

■ Focused Review

Spinal Cord Stimulation

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Spinal cord stimulation is the most common mode of neuromodulation used in managing chronic low back pain. It is minimally invasive and reversible as opposed to nerve ablation.

The basic scientific background of the initial spinal cord stimulation trials was based on the gate control theory of Melzack and Wall. It has been demonstrated in multiple studies that dorsal horn neuronal activity caused by peripheral noxious stimuli could be inhibited by concomitant stimulation of the dorsal columns. Various other mechanisms, which may play a significant role in the mechanism of action of spinal cord stimulation, include the suppressive effect of spinal cord stimulation on tactile allodynia, increased dorsal horn inhibitory action of gamma-aminobutyric acid (GABA), prevention or abolition of peripheral ischemia, and effects on human brain activity.

Spinal cord stimulation is indicated in low back pain with

radiculopathy, failed back surgery syndrome, complex regional pain syndrome, peripheral vascular disease, and ischemic heart disease.

There is substantial scientific evidence on the efficacy of spinal cord stimulation for treatment of low back and lower extremity pain of neuropathic nature. Clinical studies revealed a success rate of from 50% to 70% with spinal cord stimulation, with decreased pain intensity scores, functional improvement and decreased medication usage.

This review discusses multiple aspects of spinal cord stimulation, including pathophysiology and mechanism of action, rationale, indications, technique, clinical effectiveness, and controversial aspects.

Keywords: Spinal cord stimulation, failed back surgery syndrome, low back pain, percutaneous implantation, complications

Spinal cord stimulation for treatment of chronic low back pain has recently gained popularity. As opposed to nerve ablation, spinal cord stimulation is minimally invasive and reversible. The recent improvements in hardware design have made implantation techniques simpler and resulted in prolonged equipment longevity. Spinal cord stimulation screening trial, which is performed before permanent implantation, is a relatively minor invasive procedure, which allows patients to test its effects before final implantation. The scientific evidence has shown better outcomes with spinal cord stimulation in comparison to other modalities for treatment of some forms of low back pain.

Spinal cord stimulation is by far the most common mode of neuromodulation used in chronic low back pain. Failed

back surgery syndrome is the most common indication. The stimulating electrodes are placed in the epidural space either percutaneously or surgically depending on the severity of the accessibility of the epidural space. Consequently, the electrodes stimulate dorsal columns of the spinal cord; and, thus, the alternative term for spinal cord stimulation is *dorsal column stimulation*.

The current trend among interventional pain practitioners is to try spinal cord stimulation earlier in the course of chronic low back pain, even though for many years it was considered as a last option "when everything else failed." However, considering the relatively low cost of spinal cord stimulation trials, its low risk-benefit ratio and favorable outcome studies, spinal cord stimulation may be the best treatment option in some forms of chronic low back pain, such as failed back surgery syndrome.

Although its mechanisms of action have been attributed to Melzack and Wall's (1) "gate control theory," recent research efforts have revealed new potential mechanisms of action. It seems that spinal cord stimulation can at least partially exert its actions through modulation of neurotransmitters in the CNS.

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HISTORY

Humans opened an era of spinal cord stimulation by utilizing the electrical power of torpedo fish in 600 BC. The first attempts at brain electrical stimulation were reported in 1874. However, the first implantation of brain electrodes was not performed until 1948, for treatment of psychiatric disorder. Many attempts to use electrical CNS stimulation for treatment of pain emerged in the 1950s and 1960s based on the gate control theory of pain proposed by Melzack and Wall in 1965 (1). Two years later, Shealy and associates introduced spinal cord stimulation (2). Initial spinal cord stimulation procedures involved open intrathecal implantation of electrodes via laminotomy. The lack of adequate hardware and paucity of clinical outcome studies significantly slowed the development of neurostimulation in the 1970s.

The hardware technology has substantially improved over the years. Moreover, electrodes have become smaller in shape and easier to navigate through the epidural space; and, finally, internal pulse generators have new programming capabilities and a longer battery life span. All these technological developments led to the successful application of minimally invasive percutaneous stimulation trials for a variety of patients with low back pain.

MECHANISM OF ACTION

The basic scientific background of the initial spinal cord stimulation trials was the gate control theory by Melzack and Wall (1). Their theory proposed that stimulation of A-beta fibers modulates the dorsal horn "gate" and therefore reduces the nociceptive input from the periphery. Indeed, several studies demonstrated that dorsal horn neuronal activity caused by peripheral noxious stimuli could be inhibited by concomitant stimulation of the dorsal columns (3). However, it seems that other mechanisms may play a more significant role in mechanisms of spinal cord stimulation's action (4, 5).

Many animal studies showed a suppressive effect of spinal cord stimulation on tactile allodynia, which is mediated via A δ fibers and represents the state of central hyperexcitability (6, 7). Since allodynic animals seem to have lower extracellular levels of gamma-aminobutyric acid (GABA), one of the proposed mechanisms of spinal cord stimulation action involves increased dorsal horn inhibitory action of GABA (8-10). In those studies, intrathecal administration of the GABA_B agonist baclofen enhanced the antinociceptive action of spinal cord stimulation in an ani-

mal model, while GABA antagonists abolished the anti-allodynic effect of spinal cord stimulation. In humans, the intrathecal baclofen infusion produced significant augmentation of spinal cord stimulation effects (11). However, further studies are needed to clarify the beneficial effects of concomitant use of spinal cord stimulation and intrathecal GABA_B agonists for the treatment of certain forms of neuropathic pain syndromes.

Other putative mechanisms may also be responsible for pain relief induced by spinal cord stimulation. Recent animal and human studies revealed a potential role of adenosine in mechanisms of action of spinal cord stimulation. Intrathecal administration of adenosine_A receptor agonist was found to have a potentiating effect with spinal cord stimulation and also a synergistic effect with baclofen (4). Furthermore, the disinhibition of descending analgesia pathways originating in periaqueductal gray and/or the release of serotonin and substance P might explain the mechanism of action of spinal cord stimulation (12, 13).

Spinal cord stimulation may also abolish peripheral ischemic pain by rebalancing the ratio of oxygen supply and demand and thus preventing ischemia (5). At low levels of stimulation, spinal cord stimulation may act by suppressing the sympathetic activity via α -adrenoreceptors. However, at increased levels of stimulation, the nitric oxide-dependent release of calcitonin gene-related peptide may play a significant role in inducing vasodilatation (14). This might also explain the better survival of skin flaps during spinal cord stimulation (15). On the contrary, Kemler et al (16) reported that the use of spinal cord stimulation was not associated with increase in peripheral blood flow.

Patients with chest pain due to refractory angina pectoris respond well to spinal cord stimulation. Many possible explanations exist for spinal cord stimulation's mechanism of action in myocardial ischemia. The most likely mechanism for pain relief consists of redistribution of the coronary blood flow from regions with normal perfusion in favor of regions with impaired myocardial perfusion (17). This anti-ischemic effect of spinal cord stimulation was shown by coronary blood flow measurements and positron emission tomography. Other lines of evidence show that modulation of the intrinsic cardiac nervous system might contribute to the therapeutic effects of spinal cord stimulation in patients with angina pectoris (18). In this proposed mechanism, spinal cord stimulation may suppress the excitatory effects of myocardial ischemia on intrinsic cardiac neurons.

The effects of spinal cord stimulation on human brain activity were studied utilizing functional magnetic resonance imaging (MRI). The spinal cord stimulation produced increased activity in the human somatosensory cortex (SI and SII areas), contralateral to the side of pain and cingulate gyri. The somatosensory cortex activation becomes more pronounced with increased spinal cord stimulation activity (19). These brain areas activated by spinal cord stimulation correspond to CNS pain pathways involved in processing of somatosensory (SI, SII) and affective components (cingulate gyri) of pain. Further research may better define the role of higher CNS structures during spinal cord stimulation.

ANATOMY AND HARDWARE

For chronic low back/low extremity pain treatment, the spinal cord stimulation electrode leads are generally placed in the thoracic epidural space, with a lead tip location at the T8-10 level. An electrical field from the leads reaches the dorsal column of the spinal cord and modulates its pain transmission. The anatomical position of the spinal cord stimulation lead is critical for “coverage” and, subsequently, pain relief. Holsheimer et al measured the dorsal CSF layer thickness in thoracic areas corresponding to spinal cord stimulation electrodes’ placement and correlated results

with paresthesia perception from spinal cord stimulation coverage (20). They concluded that thickness of the dorsal CSF layer is the main factor determining the perception threshold and paresthesia coverage in spinal cord stimulation. In other words, an increasing thickness raises the threshold and reduces the coverage and vice versa. In the same study, the effects of an asymmetrical electrode position with respect to the spinal cord midline were also analyzed by computer modeling. The authors concluded that a lateral asymmetry of less than 1 mm gives a significant reduction of perception threshold and may result in unilateral spinal cord stimulation coverage.

The same group of investigators using MRI found that spinal cord midline and vertebral midline are apart by at least 1 to 2 mm in all levels investigated in 40% of patients. Further, Bartolat et al found that only 27% of paresthesia was felt symmetrically when the stimulating contacts were perfectly located at the radiological midline (21). Consequently, adequate symmetrical spinal cord stimulation coverage of the low back and lower extremity is in many cases difficult to achieve.

The permanent spinal cord stimulation hardware consists of a spinal cord stimulation lead, an extension cable, a power source, and a pulse generator (Figs. 1 and 2). Many



Fig. 1. Spinal cord stimulation lead, an extension cable, a power source/ pulse generator and patient programmer

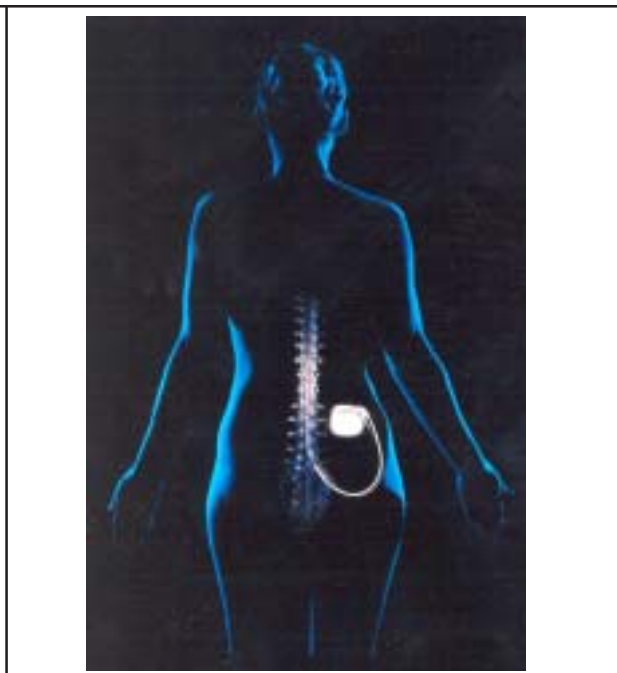


Fig. 2. Spinal cord stimulation lead, an extension cable, a power source/ pulse generator surgically placed

leads contain a removable stylet, which eases lead steering during implantation. The lead design varies in the number of electrodes from four to eight. The distance between the electrodes and the length of the leads also can differ. It is not clear if an increased number of electrodes provides better coverage, but it might be beneficial in case of lead migration. The leads with minimal space between electrodes (such as the Medtronic Quad® compact lead) might be better suited for isolated axial low back pain without a radiating component to the lower extremity. There are two types of pulse generators: (a) the completely internal pulse generator containing a battery; and (b) an internal pulse generator supplied by external power through the radiofrequency antenna applied to the skin. The implanted pulse generator is more convenient to use and can be easily adjusted by the patient using a small telemetry device. Patients can turn the stimulator on and off, and control the stimulation amplitude, frequency and pulse width. A separate external programmer allows for more complex internal pulse generator reprogramming by the physician. In case of inadequate stimulation, the physician can change polarity and number of functioning electrodes in order to provide better stimulation coverage. The batteries have to be changed every 3 to 6 years, which requires a brief visit to the operating room. The battery life depends on the time the stimulator is used and the stimulation amplitude. The externally powered internal pulse generator has an advantage over the implanted one in patients requiring higher amplitudes of stimulation, which would otherwise deplete the implanted batteries in a short period of time.

The permanent spinal cord stimulation implant can be achieved by placing the percutaneous lead via epidural needle or “paddle” lead via open laminotomy. The configuration of spinal cord stimulation electrodes varies in these two techniques. Percutaneous electrodes are the same configuration as the ones used for the stimulation trial. Paddle electrodes are larger and can be anchored directly to the dura, potentially minimizing migration.

RATIONALE

Spinal cord stimulation is not a neurodestructive procedure as opposed to neuroablation. Its effects are easily reversible. The relatively low invasiveness of a spinal cord stimulation trial (comparable to an epidural catheter placement), makes spinal cord stimulation the treatment of choice for certain forms of low back pain. In the long term, this treatment modality can be more cost effective than conservative treatment options (Table 1). Many stud-

ies have confirmed good outcomes of spinal cord stimulation for low back pain and highlighted its advantages over re-operation.

INDICATIONS

Axial vs. Radicular Pain

Generally, patients with radicular pain to the lower extremities seem to respond better to spinal cord stimulation than patients with isolated axial low back pain (22-41). However, a few studies have shown that axial low back pain in combination with bilateral leg pain also responds well to spinal cord stimulation (27, 35, 42).

Low Back Pain and Lumbar Radiculopathy

Surgically naive patients who are poor candidates for surgery may respond well to spinal cord stimulation. The chronic radicular pain in these patients is commonly of neuropathic origin. In these patients, it is important to rule out other sources of pathology, eg, facet disease, sacroiliac arthropathy, internal disc disruption, piriformis syndrome, and/or myofascial pain, before choosing spinal cord stimulation. In some cases of lumbar radiculopathy, better outcomes might be achieved by placing the spinal cord stimulation lead directly through the neural foramina (retrograde lead placement) (38).

Failed Back Surgery Syndrome

Failed back surgery syndrome is the most common indication for spinal cord stimulation placement in the United States today (37). It is defined as persistent pain after attempted surgical treatment for low back pain. Failed back surgery syndrome occurs in 20% to 40% of the more than 200,000 American patients who undergo lumbar spine surgery each year (23). For patients who fail medical management, physical therapy and nerve blocks, spinal cord stimulation may be the treatment of choice. Many studies are supporting the role of spinal cord stimulation in these patients, emphasizing its advantages over re-operation (26).

Other Indications

Spinal cord stimulation has been shown to be beneficial in many other chronic pain conditions. The literature supports the use of spinal cord stimulation in complex regional pain syndrome, peripheral vascular disease, and ischemic heart disease (43, 44, 45). The use of spinal

Table 1. Five-year medical costs of spinal cord stimulation

	Actual Costs			Present Value of SCS Savings (Costs)*	
	Chronic Maintenance	SCS Internal	SCS External	SCS Internal	SCS External
Base case					
Average charges	\$82,630	\$80,000	\$74,060	\$840	\$6460
Medicare fees	\$49,780	\$49,680	\$46,010	(\$1140)	\$2500
Spinal cord stimulation payback					
Average charges		4.3 yr	3.2 yr	4.7 yr	3.4 yr
Medicare fees		5.0 yr	3.7 yr	5.5 yr	3.9 yr
Increase of 10% in clinical efficacy (from 46% to 56%)					
Incremental savings		(\$4290)	(\$5060)	\$3990	\$4690
Charges					
Incremental savings - costs		(\$2510)	(\$3000)	\$2300	\$2760
Spinal cord stimulation payback (at 56% efficacy)					
Average charges		3.5 yr	2.5 yr	3.8 yr	2.6 yr
Medicare fees		4.1 yr	3.0 yr	4.5 yr	3.1 yr
Spinal cord stimulation payback (at 100% efficacy)					
Average charges	\$82,630	\$50,540	\$38,340	\$27,970	\$39,340
Medicare fees	\$49,780	\$32,950	\$25,100	\$14,230	\$21,550
Spinal cord stimulation payback (at 100% efficacy)					
Average charges		1.4 yr	1.0 yr	1.5 yr	1.0 yr
Medicare fees		2.1 yr	1.4 yr	2.1 yr	1.4 yr

* Present values are calculated assuming a 5% real discount rate, published in 1997. Adapted and modified from Bell et al (36). SCS – spinal cord stimulation

cord stimulation in postherpetic neuralgia, diabetic neuropathy, deafferentation pain and spinal cord injury pain is controversial.

Contraindications

Severe psychiatric diseases present major contraindications for spinal cord stimulation implantation and psychological evaluation of the candidate patient is recommended before implantation. Infection, drug abuse and coagulopathies are also contraindications for spinal cord stimulation placement. One should use caution in spinal cord stimulation placement in patients with thoracic spinal canal stenosis. This applies in particular to dual-lead systems.

TECHNIQUE

Implantation Technique

The patient is placed in prone position, with a pillow under the abdomen, to facilitate approach to the epidural space. Both trial and permanent implantation are performed under local anesthesia with light intravenous (IV) sedation as needed. Most common entry sites for the lumbar area are the T12/L1 or L1/2 spinal interspaces. Anteroposterior fluoroscopic images are obtained, making sure that the spinous processes are placed midline to the pedicles. The needle entry site is just lateral to the spinous process. The epidural space is identified by the loss-of-resistance

technique. It is recommended that the lateral fluoroscopic views be checked during needle insertion, in order to assess needle depth. The spinal cord stimulation lead is inserted in the epidural space under continuous fluoroscopic guidance. The curved lead tip can facilitate the desired lead positioning and treading. The goal is to position the lead midline to the spinous process fluoroscopic image or to its lateral margin if unilateral coverage is intended. Further, lateral positioning of the spinal cord stimulation lead can cause lead dislodgment to the lateral or anterior epidural space and, therefore, inadequate coverage. Once adequate lead position is obtained, trial stimulation is performed. It is important that stimulation paresthesias provide at least 70% to 80% overlap with the patient's pain location.

Permanent stimulator placement technique is similar to the trial. While the trial is usually done in the pain clinic setting, permanent spinal cord stimulation placement is reserved for the operating room. Under local anesthesia and IV sedation, a skin incision is made along the lumbar insertion site where the stimulator lead is placed and anchored to the skin. A separate subcutaneous pocket for a pulse generator is made in the gluteal or abdominal area. The spinal cord stimulation lead is then connected with the internal pulse generator by an extension cable tunneled under the skin. Finally, the skin and subcutaneous tissues are closed in layers.

Patients should avoid extreme activity for the first 6 to 8 weeks following permanent spinal cord stimulation implantation in order to prevent lead migration and allow for epidural scar tissue formation.

During trial and permanent lead implantation, care should be taken to obtain the best possible pain coverage ("sweet spot placement"). The spinal cord stimulation topographic coverage depends on the spinal level where the spinal cord stimulation lead tip is positioned. For low back pain and lower extremity pain, the T9-10 levels are recommended; however, there is high intersubject variations in these guidelines.

Stimulation Trial

A stimulation trial is warranted before proceeding with permanent spinal cord stimulation implantation. The percutaneous spinal cord stimulation trial is a minimally invasive procedure and can positively predict a long-term outcome in 50% to 70% of cases. The trial allows the patients to evaluate the spinal cord stimulation analgesic activity in their normal surroundings. The criteria for a

successful trial include at least a 50% pain intensity reduction, a decrease in analgesic intake and a significant functional improvement.

There is no consensus on technical approach and the length of a spinal cord stimulation trial. Minimal trial time should be 24 hours, although many centers perform 3- to 5-day trials. The initial inpatient trial allows for proper spinal cord stimulation adjustment, after which the patient is discharged home for several days of "home" trial. In cases of equivocal results, the trial time can be extended.

There are two technical approaches for spinal cord stimulation trials:

- ◆ Percutaneous Placement; Once the trial is completed, the lead is removed, and a new lead and internal pulse generator are placed (on separate occasions).
- ◆ Open Surgical Approach; The second approach is to tunnel and anchor the trial lead via surgical incision and to later internalize it for permanent spinal cord stimulation placement. This approach simplifies the final procedure and assures that stimulation coverage remains the same during both the trial period and permanent implantation. Its major disadvantage is the need for a second visit to the operating room for lead removal in case of an unsuccessful trial. The advantage of a percutaneous trial is its minimal invasiveness with a similar low risk of complications as in routine epidural catheter placement.

The percutaneous trial followed by lead placement via laminectomy is another less frequently utilized approach for spinal cord stimulation placement. In this case, a lead with wider electrodes is placed via laminotomy during permanent implantation. Wider electrodes might provide better coverage in certain patients and are less prone to migration in comparison to standard spinal cord stimulation leads (46).

CLINICAL EFFECTIVENESS

There is substantial scientific evidence on the efficacy of spinal cord stimulation for treatment of low back and lower extremity pain of neuropathic nature. Clinical studies have revealed success rates of from 50% to 70% with certain methods of spinal cord stimulation (22, 23, 24, 25). These studies have shown decreased pain intensity scores, functional improvement and decreased medication use with spinal cord stimulation treatment. The main drawback of neurostimulation is a decrease in its effectiveness over time,

seen in 20% to 40% of patients. It seems that this “tolerance’ to treatment is due to reorganization of the CNS (CNS plasticity) that takes place in neuropathic pain states. Anecdotal evidence suggests that not using the spinal cord stimulation continuously, eg, shutting it off overnight, may decrease the development of tolerance.

It has been documented that patients with failed back surgery syndrome respond better to spinal cord stimulation than the re-operation (26). Reported success rates in treating failed back surgery syndrome vary from 12% to 88%, with higher efficacy reported in recent studies (27, 28, 29). A systematic review of the literature related to spinal cord stimulation and failed back surgery syndrome by Turner et al (30) revealed that on average, 59% of patients had > 50% pain relief. The average complication rate in the same study was 42% but related to mainly minor complications (Table 2). Besides pain relief, spinal cord stimulation improves functional status in a significant number of patients, with a 25% return-to-work rate (27) and up to 61% improvement in activities of daily living (31). The reduced consumption of analgesics with spinal cord stimulation treatment varies from 40% to 84% in published reports (24, 32).

Certain psychological tests have been shown to predict outcomes in spinal cord stimulation treatment (33). Although spinal cord stimulation is an excellent treatment choice for patients with failed back surgery syndrome (34, 35), more studies are needed to further narrow down the patient selection criteria and improve long-term success rates.

OUTCOMES AND COST EFFECTIVENESS

Compared with the more conservative treatments, such as

medical regimens and physical therapy, spinal cord stimulation may appear costly. However, the overall cost can actually be lower than conservative management costs over time. If taken together, the cost of medications, emergency room visits, multiple physician visits, X-rays, and absence from work can easily surpass the cost of spinal cord stimulation implant. Bell et al have shown that for those patients for whom spinal cord stimulation is clinically efficacious, spinal cord stimulation pays for itself within 2.1 years (Table 1) (36).

COMPLICATIONS

The spinal cord stimulation complications can be divided into surgical complications and hardware complications. The most common surgical complication is infection. Wound hematoma and seroma are other commonly encountered surgical complications. Turner et al (30) performed a meta-analysis of spinal cord stimulation for failed back surgery syndrome publications and found reported a 5% incidence of infection and 9% incidence of other surgical complications. The authors also report that hardware complications include: lead migration (24%), lead failure (7%) and pulse generator failure (2%). While this analysis evaluated studies using old hardware systems, it seems that the rate of these complications is much lower currently. In our institution, we see much lower complication rates with spinal cord stimulation.

Surgical Complications

Bleeding at the internal pulse generator site (subcutaneous hematoma) is usually self-limiting and gradually reabsorbs in a few weeks. Frequent exam of the hematoma site is important, since hematoma can lead to infection.

Table 2. *Complications and hardware failure in spinal cord stimulation*

Cause	Annual Incidence (%)	Medicare Fee	Average Charge
Lead repositioning (outpatient procedure; first 3 years)	14.0	\$1475	\$2700
Lead replacement (inpatient procedure)	7.3	\$3200	\$5450
Infection requiring system removal and replacement (inpatient procedure; first year)	5.0	Itrel - \$11,950 X-trel - \$10,650	Itrel - \$19,600 X-trel - \$15,050
Generator failure and replacement (inpatient procedure)	3.1	Itrel - \$8,450 X-trel - \$4,550	Itrel - \$13,150 X-trel - \$7300

Adapted and modified from Bell et al (36).

Antibiotic prophylaxis regimens for spinal cord stimulation vary. The minimal prophylaxis should consist of preoperative antibiotic coverage, eg, cefazolin 1 g IV. However, at many institutions, prophylactic antibiotics are given up to 10 days postimplantation. Obtaining a CBC with differential urine analysis and sedimentation rate can further decrease the risk of infection by excluding patients who have any laboratory sign of infection. Usual signs of post procedural infection are increased temperature and tenderness at the incision site. Redness, swelling, and discharge at the insertion site can also occur. If infection occurs at the internal pulse generator insertion site, one should make sure to first aspirate the site for cultures before initiating antibiotic coverage and removing the hardware.

Inadequate Coverage or Spinal Cord Stimulation Malfunction

In case of spinal cord stimulation malfunction, one should obtain AP and lateral fluoroscopic images of the spinal cord stimulation lead tip, internal pulse generator and all connections to rule out lead migration, breakage or disconnection. If the cause is not found by fluoroscopy, one should analyze the internal pulse generator using the programmer. The battery status and impedance of each electrode in relation to the internal pulse generator should be checked. If two electrodes have exactly the same impedance, there might be a short circuit between them, most commonly located at the connector or internal pulse generator site. Some mechanical failures might require surgical revision and replacement of affected spinal cord stimulation components.

Decrease in Stimulation Amplitude

The decreased stimulation threshold can be caused by intrathecal migration of the spinal cord stimulation lead. If migration stays unnoticed, it can lead to serious complications such as spinal cord injury. This complication seems to be most common in patients with significant spinal canal stenosis. If intrathecal migration is suspected, the MRI of targeted spinal level should be obtained before anticipated spinal cord stimulation placement.

CONTROVERSIES

Single- vs. Dual-Lead System

Adequate relief of axial low back pain using spinal cord stimulation remains a challenge. It is not clear if spinal cord stimulation is indicated for isolated axial low back

pain or only for axial low back pain combined with lower extremity pain. If the goal of spinal cord stimulation is to cover low back pain and bilateral lower extremities pain, single- or dual-lead systems should be considered. Utilizing a dual-lead system can potentially provide “deeper” electrical field penetration in the dorsal column and therefore provide better axial low back pain coverage (42, 46). On the other hand, North et al (47) have shown that there is no advantage in using the dual over single lead for axial low back pain and that a failure rate is higher in dual electrodes (35).

Four vs. Eight Electrode System

Both four and eight electrodes were shown to be effective in treatment of low back and lower extremity pain, with no apparent advantages of one system over the other. Even though it seems that eight electrodes may have the potential advantage in case of lead migration, this has yet to be shown in clinical trials.

Internal vs. External Power Source

An internalized, fully implanted power source offers apparent advantages. It is more convenient for the patient to use, it is aesthetically more appealing, and it does not require frequent external battery changes. However, in certain situations, the external power source can be indicated. This applies to all cases where high amplitudes of stimulation are needed during the trial phase. In particular, the required stimulation amplitude should be monitored when dual-lead systems are used. Dual-lead systems tend to empty batteries faster than one lead system even at modest stimulation amplitudes; and if an internal power source is used in such cases, these patients may require frequent battery replacements.

Percutaneous vs. Laminectomy Approach

Percutaneous placement of the spinal cord stimulation lead is a less invasive procedure, minimizing immediate complications and requiring less operating room time. Since percutaneous electrodes are placed under monitored anesthesia care, adequate spinal cord stimulation coverage can be confirmed during the permanent implantation, making it a significant advantage over laminectomy style electrodes, which are generally placed under general anesthesia, eliminating the patient’s feedback on stimulation coverage.

On the contrary, laminectomy electrodes provide several

advantages over percutaneous placed ones (48):

- ◆ They are anchored to the dura with minimal chance of migration (49, 50, 51).
- ◆ They are in closer contact with epidural space, and they do not cause unnecessary posterior epidural space stimulation.

CARDIAC PACEMAKERS AND SPINAL CORD STIMULATION

The interference and inhibition of the cardiac pacemaker can be caused by spinal cord stimulation. However, spinal cord stimulation can be used in a patient with a pre-existing pacemaker if certain precautions are taken:

- ◆ Both devices should be programmed in bipolar mode;
- ◆ The spinal cord stimulation frequency should be set at 20Hz;
- ◆ Each spinal cord stimulation programming should be performed using continuous ECG monitoring. More importantly, the manufacturer's recommendations should be strictly followed, and the input of a cardiologist is recommended.

CONCLUSION

Spinal cord stimulation is an excellent treatment modality for carefully selected patients with low back and lower extremity pain. It may be a treatment of choice for patients with failed back surgery syndrome. The main advantages of spinal cord stimulation are its minimal invasiveness, reversibility and convincing studies to justify its use. In well-selected patients, spinal cord stimulation is cost effective in comparison to conservative treatment approaches. However, further studies are still needed to better identify patient selection criteria for spinal cord stimulation.

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