Peripheral Nerve Stimulation for Neuropathic Pain

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Chronic neuropathic pain affects between 8–15% of the population in the United States.[1,2] Patients suffering from chronic pain are likely to experience a diminished quality of life due to a loss of function, sleep disturbances, anxiety and depression.

A 2016 study by National Center for Health Statistics estimated that the financial impact was approximately $560 billion dollars from lost productivity, direct medical costs and disability programs.[1] Efforts to combat this health problem with narcotics have unintentionally contributed to the opioid crisis.[3] Therefore, the financial and societal impact of chronic pain underscore the importance in continued development of non-pharmacological interventions. Peripheral nerve stimulation (PNS) is a type of neuromodulation emerging as an effective non-narcotic treatment option for chronic neuropathic pain.

NEUROMODULATION

Neuromodulation is broadly defined as "the alteration of nerve activity through targeted delivery of a stimulus, such as electrical stimulation or chemical agents, to specific neurological sites in the body."[4] Neuromodulation that alters nerve activity with electrical stimulation is interchangeably referred to as neurostimulation.

LEARNING OBJECTIVES

- Define neuromodulation and neurostimulation
- Review how pain is transmitted
- Understand the Gate Control Theory
- Explain the difference between neuropathic and nociceptive pain
- List the components of a PNS system

Many conditions can be treated with neuromodulation but the most widely used application is the treatment of chronic pain with spinal cord stimulation.[4] Several clinical studies have supported the effectiveness of SCS in treating chronic pain due to failed back surgery syndrome (FBSS), complex regional pain syndrome (CRPS), diabetic peripheral neuropathy, postherpetic neuralgia and peripheral vascular disease.[5] The spinal cord stimulator was first used in 1967 but didn't gain FDA approval until 1989.[5] Before the advent of commercially available peripheral nerve stimulators, the spinal cord stimulator was the only neuromodulation treatment option for patients suffering from peripheral neuropathies and CRPS.

Peripheral nerve stimulation (PNS) is a type of neuromodulation emerging as an effective non-narcotic treatment option for chronic neuropathic pain.

Advancements in the development of peripheral nerve stimulation over the last decade has precluded the use of spinal cord stimulators for peripheral nerve pain. The FDA approval of the dorsal root ganglion stimulator (DRGS) in 2016[6] and the Peripheral Nerve Stimulator in 2015[7] changed the neuromodulation landscape with the offering of more targeted and specific neurostimulators. There are clear advantages in selecting a peripheral nerve stimulator over a spinal cord stimulator when neuromodulating a peripheral nerve. A spinal cord stimulator lead electrode measures two to three times longer than a PNS lead; and while SCS leads work well for implantation in the spinal column, they have drawbacks when employed for peripheral nerve stimulation in the extremities. Spinal cord stimulator leads have a higher tendency for lead migration and impedance when used in the periphery compared to PNS leads which are smaller and anatomically well-suited for peripheral nerves.[8]

HISTORY OF PNS

Although PNS has only recently been introduced as an accepted treatment for chronic pain in the United States, the use of electricity to treat peripheral nerve pain can be traced to the Roman Empire. In 15 AD Scribonius, physician to the Roman Emperor Tiberius, observed that a patient who accidentally stepped on an electric torpedo fish had less gout pain afterwards.[9] This led Scribonius to recommend the use of torpedo fish for chronic pain.[9] Despite this early yet anecdotal discovery, advances in PNS didn't significantly progress until the 1960's. In 1965, doctors Wall and Sweet documented the first PNS surgery. They implanted an electrode on the median and ulnar nerves of a 26-year-old woman with a history of pain and neuropathy in the fingers and hand.[1] When the nerves were stimulated, pain diminished and was replaced with a tingling feeling known as paresthesia.[1] Development of PNS continued over the next fifty years from an open procedure that was prone to complications to one that is minimally invasive and relatively safe.[10]

ANATOMY AND PHYSIOLOGY OF PAIN TRANSMISSION

A brief review of the relevant anatomy involved in pain transmission aids in understanding how neuromodulation works. Nociceptors also known as pain receptors respond to mechanical, thermal or chemical stimuli. In the skin there are A-beta, A-delta and C nociceptors whose axons terminate in the dorsal horn of the spinal column.[11] The A-beta nerve fibers respond to touch, are larger than A-delta and C fibers and have a relatively fast conduction speed.[12]

Pain transmission and modulation is accomplished by ascending and descending pathways in the spinal cord. When pain signals reach the dorsal horn from the periphery, they are transmitted by interneurons to the spinothalamic tract, the ascending pathway in the spinal cord. The signals travel up the spinothalamic tract, through the medulla and to the thalamus. From the thalamus the pain signals are relayed to the somatosensory cortex. Sensory information reaches the midbrain where the descending pathway originates. The descending pathway aids in modulating pain transmission through the release of neurotransmitters in the dorsal horn.[13] An individual's perception of pain is the result of the interplay between the pathways.

GATE CONTROL THEORY

The field of neuromodulation gained traction with the publication of Melzack and Wall's Gate Control Theory in 1965. This theory proposed that the transmission of pain signals in the dorsal horn depends on the type of peripheral nerve fiber stimulated.[14] Small A-delta and C peripheral nerve fibers inhibit cells in the area of the dorsal horn that inhibit transmission of sensory input to the somatosensory cor-

tex. This has the net effect of allowing the transmission of sensory input to the brain and thereby opening the gate for pain signals.[14] Large diameter A-beta fibers do the opposite. They excite the inhibitory cells in the dorsal horn which closes the gate to pain signals and prevents the pain signals from traveling to the brain. The balance between the input of large and small nerve fibers determines whether the gate opens or closes.[14] In theory, PNS works by activating the A-beta nerve fibers that cause the gate to close.[15] When the Gate control theory was introduced, it did not completely explain the complexities of pain transmission. It did, however, spur further research in the field[16] and elucidated how an electrical signal could decrease the feeling of pain.

CLINICAL EFFICACY OF PNS

Several research studies have proven the effectiveness of PNS in reducing neuropathic pain. One of the largest studies to date included 94 patients with neuropathies of the upper extremities, lower extremities and trunk. The results of this random double-blind study showed that the treatment group had a mean decrease in pain of 27% whereas the control group had a mean decrease of only 2.3% within three months of implantation. In addition, the treatment group reported significantly better scores in secondary outcomes such as quality of life. Participants reported no major adverse events or complications.[17]

NEUROPATHIC PAIN

Pain can be classified as either nociceptive or neuropathic. Nociceptive pain is caused when nociceptors are stimulated by noxious stimuli or tissue damage. Musculoskeletal pain like tendinitis is considered nociceptive.[18] Neuropathic pain, however, is the pain that is caused by damage or lesions of the peripheral or central nervous system and can be present in the absence of a painful stimulus.[18] Neuromodulation whether its SCS, DRGS or PNS is used only to treat neuropathic pain.

Acute injuries typically heal without long-term changes in the somatosensory system. It is the repetitive or continuous nociceptive stimulation that most often leads to neuropathic pain. Neuropathic pain stems from nerve damage due to ischemia, metabolic disorders, inflammation, autoimmune disorders, toxicity, radiation, genetic disorders and mechanical injuries such as entrapment or transection.[13] Surgical procedures contribute to the incidence of neuropathic pain and affect a significant number of patients.[19] *(See sidebar)*

Surgically-induced Neuropathic Pain (SNPP)

There are risks associated with any surgical procedure. The diagnosis or resolution of a medical condition undoubtedly outweighs these risks for most patients. Surgically Induced Neuropathic Pain is nerve damage sustained during the perioperative period that develops into chronic neuropathic pain. Inadvertent transection of a nerve during dissection, pressure injuries from positioning and stretching or bruising from retraction all contribute to the development of SNPP.[19] The patient's immune response to inflammation, an inherent consequence of a surgical wound, also plays a role in the development of SNPP.[24] Infrapatellar nerve damage from total knee arthroplasty, peroneal nerve damage due to poor positioning or sciatic nerve damage from amputation are a few examples of common surgical procedures that can lead to SNPP. The highest incidence of SNPP occurs with thoracotomy, mastectomy, amputation and inguinal herniorrhaphy. One study proposed that there is a 50% risk of developing a chronic neuropathy when undergoing an inguinal herniorrhaphy.[19]

The surgical team can take steps to minimize the patient's risk of nerve damage. The circulator should pad bony prominences and anatomical areas subjected to undue pressure and the scrub person should use the least amount of force necessary when retracting tissue. A surgeon's careful dissection and use of nerve stimulators when warranted are other preventative measures. There is some evidence that an anesthesia provider's selection of analgesics may also help decrease the incidence of SNPP.[19]

Table 1

Cause	Condition	Targeted nerve(s) for PNS
Mechanical	Phantom limb pain	Sciatic
Ischemia	Post stroke shoulder	Axillary
Mechanical	Post herniorrhaphy pain	Ilioinguinal
Radiation	Brachial plexopathy	Brachial plexus
Mechanical	Meralgia Paresthetica	Lateral femoral cutaneous
Metabolic	Diabetic neuropathy	Tibial
Autoimmune, Inflammation	CRPS	Nerves of the Lower and Upper extremities

The transition from an acute injury to chronic pain involves complex pathophysiological changes in the nervous system.[20] Alterations in ion channels and receptors in addition to the release of neuronal chemokines, neurotransmitters and neuromodulators all play a role in creating dysfunctional pain transmission.[20] Whatever the cause, PNS is often a feasible treatment. (See Table 1.)

Patients with neuropathic pain show unique symptoms not associated with nociceptive pain. Classic symptoms of neuropathic pain include shocking, shooting pains partly due to ectopic firing and hyperexcitability of the nerve.[21] Patients may also experience *allodynia*, a painful response to something that normally does not cause pain or hyperalgesia, an exaggerated response to something painful.[18] Some patients exhibit Tinel's sign which is a feeling of pins and needles when tapping on the affected area.[22] The diagnostic examination includes an evaluation of strength, tone and sensations such as cold, vibration and light touch.[23]

Once a diagnosis is determined the patient is treated first with non-narcotic medications for several weeks. If the patient doesn't satisfactorily respond, then they are referred to a pain management specialist who may treat the pain with nerve blocks, epidural injections, radio-frequency ablation or adhesiolysis.[23] Neuromodulation is not a front-line treatment for neuropathic pain and is tried only after other therapies have failed. Before moving forward to PNS, the patient must meet certain criteria: they must be able to tolerate the neurostimulation and be capable of controlling the device; their pain must be identifiable along a nerve distribution; they must be ruled out for a treatable cause like entrapment; and they have a positive diagnostic nerve block.[15]

BASICS OF PNS

There are slight variations of the PNS systems on the market each with its own merits. Despite differences in design, the goal is electrical stimulation of the nerve to disrupt the transmission of pain signals. Common features of any PNS device include:

- Patient Programmer – allows the patient to start/stop a therapy session
- External transmitter, worn on the skin – transmits signal to the internal pulse generator or antenna
- Internal pulse generator or antenna - sends the signal to the lead and electrodes
- Implanted lead with electrode contacts – stimulates the nerve

All peripheral nerve stimulators have similar therapy parameters including pulse width, frequency and amplitude. The parameter settings are highly individualized and are programmed with the patient's collaboration and feedback after the lead is implanted. During the programming phase, the clinician adjusts the pulse width, frequency and amplitude of the device until the patient reports a feeling of paresthesia in the affected area. The patient may feel a slight buzzing or tingling sensation and a decrease in pain. After the device is programmed the patient initiates therapy sessions as needed.

The Stimrouter PNS system (Bioness, Valencia CA) is used in this discussion as a representative example of the implantation method. This system features a lead with three electrode contacts at one end that is implanted near the nerve. The receiver at the opposite end of the lead delivers the signal from the transmitter. (See Figure 1.)

CASE STUDY

A 42-year-old healthy male diagnosed with mononeuropathy in the right foot is scheduled for implantation of a peripheral nerve stimulator at an ambulatory surgery center. Two years prior he sustained an injury to the tibial nerve while undergoing an Achilles tendon repair and developed SNPP. His goal is decreased pain and improved functionality so he can resume his passion for hiking.

PROCEDURAL STEPS

The patient is positioned supine on the operating room table with the foot and ankle side slightly everted and given light sedation but remains conscious throughout the procedure. The medial portion of the leg is prepped from the ankle to approximately 10 cm below the knee with Chloraprep. After the three-minute drying time, the area is draped by squaring off with four towels and applying a fenestrated drape. After the time out is performed, the Mayo stand with the implantation system is moved into position. The kit contains the insertion tools and the implant. The Mayo setup requires only the kit, a scalpel with #11 blade and a Debakey forcep. An ultrasound is used to aid in visualization of the nerve and the ultrasound wand is draped. The surgeon uses a marker and ruler to plan the incision site, and direction of electrode and receiver. The area is anesthetized with lidocaine and a stab incision is created. A flexible probe is inserted and advanced toward the tibial nerve with ultrasound guidance. When the probe is believed to be near the nerve, it is connected to a peripheral nerve stimulator. The patient is questioned if they feel stimulation. In this case, he feels stimulation along the tract of the tibial nerve. He reports a tingling sensation on the arch of his foot and into the great toe. The patient feedback verifies the correct location. Using a Seldinger technique the introducer cannula and dilator are threaded over the stim probe. The dilator and probe are removed leaving the cannula. The 15-centimeter lead is inserted into the cannula. The position of the lead is verified with ultrasound and stimulation. If the patient is still feeling paresthesia, the lead is deployed. The receiver end of the lead lies outside of the incision upon deployment. The receiver end is tunneled approximately 2-11 centimeters from the incision site. The incision is closed with skin adhesive and dressed with a sterile gauze. The patient is transported to PACU where he meets with the clinician for programming and begins the first therapy session with the nerve stimulator.

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OPEN PROCEDURE

The implantation of a peripheral nerve stimulator is sometimes accomplished through an open incision as part of another procedure. In those cases, the lead is simply positioned next to the nerve. Because the nerve is visible, ultrasound guidance and patient input is not necessary.

FUTURE IMPLICATIONS FOR PNS

In Europe, the application of PNS has grown beyond neuropathic pain. Peripheral nerve stimulation of the tibial nerve with a permanent implantable lead is now being implemented as a treatment for fecal incontinence (FI) and overactive bladder syndrome (OAB).[25] In the United States, PNS for OAB and FI are still under investigational status. Currently, percutaneous nerve stimulation without implantation of a permanent lead is an acceptable treatment for OAB. Because patients must go to their physician's office weekly for treatments, the convenience of a permanent lead would be preferable for some patients.[26]

CONCLUSION

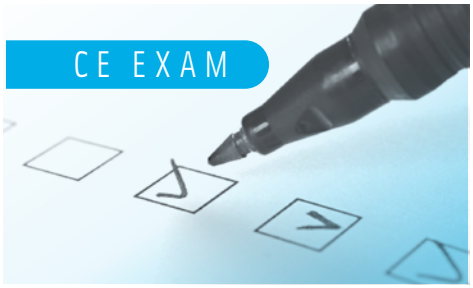
Neuropathic pain is a debilitating and widespread problem. The causes of peripheral nerve damage are varied and includes surgical procedures. Peripheral nerve stimulation is proving to be a safe and effective treatment.

ABOUT THE AUTHOR

Catherine Richmond, CST, has a Bachelors in Science in Psychology from Indiana University, a certificate in surgical technology from Bloomington Hospital (Bloomington, Indiana) and a MBA in Healthcare Management from Western Governors University. She has worked in healthcare for more than 20 years. She is currently a Field Clinical Manager for Bioness Inc.

REFERENCES

1. Dahlhamer J, Lucas J, Zelaya, C, et al. Prevalence of Chronic Pain and High-Impact Chronic Pain Among Adults — United States, 2016. MMWR Morb Mortal Wkly Rep 2018;67:1001–1006. DOI: http://dx.doi.org/10.15585/mmwr.mm6736a2
2. DiBonaventura MD, Sadosky A, Concialdi K, et al. The prevalence of probable neuropathic pain in the US: results from a multimodal general-population health survey. J Pain Res. 2017;10:2525-2538. Published 2017 Nov 1. doi:10.2147/JPR.S127014
3. Rosenblum A, Marsch LA, Joseph H, Portenoy RK. Opioids and the treatment of chronic pain: controversies, current status, and future directions. Exp Clin Psychopharmacol. 2008;16(5):405-416. doi:10.1037/a0013628
4. Neuromodulation Definitions. International Neuromodulation Society website. https://www.neuromodulation.com/neuromodulation-defined. Updated April 02, 2019. Accessed July 14, 2020
5. Gupta, Guarav. Spinal Cord Stimulation. Medscape website. https://emedicine.medscape.com/article/1980819-overview. Updated August 27, 2018. Accessed July 14, 2020.
6. Timothy R. Deer & Jason E. Pope (2016) Dorsal root ganglion stimulation approval by the Food and Drug Administration: advice on evolving the process, Expert Review of Neurotherapeutics, 16:10, 1123-1125, DOI: 10.1080/14737175.2016.1206817
7. Implantable Nerve Stimulation for the Treatment of Chronic Pain. Wellmark website. https://www.wellmark.com/Provider/MedpoliciesAndAuthorizations/MedicalPolicies/policies/Implantable_peripheral_nerve_stimulation.aspx. Updated October 2019. Accessed July 14, 2020.
8. Baldeschi G, Dario A, De Carolis G, et al. Peripheral Nerve Stimulation in the Treatment of Chronic Pain Syndromes From Nerve Injury: A Multicenter Observational Study. Neuromodulation 2017; 20: 369–374. DOI: 10.1111/ner.12539
9. Krames E, Peckham P, Rezai A. Neuromodulation A Historical Perspective In: Neuromodulation. First Edition. San Diego, CA: Elsevier; 2009: pages 29-30.
10. Deer T, Levy R, Verrills P. Perspective: Peripheral Nerve Stimulation and Peripheral Nerve Field Stimulation Birds of a Different Feather. Pain Medicine 2015; 16: 411–412. Accessed June 29, 2020
11. Purves D, Augustine GJ, Fitzpatrick D, et al., editors. Neuroscience. 2nd edition. Sunderland (MA): Sinauer Associates; 2001. Nociceptors. Available from: https://www.ncbi.nlm.nih.gov/books/NBK10965/
12. Ascending Pain Pathways. The Brain from top to Bottom website. https://thebrain.mcgill.ca/flash/d/d_03/d_03_cl/d_03_cl_dou/d_03_cl_dou.html. Accessed July 13, 2020.
13. Yam M, Loh Y, Tan C, et al. General Pathways of Pain Sensation and the Major Neurotransmitters Involved in Pain Regulation. Int J. Mol. Sci. 2018, 19, 2164. DOI:10.3390/ijms19082164
14. Mendell LM. Constructing and deconstructing the gate theory of pain. Pain. 2014;155(2):210-216. doi:10.1016/j.pain.2013.12.010
15. Raghavendra N, Banik R. Current Innovations in Peripheral Nerve Stimulation. Volume 2018 Article ID 9091216. https://doi.org/10.1155/2018/9091216
16. Moayedi M, Davis D. Theories of pain: from specificity to gate control. J Neurophysiol 109: 5–12, 2013. DOI:10.1152/jn.00457.2012
17. Deer T, Pope J, Benyamin R, et al. Prospective, Multi-Center, Randomized, Double-blinded, Partial Crossover Study to Assess the Safety and Efficacy of the Novel Neuromodulation System in the Treatment of Patients with Chronic Pain of Peripheral Nerve Origin. Neuromodulation 2016; 19: 91-100. DOI:10.1111/ner.12381
18. Colloca L, Ludman T, Bouhassira D, et al. Neuropathic Pain. Nat Rev Dis Primers: 3, 2017; 17002. DOI:10.1038/nrdp.2017.2
19. Boorsook D, Kussman B, George E, et al. Surgically-Induced Neuropathic Pain (SNPP): Understanding the Perioperative Process. Ann surg. 2013; 25(3): 403-412. DOI:10.1097/ SLA.0b013e3182701a7b.
20. Feizerfan A, Sheh G. Transition from acute to chronic pain. Continuing Education in Anaesthesia, Critical Care & Pain 2015; 15(2): DOI:10.1093/bjaceaccp/mku044
21. Baron, R. Mechanisms of Disease: neuropathic pain—a clinical perspective. Nat Rev Neurol 2006; 2, 95–106: https://doi.org/10.1038/ncpneuro0113
22. Seladi-Schulman J. Tinel's Sign.Healthline website. https://www.healthline.com/health/tinels-sign#test. Updated Septermber 18, 2018. Accessed July 14, 2020.
23. Bates D, Schultheis B, Hanes M. A Comprehensive Algorithm for Management of Neuropathic Pain. Pain Medicine, 20, 2019, S2–S12: DOI: 10.1093/pm/pnz075
24. Staff N, Engelstad J, Klein C, et al. Post-surgical inflammatory neuropathy. Brain, 2010; 133(10) 2866–2880, https://doi.org/10.1093/brain/awq252
25. Bioness StimRouter® Neuromodulation System Receives CE Mark Approval for the Treatment of Fecal Incontinence. https://www.prnewswire.com/news-releases/bioness-stimrouter-neuromodulation-system-receives-ce-mark-approval-for-the-treatment-of-fecal-incontinence-300981289.html Updated January 6, 2020. Accessed July 14, 2020
26. Lane G, Draayer Y, Barboglio-Romo P, et al. A prospective observational cohort study of posterior tibial nerve stimulation in patients with multiple sclerosis: design and methods. 20:58https://doi.org/10.1186/s12894-020-00629-y



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1. **The following are characteristics of A-beta nerve fibers except:**
   - **a.** Large diameter
   - **b.** Inhibits interneurons in the dorsal horn
   - **c.** Fast conduction speed
   - **d.** Primarily respond to touch

2. **Which condition is not usually treated by neuromodulation?**
   - **a.** Meralgia paresthetica
   - **b.** CRPS
   - **c.** FBSS
   - **d.** Rheumatoid arthritis

3. **Surgically Induced Neuropathic Pain is due to:**
   - **a.** Patient's immune response
   - **b.** Transection of a nerve
   - **c.** Poor positioning
   - **d.** All of the above

4. **_____ is a painful response to a non-painful stimulus.**
   - **a.** Allodynia
   - **b.** Hyperalgesia
   - **c.** Hypoalgesia
   - **d.** Paresthesia

5. **This component of a peripheral nerve stimulator sends a signal to the IPG or antenna.**
   - **a.** Patient programmer
   - **b.** External transmitter
   - **c.** Electrode
   - **d.** Implanted lead

6. **Which nerve is likely affected when a patient develops SNPP after an AKA?**
   - **a.** Greater occipital
   - **b.** Suprascapular
   - **c.** Lateral femoral cutaneous
   - **d.** Sciatic

7. **PNS can treat OAB by stimulating the _____ nerve.**
   - **a.** Lateral femoral cutaneous
   - **b.** Saphenous
   - **c.** Tibial
   - **d.** Cluneal

8. **Which is not a disadvantage of implanting a spinal cord stimulator lead on a peripheral nerve?**
   - **a.** Limited parameter settings are available
   - **b.** Impedence
   - **c.** Lead migration
   - **d.** Large size

9. **The settings that are adjusted during programming of the stimulator include:**
   - **a.** Amplitude
   - **b.** Frequency
   - **c.** Pulse width
   - **d.** All of the above

10. **Which statement is true about neuromodulation?**
    - **a.** Neuromodulation is primarily used to treat nociceptive pain.
    - **b.** Neurostimulation modulates nerve activity through chemical agents.
    - **c.** Neuromodulation is most often used to treat chronic pain.
    - **d.** Dorsal root ganglion stimulation preceded the use of spinal cord stimulation.

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