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# Peripheral Nerve Stimulator as an Alternative to Spinal Cord Stimulator for Management of Refractory Lower Extremity Neuropathic due to Lumbar Post Laminectomy Syndrome (PLS): Case Report

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## Abstract

Neuropathic pain in lower extremities related lumbar post laminectomy syndrome (PLS) is a vague term defined clinically as persistent or recurrent neuropathic pain following otherwise anatomically successful surgery on the lumbar spine. We present a case of refractory chronic right lower extremity neuropathic pain of the foot in a 22-year-old man who underwent surgical posterior decompression of L1 laminae and Right transpedicular decompression with posterior T12-L2 fusion. The pain was refractory to conventional medications and adjuvants like anticonvulsants, antidepressants, muscle relaxants including common interventional pain procedures on the spine and lower extremity. Spinal cord stimulation is being increasingly used for such complex refractory pain but it comes with its inheritent risk in addition to cost and long term sequele. To avoid the limitations of spinal cord stimulator and patient refusal we placed Bioness Stim Router Permanent Placement of PNS Leads, percutaneously under ultrasound guidance to the right tibial and right peroneal nerves for the right lower extremity neuropathic pain of the foot. Patient reported more than 90% decrease in pain severity associated with significant reduction in overall oral analgesics post implantation.

**Keywords:** Neuropathic Pain; Anticonvulsants; Antidepressants; Lumbar Spine

## Abbreviations

PLS: Post Laminectomy Syndrome; PNS: Peripheral Nerve Stimulation; SCS: Spinal Cord Stimulation; IPG: Impulse Generator; MRI: Magnetic Resonance Imaging; OR: Operating Room; CPN: Common Peroneal Nerve; TN: Tibial Nerve; mA: Milli Ampere; FBSS: Failed Back Surgery Syndrome; IASP: International Association for the Study of Pain; PAG: Periaqeductal Grey; RVM: Rostral Ventromedial Medulla; CNS: Central Nervous System; NSAIDs: Non-Steroidal Anti-Inflammatory Drugs.

## Introduction

While the use of peripheral nerve stimulation (PNS) originated more than five decades ago, this therapy has only recently experienced resurgence after falling out of favor for many years. After the publication of the gate control theory in 1960 by Melzack, et al. [1], published Case series demonstrating decreases in pain perception throughout the entire episode of peripheral electrical stimulation of the selected area [1,2]. Shelden compared electrical stimulation over compression technique for intractable trigeminal neuralgia, reported higher success rate with electrical stimulation over compression method [3]. After its initial exciting report, there were multiple publications on PNS and it failed to gain popularity among the clinicians as treatment was mired by suboptimal outcomes, poor long-term success rates, and often encountered with complications such as nerve damage or stimulation-induced fibrosis. Many of the adverse occurrences could be attributed to technical problem like inappropriate placement, lead migration, inadequate pulse generator in addition to proper patient selection [3-6].

In the 1990s, Weiner and Reed revived the interest in PNS after their successful demonstration of placement of a percutaneous electrode insertion in the vicinity of the greater occipital nerves to treat occipital neuralgia [7]. More recently, the emergence of a minimally invasive percutaneous approach, typically performed under ultrasound, has led to even more widespread interest in PNS as an alternative to neurosurgically implanted more invasive spinal cord stimulation (SCS) systems [7-10]. For PNS there are multiple options exist for the placement of electrical leads which are stimulated directly with an external or implanted power source/impulse generator (IPG) or hybrid systems powered via an external power source. Neuromodulation of peripheral nerves to treat pain is an area of great intellectual activity and is still evolving. While spinal cord and deep brain stimulation being more invasive and placed within the central nervous system have greater current public and clinical awareness than peripheral nerve stimulation (PNS), the PNS antedates both. Neuromodulation often provides an opportunity to reduce or eliminate the use of opioids to treat complex chronic pain [9-11]. Here we are reporting a case of intractable neuropathic pain refractory to conventional analgesics and common interventional pain procedure which was successfully treated with peripheral neuromodulation.

## Case Report

22 years old male with past medical history of spinal trauma resulting from a fall from heigh causing burst fracture of L1 vertebra which was treated surgically by posterior decompression of L1 laminae and Right transpedicular decompression with posterior T12-L2 fusion. After few weeks, he presented with back pain and radicular pain on the right leg pain associated with weakness and numbness. He reported the pain as severe (Numerical Rating Scale NRS- 9/10 in severity) and burning in character. Physical exam revealed tenderness to palpation in area of lumbar paraspinal area bilaterally and positive facet loading bilaterally and right foot drop with variable sensory deficit over the right L2-5 distribution. Patient was advised for magnetic resonance imaging (MRI) which didn't reveal any direct cord compression and intact spinal implants and posterior instrumental fusion T12, L1, L2, L1 decompressive laminectomy, chronic L1 vertebral body and right L1 transverse process fracture. Initially he was prescribed simple analgesic Ibuprofen (Nonsteroidal Analgesics (NSAIDs), Tramadol (opioid) along with Pregabalin (Anticonvulsant), Cymbalta (Antidepressant) and baclofen (muscle relaxant), he was also advised for physical therapy for 2 weeks. He demonstrated moderate reduction in pain score (5/10) and slight improvement in right leg weakness. Considering the persistent pain despite NSAIDs and other adjuvants, He was offered a trial of radiofrequency ablation (RFA) of medial branch of dorsal root of Right L3, 4 and 5 Lumbar spinal nerve after a successful diagnostic block with local anesthetic. Unfortunately, the results of RFA were unsatisfactory as there was no significant pain relieve contrary to our expectation. After failed attempt with RFA, he had undergone other interventional procedures like lumbar sympathetic and caudal injection with steroid (Depomedrol) which temporarily relieved his pain lasting for few days. Patient was also given option of spinal cord stimulation as an alternative option, but considering the more invasive nature of the procedure and long term sequele, patient refused to go ahead with spinal cord stimulation but willing for peripheral nerve stimulation. We decided to proceed to peripheral nerves stimulator in the right Common peroneal nerve and tibial nerve in popliteal fossa under ultrasound guidance. Patient was cleared by psychiatric evaluation for the procedure. The risks and benefits of the procedure were described to the patient, and informed consent was obtained.

The patient was brought into the Operating Room (OR) suite and placed prone position on the procedure table. Standard monitors were applied (Non-invasive blood pressure, pulse oximetry, and ECG) and closely monitored during the procedure. An intravenous line was placed prior to the procedure. Patient was sedated by Midazolam 2 mg intravenously, Antiseptic skin preparation done with chlorohexidine and alcohol, sterile drapes placed on the right popliteal fossa and upper back of thigh, and sterile technique was strictly observed throughout the procedure including sterile probe cover for the ultrasound transducer.

A linear high frequency probe (20 Hz) was used for scanning the popliteal area in horizontal transverse plane, the

ultrasound transducer was adjusted by sliding and tilting until the bifurcation of the common peroneal nerve (CPN) and tibial nerve (TN) was seen. A bioness StimRouter Neuromodulation System with dual stimulation leads with three electrical stimulation (tetrapole) was chosen for the peripheral neuromodulation.

Inplane technique with transducer in transverse plane was used for needling and placement of stimulating leads. After subcutaneous infiltration with lignocaine 1%- 2 ml, a nick was given at the needle entry point. A 18 G spinal needle was used as a conduit for lead placement. Initial we targeted the CPN, the spinal needle was inserted into the skin and subcutaneous tissues and directed at towards the CPN nerve (Figure 1) and small pocket was created using 5 ml of dextrose 5% water solution, after confirming the spread of solution around the CPN, the stimulating lead was placed he spinal needle was then removed and the stimulating lead (tetrapole) placement confirmed with electrical stimulation with 0.7 milli ampere(mA), Same steps were followed for the TN lead placement (Figure 2&3). The stimulating leads were tunneled out laterally (Figure 4). Electrical stimulation was checked before and after tunneling of the stimulating leads. A single 3.0 proline suture was used to approximate both the incisions and glued with derma bond at both exit sites. These sites were then covered with transparent Tegaderm. The stimulating leads are connected to the external pulse generator, after final confirmation with electrical stimulation and stimulation established at 2 Hz frequency. The patient tolerated the procedure well and there was no immediate complication encountered. Patient reported significant pain relief immediately. His oral analgesics dose was made half Meloxicam, Pregabalin and Cymbalta along with Tramadol as needed. In the Flow up Patient reported that pain control has been adequate with minimal use of opioid prescription provided to patient. Patient reports more than 90% decrease in their baseline pain score since implantation of peripheral nerve stimulator. Patient used to use the stimulation around 18-20 hrs. a day and was very satisfied with the device. His oral medication has been reduced on biweekly basis and he is currently taking on pregabalin and tramadol as required.

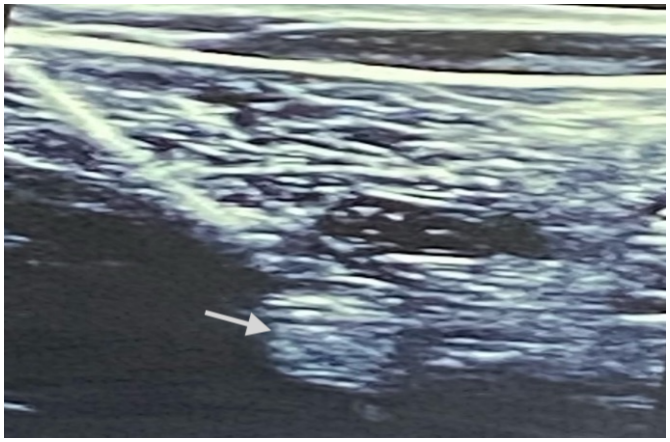

**Figure 1:** Showing Introducer of the lead close to CPN (white arrow).

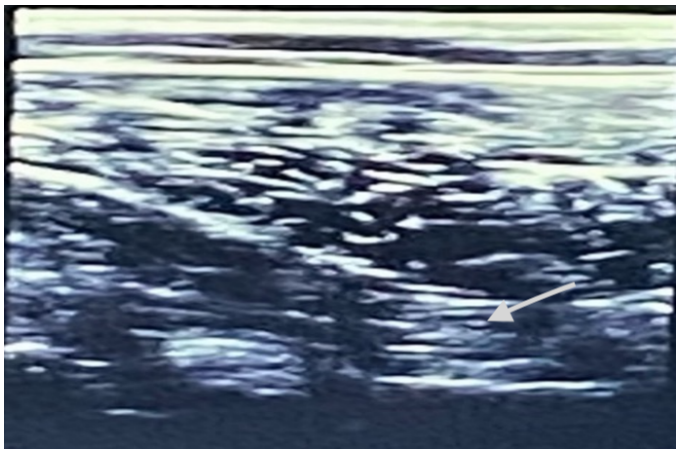

**Figure 2:** Showing Second Introducer Close to TN (white arrow).

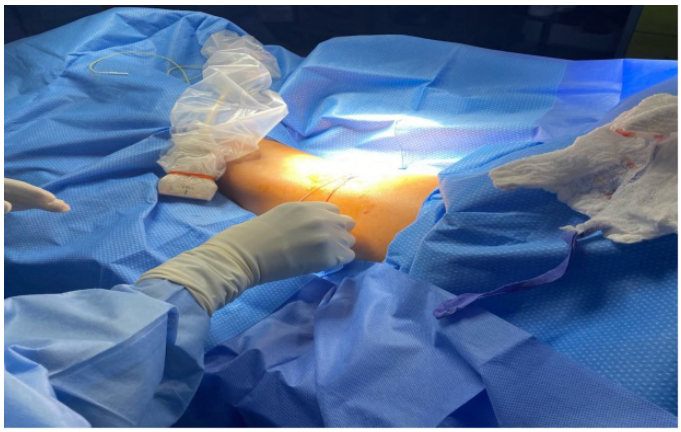

**Figure 3:** Showing Two Leads Before Tunnelling.

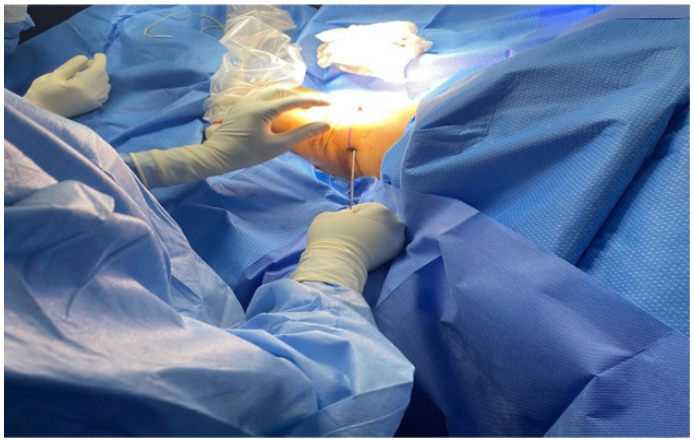

**Figure 4:** Showing Tunnelling of the Lead.

## Discussion

Post laminectomy syndrome (PLS) or failed back surgery syndrome (FBSS) is a vague term defined by the International Association for the Study of Pain (IASP) as persistent or recurrent back pain, with or without referred or radiating pain, that is located mainly in the lower limbs after anatomically successful surgery on the lumbar spine [12]. It has a high prevalence rate, since over 300,000 spinal fusions are performed annually in the United States and out of which 10-40% may actually develop PLS [12]. Possible pathophysiologic causes for PLS are myriad and may include, but not be limited to, epidural scarring, dural sac deformity, arachnoiditis, or persistent spinal instability [13]. Symptoms of PLS may include a various combination of axial pain, radicular pain, diffuse lower extremity pain, bladder, bowel and sexual dysfunction. PLS has traditionally been treated with repeat surgery, pharmacologic management, or interventional pain therapies such as epidural steroid injections, facet denervation, or spinal cord stimulation (SCS) [12,13]. The success of these therapies are variable and depends largely on the specific pathophysiology at play, but in many cases, treatment is no more than palliative therapy. Our patient showed inadequate clinic response to medications and interventional pain procedures. The next step to manage refractory neuropathic pain was neuromodulation namely SCS, a form of neuromodulation therapy used for various chronic painful condition of trunk and limbs [6]. Although, SCS has been used effectively over five decades, despite improvement in technology, physician training and better patient selection, it still carries risk of short and long term complications like post spinal headache, epidural hematoma, infection, fibrosis, nerve injury, wound infection [5]. We have used an alternative form of neuromodulation to treat neuropathic related to PLS, subcutaneous peripheral nerve stimulation (PNS) which has been in clinical use since 1965 and has demonstrated reasonable success rate as a treatment for numerous neuropathic conditions [11]. However, recognizing the limitations of spinal cord stimulator and emerging evidence supporting the use of PNS to manage post laminectomy neuropathic pain, we decided to procced with peripheral nerve stimulator. Although

systematic reviews of SCS for the treatment of chronic low back pain have been published [11,14]. Evidence supporting PNS for the treatment of this condition is still emerging. In a multicenter RCT, Eldabe et al compared 38 patients with failed back surgery syndrome (FBSS) who received subcutaneous PNS plus optimized medical management with those who received optimized medical management alone (n = 36) [14]. In the PNS arm, a maximum of two leads were placed subcutaneously in the vicinity of pain [14]. Patients were evaluated at baseline and periodically at one, three, six, and nine months after study arm assignment. The primary endpoint was more than 50% reduction in pain at six months and more than 30% reduction in pain at nine months. A significant proportion of patient reported greater reduction on pain in the PNS combined with optimal medical management at nine months 39.3% as compared to only 1.7% of patients who received optimal medication management alone ($P < 0.0001$) [14].

### Mechanism of Action of PNS

Although the exact mechanism of action of PNS is unknown, there is evidence that both central and peripheral mechanisms contribute to its analgesic effect. The peripheral and central sensitization after an initial neural injury play a critical role in the development of chronic neuropathic pain. The central sensitization and wind-up phenomenon is a complex mechanism involving both ascending afferent and descending efferent pathways. The inflammatory cascade leads to hyperexcitability of afferent while altered modulation from the Periaqeductal grey (PAG) and rostral ventromedial medulla (RVM) leads to increased pain signal transmission to thalamus and sensory cortex [15]. Like SCS, PNS is thought to provide analgesia via modulation of gate-control theory of pain, as described by Melzack, et al. [1]. As per this theory, the stimulation of large-diameter, low threshold, non-nociceptive Aβ fibers results in the excitation of inhibitory dorsal horn interneurons that are involved in the processing and transmission of nociceptive information from the Aδ and C nerve fibers, thus inhibiting pain signal transmission from the spinal cord to higher centers in the central nervous system (CNS) [15,16]. Moreover, the PNS decreases central sensitization and hyperalgesia by reducing excessive peripheral nociceptive activity in the spinal cord, inhibiting the wide dynamic range neurons in the dorsal horn, and decreasing Aβ fiber-induced activity in the medial lemniscal pathway in the brain [14,16]. Additionally, animal research has also demonstrated that the analgesic effects of the PNS may involve either serotonergic (5HT2, 5HT3), GABAergic, and glycinergic pathways [17,18]. On a molecular level, the PNS has been shown to modulate the local microenvironment by changes in the biochemistry of neuron by downregulating neurotransmitters, local inflammatory mediators and upregulating the endogenous endorphin. Electrophysiological studies have also demonstrated reduced spontaneous electrical discharge known as ectopic discharges with PNS. A human study by Torebjork and Hallin has demonstrated that repeated electrical stimulation of intact radial and saphenous nerves resulted in excitation failure of A and C fibers [19]. The concept of peripheral reconditioning of the central nervous system through longer-term changes in central plasticity has also been proposed [20].

## Conclusion

Our case contributes to a growing body of evidence that supports the use of PNS to treat well-localized neuropathic pain. It is unique in that it represents a simple and relatively safe alternative to SCS in the treatment of a prevalent condition.

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