

Spinal Cord Stimulation for the Neuropathic Pain Caused by Traumatic Lumbosacral Plexopathy after Extensive Pelvic Fracture

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The neuropathic pain caused by lumbosacral plexopathy as a sequela to extensive pelvic and sacral fractures is rare because many posttraumatic cases remain undiagnosed as a result of the high mortality associated with these types of injury and because of the survivors of multiple trauma, including pelvic fractures, frequently have an incomplete work-up. Although surgical treatments for medically refractory lumbosacral plexus avulsion pain have been reported, an effective surgical technique for pain relief in lumbosacral plexopathy has not been well documented. We describe the effectiveness of spinal cord stimulation (SCS) in a patient suffering from severe neuropathic pain caused by lumbosacral plexopathy after an extensive pelvic fracture.

KEY WORDS : Neuropathic pain · Lumbosacral plexopathy · Pelvic fracture · Spinal cord stimulation.

Introduction

Post-traumatic lumbosacral plexopathy is a relatively rare clinical entity that usually results in severe neurological deficits^{3,8)}. As the post-traumatic lumbosacral plexopathy is frequently undetected after pelvic and sacral fractures due to several clinical limitations^{3,7,8)}, there are only a few reports on the neuropathic pain resulting from traumatic lumbosacral plexopathy as well as its effective management.

Spinal cord stimulation(SCS) has been used to treat intractable pain syndromes for more than 30years¹³⁾. The best established application for SCS, which is based on more than 30years of clinical experience, is neuropathic pain¹¹⁾. Although the analgesic effect of SCS for treating the various peripheral neuropathic pain has been reported^{5,11,13,20)}, its effectiveness in the lumbosacral plexopathy has not been well known.

Case Report

History and examination

A 23-year-old male patient was referred for relief of bilateral

lower extremity pain. Fifteen months ago, he was crushed by the blade of a crane and was transferred to the emergency unit. Severe respiratory distress from a pneumothorax as a result of multiple rib fractures and a rupture of right-sided hemi-diaphragm was detected, and an emergent laparotomy was performed to repair the ruptured diaphragm and closed chest tube drainage. The patient also showed evidence of hypovolemic shock from retroperitoneal hemorrhage due to an extensive pelvic fracture and left femur fracture and was managed with a transfusion and fluid therapy. Three days after the accident, a closed reduction with an internal fixation of the left femur fracture and external fixation for the unstable pelvic and sacral fracture were performed (Fig. 1).

Subsequently, he had been treated with artificial ventilation for the acute respiratory distress syndrome for 3weeks. Continuous hematuria through an indwelling foley catheter with resultant anemia developed, and pelvic angiography was performed under the suspicion of a bladder rupture and to search for the cause of the retroperitoneal hemorrhage. An interventional coiling was performed in the right internal iliac artery to control the retroperitoneal hemorrhage. For the following months,

• Received : February 3, 2005 • Accepted : June 1, 2005

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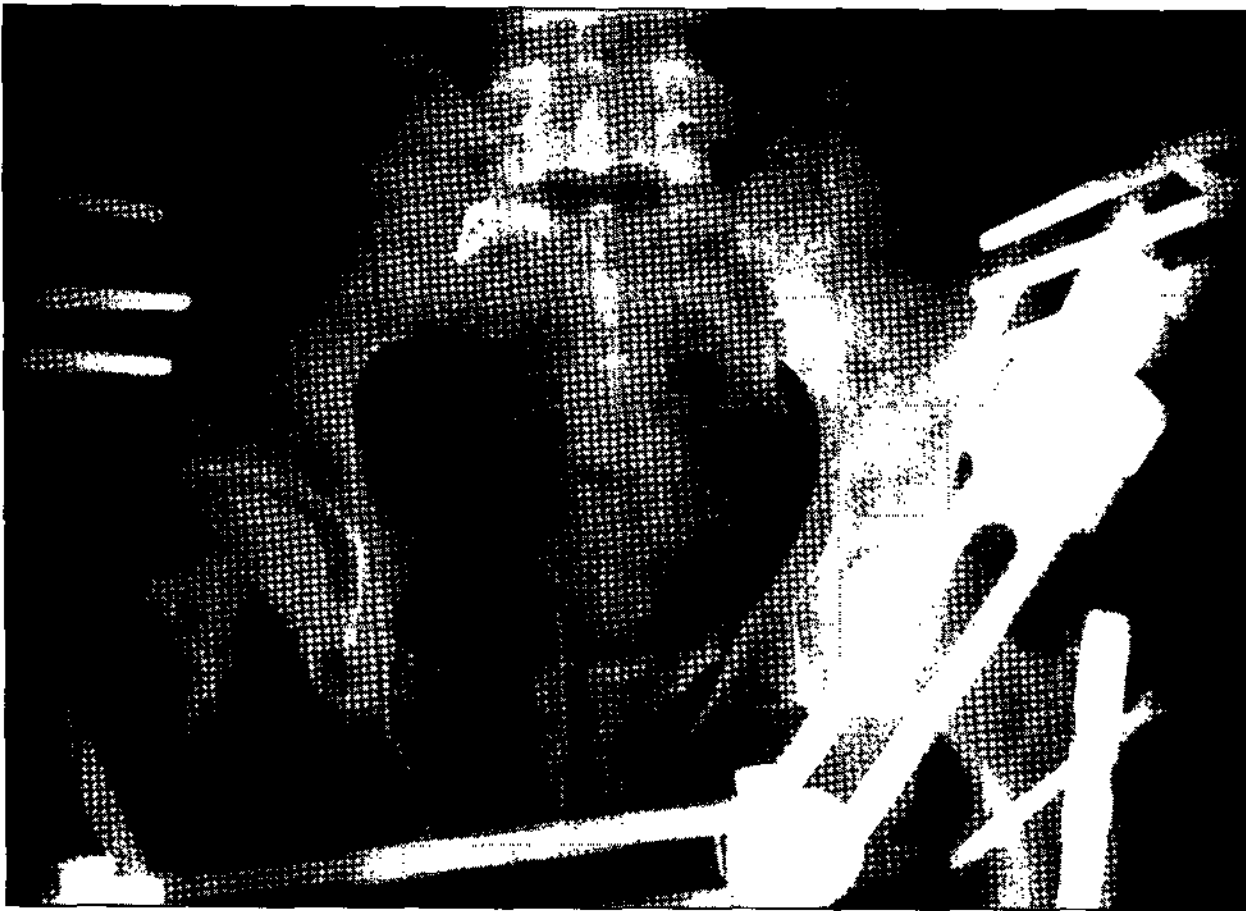


Fig. 1. A pelvic x-ray film showing extensive pelvic fractures, i.e. bilateral superior and inferior pubic rami with displacement and sacral fracture dislocation of the right sacroiliac joint. Note the external fixator for the unstable pelvic fractures and a thrombogenic coil for the retroperitoneal bleeding in the right retroperitoneal cavity.

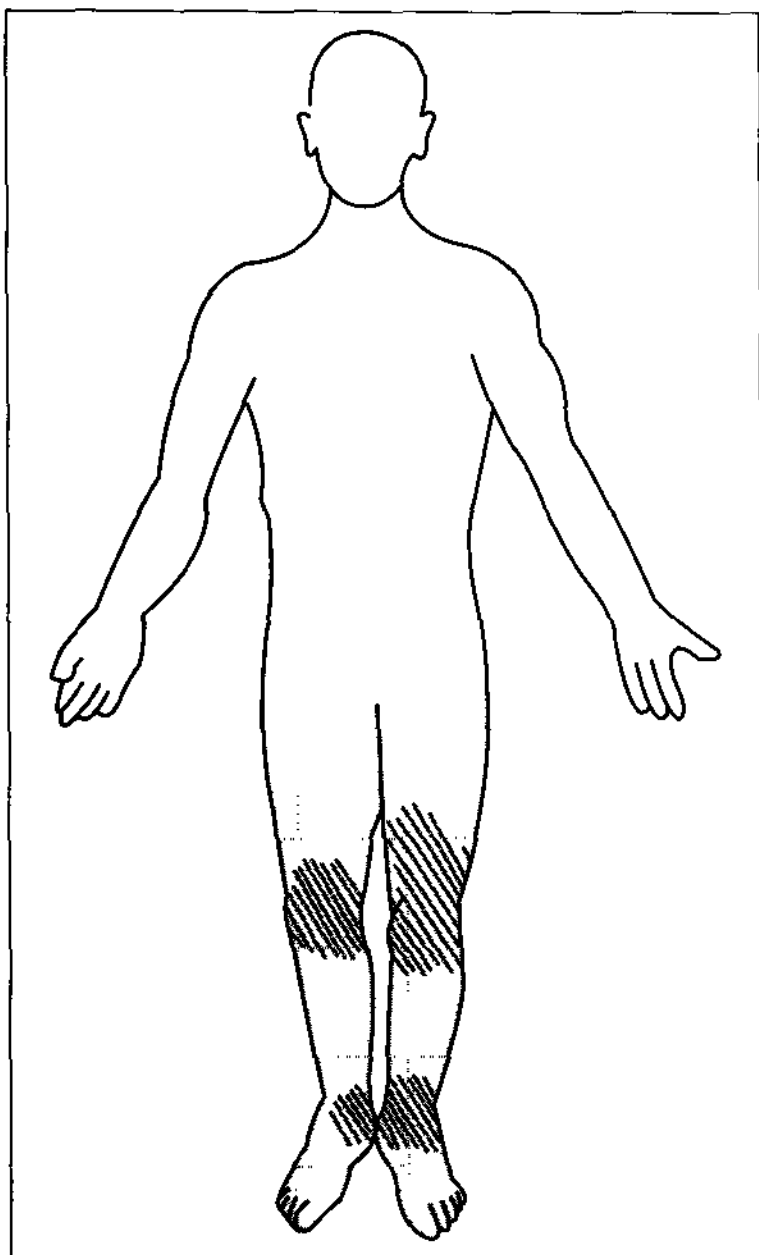


Fig. 2. Schematic drawing of the pain distribution after the traumatic lumbosacral plexopathy.

the patient's general condition stabilized, but he was bedridden due to severe bilateral leg weakness and he gradually developing leg pain.

Initially, he was paraplegic with marked sensory loss in both legs. However, the sensory loss was partially recovered. Two months after injury, he began to feel deep pressure-like pain in his legs, which was more severe in his left leg and intermittent electricity-like pain in both lower

extremities were evident (Fig. 2). Despite best maximal medical treatment, his pain gradually worsened, and eventually he could barely sleep at night due to severe deep pressure-like pain. Local anesthetic blocks and lumbar sympathetic blockade were ineffective. He suffered two kinds of intractable pain, the constant deep pressurelike pain in his knees and ankles and intermittent electricitylike pain in his both legs. The constant pain persisted all the day and night, and this pain aggravated during night, subsequently 2~3times of Demerol injection were required at night.

At the time of the referral for pain control, his medications consisted of gabapentin 2400mg, amitriptyline 50mg, tegretol

800mg, tramadol 400mg, and morphine 90mg per day. Despite of these medications, the pain relief was not obtained and his VAS was 8/10. An examination indicated muscle atrophy in both legs with only grade 2 motor power in his distal legs. Hypesthesia and hypoalgesia were noted below the L2 dermatome. The urinary and rectal functions were preserved. Plain spine x-rays and MRI of the cervical, dorsal, and lumbar spine did not show any evidence of spinal cord injury or fracture. A lumbar myelography was normal without evidence of a pseudomeningocele or root avulsion. Somatosensory evoked potential of the lower leg did not show any discernible wave pattern. The diagnosis of lumbosacral plexopathy as a result of traction injury was made after electromyography(EMG) examination. Considering that this type of chronic neuropathic pain of a benign origin is notoriously treatment-resistant and he had shown a tedious but objective motor recovery, neuromodulation such as SCS instead of a neuroablative technique such as DREZ lesioning was adopted.

Surgical procedure

The patient was placed in the semilateral position exposing his back and spine in the operating field. With the local infiltration of 2% lidocaine, an approximately 5cm-sized linear skin incision was made over the T12-L1 spine level and the dissection was carried down to the fascia and muscles. The inferior portion of spinous process and lamina of T12 were removed, and the ligamentum flavum and epidural fat were excised. The laminotomy electrode (3587A, Resume II[®], Medtronic, MN) was inserted into the posterior epidural space and the electrode was advanced to the T10-T11 epidural space under fluoroscopic guidance. Intraoperative electric stimulation was performed through a screener (model 3625, Medtronic, MN) and the paresthesia coverage was evaluated. At the T10-T11 level, paresthesia initially arose in the dorsum of the foot and, with increasing the intensity of stimulation (1' - 2' +, 50Hz, 120 μ sec, up to 2.5volts), paresthesia ascended to the knee and anterior thigh, and finally to the buttock. Because the sole and posterior leg were not covered with paresthesia with the T11-T12 laminotomy electrode, a second laminotomy electrode was introduced into the epidural space below the first electrode level under the fluoroscopic vision. The location of the second, inferior electrode was determined (T12-L1) according to the intraoperative stimulation findings that elicited paresthesia over the bilateral sole and posterior calf and thigh. The final electrodes positions were confirmed to be at the level of the T11 and T12-L1 space. Confirming that the whole painful legs were covered with stimulationinduced paresthesia, the electrodes were fixed to the paraspinal muscles and fascias, and the wound was closed in layers (Fig. 3).

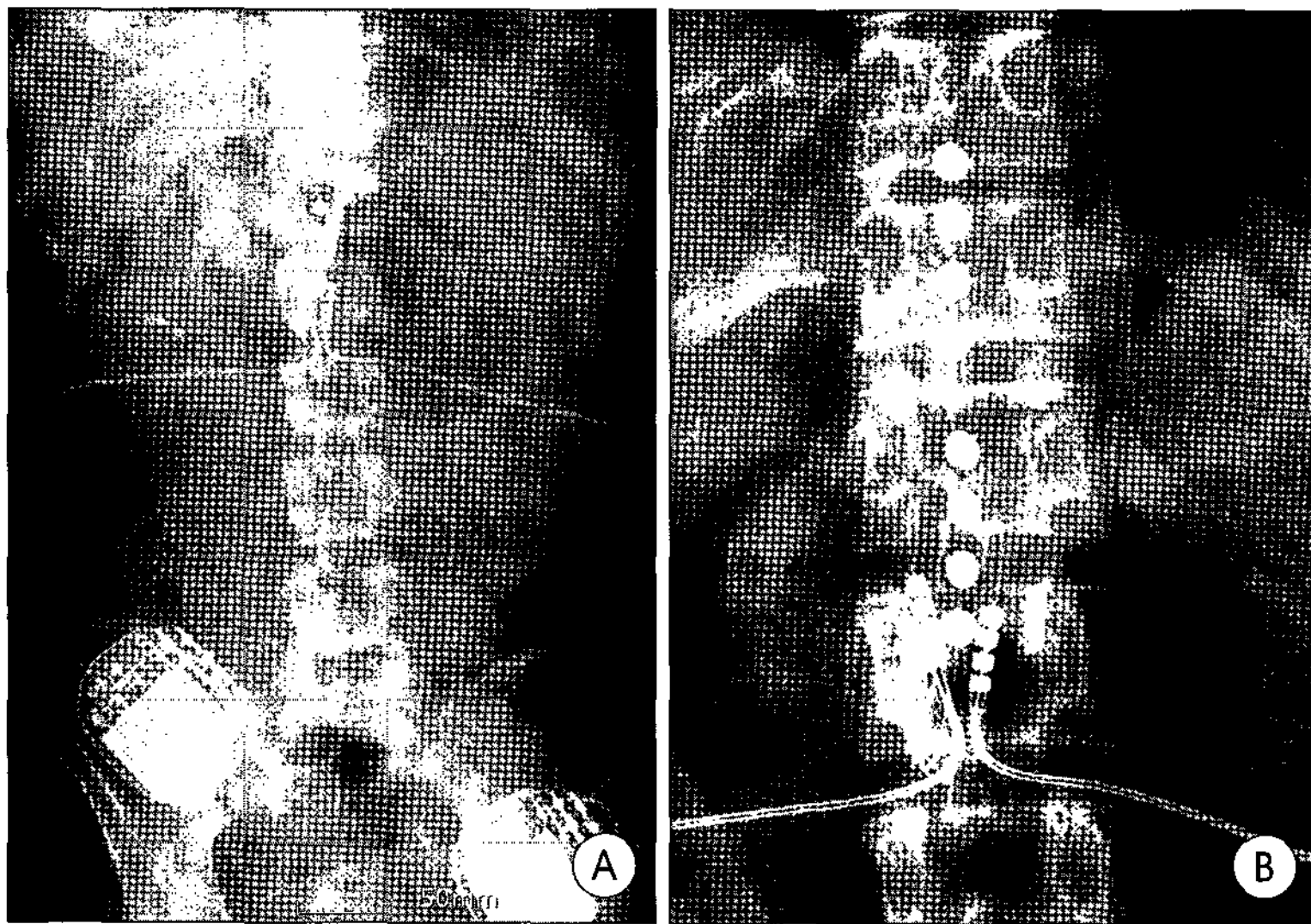


Fig. 3. Postoperative spine X-ray film showing the placement of the two laminotomy electrodes and the two pulse generators implanted subcutaneously in the bilateral lower quadrants (A) and the location of the electrodes in the thoracolumbar area; the upper one centered in the T11 and lower one in the T12-L1 epidural space (B).

Results of the pain relief

During the 3 days of trial stimulation his pain was significantly relieved (more than 70%). After inducing general anaesthesia, two implantable pulse generators (model 7425, Itriel III[®], Medtronic, MN) were implanted in the anterior lower abdomen and continuous stimulation was given. The chronic stimulation parameters are 1.5~2.4v, 150~210 μ sec, and 30~50Hz. The intermittent lancinating, electric shocklike pain responded much better than the deep pressure-like pain with the SCS. His VAS improved from 8~9 to 3~4 after SCS and this analgesic effect proved to long-lasting until 12 months postoperatively. His medications were decreased to gabapentin 900mg, amitriptyline 10mg, tramadol 150mg a day.

Discussion

Lumbosacral plexopathy in pelvic fractures

Lumbosacral plexopathy is a well-defined clinical entity with a frequency of occurrence that varies significantly between observers^{3,7,8}. The clinical presentation of lumbosacral plexopathy includes the motor and sensory deficit in the distribution of the multiple nerves originating from this plexus i.e. the sciatic, femoral, obturator, and superior and inferior gluteal nerves⁸. The proximity of the lumbosacral plexus to the sacral bone and the sacroiliac joint suggest a higher incidence of lumbosacral plexopathy with injury to these structures. In several studies on of trauma patients, a correlation was observed between the occurrence of lumbosacral plexopathy and the type of pelvic fracture^{6,8,19}, and the highest incidence of lumbosacral plexopathy was found among the patients with unstable pelvic fractures completely disrupting the sacroiliac

complex^{6,8,19}. An electrophysiologic study using nerve conduction and EMG performed no sooner than 3~4 weeks after the inciting event is essential for making final diagnosis⁸. Lumbosacral plexus avulsion with resultant lower extremity deafferentation pain is being encountered increasingly as more patients with extensive pelvic or sacral fractures survive their initial trauma¹⁷.

Mechanisms of spinal cord stimulation

SCS is a clinical application of the "gatecontrol" theory presented by Melzack and Wall¹⁰, who postulated that activity in the large cutaneous afferents inhibit the activity of the neurons in the dorsal horns, which then transmit noxious information. Since the introduction of SCS by Shealy, et al.¹³, this technique has been applied increasingly in the treatment of several types of chronic, intractable pain^{5,11,13,20}. Despite the extensive use of SCS for at least 30 years, knowledge of the physiological and biochemical mechanisms behind the beneficial effects of these methods is still incomplete⁹. In the neurogenic pain, the suppressive effect on the hyperexcitability of the dorsal horn neurons of the wide-dynamic range (WDR) cells and biochemical changes in the dorsal horn transmitters and neuromodulators (GABA, glycine, adenosine, 5-HT) have been demonstrated⁹. Antidromic activation of the dorsal column fibers may mimic the normal afferent inflow to these cells and produce the presynaptic inhibition of the pathologic afferent signals, and postsynaptic inhibition of the WDR cells through the interneurons activated from the dorsal column collaterals can occur. Supraspinal circuits such as the spinal descending pathways originating in the dorsal column nuclei, which terminate deep in the dorsal horn, can be activated by SCS and may play an important role in pain modulation^{9,18}.

Spinal cord stimulation in lumbosacral plexopathy

The reason we considered SCS for this patient was that SCS is one of the safest, non-destructive procedures available today for the long-term management of chronic pain¹⁴. Although DREZ lesioning of the conus medullaris has been reported to be effective in controlling neuropathic pain of lumbosacral plexus avulsion^{4,16}, its efficacy on controlling the neuropathic pain of lumbosacral plexopathy has not been reported. In addition, extensive DREZ operation at the lumbosacral segments inevitably result in leg hypotonia or sphincter disturbances¹⁵. In our opinion, the DREZ procedure was not appropriate for this patient because the urinary continence

was preserved and his cutaneous sensation was maintained, though severely hypesthetic and hypalgesic, and he has shown a slow, but steady recovery of the motor power despite the severe paraparesis.

We used a laminotomy electrode instead of a percutaneous electrode because the insulated, laminotomy electrode has several technical advantages that would double the battery life and might be associated with an improved clinical outcome¹². In a prospective, randomized study of patients with chronic lumbosacral pain syndrome, the overlap of pain by paresthesias and the calculated overlap (from the graphic data entered by the patients) were significantly better for the insulated electrode¹². The insulated, laminotomy electrode required a significantly lower amplitude than the percutaneous electrode¹², and this can be an additional benefit to the patient because the current SCS devices are not covered by national insurance in our country. Other advantages of the laminotomy electrode is their insulated dorsal surface, more inherent stability in the dorsal epidural space, and their lesser propensity to migrate².

It is generally accepted that in order to obtain a successful treatment of chronic pain by SCS, the stimulation-induced paresthesia must cover completely the painful area^{1,2,11,12,14}. In order to overlap both the anterior and posterior side of bilateral legs including the feet with paresthesia, we placed two laminotomy electrode from the T11 to L1 spine level according to the sensory mapping results of the SCS¹, and inserted the electrodes under local anesthesia to obtain an accurate sensory feedback from the awake patient as much as possible.

Conclusion

Spinal cord stimulation is effective for alleviating the chronic peripheral neuropathic pain caused by lumbosacral plexopathy following an extensive pelvic bone fracture.

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