

Spinal Cord Stimulation for the Treatment of Intractable Pain from Failed Back Surgery Syndrome

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ABSTRACT

Introduction: Failed back surgery syndrome (FBSS) is a debilitating chronic neuropathic pain condition, affecting approximately 10-40% of patients after lumbosacral spine surgery. Treatment of FBSS is challenging as conservative therapies and repeat surgery often fail in providing adequate pain relief. Spinal cord stimulation (SCS) has been proven to be a successful therapeutic option in FBSS patients.

Case Report: A 23 year-old male presented with persistent low back pain and bilateral radicular pain after spinal surgery. He was also wheelchair-bound because of his severe pain. Conservative treatment with oral analgesics and interventional pain procedures were unsuccessful in improving his pain. Subsequently, we performed a permanent implantation of epidural leads for SCS after a successful trial of SCS. He reported good coverage of pain without complications after the procedure. Three months after surgery, and after intensive physiotherapy and rehabilitation, he could walk and return to work.

Conclusion: We describe our first successful case of treating intractable pain from FBSS in a patient after implantation of SCS leads in our institution.

Keywords: failed back surgery syndrome, intractable, low back pain, spinal cord stimulation

INTRODUCTION

Failed back surgery syndrome (FBSS) is a chronic neuropathic pain condition involving the back and legs¹ in 10–40% of patients who have undergone lumbosacral spine surgery^{2,3}. Compared with nociceptive pain, neuropathic pain is less responsive to analgesic drugs and other conventional medical management^{1,4}.

Spinal cord stimulation (SCS) describes the use of pulsed electrical energy near the spinal cord to control pain^{5,6}. It is a technically challenging interventional pain management technique. SCS has proven to be an effective therapeutic modality for the treatment of certain chronic pain syndromes, including FBSS, pain associated with peripheral vascular disease, peripheral neuropathies, multiple sclerosis and complex regional pain syndrome⁷. We describe a case of FBSS in a patient who reported significant reduction in pain after SCS.

CASE REPORT

A 23-year-old male developed low back pain in 2006 after exercises during national service. He experienced an acute exacerbation of low back pain in February 2007. He had neither radicular symptoms nor bowel or bladder incontinence. Subsequently, he was started on Gabapentin 300mg TDS and Baclofen 10 mg BD. In addition, he was referred to a rehabilitation physician who advised him for ultrasound, traction and physiotherapy. His pain gradually improved and he was discharged from hospital and returned to normal activities.

In June 2007, he developed an acute exacerbation again. He complained of low back pain, associated with radicular pain and weakness in his left leg. Pain score was 6–7/10. He was unable to weight bear. A magnetic resonance imaging (MRI) showed degenerative disc disease at L4/5, L5/S1 levels with posterior annular tear and focal central disc



Fig. 1. A magnetic resonance imaging (MRI) showed degenerative disc disease at L4/5 and L5/S1 levels with posterior annular tear and focal central disc protrusion.

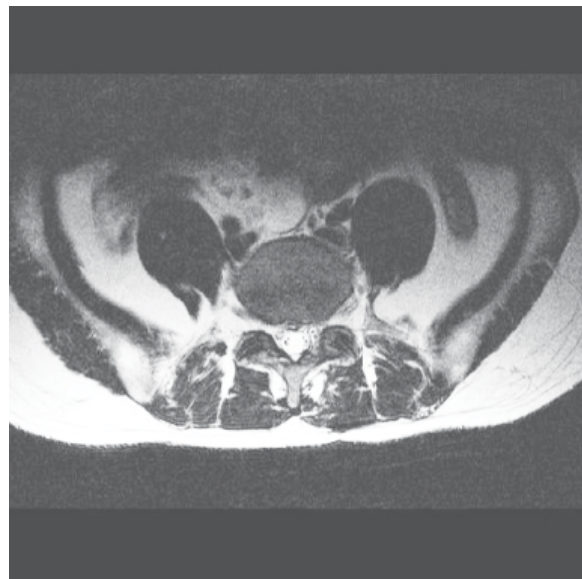


Fig. 2. A magnetic resonance imaging (MRI) showed degenerative disc disease at L4/5 levels with posterior annular tear and focal central disc protrusion.

protrusion (Fig. 1 and 2) He received a left L4 transforaminal and L5/S1 interlaminar epidural steroid injection by the pain management team. He reported partial relief of symptoms and was discharged the same day.

As his pain symptoms did not completely resolve, he underwent a left L4/5, L5/S1 foraminotomy and microdisectomy in August 2007. However, his low back pain and bilateral radicular symptoms persisted even after surgery. Pain was more severe on the left side. He could ambulate with crutches, but he could not return to work.

In January 2008, his back and leg pain worsened at pain score 7/10. Physical examination showed motor weakness of lower extremities (Gr4/5), reduced sensation to pinprick and light touch in L4, L5 and S1 bilaterally. Deep tendon reflexes were normal. Subsequently, he became wheelchair-bound. He was treated conservatively with adjustment of pain medications. He was prescribed Pregabalin 150 mg BD, Nortriptyline 25 mg ON, Anarex 2 tablets TDS, Baclofen 10 mg TDS, and Oxycodone 5 mg PRN for pain every 4 hourly. A repeat MRI of his lumbar

spine showed a recurrent left paracentral disc protrusion at L5/S1, which severely narrowed the left lateral recess. At L4/5 there was enhancement of the posterior annulus at the site of the previous microdisectomy. Enhancement in the anterior and left lateral epidural space was also suspicious of early epidural fibrosis. He was deemed unsuitable for a repeated surgery. Following that, he underwent bilateral L4/5 transforaminal neuroplasty with only marginal improvement in pain and function.

Finally, he was offered spinal cord stimulation. Trial leads insertion was performed in May 2008. The epidural space was accessed at L1/2 and T12/L1 levels. Two leads were advanced under fluoroscopic guidance to the T9 vertebral level in the dorsal epidural space. With stimulation, he reported good coverage of pain involving 80% of the right leg and 40–50% of the left leg. Therefore, we proceeded with permanent implantation of spinal cord stimulator. Implantation was performed with dual octrode leads, and skin entry point was through L1/2 and T12/L1 levels. The epidural leads were placed at T10–12 level and they were anchored at the interspinous ligaments. (Fig. 3,

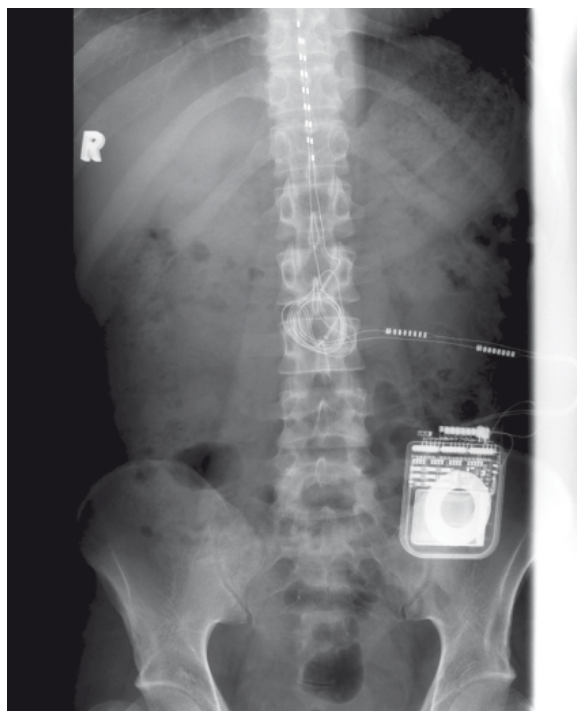


Fig. 3. Radiograph showing the spinal cord stimulator system consisting of a subcutaneous implantable pulse generator with dual octrode leads placed at T10-12 level.

4 and 5) Extension wires were then tunnelled under the skin to a subcutaneous pocket at the left abdominal wall where the implantable pulse generator was located. He was discharged home a week later with full instructions on how to use the SCS system.

At follow-up 2 weeks after surgery, he continued to report good coverage of pain in both legs although his motor and sensory deficits remained. Pain score was 3/10. He was referred for physiotherapy and started ambulating with crutches. In addition, he was doing well with cycling, stairs, and leg strengthening exercises with weights. His medication intake was reduced to Pregabalin 150 mg ON and Nortriptyline 25 mg ON. By 3 months after permanent spinal cord stimulator implantation, he could walk with a walking stick and returned to work in an advertising based business. By 1 year after the procedure, his pain improvement was sustained. Pain score was 4/10. He claimed that he was able to travel by bus and train independently. His symptom control was good even at 2 years.



Fig. 4. Implantation was performed with dual octrode leads and the epidural space was accessed at L1/2 and T12/L1 levels.

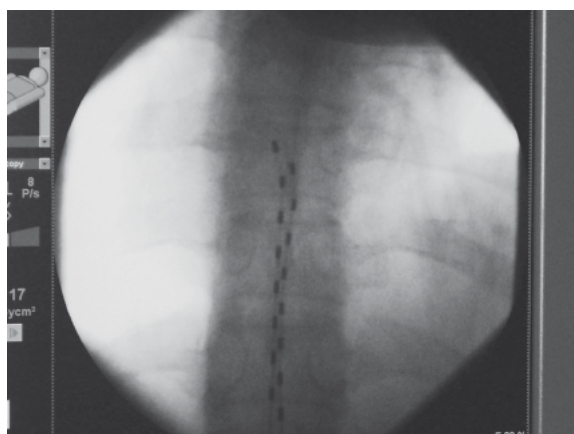


Fig. 5. Anterior fluoroscopic view of dual octrode leads placed at T10-12 level.

DISCUSSION

Treatment of FBSS is a challenge as conventional medical therapies as well as repeat surgery are often unsuccessful in providing adequate pain relief. In medically refractory FBSS patients, where recurrent neuropathic pain persists despite surgery and analgesics are no longer effective or produce intolerable side effects, SCS has proven to be a particularly successful therapeutic option that should be considered even before strong opioids. FBSS is the most frequent indication for SCS, with neuropathic leg pain component being a good responder⁸.

We report a successful case using SCS to treat a patient with intractable neuropathic pain from FBSS. This technique was first applied in the intrathecal

space and finally in the epidural space as described by Shealy in 1967⁶. In the present day, SCS involves the implantation of leads in the epidural space to transmit pulsed energy across the spinal cord or near the desired nerve roots. The gate control theory proposed that painful peripheral stimuli carried by C-fibres and lightly myelinated A-delta fibres terminate at the substantia gelatinosa of the dorsal horn. Large myelinated A-beta fibres responsible for touch and vibratory sensation also terminate at the "gate" in the dorsal horn. It was hypothesised that their input could be manipulated to "close the gate" to the transmission of painful stimuli. As an application of the gate control theory, the implantation of spinal cord stimulator device was developed for the treatment of chronic pain.

The neurophysiological mechanisms of action of spinal cord stimulation are not completely understood. However, recent research has given an insight into effects occurring at the local and supraspinal levels, and through dorsal horn interneuron and neurochemical interactions^{9,10}. Experimental evidence points to SCS having a beneficial effect at the dorsal horn level by favourably altering the local neurochemistry, thereby suppressing hyperexcitability of the wide dynamic range interneurons¹¹.

Numerous studies, representing up to 10 years of follow-up, have assessed the efficacy of SCS in reducing the pain associated with FBSS^{12–16}. A long-term retrospective review of 254 patients selected for dominant neuropathic pain in the leg found that 68% of patients had a good to excellent response to SCS and that pain improved significantly by up to 57%. As a result, concomitant pain medication was reduced by more than 50%¹³.

The efficacy of SCS at reducing intractable low back pain was assessed in a recent multicentre prospective study by Barolat *et al*¹⁴. Preoperatively, all patients had pain in their back and legs, with more than 50% of their pain in their low back. Following 12 months of treatment with SCS, the majority of patients reported fair to excellent pain relief in both the low back (68.8%) and legs (88.2%). In a systematic review, around 62% of FBSS patients treated with SCS achieved 50% pain relief or more. In addition, 53% of patients no longer required analgesics¹⁷. Additionally, a recent systematic review by Frey *et al* evaluating the effectiveness of SCS in relieving chronic intractable

pain of failed back surgery syndrome indicated the evidence to be Level II-1 or II-2 for clinical use on a long-term basis¹⁸.

SCS also has been shown to significantly improve the quality of life (QoL) of patients with FBSS^{13–15}. In a recent cost-effectiveness study comparing SCS with conventional pain therapy (CPT) in 104 FBSS patients over 5 years, the QoL was found to improve by 27% in the SCS group, compared with only 12% in CPT group⁷. In another study specifically designed to evaluate patient satisfaction after SCS, 70% of patients declared that they were satisfied with the outcome of their treatment¹⁹.

The literature shows that the incidence of side effects experienced with SCS is relatively low. Most of these were reversible and mainly due to electrode or lead problems. No serious adverse events and no neurological complications were reported.

Evidence suggests that early treatment of neuropathic back and leg pain with SCS yields the best results. The sooner SCS is implemented after the first failed back surgery, the better the outcome. In a study of 235 patients treated over 15 years, the success rate of SCS dropped from 93% in patients who had a 3-year delay between surgery and implantation to 9% for those who had a 12-year delay. Thus, SCS should be considered early in the management of FBSS, prior to a second operation and before the use of high-dose opioid analgesics²⁰. A randomised controlled trial by North *et al* demonstrated that compared with re-operation, SCS provides effective pain relief for many years¹⁶. A prospective randomised controlled multicentre trial showed that selected FBSS patients reported sustained pain relief, clinically important improvement in functional capacity and health-related quality of life, and satisfaction after 24 months of treatment²¹.

Trial stimulation is essential to allow doctors and patients to assess SCS for efficacy and paraesthesia sensation before permanent implantation. Psychological evaluation is also vital and as much emphasis should be given to identifying potential adjunct therapies (e.g. treatment of major depression or drug dependence) that could enhance the success of SCS therapy as to identifying "predictors" of success. Some investigators have suggested that screening for patients with personality disorders, somatoform

disorder, or hypochondriasis may improve success rate of SCS. To have realistic expectations of pain, it is crucial that patients have a full understanding of what SCS entails⁸.

CONCLUSION

We describe our first successful case of treating intractable pain from FBSS in a patient after implantation of SCS in our institution. SCS is the treatment of choice in well-selected patients with medically refractory FBSS and should be considered when analgesics are no longer effective or cause intolerable side effects.

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