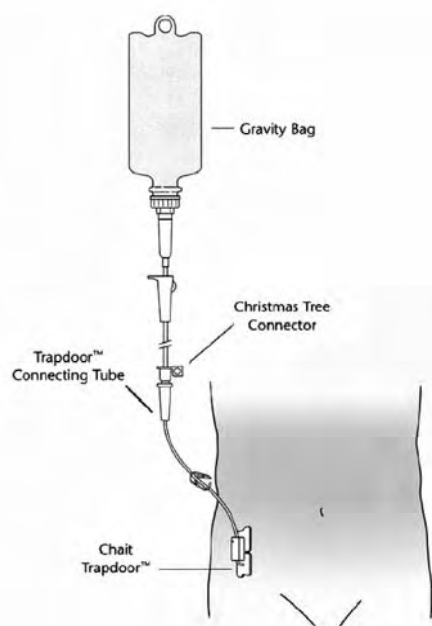


Chait/ACE set-up

Most enemas get up about halfway and then fall down. An irrigation schedule can be every one, two or every third day and evacuation occurs within about 15-30 minutes. One can use tap water, saline or phospha-soda. It requires no additional assistance. Folks can sit on the commode and be clean and worry free after 30 minutes and until the next time.

In conclusion, the effects of TM on pelvic function are highly variable. It is possible to diagnose voiding dysfunction and plan appropriate therapy. Our goals in therapy are to preserve the safety of the kidneys and this is ordinarily not hard to do. Continence, independence and maximizing quality of life are the other goals of therapy. Our progress has been incremental, but it is real. We need to be our own best advocates, and that requires that you become educated about your condition and about possible therapy options. One of the goals of the TM Center at Johns Hopkins is to be that place of information transfer. The road is long. I believe that in our lifetime, many of these conditions will be better understood, and we will have medicines and therapies to better restore function.

Interventional Approaches to Neuropathic Pain

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Adapted from a presentation given at the 2006 Rare Neuroimmunologic Symposium

I am a pain medicine specialist, anesthesiologist, and the director of the pain treatment center and pain fellowship program at Johns Hopkins. My article focuses on interventions that we typically use to treat neuropathic pain. As an introduction, I will distinguish acute versus chronic pain and nociceptive versus neuropathic pain and will describe the mechanisms behind the various types of pain. I will then highlight the interventional strategies or injections that we use to help relieve neuropathic pain.

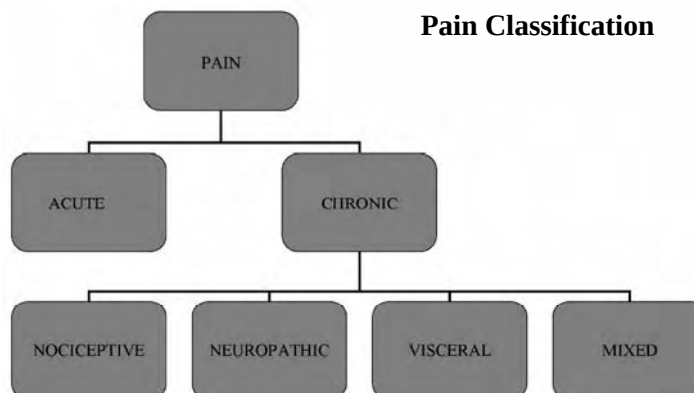
Pain warns of threatened or ongoing tissue damage. The International Association for the Study of Pain defines pain as, "an unpleasant sensory and emotional experience associated with potential or actual tissue damage." In general, tissue that is injured from surgery, trauma, or even disease processes release inflammatory products such as prostaglandins or histamine. These products trigger pain signals that travel from the body to the spinal cord and then to the

brain where the messages are interpreted as painful.

Pain can be classified as both acute and chronic. Subsumed under chronic pain are nociceptive pain, neuropathic pain, visceral pain, and then a mixture of all three types of pain. For instance, some patients who suffer from low back pain experience both nociceptive and neuropathic pain; however, it can be difficult to distinguish the mechanisms that are responsible for particular types of pain.

Acute pain (next page) can be viewed as an unpleasant reaction or sensation due to some type of tissue damage that may be related to surgery or even an injury, such as spraining an ankle. Acute pain is physiologically normal and serves a protective role. The degree of pain a patient experiences typically corresponds to the extent of tissue damage. Acute pain treatment outcomes are good; that is, most acute pain can be successfully treated with medications and patients usually do not suffer from persistent pain. For example, we often use oral or intravenous opioids and/or epidural anesthesia intra-operatively and post-operatively with good pain control and minimal side effects.

Chronic pain poses more of a challenge in understanding the disease and treating the symptoms. The International Association for the Study of Pain classifies or describes chronic pain as, "pain that persists after the

Pain Classification

The Neural Pain Pathway

Pain-sensing neurons
"nociceptors"
are stimulated by



Force



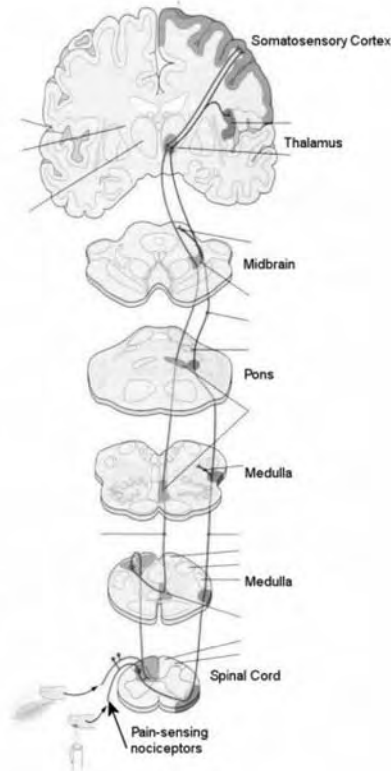
Heat



Cold



Chemicals



expected healing time of injury.” Patients who suffer from chronic pain often describe symptoms that are out of proportion to anything that physicians would detect on physical exam. We believe that chronic pain serves no clinical benefit. Instead, it often leads to psychosocial struggles such as depression, anxiety, fear, and other comorbidities. Pain experts generally agree that chronic pain begins anywhere from 3-6 months of persistent pain and results from a variety of mechanistic changes to the nervous system (1, 2). Treating chronic pain requires multimodal therapy: injections, medications, physical therapy, and psychological interventions in order to achieve the best outcome. In our pain clinic, we treat non-malignant (non-cancer), chronic pain patients, as well as patients suffering from cancer-related pain. We focus on providing an array of therapies that will maximize pain relief, increase mobility, allow patients to re-engage socially, and enjoy an enhanced quality of life.

Nociceptive pain may result from mechanical, thermal or chemical excita-

tion of nociceptors (pain receptors). Nociceptive pain is often considered acute, though several chronic pain states, such as arthritis and sickle cell crises may be nociceptive in nature. Nociceptors are located throughout the body. They are widely distributed in the skin, subcutaneous tissue, bone, muscle, connective tissue, viscera (organs), and blood vessels. When patients experience nociceptive pain, they often describe it as aching, throbbing or sometimes sharp. This pain is typically responsive to opioids, such as morphine, fentanyl, or hydromorphone (dilaudid).

Neuropathic pain differs from nociceptive pain. The International Association for the Study of Pain defines neuropathic pain as, “pain that is initiated or caused by a primary lesion or dysfunction in the peripheral or central nervous system or both.” Neuropathic pain continues without ongoing tissue damage and despite tissue healing. There are several clinical characteristics that are associated with neuropathic pain.

For example, patients describe this type of pain as burning or electric-like in sensation (3). There tends to be a delay in onset after injury. Patients also describe neuropathic pain as shooting, stabbing, or shock-like, or even a continuous aching sensation. Upon examination, physicians frequently detect a phenomenon called allodynia, or pain from a stimulus (like a cotton swab) that does not normally evoke pain. Furthermore, clinicians may also uncover another feature of neuropathic pain called hyperalgesia, or an exaggerated, quite painful response to a stimulus (like a needle prick) that normally provokes pain (4).

Origins of Neuropathic Pain

Peripheral nerve trauma

- Entrapment neuropathies
- Nerve transection
- Amputation or stump pain
- Neuroma

Other Mononeuropathies

- Diabetic neuropathy
- Malignant nerve/plexus invasion
- Connective tissue disease

Central sensory deficits

- Infectious
- Chemical
- Ischemic insults
- Disease (Multiple Sclerosis, Cerebral Palsy)

Polyneuropathies

- Diabetic
- Alcoholic
- Nutritional
- Infectious (HIV)
- Chemical (Chemotherapy)
- Idiopathic/Genetic

Root/dorsal root ganglion

- Post herpetic neuralgia
- Trigeminal neuralgia
- Prolapsed disc/compression
- Arachnoiditis
- Tumor compression
- Root avulsion
- Surgical (Rhizotomy)

There are multiple origins of neuropathic pain that range from peripheral nerve trauma, such as amputation or

Prevalence of Pain in the US (5)

<u>Condition:</u>	<u>Number of Cases</u>
Diabetic Neuropathy	600,000
Post-herpetic neuralgia	500,000
Cancer pain	200,000
Spinal cord injury	120,000
CRPS	100,000
Multiple sclerosis	50,000
Phantom pain	50,000
Post-stroke	30,000
HIV-related	15,000
Trigeminal neuralgia	15,000
Low back pain	2,100,000

nerve injury, to diabetic neuropathies and infectious or chemical causes. Chemotherapy-induced neuropathies often resolve, though sometimes they require specific pain-relieving medications, if the neuropathy persists chronically. Spinal cord injury or nerve root injury may result from trauma, disease processes (cancer or multiple sclerosis, for instance), or sometimes following surgery. Post-herpetic neuralgia (shingles pain that persists) represents another classic neuropathic pain state, as well as trigeminal neuralgia. Arachnoiditis can develop after multiple spine surgeries, such as spinal fusion and may cause chronic low back and leg pain.

The prevalence of low back pain is very high in the United States and it is significantly higher than cases of pain associated with other conditions. The cause of low back pain is tremendously difficult to establish and it is equally difficult to determine whether low back pain is caused by nociceptive or neuropathic pain mechanisms. The prevalence of neuropathic pain is highest for diabetic neuropathy, then postherpetic neuralgia (persistent shingles pain), cancer-related pain, spinal cord injury, and complex regional pain syndrome (also known as reflex sympathetic dystrophy). The incidence or number of newly diagnosed cases of

neuropathic pain is 50% or greater in patients with AIDS, cancer, and diabetes, whereas it is only 28% in patients with multiple sclerosis.

Clinical Manifestations of Neuropathic Pain

Allodynia: pain due to stimulus that does not normally produce pain

Analgesia: absence of pain in response to stimulation that would normally be painful

Hyperalgesia: an increase in response to stimulus that is normally painful

Hyperesthesia: increase sensitivity to stimulation

Hyperpathia: abnormally painful reaction to a stimulus as well as an increase threshold

Hypoalgesia: diminished pain in response to a normally painful stimulus

Hypoesthesia: decreased sensitivity to stimulation

Paresthesia: an abnormal sensation, whether spontaneous or evoked

Dysesthesia: an unpleasant, abnormal sensation whether spontaneous or evoked

This chart identifies and defines the various clinical manifestations of

neuropathic pain. Neuropathic pain is difficult, but rarely impossible to treat. Researchers struggle to link symptoms that patients exhibit to specific mechanisms in the nervous system that may explain the symptoms. A single mechanism may be responsible for multiple symptoms in one patient, or the same symptom (burning, for instance) seen in different patients may be due to different mechanisms. In fact, multiple mechanisms may exist simultaneously in a single patient and may change over time (6). As we unravel these mechanistic intricacies, targeted medical or perhaps procedural therapies may be developed to treat elements of the dysfunctional nervous system.

Neuropathic pain is associated with distinct cellular and molecular mechanisms that incorporate ion channels, cytokines, and neuropeptides. Pain results from abnormal communication between the peripheral and central nervous system. Specifically, pain may derive from aberrant relationships between large and small fibers, and sympathetic fibers in the nervous system.

Mechanisms of Neuropathic Pain

Brain

- Altered “gating”
- Molecular Changes
- Gene expression changes
- Receptive field changes

Spinal cord

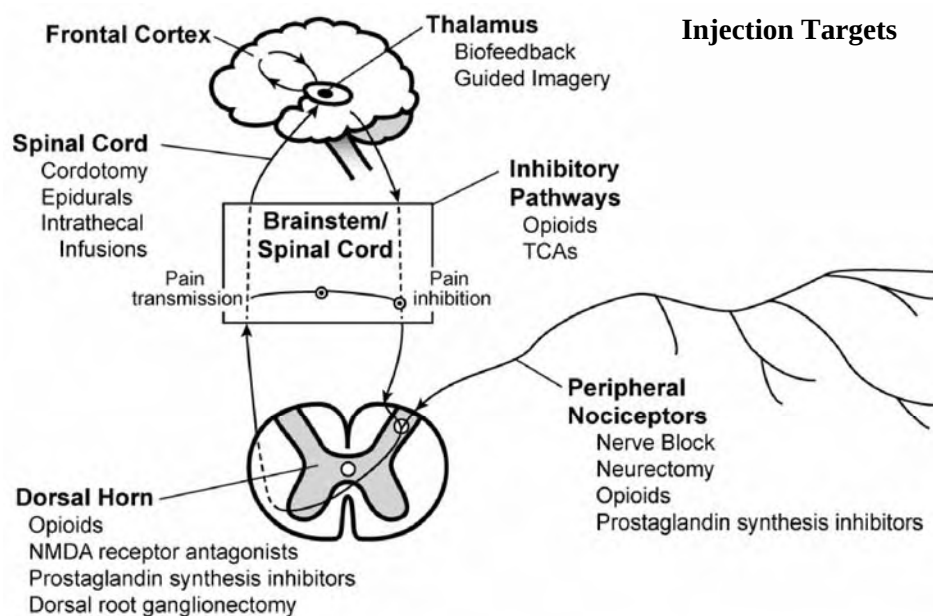
- Altered “gating”
- Dorsal horn denervation
- Hypersensitivity
- Gene expression changes
- Receptive field changes

Peripheral Nerve Fibers

- Ectopic discharges
- Mechano-sensitivity
- Ephaptic cross talk

Sympathetic Fibers

- Cross talk
- sprouting



sulting from previous spine surgery, or neuropathic pain stemming from persistent shingles pain or complex regional pain syndrome (RSD).

Neuropathic Pain Syndromes

- **Post-herpetic Neuralgia (PHN)**
- **Complex Regional Pain Syndrome (CRPS)**
- Peripheral (Small Fiber) Neuropathies: Diabetic
- Ischemic Limb Pain
- Angina Pectoris
- Cancer Related (Chemo/RTX)
- Trigeminal Neuralgia
- Phantom Limb Pain
- Post Stroke Pain
- HIV Neuropathy
- Spinal Cord Injury
- Neuroradiculopathy

There are multiple neuropathic pain syndromes. I will focus on two of these syndromes for which we have some evidence of efficacy for interventions: post-herpetic neuralgia and complex regional pain syndrome.

Postherpetic neuralgia (PHN) is persistent, chronic shingles pain and it is caused by the herpes zoster virus. Typically, patients experience a rash followed by vesicle formation, then scab development, and finally continued pain. Patients often describe pain

that is burning or electric-like. The pain courses along a certain dermatomal distribution and typically lasts greater than one month after the rash heals. Often the chest and face are affected. It is more common in women, and unfortunately, there is an increased risk of developing post-herpetic neuralgia as we age. The risk of having continued pain at 12 months is almost five times higher in patients who are 80 years of age compared to those less than 80 year of age. In fact, almost 50% of patients greater than 70 years of age describe pain lasting greater than one year after the onset of the PHN rash (7).

What is the evidence for the efficacy of injection therapy in the treatment of PHN? There are four blocks that we typically use: intrathecal steroid injections, epidural steroid injections, sympathetic blocks, and spinal cord stimulation.

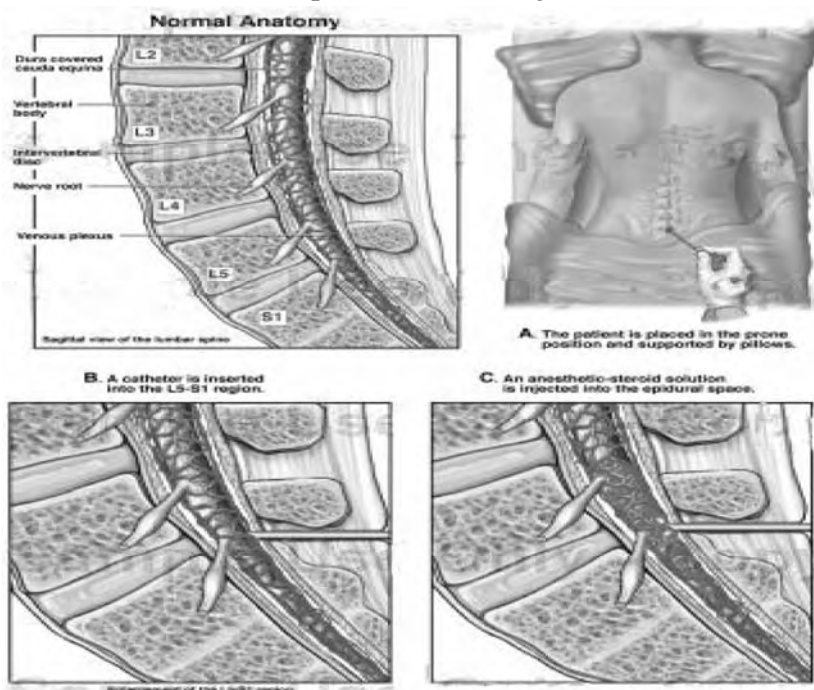
Intrathecal steroids were studied by Kotani et al in 2000. He studied 277 patients that had intractable post herpetic neuralgia for three years. The study was of good quality and consisted of a randomized, double blind, controlled trial. The groups in the trial included those who were in-

jected with lidocaine (3cc of 3% lidocaine) intrathecally, those who received lidocaine plus steroid (3cc of 3% lidocaine with methylprednisolone), and those that received no injection (control group). Ninety percent of the patients in the lidocaine plus steroid (injected into the cerebrospinal fluid) group reported relief. They reported good to excellent relief and a decrease in their use of anti-inflammatory (NSAID) drugs. These patients described the same degree of relief even two years later and with no side effects. Despite the favorable outcome of this treatment, many pain physicians do not use intrathecal therapy (intrathecal local anesthetic and steroid) to help treat this condition, because of reports of chemical meningitis, chronic arachnoiditis, and transverse myelitis that may result from repetitive injections of steroid (methylprednisolone) intrathecally.

Some studies that examine treatments of PHN suffer from methodological problems or low numbers of study patients. Nevertheless, I believe that they serve as a springboard for future studies of higher quality. Most of these studies at least illustrate some positive effects on patients who suffer from chronic pain conditions and therefore, help guide our therapy.

Kikuchi et al (1999) studied the efficacy of epidural steroids compared to intrathecal steroids in patients who had post herpetic neuralgia for greater than one year. The study involved just 25 patients. It was a randomized, controlled, single-blind study for four, weekly epidural or intrathecal injections. He evaluated continuous pain, lancinating (shooting) pain, and allodynia before treatment, at the end of treatment, a week later, and 24 weeks later. He found that there was significant pain relief in the patients who received the intrathecal steroids, similar to Kotani's study. There was minimal relief in those who received epidural steroids, however.

Lumbar Epidural Steroid Injection



The evidence more strongly demonstrates that patients with acute herpes zoster (shingles) typically do benefit from either intrathecal or epidural steroids that help reduce pain in that acute phase. The steroids may reduce neuronal inflammation that is associated with the acute phase of herpes zoster and may exert a membrane stabilizing effect on painful nerve transmission.

These are images showing an epidural steroid injection. The patient lies on his/her belly. The procedure can be done with fluoroscopy (x-ray) or just at the bedside. The image on the left is the spinal cord with exiting nerves. Under fluoroscopy, the patient lies face down and a small needle is inserted into the epidural space usually around lumbar level 4-5 or 5-1. Local anesthetic is used to numb the area of needle insertion. Contrast is injected to outline the epidural space, and then a small amount of local anesthetic along with steroid is injected into the epidural space. Complications are rare if performed by an experienced pain physician.

If patients suffer from post-herpetic

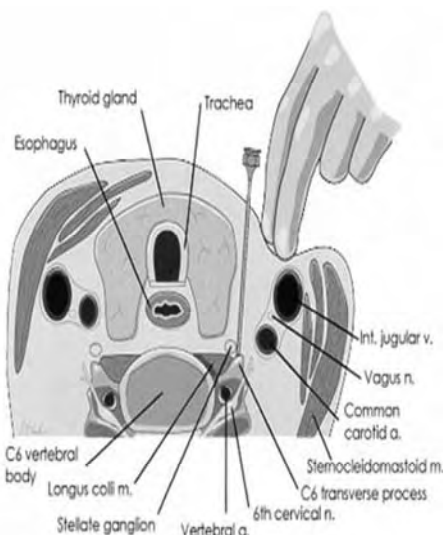
neuralgia at the thoracic (chest) level, we may offer intercostal blocks (nerve blocks under the ribs) or thoracic epidural steroid injections. Patients with postherpetic neuralgia in the face may benefit from a stellate ganglion block which blocks certain nerves that supply the face, scalp, ear, and neck.

Is there evidence that sympathetic blockade with local anesthetics is helpful in patients who have post-herpetic neuralgia or acute herpes

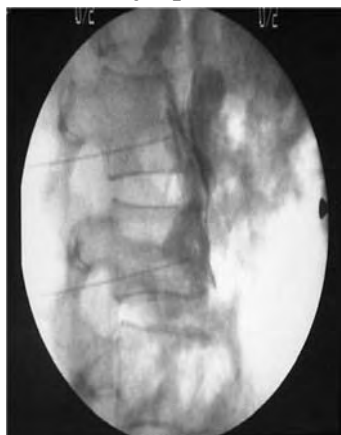
zoster (shingles)? Wu et al (2000) and Opstelten et al (2004) conducted reviews of the literature and found that sympathetic blocks are widely used for the prevention and treatment of post-herpetic neuralgia and for the treatment of acute herpes zoster. However, the studies relating to these treatments were of low quality (lack of randomized, controlled trials). Case reports suggest that sympathetic nerve blocks may provide considerable relief in acute herpes zoster, but may only offer short-lived relief in PHN. However, sympathetic blocks may be a worthwhile strategy to pursue, if pain is inadequately controlled by medications. Since the severity of pain during an acute herpes zoster attack is a risk factor for progression to PHN, sympathetic blockade may lower the incidence of PHN by reducing pain severity.

The graphic demonstrates how this injection is performed. The stellate ganglion is one of several structures that compose the sympathetic nervous system. This local anesthetic block interrupts sympathetic outflow to the face, head, upper arm, ear, and neck. By blocking sympathetic nervous system transmission, pain signals that travel with these nerves can also be blocked. Patients who suffer from neuropathic pain in the previously men-

Sympathetic Block: Stellate Ganglion Block



Sympathetic Block: Lumbar Sympathetic Block



tioned regions may benefit from this procedure. It can be performed at the bedside or under x-ray guidance. When performed at the bedside, the patient lies on his/her back and the physician palpates the cricoid cartilage, and then moves laterally and away from the trachea and large vessels of the neck. A small, thin needle is inserted to the transverse process of C6 followed by injection of local anesthetic. The procedure is similarly performed under x-ray guidance, though the needle may be positioned at C6 or C7 and contrast is injected to further verify that the needle is not located in a blood vessel, and to confirm proper spread of the solution.

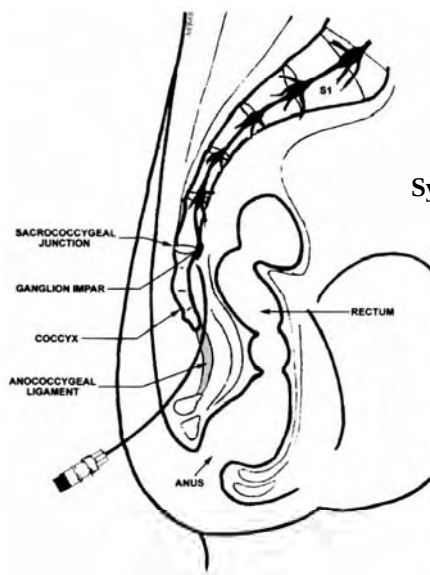
The x-ray image on the right demonstrates a needle positioned on the transverse processes of C6, contrast (dark material) spread moving down toward the upper chest, and then subsequent injection of local anesthetic solution to block the stellate ganglion.

The next graphic shows an x-ray image of a lumbar sympathetic block. This injection is performed for patients who have neuropathic pain in the lower extremity. It is typically performed under x-ray guidance at the level of the low back. The needles in the image on the left are placed at L2 and L3. Patients are positioned face down during the procedure after which we insert a thin and long needle (7 inches) to contact the anterior-lateral



aspect of the vertebral body. The image on the left shows injection of contrast to verify proper needle location before local anesthetics are then injected.

Skin temperature is measured while performing these sympathetic blocks. We expect an increase in temperature as a result of interrupting sympathetic outflow to the skin. In other words, blood vessels dilate and release heat when parts of the sympathetic nervous system are blocked with local anesthetics. In order to confirm that the block is performed properly, we measure skin temperature. The image on the right demonstrates that one foot is warmer than the other after performing a sympathetic block.

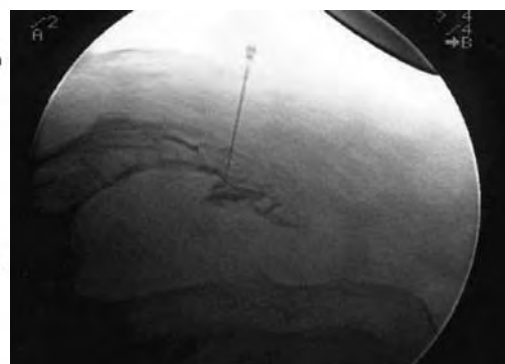


The ganglion impar represents the terminal end of the sympathetic chain (sympathetic nervous system). It is located near the sacral and coccygeal region of the spine as noted in this image. This structure can be blocked for patients suffering from neuropathic pain in the rectal, anal, perineal, and parts of the vaginal and urethral areas. A 3½ inch, 22 or 25 gauge needle is inserted through the anococcygeal ligament or the sacrococcygeal junction and gradually reaches the retrocecal space. The x-ray image on the right depicts a needle positioned through the sacrococcygeal junction and contrast spread in the retrocecal space. We want to avoid needle insertion into the colon or rectum. About 6-7 cc of local anesthetic and steroid are then injected to block the ganglion impar.

Neuromodulation (electrical stimulators and drug pumps) can be useful for alleviating neuropathic pain. A spinal cord stimulator delivers small doses of electricity to a certain area of the spinal cord in an attempt to block the transmission of painful sensations. Drug pumps serve a similar purpose, though specific medications are used instead of electricity.

The image on the next page shows a drug pump (intrathecal pump). Such a pump may contain morphine, hydromorphone, or bupivacaine. It is implanted underneath the skin and a tube is tunneled to the fluid-containing space surrounding the spinal cord,

Sympathetic Block: Ganglion Impar Block



Implantable Devices

Intrathecal Pump



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called the intrathecal space. Medications are therefore delivered from the pump directly to the spinal cord. The image on the right illustrates a spinal cord stimulator. This device contains a receiver and electrodes. The receiver is implanted underneath the skin and the electrodes are placed in the epidural space and directly on top of the spinal cord. Patients feel a buzzing or comfortable humming sensation when the stimulator is activated.

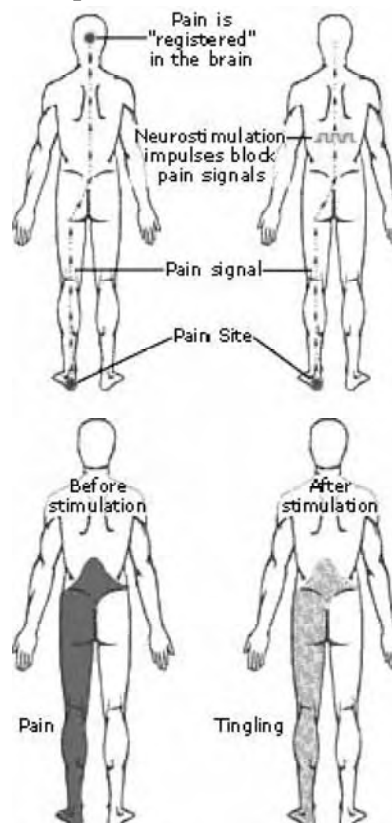
The studies on the use of spinal cord stimulation (SCS) for the treatment of post-herpetic neuralgia have provided mixed results. For instance, Meglio et al (1989) and Harke et al (2002) both reported that patients with PHN derived relief from stimulation; however, Kumar et al (1996) noted that just two of eight patients had pain relief at 7 years of continuous treatment with spinal cord stimulation. In sum, studies do demonstrate satisfactory relief over a 2-4 year period, though the level of evidence is less strong. However, SCS

may offer an alternative approach to pain control in patients who have unrelenting and intolerable pain from PHN.

Reflex Sympathetic Dystrophy (RSD) is a type of neuropathic pain. The condition has been re-named Complex Regional Pain Syndrome (CRPS). It predominates in women (60-81%) and often appears in early adulthood (36-42 years). The syndrome is typically caused by some type of injury such as a fracture, strain or a sprain. Some patients present with this syndrome following surgery or even spontaneously. The pain is reported as intense, with aching, burning, or shooting qualities. CRPS often occurs in the extremities: legs or arms. Clinical manifestations include allodynia and hyperalgesia, swelling, color and temperature changes, sweating changes, decreased range of motion, weakness, tremor, and nail and hair changes (8).

There are three interventional strate-

Spinal Cord Stimulation



gies used to treat CRPS: sympathetic blocks, spinal cord stimulation, and drug (intrathecal) pumps. Sympathetic blocks are frequently used to help treat this disease process. The quality of literature on lumbar sympathetic and stellate ganglion blocks in CRPS is limited, however. Yet, sympathetic blocks such as stellate ganglion blocks for arm pain and lumbar sympathetic blocks for leg pain can offer meaningful relief and can facilitate compliance with physical and occupational therapy. Many patients with CRPS may otherwise never move their leg or arm due to severe pain. Therefore, the reduction in pain associated with sympathetic blocks often permits individuals to participate in physical therapy and reduce their level of disability.

Spinal cord stimulation may also be considered for the treatment of CRPS. I have had a reasonable degree of success in using spinal cord stimulation for treating pain in patients who suffer from uncontrolled CRPS. It is typically considered in patients who are failing all other treatments such as nerve blocks, physiotherapy, or medications.

Overall, the literature provides moderate evidence that spinal cord stimulation effectively reduces pain in CRPS patients and promotes some benefit in function. For instance, Kemler et al (2000) in a high quality study examined the use of spinal cord stimulation in patients who had CRPS (9). Some patients received spinal cord stimulation plus physical therapy and the other group just engaged in physical therapy. He found that at the 6 month and one year follow up, pain was significantly reduced in the spinal cord stimulator group; however, he later found that patients in the spinal cord stimulator group reported less pain relief at both the 3 year and 5 year follow up.

In a more recent study, Harke et al (2005) found significant improvement

in quality of life and functional status at a 3 year follow up among CRPS patients who were using spinal cord stimulation as a treatment modality (10). Specifically, patients reported decreased pain and disability, improved functional status, and a reduction in medication use.

The effects of spinal cord stimulation may change over time. That is, some studies have reported that the beneficial effects of stimulation lessen in time for patients with CRPS, and PHN (11, 12). In general, I would say that many patients with neuropathic pain who use spinal cord stimulation as a treatment report that the therapy has improved their quality of life and lessened their disability for the period of time the device was used.

Intrathecal pain pumps with an opioid (morphine, for instance) may be helpful in controlling intractable pain associated with CRPS, though the literature fails to support this treatment with high quality studies. However, van Hilten et al (2000) showed that patients with contractions (arm or leg in fixed or rigid position) associated with CRPS demonstrated complete or partial relief of these symptoms after an agent called baclofen was infused through a catheter and an implanted pump (13). Some patients reported reduced pain and fewer sensory disturbances as well.

In general, pain specialists consider implantable pain pumps only in select patients and in those individuals who fail all other therapies.

Neuropathic and chronic pain can be best treated with multimodal therapy. Pharmacological treatments are helpful, and certain interventional/procedural approaches can offer significant relief. Complementary medicine, such as acupuncture and pain psychology can also be helpful. Depression and anxiety often co-exist with chronic pain and neuropathic

pain. Behavioral therapies can aid in reducing the impact of pain on a person's life and should be considered with any treatment strategy.

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Fatigue and Transverse Myelitis

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Adapted from a presentation given at the 2004 Rare Neuroimmunologic Disorders Symposium

This article is about fatigue in transverse myelitis. I have never really considered transverse myelitis a disease. I have considered TM a symptom of some other condition; in much the same way as seizures end up being called epilepsy, if you are uncertain of the cause. There are numerous causes of transverse myelitis. These include:

- Parainfectious - viral and bacterial
- Postvaccinal
- Autoimmune: Lupus, Sjogren's, Sarcoid, Multiple Sclerosis
- Paraneoplastic syndrome
- Vascular

Fatigue can be associated with any and all of the diseases associated with Transverse Myelitis. Not every case of transverse myelitis, however, has fatigue associated with it. I don't know which causes are associated with fatigue and which are not; I am not sure that anyone has that answer. Multiple Sclerosis is the prototype for fatigue in this disease group, so I am going to focus on the relationship between MS and fatigue. I recognize that this case may not fit each and every person with their individual form of transverse myelopathy or myelitis.

Fatigue is the single most common symptom that we see in multiple sclerosis. It is the most disabling symptom that we see in multiple sclerosis. If you have a transverse myelopathy