasia/coagulopathy, local infection/sepsis, patient refusal

### **Celiac Plexus/Splanchnic Nerve Block**

Anatomy – sympathetic supply to the abdominal viscera arises in the antero-lateral horn of the spinal cord; preganglionic fibers from the spinal segment T5 – T10 give rise to the greater splanchnic, T11 – T12 lesser splanchnic, T12 least splanchnic; these lie along the thoracic vertebrae and then join the celiac plexus; also receives some parasympathetic innervation from the vagal nerves; the celiac ganglion is a meshlike structure that lies in front of the aorta, retroperitoneally; measures 1 – 4.5 cm at the level of L1, from there postganglionic fibers supply the abdominal viscera; pain transmitted via the celiac plexus is primarily from the upper abdomen, including pancreas, diaphragm, liver, spleen, stomach, small bowel, ascending and proximal transverse colon, adrenal glands, kidneys, abdominal aorta, and mesentery

Technique – multiple approaches including posterior, anterior, and open (surgical); posterior approach can be retrocrural, transcrural, or transaortic, where the needle lies in front of the aorta, transcrural is celiac plexus block, retrocrural is splanchnic nerve block (\*for the most part, we traditionally perform the retrocrural, splanchnic nerve block here at Stanford secondary to distorted anatomy of invasive tumors often limiting the transcrural approach); all provide sympathetic blockade, but splanchnic is better when tumor invasion distorts anatomy; posterior approach is the classic approach – patient is prone, either fluoroscopy or CT-guided (we perform with fluoroscopy); difference between transcrural and retrocrural is final needle position; if the needle tip ends up at the level of the T12 vertebra in the lower third of the anterior lateral area, then it is transcrural; if the needle is at the middle to upper third of the L1 vertebra, it is retrocrural; the needle is usually inserted at the edge of a triangle formed by the T12 rib, L1 transverse process, and tip of the T12 spinous process; needle directed according to retrocrural or transcrural approach; bilateral needles should be used; position confirmed by AP and lateral views with contrast spread; following confirmation of dye spread, either local anesthetic (8-15 ml) on each side, or alcohol or phenol for neurolysis; before lytic block, local should be injected first to ensure there is no intravascular, intrathecal, or epidural spread as confirmed by spinal analgesia – then inject the neurolytic agent

Indications – abdominal visceral pain, including malignancy (gastric malignancy, pancreatic ca), acute/chronic pancreatitis, biliary sphincter disorder with pain, abdominal angina; (\*here at Stanford, secondary to risks associated with neurolysis, we traditionally only perform neurolysis in cancer patients)

Complications – mechanical – injury to the blood vessels (AORTIC DISSECTION), retroperitoneal hemorrhage, injury to kidney, ureter, lung, pleura (pneumothorax, hemothorax); PARAPLEGIA secondary to intravascular/intrathecal injection or trauma to the blood supply to the anterior spinal artery (from the artery of adamkeiwicz); 1 /683 risk of serious adverse outcome (ie. Paraplegia) with neurolytic block; pharmacologic – hypotension and diarrhea secondary to sympathetic blockade, intravascular injection - seizures

### **Lumbar Sympathetic Block**

Anatomy – preganglionic flow to the lower extremities arises from the dorsolateral part of the spinal cord (lower thoracic and upper 2 lumbar segments); fibers synapse into the lumbar sympathetic ganglion, which is located in the anterior lateral surface of the L 2- L4 vertebrae, anterior to psoas, most postganglionic sympathetic fibers accompany nerve roots to the lower extremity

Technique – patient prone, with fluoroscopic guidance, avoiding the transverse process of L2 or L4, a 5 inch spinal needle is advanced so that the final position of the tip of the needle is in front of and just lateral to the L2 vertebral body (in the midfacetal line) or at the superior third of the L3 vertebral body (most individuals the sympathetic ganglia is located); usually needle entry point should lie 7 – 10 cm lateral to the spinous process of L2 vertebrae; injection of dye confirms spread anterior and lateral to the vertebral body on both AP and lateral views; to confirm sympathetic blockade, monitor temperature of lower extremity, use of 3 – 5 ml of local anesthetic (avoid larger volumes to avoid spread to somatic blockade and confuse the picture)

Indications – complex regional pain syndromes in lower extremities, vascular disorders of the lower extremity (ischemia), sympathetically maintained neuropathic pain of the lower extremities

Complications – mechanical – infection, trauma to the lumbar nerve and disc, intravascular, intrathecal, and epidural injection, hematuria from renal trauma; pharmacologic – intravascular or intrathecal injection of local or neurolytic, hypotension, paraplegia, genitofemoral neuralgia with neurolysis; sympatholysis can be performed with RFA, neurolytic (careful to limit possible neurolytic spread to somatic nerves)

### **Superior Hypogastric Plexus Block**

Anatomy – plexus located retroperitoneally in the lower third of the 5<sup>th</sup> lumbar vertebral body and the upper 3<sup>rd</sup> of the sacrum in close proximity to the bifurcation of the common iliac vessel; supplied by the lumbar aortic and celiac sympathetic plexus; some parasympathetic contribution from ventral root of S2-S4; supplies the pelvic organs, sigmoid colon, rectum; communicates with the inferior hypogastric plexus

pain pathways - analgesia to the organs in the pelvis is possible because the afferent fibers innervating these structures travel in the sympathetic nerves, trunks, ganglia, and rami (therefore, a sympathectomy for visceral pain is analogous to a peripheral neurectomy or dorsal rhizotomy for somatic pain)

Indications – treatment of pelvic pain, including malignancy, endometriosis, and pelvic inflammatory disease/adhesions (\*traditionally, neurolysis at Stanford usually only in malignancy secondary to risks associated with neurolysis)

Technique – prone position on a pillow to reduce lumbar lordosis, xray beam turned 45 degrees posterolateral view at the level of the L5 vertebra; then cephalocaudal view is used to avoid the iliac crest, with the view of the anterior lateral part of the L5 vertebra identified; gun barrel technique, needle inserted so that the tip lies in front of the vertebral body of L5 (usually need to bend needle tip to avoid TP of L4 or L5); position confirmed by AP and lateral views of contrast dye spread; then 3 – 10 ml local or neurolytic on each side

Complications – infection, trauma to nerves/disc, intrathecal/epidural injection, intravascular/intrathecal injection

### **Ganglion Impar (Ganglion of Walther) Block**

Anatomy – solitary retroperitoneal structure located at the level of the sacrococcygeal junction and marks the termination of the paravertebral sympathetic chain

Indications – sympathetically maintained perineal pain, visceral pain

Technique – transsacrococcygeal ligament - needle inserted through the ligament until it lies just a few millimeters in front of the curvature of the sacrum, confirmed by dye spread followed by local anesthetic or neurolytic

Complications – caudal/epidural injection, injury to rectum/periosteum, infection

### **References:**

Boas, Robert A. “Sympathetic nerve blocks: In search of a role.” *Regional Anesthesia and Pain Medicine* 23(3): 292-305, 1998.

Day, Miles. “Sympathetic Blocks: The evidence.” *Pain Practice*, Volume 8, Issue 2, 2008 98–109.

De Leon-Casasola, Oscar A. “Critical Evaluation of Chemical Neurolysis of the Sympathetic Axis for Cancer Pain.” *Cancer Control*. March/April 2000, Vol. 7, No.2.

Malmqvist EL, Bengtsson M, Sorensen J. “Efficacy of stellate ganglion block: A clinical study with bupivacaine.” *Regional Anesthesia*. 1992; 17: 340-347.

Mercadante, Sebastiano. “Celiac plexus block: a reappraisal.” *Regional Anesthesia and Pain Medicine* 23(1): 37-48, 1998.

## **TRANSCUTANEOUS NERVE STIMULATION**

*Stephen Coleman, MD*

### **Technique**

- High frequency (>50 Hz)
- Stimulate at increasing amplitude until patient feels comfortable tingling.
- Low frequency (<10 Hz)
- Stimulate and increasing amplitude until patient feels tapping without motor contraction (low intensity).
- Stimulate at increasing intensity to produce motor contraction yet below noxious level (high intensity).
- Pulse duration and amplitude are variable.
- Theories of TENS

- Gate control theory
- Peripheral stimulate large afferent A-beta fibers inhibits nociceptive transmission in the dorsal horn. This theory supports a segmental effect at the spinal cord level.
- Contrary data include reduction in TENS analgesia in spinalized animals and antihyperalgesic effects outlast stimulation time by 8 to 24 hours.
- Neurotransmitter theory
- Blockage of adenosine receptors with caffeine significantly reduced effect of TENS
- Release of endogenous opioids and serotonin. Opioid agonists are increased in CSF after TENS.
- Clinical Studies (inconclusive)
- Studies included heterogeneous populations
- Many studies were done without placebo
- TENS may have a placebo effect.
- Stimulation parameters (frequency, intensity, pulse duration) not standardized.
- Placement of electrodes not standardized
- Outcome measures not standardized.

**Note:**

Sluka K, Walsh D. Transcutaneous Electrical Nerve Stimulation: Basic Science Mechanisms and Clinical Effectiveness. *Journal of Pain*. 2003;4(3):109-121. [PDF](#)

Johnson M, Martinson M. Efficacy of Electrical Nerve Stimulation for Chronic Musculoskeletal Pain. *Pain*. 2007;130:127-165. [PDF](#)

**Questions**

1. What are the variables controlled when using TENS?
2. Frequency, amplitude, pulse width, location, duration of treatment and frequency of treatment.
3. Clinical efficacy well documented in randomized clinical trials? What are some of the reasons that may account for the lack of efficacy in clinical trials.
4. Describe theories to explain TENS effects

**SPINAL CORD STIMULATION**

*Einar Ottestad, MD*

SCS is an adjustable, nondestructive, neuromodulatory procedure that delivers therapeutic doses of electrical current to the spinal cord for the management of neuropathic pain.

**Indications:**

- Failed back surgery syndrome
- Complex regional pain syndrome
- Peripheral neuropathy
- Phantom limb pain
- Postherpetic neuralgia
- Deafferentation pain
- Abdominal pain
- Pelvic pain
- Spinal stenosis

- Radiculopathy
- Anginal pain
- Peripheral vascular disease

### **Mechanism of action:**

- General
  - Gate control theory: Activation of A beta afferents inhibits transmission of C fibers and A delta fibers
  - Antidromic action potentials pass caudally in dorsal column to activate spinal segmental mechanisms in dorsal horns
  - Ascending action potentials to the brainstem activate descending inhibitory pathways
  - Changes to neurotransmitters such as release of serotonin, substance P, GABA within dorsal horn
- Specific
  - Angina:
  - Redistribution of myocardial flow
  - Inhibits activity of spinothalamic tracts evoked by cardiac sympathetic afferents
  - Less ST depression/EKG evidenced ischemia, less use of nitrates, less pain
  - More exercise capacity, QOL, time to angina
  - 5-year survival similar to CABG, less cost/complications
  - Decrease in NYHA grade
  - Does not conceal acute MI or true ischemia

### **PVD**

- Increased blood flow
- Inhibition of sympathetic outflow
- Higher level of limb salvage
- More favorable outcome than arterial reconstruction in one study

### **Contraindications:**

- Sepsis
- Coagulopathy
- Previous surgery obliterating spinal canal
- Local infection
- Psychological comorbidity

### **Anatomy:**

- C2 – Occipital and jaw
- C2-4 – Shoulder
- C5-6 – Arm/hand
- Low cervical-high thoracic –Angina
- T5-7 – Abdominal pain
- T8-10 – Axial LBP, FBSS, radiculopathy
- Low thoracic high lumbar – Pelvic pain
- Sacral – Pelvic pain

### **Procedure:**

- Percutaneous: Trial and implant
- Prone, local anesthetic, IV sedation

- Fluoroscopic guided entrance 2-3 levels below target
- LA, 14g Tuohy paramedian, LOR technique
- Live fluoro-guided advancement of lead to target level
- Lateral fluoroscopy to confirm dorsal vs. ventral epidural space
- Connect to programmer and confirm appropriate paresthesia
- Steri-strip/tegaderm; 3-5 days with external IPG

### **Implant:**

- Prone, LA, IV sedation
- 3-4 cm vertical incision and dissection to spinous processes
- Tuohy, lead placement, confirmation of paresthesia
- Anchor lead to interspinous ligament
- Pocket creation on lateral buttock
- Lead extension connected
- Tunnel extension from back incision to pocket
- Connect extension to pocket and to IPG
- Close both incisions

### **Surgical:**

- Laminotomy or laminectomy by ortho/neuro spine surgeon
- General anesthesia
- Paddle leads can be more effective

### **Complications:**

- Migration ~ 11%
- Usually in first few days
- Fibrosis eventually secures lead (4<sup>th</sup> week)
- Infection ~ 3-5%
- CSF leak
- Seroma/hematoma

### **Results:**

50-60% of patients obtain 50% pain relief. Goal is not NO pain.

With time, SCS might need to be reprogrammed or voltage increased as scar tissue forms.

Patient has PDA that can turn SCS on/off, change voltage, change programming

Rechargeable IPGs available.

### **Questions:**

1. Can a patient who is s/p thoraco-lumbar scoliosis surgery have a percutaneous SCS trial for L3 radiculopathy?
2. Can a S3 radiculopathy be covered by SCS? How?
3. Patient X is getting surveillance MRI for h/o brain meningioma s/p resection. Should she get a SCS for right leg CRPS?

### **References:**

Falowski S, et al. Spinal cord stimulation: an update. *Neurotherapeutics* 2008; 5:86-99.

Taylor R, et al. Spinal cord stimulation in the treatment of refractory angina: systematic review and meta-analysis of randomized controlled trials. *BMC cardiovascular disorders* 2009; 9:13.

## RADIOFREQUENCY

Joshua Pal, MD

By utilizing a specialized RF lesion generator machine, and special needles, we can create defined, well localized, and predictable fields of radiofrequency energy around the tip of RF needles.

The goal is to apply these fields to sensory (not motor) nerves to reduce pain.

We can monitor the impedance and temperature at the needle tip to confirm that the correct amount of RF energy is being delivered to a relatively circumscribed field of tissue

Similar to nerve stimulator guidance in regional anesthesia, We optimize needle placement by trying to elicit sensory stimulation of the target nerve at around, but preferably below, 0.2-0.5 volts (at a stimulation frequency of 50 herz) and confirm. In order to avoid damaging a motor nerve, we confirm a lack of motor stimulation with final needle placement, usually below 2 volts .

RF energy delivered in "pulses" of 20msec bursts twice per second and is not typically painful (PulsedRF, or PRF) (eg, 40 degrees celsius for 60-120 seconds) to nerves does not denature tissue or heat up tissue enough to cause a thermal lesion. The neurodestructive threshold has been thought to be 45 degrees celsius. PRF of peripheral nerves is sometimes performed with 45-60 volts at 2 herz for four minutes PRF has (controversially) been shown to "modulate" target neural structures and decrease nociceptive transmission (it is theorized that the PRF alters gene expression within the neural structure).

It is not typically painful. It is thought unlikely to damage nerves and therefore safer. Uses include PRF of the occipital nerves, ilioinguinal nerves, DRGs, medial branch nerves, gasserian ganglion, peripheral nerves, facial nerves, intercostal nerves, etc

Continuous RF energy of a higher temperature for a longer amount of time (eg, 80 degrees for 80 seconds) creates a "burn" that denatures tissue, including the target nerve. It is typically painful, and often local anesthetic is administered through the needle to prevent pain with lesioning. Great care must be taken to ensure no adjacent structures are affected by the RF field. Sensory and motor testing is often carried out to confirm that adjacent neural structures are not close enough to the needle tip to be effected by the RF lesion.

Sometimes PRF is performed first in the hopes that this safer procedure will be effective; if it is not, sometimes the procedure is repeated with continuous RF.

Patient Name \_\_\_\_\_

Examiner Name \_\_\_\_\_ Date/Time of Exam \_\_\_\_\_



**STANDARD NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY**



**MOTOR**  
KEY MUSCLES (scoring on reverse side)

	R	L	
C5	<input type="checkbox"/>	<input type="checkbox"/>	Elbow flexors
C6	<input type="checkbox"/>	<input type="checkbox"/>	Wrist extensors
C7	<input type="checkbox"/>	<input type="checkbox"/>	Elbow extensors
C8	<input type="checkbox"/>	<input type="checkbox"/>	Finger flexors (distal phalanx of middle finger)
T1	<input type="checkbox"/>	<input type="checkbox"/>	Finger abductors (proximal finger)

UPPER LIMB TOTAL (MAXIMUM)  +  =  (25) (25) (50)

Comments:

L2	<input type="checkbox"/>	<input type="checkbox"/>	Hip flexors
L3	<input type="checkbox"/>	<input type="checkbox"/>	Knee extensors
L4	<input type="checkbox"/>	<input type="checkbox"/>	Ankle dorsiflexors
L5	<input type="checkbox"/>	<input type="checkbox"/>	Long toe extensors
S1	<input type="checkbox"/>	<input type="checkbox"/>	Ankle plantar flexors

Voluntary anal contraction (Yes/No)

LOWER LIMB TOTAL (MAXIMUM)  +  =  (25) (25) (50)

**SENSORY**  
KEY SENSORY POINTS

0 = absent  
1 = impaired  
2 = normal  
NT = not testable

	LIGHT TOUCH		PIN PRICK	
	R	L	R	L
C2				
C3				
C4				
C5				
C6				
C7				
C8				
T1				
T2				
T3				
T4				
T5				
T6				
T7				
T8				
T9				
T10				
T11				
T12				
L1				
L2				
L3				
L4				
L5				
S1				
S2				
S3				
S4-5				

TOTALS:     (MAXIMUM) (50) (50) (50) (50)

Any anal sensation (Yes/No)

PIN PRICK SCORE (max: 112)

LIGHT TOUCH SCORE (max: 112)

NEUROLOGICAL LEVEL: The most caudal segment with normal function

NEUROLOGICAL LEVEL	R	L	COMPLETE OR INCOMPLETE? (Example - Any sensory or motor function in S4/S5)	ZONE OF PARTIAL PRESERVATION (Caudal extent of partially denervated segments)	R	L
SENSORY	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>	<input type="checkbox"/>
MOTOR	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>	<input type="checkbox"/>

ASIA IMPAIRMENT SCALE:

• Key Sensory Points

Therapeutic use of continuous RF is limited to primarily denervating some of the posterior innervation of the sacroiliac joint, the medial branch nerves that innervate facet joints (they also innervate multifidus musculature, and the loss of innervation to this musculature seems to be well tolerated), and there is support for gasserian ganglion continuous RF for trigeminal neuralgia (however a side effect is the development of anesthesia dolorosa).

RF or PRF treatment is most often preceded by positive diagnostic local anesthetic blocks of the target nerve. Some centers proceed straight to continuous RF of the cervical and lumbosacral medial branch nerves without the diagnostic block (ie, at the Palo Alto VA).

Medicare may move to requiring two diagnostic block tests

Efficacy occurs in 1-2 weeks, but may take as long as 4 weeks, and pain is sometimes worse before it improves. The evidence level for efficacy of continuous RF for facet arthropathy in the cervical spine is class 1, in the lumbar spine it is thought by some bodies to be class 1, and there are few studies

looking at denervating the posterior aspect of the SIJ and posterior annulus of lumbar intervertebral disks with larger lesions using cooled-tip RF probes (Bayliss system).

It is interesting to note that MRI imaging, PET/CT, and physical exam are thought to poorly predict outcome of either PRF or continuous RF.

Diagnostic blocks do predict efficacy.

Side effects:

In general these are very safe procedures with very rare adverse events (the exception may be RF of the gasserian ganglion, with the potential side effect of anesthesia dolorosa). This is part of the reason why at the VA we proceed straight to RF with no diagnostic blocks.

The worst-case scenario during the commonly performed RF of spinal medial branches is injury to the nerve root (this is why we confirm lack of dermatomal motor response with final needle placement).

Transient side effects include worsened pain, with a subtype being a mild-moderately painful "neuritis" for a couple weeks, which is sometimes appreciated as a "sunburn" type feeling in the area.

## **Other General Components**

### PHYSICAL EXAMINATION

*Joshua Pal, MD*

This section is designed to give you a rough sense of expectations for what to present and what to put into your note in regards to PE. It is not intended to replace review of a text that has good pictures and explanation of physical exam techniques, as well as a discussion of the relative predictive value of response to treatment (ie, both MRI imaging and P.E. tests for painful sacroiliac joint arthropathy and facet arthropathy are notoriously poor at predicting favorable response to diagnostic nerve block)

When you go into a room with a *new evaluation*, you are expected to perform the following physical exam elements, summarize them in your presentation, and document them in the chart if you are dictating.

#### **Neuro Exam:**

(For a great visual and conceptual presentation of the neuro assessment, see the ASIA impairment scale, which may be copied freely per ASIA...we will include it at the end of this section) (For nomenclature, muscle stretch reflexes = "deep tendon" reflexes)

First, document the pt's sensation to ice at their most painful site. Reduction in sensation to cold is indicative of potentially a sensory neuropathy either at the nerve root or peripheral.

#### **Lower back/LE pain:**

Examine the achilles and patellofemoral tendon reflexes and their symmetry, and L1-S1 dermatome sensation to light touch. Also check for pathological reflexes, including clonus (usually more than 3 beats is pathologic) and Babinski's (confirm no upgoing toes).

### **For neck/UE pain:**

Examine the brachioradialis, biceps, and triceps tendon reflexes and their symmetry, and C5-T1 dermatomes sensation to light touch. Also check Hofman's sign, Spurling's maneuver, and Lhermitte's sign. If they have hand or forearm pain, assess for CTS.

If you suspect *myelopathy* in the C/S, or *central pain* (ie post-stroke, MS) check all of the above. If you suspect *diabetic peripheral neuropathy*, assess at least position sense, and maybe vibration sense, of the great toes.

You may also wish to document whether the pt displays "pain behavior", as a means to convey your impression of the patient's presentation to the next person who reads your note. Note: these are not Waddell's signs, but are instead a subjective assessment of whether the pt is very vocal and colorful in their body language in conveying their suffering. Ask your attending their thoughts on this if you suspect your patient is displaying pain behaviour, and how this may or may not change management.

Document sedation, or lack of sedation, if on a relevant pharmacological regimen. Document affect and presence or absence of depression, anxiety, and risk factors for sleep apnea (anxiety/depression, sleep apnea, and hypothyroidism can exacerbate suffering as well as potentially nociception....testosterone deficiency can result from chronic high-dose opiate use, etc)

### **MSK Exam:**

LBP / LE pain/ hip pain/buttock pain/groin pain:

Depending on the pt's presentation, perform the following:

- assess for piriformis syndrome
- perform FABER's and Patrick's maneuvers
- externally rotate the hips, and perform scouring maneuvers with the pt supine
- pain with palpation over the superior aspect of the SIJs bilaterally, as well as the gluteus medius muscles (they overlap, and it is sometimes difficult to determine whether its pain from the SIJ or with palpation of the gluteus medius)
- relative tone of the lumbar paraspinals
- pain with lumbar facet loading maneuvers, pain with palpation of the lumbar facets (L3-L5 area), or do both combined
- straight leg raises bilaterally, and note the degree at which pain radiating below the knee is elicited, and whether its ipsilateral or contralateral (if greater than 45 degrees, not a true positive)
- pain with palpation of the greater trochanteric bursae
- for groin pain, thigh flexion against resistance

Neck pain/Headache/UE pain/shoulder pain/pain between the shoulder blades

- relative tone of the cervical paraspinals and trapezius
- pain with cervical facet loading maneuvers, pain with palpation of the lumbar facets (C5-T1 area), or do both combined
- assess for whether tapping over the proximal aspect of the occipital nerve evokes pain radiating anteriorly (occipital neuralgia)
- assess for scapular winging (sometimes this is subtle if their most painful area is between their scapulae and worse at the end of the day, but may be a clue for notalgia paresthetica)

- assess for whether the patient has tender points versus trigger points (trigger points refer pain when palpated)...these are often found in the trapezius or adjacent to the superomedial corner of the scapula, where many neck/shoulder muscles insert)
- assess for external rotation of the shoulder (rule out frozen shoulder, which can develop secondarily)

When you go into a room with a *return/follow-up patient*, it is appropriate to perform a focused physical exam of the painful area. While learning chronic pain management, it is probably helpful to do at least this for your own learning. However, the attendings and fellows may not always do this because it is unlikely to change their management if the patient's verbal report is unchanged from the initial visit. Some believe that patients may have greater satisfaction with their visit if they are physically examined (ie, chiropractic care gets very high patient satisfaction scores even though it does not have very high pain reduction scores, which may be from a high "touch factor", which may also create a greater placebo response...but this is purely conjecture).

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## OUTCOMES IN PAIN MEDICINE

*Sean Mackey, MD, PhD*

### Background

Why is it important to evaluate outcomes in pain medicine? This question has become much more relevant in today's environment of evidence based medicine (EBM). Soon – if in fact the time has not already arrived - we will no longer be able to say "In my experience..." and expect our colleagues, payers, and even patients to accept the most erudite explanation without responding with a demand for published evidence. Today we are expected to be able to reference multiple randomized controlled trials to support our decision for a particular test or therapy. Unfortunately, in the realm of Pain Medicine, the field is sorely lacking a large quantity of these studies. Nonetheless, it is incumbent on us to accurately measure what we can to so that we can address the following questions:

- How do you know that you have helped a patient with chronic pain?
- How do you know when a certain treatment is more appropriate than another for a specific disease?
- How do you describe the unique benefits of the services you offer?

The purpose of this article is to review the multiple dimensions of pain and the current tools we have to measure that experience. The focus will be on self-administered tools that are readily available for a clinical environment.

## **Pain – A Multidimensional Experience**

Pain has been defined by the International Association for the Study of Pain (IASP - 1979) as “an unpleasant sensory or emotional experience associated with actual or potential tissue damage, or described in terms of such damage.” This defines pain as a subjective experience; therefore, unlike many diseases such as hypertension or diabetes, there is no objective measurement for a patient’s pain. We must do our best to correlate objective data (physical exam findings, imaging results, lab tests) with the patient’s subjective reporting. Further complicating the problem is the fact that pain is often confused with the concept of nociception - the neural signals generated and transmitted to the spinal cord and brain in the face of stimuli that are potentially or actually tissue-damaging. Pain, in contrast, requires a functioning brain to process these nociceptive signals and translate them into a subjective experience.

This contrast is particularly important in chronic pain conditions where there is a lack of objective tissue damage but pain is still present. These neuropathic or non-nociceptive conditions are often the result of dysfunction or damage within the spinal cord, brainstem, or brain. It is this damage to the peripheral and central nervous system that warns us of actual or potential tissue damage. However, with neuropathic pain, these signals have become maladaptive in that they impart no beneficial survival value. Chronic pain becomes insidious in that it is often associated with depression, anxiety, decreased libido, impaired physical functioning, altered appetite, and sleep disturbances -- an overall reduction in the patient’s quality of life. It is clear that pain, both acute and chronic, can impact a patient in many domains that no single pain score can properly encompass. Therefore, to fully capture as much of the pain experience as is possible, it is necessary to characterize these domains in the greatest possible detail.

## **Qualities of a Good Outcome Instrument**

Some of the many attributes an instrument should possess to dependably capture a patient’s pain experience are listed below. Furthermore, when a clinician chooses an instrument to measure pain outcomes, he or she should provide literature support for the following essential features:

- **Reliability**—The instrument should be reliable. It should be stable over time. It should have test-retest reliability: when patient’s status does not change...provide similar score when the patient’s status does not change (test-retest reliability; Pearson’s  $r$ ). If the scale is rated by clinicians, rather than patient self-reported, then clinicians observing the same patient should provide very similar scores (inter-rater reliability; Cohen’s  $\kappa$ ). If the scale contains multiple items, they should measure the same construct and correlate with one another (internal reliability; commonly tested with Cronbach’s  $\alpha$ ).
- **Validity**—Second, the scale should be valid; it should measure what we think it measures. In particular, the scale should agree with other indicators of pain (convergent validity) and be distinguishable from other related conditions such as anxiety and depression (discriminant validity).
- **Other Factors**—The reliability and validity of a scale depend upon several other factors: (1) The scale should be feasible to administer and score, not burdensome for either patient or clinician, (2) it should be sensitive to change, so that the score reflects true improvement or deterioration in the patient’s status, and (3) the scale must accurately reflect the target patient population, so that scores do not tend to clump in a restricted area of the scale, or go off the charts.” When analyzed with statistical

techniques such as regression, outcomes measures should preferably be at least interval in scale (Table 2). All points should be equidistant, although this criterion is hard to meet, as measures of pain are ordinal. For example, at first glance, a 0 to 10 measure of pain seems to have 10 equally spaced intervals. However, in the patient's experience, the "distance" between 0 and 1 is likely quite different from that between 9 and 10. Despite this issue, many pain measures are treated as having an interval scale, which allows the computation of means greater flexibility in choosing statistical tests. In general, this approach is acceptable, but the measure should have at least 5 distinct points.

### **Tools to Measure Pain**

Any tool used to measure pain should be consistent with the practice setting and provider, and patient needs. In a busy primary care setting, or even some subspecialty environments, it may be unreasonable to have the patient fill out multiple outcome forms if the facility lacks both sufficient staff to process the information and the infrastructure necessary to analyze and interpret the data. The instruments need to be reliable and valid for the population of interest. For instance, many practitioners combine general purpose instruments with disease specific surveys (e.g. Oswestry Disability Scale [1] for low back pain).

#### *Self-Reported Unidimensional Instruments*

Historically, the methods and instruments used to measure pain assumed pain was a single quality that varied only in intensity. These methods have included visual analog scales, numerical rating scales, and verbal rating scales. These methods have been used effectively in hospital clinics and acute settings to provide useful information about pain and analgesia.

#### **Visual Analog Scale**

The visual analog scale (VAS) is a relatively simple and efficient instrument to administer. It is typically a 10-cm line anchored at one end by the label "no pain" and the other end by a label such as "worst pain imaginable." The patient marks the line to indicate their pain intensity, and the clinician measures the length of the line on a 101-point scale [2]. Slide rule-like devices are also available that make measurement easier [3]. The device has a line on the patient side and a numeric score on the clinician side.



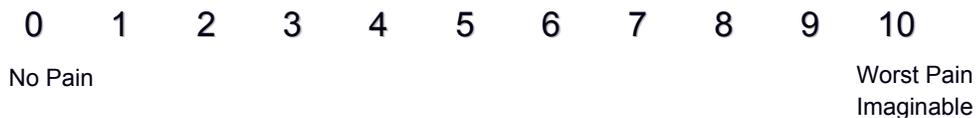
The advantages of the VAS are that it has many - in fact an infinite number of - response categories; average group scores can be treated as ratio data [4] and there is good evidence for validity. The weaknesses and limitations are that it can be more time consuming than other instruments. Some patient populations, particularly elderly people, have difficulty using the VAS resulting in higher failure rates [2, 5].

#### **Verbal and Numerical Rating Scales**

Verbal rating scales (VRS) consist of a series of verbal (categorical) descriptors ordered from least to most intense (i.e., none, mild, moderate, severe). The advantages of the VRS are that it is very easy to

administer and elderly patients may make fewer errors compared with a VAS [6]. Significant disadvantages are that it has relatively fewer response choices and the categorical nature of the instrument often requires more sophisticated statistical analysis techniques than measures made with the VAS.

The numerical rating scale (NRS) is the most commonly used unidimensional instrument. While variations exist, it typically consists of a rating scale from 0 to 10, with the left-most 0 meaning “no pain” and the right-most 10 meaning “worst pain imaginable.” Patients respond either orally or in writing with the number that best represents the intensity of their pain. A similar scale with 0 to 100 is also commonly used; however, the 0 to 10 version provides the same quality of information, while being more easily understood by patients. The NRS minimizes both patient and staff burden during data collection and computation. Patients are highly compliant when using this measure.



In busy clinical settings where there is an effort to minimize staff and patient burden during data collection, the simplest and most frequent approach is to use a numeric rating scale (NRS). There are typically an adequate number of pain response categories, and compliance with the measurement task is high. It can also, in contrast to the VAS, be administered via an interview. One of the primary weaknesses, however, is that the scores can not necessarily be treated as ratio data.

### *Self-Reported Multidimensional Instruments*

Except for the most simple and brief pain experiences, a more comprehensive assessment beyond the VAS, NRS or VRS is required. This assessment should include intensity and quality of the pain, interference with function, and effect on daily living and quality of life. Pain location is usually assessed by having the patient mark a body drawing. Measuring interference from pain will aid both clinical assessment as well as treatment efficacy. A patient may report a low pain score but also have low physical functioning. Similarly, if a patient reports favorable or effective pain relief from treatment, but no improvement in physical activity, then the report is either spurious or an incomplete indicator of the treatment effect if functional rehabilitation is one of the primary goals. A growing body of literature supports the importance of measuring physical functioning with several available instruments. These range from general purpose surveys (e.g. Brief Pain Inventory [7], Sickness Impact Profile [8], and West Haven-Yale Multidimensional Pain Inventory [9]) to condition specific measurements of function (e.g. Roland-Morris Questionnaire, Oswestry Questionnaire [1]). A sampling of these instruments are described below.

### **The McGill Pain Questionnaire**

The McGill pain questionnaire (MPQ) was developed by Melzack and Torgerson [10] to specify the qualities of pain and remains one of the more extensively tested multidimensional scales available. Pain is scaled in three dimensions: sensory, affective, and evaluative. The questionnaire consists of 20 sets of words that describe the quality of pain. Each set of words has from two to six words that vary in intensity for the quality of pain measured by the set. Multiple studies have supported the reliability and concurrent validity of the instrument [11]. It has been translated into multiple languages and has been

used in more than 500 acute and chronic pain studies. It does take 5 to 15 minutes to complete, thereby putting an additional burden on the patient relative to the VAS or NRS.

As a clinical tool, the MPQ helps characterize the quality of the patient's pain in both its affective and sensory effect. It has shown remarkable consistency in the words chosen by patients suffering from specific pain syndromes. The Short-Form McGill Pain Questionnaire (SF-MPQ) [12] was developed for research purposes and situations where the standard MPQ takes too long to administer, but the information derived from NRS and VAS are inadequate. It consists of 15 words from the sensory (n=11) and affective categories from the standard long form. The present pain intensity and VAS are also presented to provide overall assessment of pain intensity.

### **Brief Pain Inventory**

The Brief Pain Inventory (BPI) was developed Pain Research Group of the WHO Collaborating Centre for Symptom Evaluation in Cancer Care [7] with the goal of measuring both the intensity of pain (sensory dimension) and the interference of pain in the patient's life (reactive dimension). The BPI has been used extensively for many pain syndromes (although most data pertain to cancer pain) and typically takes 5 to 15 minutes to complete. It has been validated in a number of countries and languages and demonstrates good sensitivity to treatment effects (mostly in pharmacological treatments).

### **West Haven-Yale Multidimensional Pain Inventory**

The West Haven-Yale Multidimensional Pain Inventory (WHYMPI) is an alternative to the BPI that has been used for a variety of pain conditions in multiple countries. It is composed of 56 items with three parts: (a) five dimensions covering the experience of pain and suffering, interference with family, social and work functioning; (b) the patient's perception of the degree to which the spouse or significant other displays solicitous responses to pain behaviors; and (c) the degree to which the patient engages in common daily activities. Patients respond to the questions on a 7 point scale. The WHYMPI has been validated for multiple pain syndromes and is sensitive to treatment effects. It is broader in approach than the MPI and is a valuable assessment tool for both the clinician or researcher who wants to assess multiple dimensions of adaptation to chronic pain.

### **Medical Outcome Study 36-Item Short-Form Health Survey**

It is well recognized that functional limitations are only one aspect of overall perceived health, well-being and quality of life. The most widely used instrument to measure multiple dimensions of quality of life is the Medical Outcome Study 36-Item Short-Form Health Survey (SF-36) [13]. It consists of 8 subscales including: Physical Functioning; Role Limitations due to Physical Problems; Social Functioning; Bodily Pain; Role Limitations due to Emotional Problems; General Mental Health; Vitality; and General Health Perceptions. It was intended to be used for large heterogeneous patient populations, however it has been used for almost every disease condition imaginable. It is relatively easy to administer, taking approximately 10 minutes to complete. The scores can be compared to normative data for the U.S. adult population. While it is widely used, there are problems with its use as a sole chronic pain assessment tool. It only has two questions related to pain and there are concerns about its sensitivity to change and the usefulness of some of the scale scores.

### **Treatment Outcomes of Pain Survey**

The Treatment Outcomes of Pain Survey was meant to be an extension of the SF-36 specifically designed for patients with chronic pain [14, 15]. TOPS derived many of its questions from the SF-36, MPI, BPI, Fear Avoidance Beliefs Questionnaire (FABQ) [16], and Work Limitations Questionnaire [17]. It is a self-administered questionnaire (6 or 10 pages with 63 or 98 questions respectively) with the following core domains: Pain Symptoms; Functional Limitations; Perceived Family/Social Disability; Objective Family/Social Disability; Patient Satisfaction Outcomes; Fear Avoidance; Passive Coping; Solicitous Responses; Work Limitations; and Life Control. TOPS also captures demographics, patient satisfaction with treatment and substance abuse information. The scale scores have been found to be sensitive to change and it has been used in several large academic and private pain centers with good validity.

### **Measurement of Pain in Children**

Pain instruments used with children must be compatible with children's rapidly developing cognitive abilities. The clinician must take care to ensure that the child can meaningfully respond to the scale and understands its intervals. Some researchers advocate calibrating the child's ability to use the scale by having the clinician ask him or her to rate past painful experiences. For example, "Have you ever been stung by a bee? If so, where that pain would rate on the pain scale?" By testing a child's response to several events with assumed differing intensities of pain, the clinician can assess the child's understanding of the measure.

Many methods for assessing pain in children involve modifying adult scales. For example, the Colored Analog Scale (CAS) is simply a VAS with gradually increasing red coloring to indicate increasing intensity of pain. Many instruments are like a VRS or an NRS, but they replace the word descriptions with faces displaying increasingly negative expressions and emotions. An example is the Wong-Baker FACES Pain Rating Scale. A major disadvantage of the scales is difficulty separating pain from other sources of sadness, anxiety, and distress.

### **Assessment of Emotional Functioning**

There is a well established relationship between pain and emotional distress, and evidence of relative independence. Anxiety, fear, depression and anger all contribute to worsening pain and poorer treatment outcomes. Measurement of depression include instruments such as: Beck Depression Inventory [18], Hamilton Rating Scale for Depression [19], and Zung Self-Rating Depression Scale [20]. Anxiety measures include: State-Trait Anxiety Inventory, Pain Anxiety Symptoms Scale [21] and Fear-Avoidance Beliefs Questionnaire [16]. Finally, anger measures include: Buss Durkee Hostility Inventory [22], Anger Inventory and State-Trait Anger Expression Inventory. With appropriate staffing and resources, one or more of these instruments can provide valuable insight into the emotional functioning of the patient with chronic pain and may guide appropriate therapy.

### **Pain Measurement for Clinical Trials Design**

Representatives from academia, industry and government have been recently meeting to address the need of developing accurate pain measurements for clinical pain trials. They have developed the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT) with two publications [23, 24] outlining their recommendations. They have recently identified 6 core outcome domains that should be considered when designing clinical trials. These core domains include: pain; physical functioning; emotional functioning; participant ratings of improvement; symptoms and adverse

events; and participant disposition. They have recommended several specific outcome instruments to measure these domains, many of which have been summarized in this article.

## Conclusion

The measurement of pain remains a difficult problem, but there is considerable interest and research in theory and technological development. Most clinicians now recognize that pain is a multidimensional experience requiring assessment extending well beyond the simple 0 to 10 pain scores we frequently use in the acute setting. Sensory, emotional, functional, motivational, and cognitive aspects of our patients' pain all deserve appropriate evaluation so that we can confidently and accurately answer the questions posed in the opening paragraphs of this article.

Given the multiple approaches and instruments related to assessing pain outcomes, deciding upon the correct tool for any given patient or study is difficult. Each scale described above represent a compromise between factors of sensitivity, statistical robustness, comprehensiveness, quickness, ease of use, etc. When you choose an instrument, be sure you know what critical construct it measures. In an acute pain context, for example, pain intensity may be the most important variable, whereas in the functional rehabilitation of a chronic pain patient, global physical activity or satisfaction with life may be more important variables than pain intensity.

It is tempting to administer an excessive number of instruments to tap into all possible aspects of pain. However, patient compliance may drop as the total questionnaire packet length increases in size; therefore, a trade-off exists between the quality and quantity of data. Even if a patient completes a packet, the patient's responses may be made more hastily and less reliably in its latter parts. Moreover, a patient's tolerance to questionnaire length can differ drastically depending on his or her level of interest in the questionnaire's subject, reading level, the degree to which the pain condition is distracting, temperament, and degree of rapport with the administrator of the questionnaire. Fewer than 75 items (responded to in under 25 minutes) or about 5 pages should be acceptable to a majority of patients. This restriction often forces clinicians to make tough decisions regarding what measures are absolutely critical to the study. For measures completed every day, fewer than 5 minutes is preferable. One helpful way to balance data quality with feasibility is to administer quick, unidimensional measures daily and more comprehensive, multidisciplinary questionnaires every two weeks.

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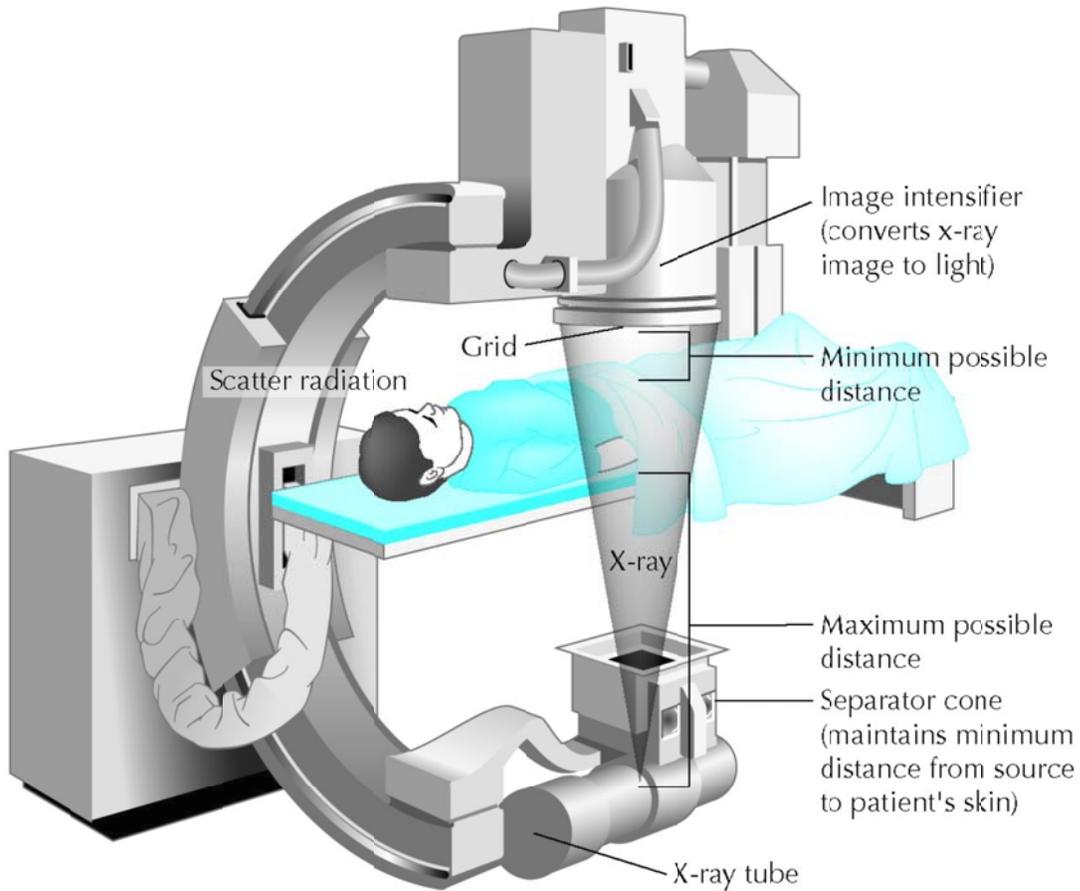
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## RADIATION RISK AND INTERVENTIONAL PAIN PROCEDURES

*Michael Leong, MD*



**Figure 1.** The C-arm fluoroscopic configuration. Maintaining a large “source-to-subject” and a small “subject-to-image intensifier” distance improves image quality and reduces patient dose and scattered radiation levels within the suite.

**Table 2. Annual maximum target area/organ permissible radiation dosages**

Area/organ	Annual maximum permissible dose in roentgen-equivalent man
Thyroid	50
Extremities	50
Lens of the eye	15
Gonads	50
Whole body	5
Pregnant women	0.5

Fig 1 & Table 2 from: Broadman L, et al. Radiation risk management during fluoroscopy for interventional pain medicine physicians. *Current Pain and Headache Reports*. 2004;8:49-55. [PDF](#)

**Table 3. Minimum Target Organ Pathologic Radiation Dose and Its Effects**

Target Organ	Dose*(rad)	(Gy)	Results
Eye lens	200	(2)	Cataract formation
Skin	500	(5)	Erythema
Skin	700	(7)	Permanent alopecia
Whole body	200 to 700	(2 to 7)	Death from infection due to hematopoietic failure (4 to 6 weeks)
Whole body	700 to 5,000	(7 to 50)	Death from GI failure (3 to 4 days)
Whole body	5,000 – 10,000	(50 to 100)	Death from cerebral edema (1 to 2 days)

\*Minimum dose that may produce the results (e.g., cataract, permanent alopecia).

From: Fishman S, et al. Radiation safety in pain medicine. *Reg Anes and Pain Med*. 2002;27:296-305. [PDF](#)

See also:

Botwin K, et al. Radiation exposure of a spinal interventionalist performing lumbar transforaminal epidural steroid injections. *Arch Phys Med Rehab*. 2002;83:697-701. [PDF](#)

Most interventional pain procedures utilize fluoroscopy for:

- Visualization of bony structures and neural foramina
- Safety of patient and documentation of placement of block
- Reimbursement

Any practitioner performing the procedure needs to understand exposure and safety to self, other room personnel, and for the patient.

Physics:

- Time - duration exposed to radiation
- Distance – space between the individual and x-ray source
- Exposure – quantity of radiation intensity measured in Roentgen ® or Coulomb / kg
- Radiation – energy absorbed by the individual in Radiation Absorbed Dose (Rad) or SI unit (Gray)
- Radiation – causing cellular damage in Radiation equivalent man (REM) or Sievert (Sv) in SI units; the later being a quality factor with variable impact on different tissues

#### Controlling exposure

- Decrease time – use freeze frame feature and minimize beam on time
- Increase distance – a major source of radiation is the patient acting a conduit for scattered radiation
- Inverse square law – the fall off of scatter intensity; doubling the distance produces ¼ exposure; at a distance of 10 or more feet, irradiation will be negligible; radiation exposure is not just along the path of the beam but everywhere within the suite
- Cross-table lateral – scatter of radiation is 2 to 3x at the entrance surface of x-ray tube then exit surface of image intensifier

Automated timer on the C-arm Fluoroscopy machines is a 5 minute limit

Shielding with lead: body, thyroid, eyes (min lead 0.35 mm attenuate scatter), surgical gloves, lead barriers; the patient – reproductive organs

Awareness of the room personnel: call “picture” to warn everyone in the room, including yourself that you are about to take an image and to move all unnecessary objects, such as your fingers out of the beam.

Collimation – making the image as small as possible and hence the area of exposure

#### Personal dosimeters

Certain procedures with multiple levels, such as radiofrequency of multiple cervical medial branches or spinal cord stimulation trials may require prolonged radiation exposure.