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Peripheral Field Nerve Stimulation in Refractory Subcutaneous Persistent Postsurgical Pain

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Background: The mechanistic underpinnings of nerve stimulation technology is an area of active debate in interventional pain literature. Whether the technology is transcutaneous, subcutaneous, or directly on the spinal cord/dorsal root ganglion, there are ample theories without substantive evidence. Although, these technologies have been proven to be invaluable for pain relief. Direct spinal cord stimulation is purported to be effective for peripheral pain through centrally mediated stimulation. However, in select cases, there is evidence for superior analgesia from a peripherally directed device, such as a subcutaneously placed peripheral field nerve stimulator (PFNS), when compared to spinal cord stimulators (SCS).

Case Report: An 81-year-old man was referred for left upper thoracic pain exacerbated by lipoma excision with diagnostic imaging unsupportive of musculoskeletal etiology. The patient was found to have soft tissue tenderness to palpation worsened by activity. He failed numerous conservative treatments and procedures. An epidural SCS was trialed, with appropriate paresthesia mapping, but was unsuccessful in providing significant relief. Ultimately, a PFNS was trialed and found to provide adequate relief. A PFNS was later implanted, resulting in successful pain relief.

Conclusion: The case demonstrates the importance of developing evidence-based guidelines for the application of PFNS. Additionally, it is important to delineate the shared and unique targets of nerve stimulator technologies so that patients may minimize risk through trial-and-error procedures.

Key words: Surgical procedures, operative, spinal cord stimulation, refractory pain, paroxysmal nerve pain, intractable pain, electric stimulation therapy, back pain without radiation,transcutaneous electric nerve stimulation

BACKGROUND

Persistent postsurgical pain (PPP) is defined as pain that arises or increases in intensity following a surgical procedure and outlasts normal healing time. PPP, or, chronic postsurgical pain (CPSP), is thought to be related to maladaptive neurologic plasticity. Surgical insult to sensory neurons induces a neurohumoral response with subsequent release of inflammatory mediators such as TNFα (1). Mediators present following injury may result in altered gene expression in neurons with a lowered

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threshold for firing and spontaneous firing of sodium channels in damaged neuronal tissue. Rapid and disorganized firing of the involved neurons may alter gene transcription in the dorsal root ganglion and result in maladaptive integration of peripheral nerve signaling. Such maladaptive transcription can lead to an imbalance of inhibitory and excitatory responses to stimuli, which may result in a pathological perception of nonnoxious stimuli as painful (1).

Examples of PPP that have been reported include postthoracotomy, postmastectomy, posttrauma, postherniorrhaphy, and postamputation pain (2,3). PPP may result from the excision of cutaneous soft tumors, with a 9% incidence of PPP following melanoma resection. A risk factor for PPP that has been identified and is relevant to our patient is severe acute pain following the initial surgical procedure (1,4). The treatment of acute postsurgical pain by peripheral nerve stimulators (PNS) is being investigated in a clinical trial by Ilfield et al (5) (NCT02898103), results pending.

Stimulation of Peripheral Nerves for Neuropathic Pain

The gate theory, presented in 1965 by Melzack and Wall (5), is commonly identified as the explanation for peripheral stimulator-mediated pain relief. Gate theory proposes that larger, myelinated nerve fibers such as Aβ are preferentially stimulated. Preferential stimulation limits pain afferents that are smaller (Aδ) and unmyelinated (C fibers) from transmitting a significant proportion of signals to the central nervous system. However, this is an active area of study; other theories have arisen involving the insular cortex, blood flow, triggered endorphin release, and alterations in depolarization and neurotransmitter release (6).

Nerve stimulators are proving to be well-suited to treat chronic neuropathic pain, with many devices from which to choose. As our patient failed transcutaneous electrical nerve stimulation (TENS) and spinal cord stimulation (SCS), but ultimately had relief from a subcutaneous PNS, we believe a review of peripheral nerve stimulation is appropriate.

PNS is a term used to describe an implantable device that electrically stimulates a named nerve. There are 2 common approaches for implantation. The minimally invasive technique involves subcutaneous implantation determined by maximal relief. Alternatively, the nerve may be surgically exposed with the stimulator adjacently placed. This differs from peripheral subcutaneous field stimulation (PSFS/PFNS) in which pain is controlled without a named nerve target. Percutaneous electrical nerve stimulation (PENS) is a method by which needles are placed paravertebrally, generally in the vicinity of peripheral nerves. These needles emit rapid alternating currents of low frequency (2 Hz) for small motor afferents, and high frequency stimulation (100 Hz) for small motor afferents and larger diameter beta afferents. TENS differs from the other approaches because the skin remains intact and in contact with the device. The settings limit the user to only low frequency or high frequency without rapid alternating frequencies. Nonneural elements of pain modulation are thought to have a greater role in the modalities that are more invasive than TENS (7). Our patient did not experience relief from TENS. This is an observation that has been supported in other cases of patients whose pain was ultimately relieved with SCS, an additional modality that our patient failed (8).

PNS differs from spinal cord stimulator placement, including procedural risks and presumed mode of action. The effects may be partially mediated by peripheral stimulation as suggested by the gate theory (9). PNS may share some of the mechanisms of SCS.

Investigators have found that SCS is not universally more effective as a treatment for chronic pain than PNS. Indeed, there is evidence of improved outcomes when combining SCS with PNS (6). The present case report demonstrates that PFNS alone was superior to SCS for refractory chronic upper back pain.

Case

An 81-year-old man presented as a referral for chronic midline thoracic pain of 9 years' duration. The pain developed gradually with no identified inciting event. The patient's past medical history included a prior T10 compression fracture, chronic obstructive pulmonary disease, mild nonischemic cardiomyopathy, mitral valve prolapse, gastroesophageal reflux disease, benign prostatic hyperplasia, hypertension, and nephrolithiasis. His surgical history includes multiple lipoma excisions, T10 kyphoplasty, lymphadenectomy, melanoma excision of the left shoulder, cholecystectomy, appendectomy, and an ankle/foot surgery. He is an ex-smoker with current chewing tobacco use.

The patient arrived in the office after having exhausted several treatment modalities since first seeking medical attention for this issue in 2012. He described an intermittent, nonradiating, severe, and aching and burning pain rated 7 of 10 on the Numeric Rating Scale (NRS) that was ameliorated by opioid medications and

heat application but exacerbated by bending and cold exposure. Medical records from an outside hospital included additional symptoms of left-sided, subscapular, T8-T9 dermatomal pain and pain medial, lateral, and deep to the former site of an excised lipoma that was moderate and bandlike on activity. The pain predated the lipoma excision.

History of a compression fracture and T8-T9 dermatomal pain prompted computed tomography (CT) imaging of the thoracic back in 2012. T9-T10 central disc protrusion was noted with no thoracic vertebral abnormality or focal spinal or neural foraminal stenosis. Subsequently, a 2016 CT scan revealed an absence of compression fractures, destructive bone lesions, or high-grade stenosis. A kyphoplasty at T10 was again noted with small disc protrusion, the greatest of which was at T9-T10, causing minor thecal sac effacement without cord compression.

Considering the patient's known history of degenerative disease and kyphoplasty, the back pain was initially attributed to underlying axial skeleton pathology. This etiology was ruled out after the physical exam findings and pain description were inconsistent with axial pathology. Treatment for subcutaneous pain was initiated. After failing conservative therapy with heating pads, cupping, dry needling, taping, opioids, acetaminophen, duloxetine, tramadol, physical therapy, lipoma excision, TENS unit, and increasing gabapentin dosages, procedural interventions were started. Facet joint injections, epidural steroid injections at the T9-T10 level, trigger point injections, and botulin toxin injections were attempted. All had failed. We trialed a SCS with leads at the superior border of the T5 vertebrae. Again, the patient did not achieve adequate pain relief. Considering his pain was lateral to the midline at a prior excisional site, we hypothesized that the pain was a form of peripheral neuralgia, and we trialed the percutaneous placement of 2 peripheral field stimulator leads, one lateral and one medial to the lipoma excision site, at a one-cm depth. We used the tonic paresthesia setting with the Spectra Wavewriter IPG (Boston Scientific, Marlborough, MA). The trial provided more than 80% relief but lacked coverage medial to the scar. The success from the trial encouraged us to proceed with a permanent implant. Lead placement was adjusted laterally to improve coverage (Figs. 1,2). The implantation and recovery went well. The patient reported more than 80% sustained relief of the target areas.

The patient continues using 600 mg of gabapentin daily, 20 mg of paroxetine daily, and a half tablet of hydrocodone 5 mg-acetaminophen 325 mg twice daily as needed to augment the effects of the PFNS. The patient reports that his use of hydrocodone-acetaminophen is sporadic and that he has periods outlasting a week during which none is required. The patient's device was interrogated 5 months following implantation and the patient declared 0 of 10 on the NRS at that time. The settings included a voltage range of 3.7 to 4.0 with higher stimulation via the medial lead.

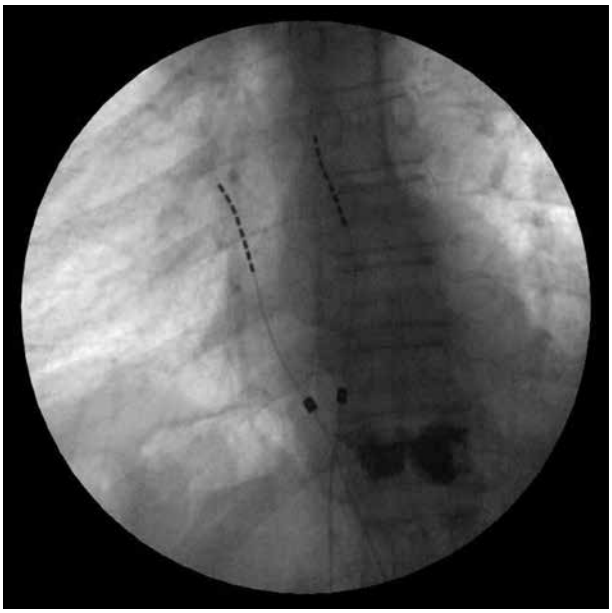

Fig. 1. Two 8-electrode leads were placed (radiopaque vertebrate is T10)

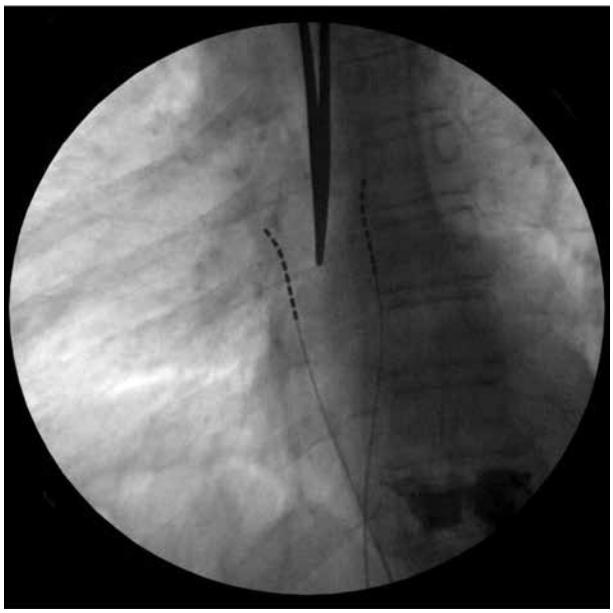

Fig. 2. Two 8-electrode leads were placed with clamp indicating point of maximal pain (radiopaque vertebrate is T10)

Discussion

Currently, there are no evidence-based clinical practice guidelines for the application of PNS. However, electrical peripheral stimulation treatments such as TENS and PSFS have been widely used as adjunct treatments for peripheral neuralgias. Furthermore, there is controversy regarding the best sites for SCS lead placement (9). We trialed our SCS leads at the superior border of T5 to alleviate pain over ribs 6-9; however, our SCS trial proved unsuccessful. Our lead placement may be considered by some sources to be too caudal to reap maximal effect.

This case describes treatment for a patient who failed multiple modalities of conservative therapy, spinal injections, and a SCS trial before having pain relief from PFNS. Although reported to have peripherally mediated effects in addition to centrally mediated, SCS proved inferior to PFNS.

Future research will be instrumental in helping to delineate the mechanistic similarities and differences between SCS and PFNS and their respective indications to address chronic pain.

Now that chronic postsurgical pain and relevant diagnoses are being included in the International Classification of Diseases, 11th Revision, increased research directed at effective treatments for these recognized diagnoses is anticipated (3).

Conclusion

This case describes a patient who failed multiple modalities of therapy, including SCS, before experiencing pain relief from a PFNS.

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REFERENCES

1. Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: Risk factors and prevention. ***Lancet*** 2006; 367:1618-1625.
2. Simanski CJP, Althaus A, Hoederath S, et al. Incidence of chronic postsurgical pain (CPSP) after general surgery. *Pain Med* 2014; 15:1222-1229.
3. Scholz J, Finnerup NB, Attal N, et al. The IASP classification of chronic pain for ICD-11: Chronic neuropathic pain. *Pain* 2019; 160:53-59.
4. Schug SA, Bruce J. Risk stratification for the development of chronic postsurgical pain. *Pain Rep* 2017; 2:e627.
5. Ilfeld BM. Percutaneous Peripheral Nerve Stimulation (Neuromodulation) for Postoperative Analgesia (pPNS). ClinicalTrials.gov identifier: NCT02898103. https://clinicaltrials.gov/ct2/show/NCT02898103. Dated Updated 04/05/2019.
6. Melzack R, Wall PD. Pain mechanisms: A new theory. *Science* 1965; 150:971-979.
7. McRoberts WP, Wolkowitz R, Meyer DJ, et al. Peripheral nerve field stimulation for the management of localized chronic intractable back pain: Results from a randomized controlled study. *Neuromodulation* 2013; 16:565-575.
8. Perryman LT. Peripheral nerve stimulation: From nerve blocks to wireless neuromodulation. *Clin Neuro Neurological Res Int J* 2019; 2:180007.
9. Goroszeniuk T, Kothari S, Hamann W. Subcutaneous neuromodulating implant targeted at the site of pain. *Reg Anesth Pain Med* 2006; 31:168-171.
10. Jensen MP, Brownstone RM. Mechanisms of spinal cord stimulation for the treatment of pain: Still in the dark after 50 years. *Eur J Pain* 2019; 23:652-659.