SPECT. See COMPUTED TOMOGRAPHY, SINGLE PHOTON EMISSION.

SPECTROFLUORIMETRY. See FLUORESCENCE MEASUREMENTS.

SPECTROPHOTOMETRY. See COLORIMETRY.

SPEECH REHABILITATION. See LARYNGEAL PROSTHETIC DEVICES.

SPINAL CORD STIMULATION

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INTRODUCTION

Spinal cord stimulation (previously known as dorsal column stimulation) is a minimally invasive technique used primarily to treat chronic, refractory neuropathic pain. It is

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based upon Melzack and Wall's gate control theory (1), and was first introduced by Shealy in 1967 (2). Neuropathic (nerve injury) pain has many etiologies, including trauma, stroke, diabetes, infection [e.g., human immunodeficiency virus (HIV), or shingles], and cancer. Unfortunately, nerve injury pain can be extremely difficult to manage. Many types of therapy have been used for neuropathic pain including medications such as antiinflammatories, opiates, and antiepilepsy drugs. Physical therapy and psychologically based approaches have also been tried with variable success. Spinal cord stimulation (SCS), transcutaneous electrical nerve stimulation (TENS), and peripheral nerve stimulation (PNS) are all forms of neuromodulation that are used for nerve injury pain.

Spinal cord stimulation is typically reserved for patients with refractory neuropathic pain, whereas deep brain stimulation is currently used for patients with movement and some pain disorders. In SCS, a lead is percutaneously inserted into the epidural space, and an electric field is applied in the vicinity of the spinal cord. The electric field depolarizes neural elements or in some way modifies the function of the nervous system. The goal is for the patient to experience a pleasant paresthesia, often described as "tingling", in the area of their pain. After an initial successful trial, a permanent stimulator can be implanted that the patient controls with a hand-held device.

This article reviews the equipment used in spinal cord stimulation, patient selection, and the possible mechanisms of this therapy. The process of inserting a stimulator will be described with possible complications, and the effectiveness of this therapy will be analyzed. Finally, possible future uses of SCS will be discussed.

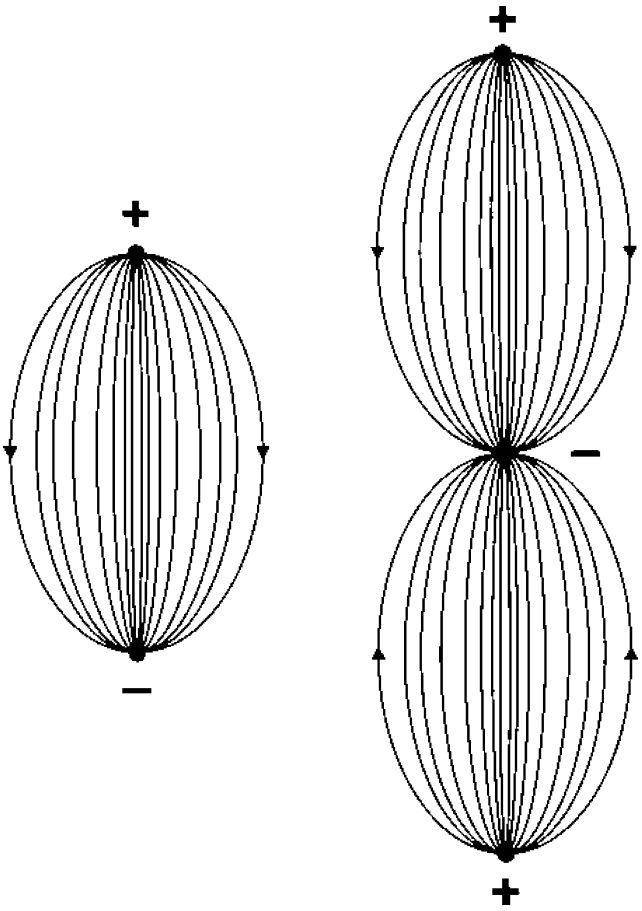

Figure 2. Current distribution between electrodes on SCS leads.

EQUIPMENT

Medtronic, Inc. (Minneapolis, MN), Advanced Neuromodulation Systems, Inc. (Allen, TX) and Advanced Bionics (Valencia, CA) are the primary manufacturers of spinal cord stimulators. There are two types of implantable leads available: the paddle (surgical) lead and the tubular percutaneous lead (see Fig. 1). The percutaneous lead can have four or eight contact points (electrodes), whereas the surgical leads are available with two, four, or eight electrodes. Each electrode can be programmed to function as an anode or cathode for the electrical current used in stimulation (see Fig. 2). The paddle lead is shielded on one side, such that stimulation is produced only on the side with the electrodes (see Fig. 3). This finding has the advantage of directing the entire electrical field toward the spinal cord, as opposed to the percutaneous lead that produces an electrical field circumfrentially around the lead, including away from the spinal cord. Hence, the paddle lead can produce SCS at lower amperage, prolonging battery life. The surgical lead also has the potential advantage of greater stability (less likely to move postimplantation) as it is sutured to surrounding tissue (3). However, the paddle lead requires a minilaminotomy, whereas the percutaneous lead is placed less traumatically via a 15 gauge touhy needle.

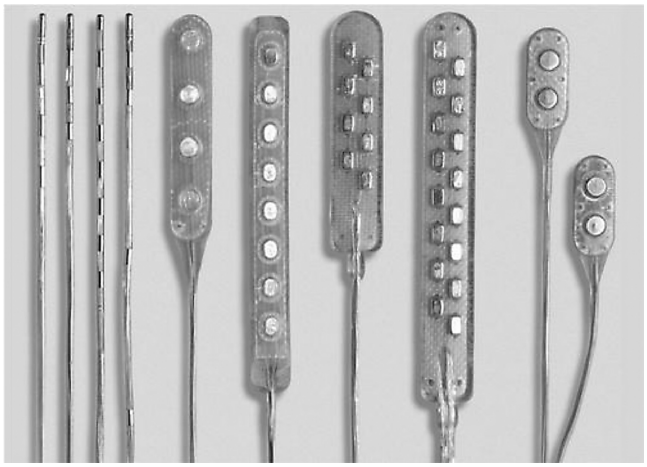

Figure 1. Various percutaneous and paddle leads. (Used courtesy of ANS, Inc.)

The percutaneous lead is made of inert polyurethane with an outside diameter of ~1.3 mm. On its distal end, it has four or eight electrodes made of platinum iridium. These are spaced 4, 6, or 12 mm apart. The electrodes are 3–6 mm long. The plate lead has a two- or four-midline circular or eight parallel rectangular electrodes. There are several options to provide current to the electrodes. An implantable pulse generator (IPG) can be placed subcutaneously (usually in the low abdomen), similar to a

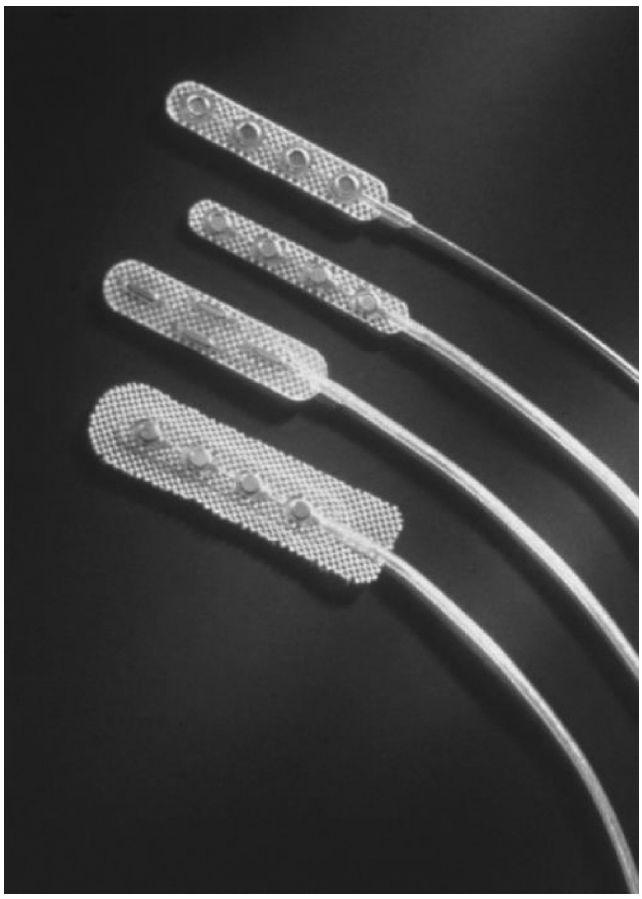

Figure 3. Examples of surgical (paddle) leads. (Used courtesy of Medtronic, Inc.)

pacemaker generator. Recently, a rechargeable IPG has become available. Depending on use, the Pt will percutaneously recharge the IPG every few days to weeks. Finally, a radio frequency (rf) receiver can be placed subcutaneously and powered by an external rf transmitter coil that is held over the device (see Fig. 4). In either case, a cable is tunneled subcutaneously from the power source to the lead in the spine.

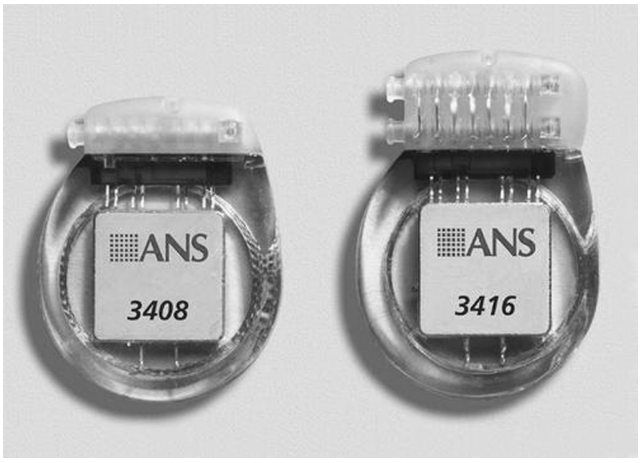

Figure 4. Implantable rf receivers. (Used courtesy of ANS, Inc.)

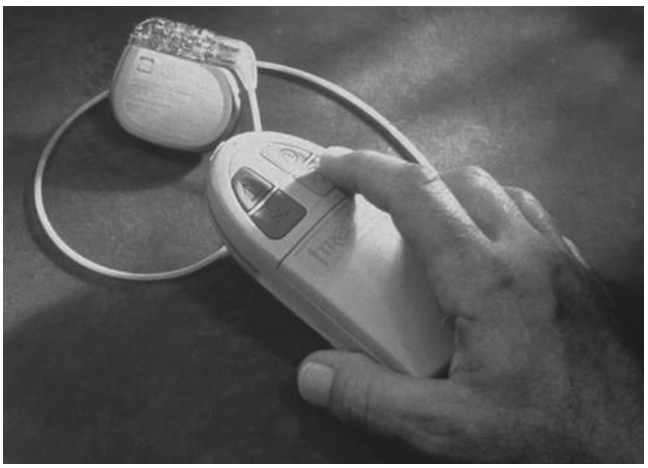

Figure 5. Implantable IPG with hand-held controller. (Used courtesy of Medtronic, Inc.)

The IPG provides pulses of electrical current that are rectangular and biphasic. Programmable features include pulse width, amplitude, and rate. Rates generally used are 30–80 Hz, amplitude is 0–12 V, and pulse width is 200–450 μs. Battery life varies depending on rate, amplitude, and time the device is used. Unless the patient uses the stimulator constantly with high rate and amplitude, the battery should last at least several years. One obvious advantage of an rf receiver system is that there is no need to periodically replace the battery. The energy source of the IPG is a hermetically sealed silver vanadium oxide cell. The power source and electronics are sealed in an oval shaped titanium shield (see Fig. 5).

PATIENT SELECTION

As noted earlier, spinal cord stimulators are used primarily for neuropathic pain. Although there are many types of neuropathic pain, chronic unilateral lower extremity neuropathic pain seems to respond best to this type of therapy. A typical patient may have the failed back surgery syndrome with residual leg pain, or the patient may have some type of neural compressive lesion that is not operable and refractory to medical management. Spinal cord stimulation may also be indicated for chronic arachnoiditis, complex regional pain syndrome, peripheral neuropathy of the lower limb, and phantom limb syndrome. Patients with idiopathic pain, mechanical low back pain, or other forms of nociceptive pain have a lower success rate when compared to those with neuropathic conditions. Clinical experience and studies have shown that SCS is most efficacious when the entire painful area is covered with paresthesia. The more diffuse the patient's pain, the more difficult it will be to cover with SCS.

Selection for SCS often includes psychological screening. Patients with untreated depression, anxiety, or drug abuse issues are not good candidates. Obviously, the patient needs to be able to understand how to use the stimulator. A trial of stimulation is probably the best predictor of long term success (4). Although the value of

psychological testing in predicting success with SCS is controversial, there is no question that patients with chronic pain are best managed with a multidisciplinary approach. This may include physical therapy, psychological and spiritual support, medications, and surgical procedures as indicated.

MECHANISMS

Both animal and human research studies have provided a partial understanding of the mechanisms of SCS (5). Melzack and Wall's gate control theory (1) suggested that stimulation of large cutaneous A-β fibers would inhibit nociceptive input from the smaller A-δ and C fibers. Since SCS has been shown to be more effective for neuropathic pain than nociceptive pain, the mechanism must include more than simple inhibition of nociceptive input. Endorphins or other endogenous opiates do not seem to be involved. In patients with ischemic lower extremity pain or refractory angina, the mechanism of SCS appears to be an increased local blood flow (i.e., microcirculation). This may be due to both inhibition of the sympathetic nervous system and activation of vaso-active chemicals (6).

Animal studies have supported the contention that A-β fiber stimulation is one of the mechanisms of SCS. Animal models of neuropathic pain can be created by lesioning the sciatic nerve, which creates tactile allodynia in the animal, a phenomenon mediated by A-β fibers. Spinal cord stimulation has been seen to suppress this sign. Another effect of SCS is on wide-dynamic range neurons. Wide-dynamic range (WDR) neurons are second order neurons in the dorsal horn of the spinal cord. They receive input from a variety of sensory neurons. In the face of continuous stimulation from injured neurons, the WDR neurons will "wind-up," that is, fire at lower depolarization thresholds. Spinal cord stimulation may decrease this WDR response while simultaneously decreasing the central excitatory neurotransmitters glutamate and aspartate. γ-Aminobutyric acid (GABA), a central inhibitory neurotransmitter, is simultaneously released; therefore, SCS may have beneficial effects on both excitatory and inhibitory pain mechanisms (7).

Recent computer modeling of SCS has led to a greater theoretical and empirical understanding of the interaction of current with spinal structures (8). These models demonstrate how the depth of cerebral spinal fluid and the distance of the electrodes from both the dorsal columns and dorsal roots can affect the patient's paresthesia perception.

IMPLANTATION TECHNIQUE

Before a spinal cord stimulator is implanted, the patient needs to be informed of potential risks. These include infection, bleeding, nerve damage, allergic reaction, and failure of the stimulator to adequately cover or reduce the patient's pain. The patient may experience swelling around the site of the generator and a seroma may develop requiring drainage. If the lead or the generator becomes infected, it may have to be removed. Lead displacement, fracture, or movement can occur such that an initially adequate pattern of stimulation becomes inadequate. Lead and battery revision may become necessary at some point. After placement of a SCS, the patient is instructed not to drive an automobile with the device turned on. Furthermore, they should not undergo a magnetic resonance imaging (MRI) scan or any type of diathermy.

Once consent has been obtained, a trial of SCS is performed. This consists of placing a trial lead in the epidural space, and if adequate coverage of the patient's area of pain can be achieved, allowing the patient to use the stimulator on an outpatient basis for 5–7 days. In 1993, Barolat et al. published a database of 106 patients in whom they had placed spinal cord stimulator leads (9). The electrodes were placed between the C1 and L1 spinal levels for chronic pain management, and the areas the patients felt stimulation were mapped. These maps provide a guideline as to which body areas will be stimulated by implanted electrodes. Barolat also noted that certain body areas were difficult to cover with paresthesia, including the low back, neck, and perineum. Clinical experience has shown that patients with bilateral extremity pain or pain in both the low back and legs may require bilateral lead placement to obtain adequate coverage. The placement of more than one lead in the epidural space allows not only wider paresthesia coverage, but also the use of complex stimulation programs that can be tailored to meet the patient's needs (see Fig. 6). With bilateral eight electrode leads, the possible stimulation combinations (anodes and cathodes) reach the thousands.

To insert the lead, the patient is placed in the prone position and sedated. The operative area is sterilely prepped and draped, and the skin is anesthetized with local anesthetic. When treating lower extremity pain, the puncture site is usually at the L1-2 level. The epidural space is entered with a touhy needle, through which a lead is advanced in a cephalad direction. Using fluoroscopy, the lead is observed to move up the spinal canal until it reaches approximately the T9–T10 level. The lead can be manipulated to direct it slightly to the side corresponding to the patient's pain. For upper extremity pain, the skin is usually punctured at T1–T2, and the tip of the lead is placed at the C3–C4 level. The presence of scar tissue or other anatomic barriers can make lead placement difficult and occasionally impossible.

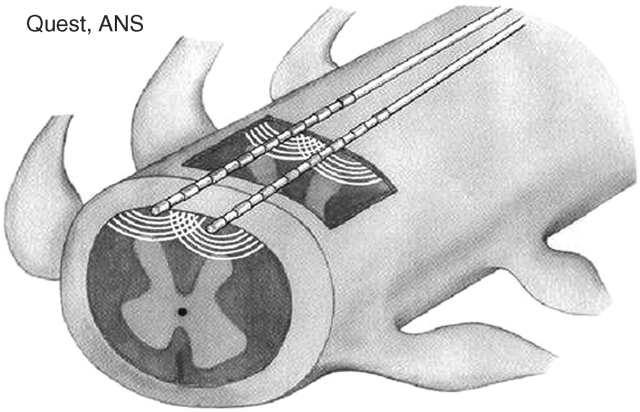

Figure 6. Dual leads allow winder parasthesia coverage. (Used courtesy of ANS, Inc.)

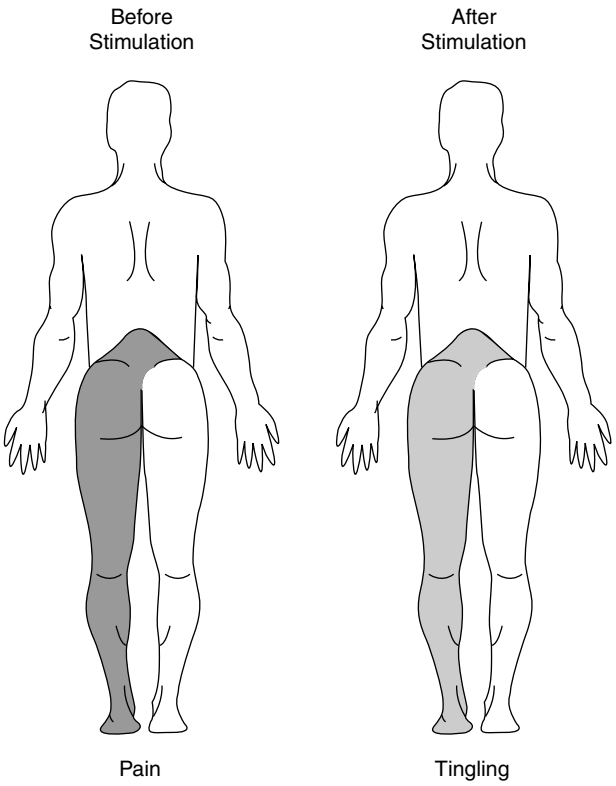

Figure 7. SCS covers painful area with pleasant paresthesias. (Used courtesy of Medtronic, Inc.)

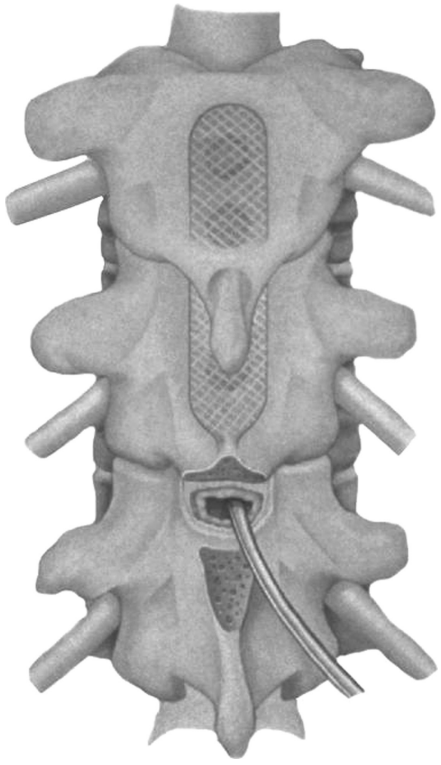

Figure 8. Paddle lead surgically inserted into the epidural space. (Used courtesy of Medtronic, Inc.)

Once the lead is felt to be in proper position, it is attached via a cable to the trial generator. This is an external programmer that allows various combinations of electrodes to be stimulated in an effort to cover the patient's pain with pleasant paresthesia, usually described as "tingling" (see Fig. 7). If the amplitude is set too high, the patient may experience discomfort or muscle stimulation. The patient must be awake enough at this point to answer questions and describe where they feel the stimulation. It is not unusual to need to adjust the position of the lead(s) several times before adequate coverage is obtained.

Once adequate coverage is obtained, the trial lead is secured with tape and/or suture. After recovery from anesthesia, the patient is given instructions as how to operate the stimulator. The patient is allowed to turn the device on or off, and can adjust the amplitude and rate to comfort. Reprogramming the pulse width and lead combinations is generally reserved for the pain specialist. The patient is told not to drive a car with the stimulator turned on, and excess twisting or raising the arms above the head is discouraged. As noted above, the patient will return to the clinic for removal of the trial lead in 5–7 days; however, the patient is encouraged to call sooner should anything change with the function of the device.

During the follow-up visit, several decisions are made. The patient is asked if the stimulator continued to cover their painful area, and if so, did it reduce the discomfort. Ideally, the patient obtained at least a 50% reduction in their pain during the trial. If the patient has received significant pain relief and they want to proceed with permanent implantation, the type of lead (surgical vs. percutaneous) is selected. Placement of the surgical lead requires a minilaminotomy, which includes removal of part of the inferior portion of the lamina and a portion of the *ligamentum flavum*, followed by insertion of the paddle lead into the exposed epidural space (see Fig. 8). Villavicencio et al. followed 27 patients who underwent placement of SCS leads (3). Patients who had electrodes placed via a laminectomy had significantly better long-term effectiveness than the patients with percutaneous leads. Nonetheless, permanent placement of percutaneous leads remains a viable and effective option that does not require a minilaminotomy. The placement of the permanent SCS lead requires that a generator (IPG) be inserted in a subcutaneous pocket, usually in the low abdomen or over the buttock. A cable is tunneled under the skin to the lead inserted in the spine. After permanent implantation, the spinal cord stimulator is controlled with a hand-held device.

OUTCOMES

A number of studies have looked at outcomes after SCS. Van Buyten et al. described 10 years of experience with SCS in 254 patients, 217 of whom had permanent stimulators placed (10). Before the study began, 10% of the patients had died and another 10% had undergone explantation. Reasons for explantation included ineffectivity (4.6%), infection, allergy, and recovery from pain. An independent review of the remaining patients who could be contacted and would participate in the study ($n = 123$) showed that 68% of them graded the treatment as excellent to good (excellent, very good, good, moderate, weak, no

improvement, worse). After excluding retirees and others not pursuing a career, 31% of the patients who had been working before their pain began had returned to work. The authors noted that their success rate is one of the highest reported.

Kay et al. published another retrospective study of SCS covering 13 years (11). Of 70 patients treated with SCS, there were 72 surgical revisions, including electrode (32), connecting cable (6), or generator revision (22). Battery depletion was the single most common indication for generator revision (16/22). Of the 72 revisions, 12 were for explantation. Of 48 patients who responded to a questionnaire, 60% rated their pain relief as substantial (>50%).

Bhdrakant et al. prospectively studied 29 patients over a 2-year period (12). The primary indication for SCS was failed back syndrome. Four of the 29 failed to obtain relief during the SCS trial. Of the 25 patients with permanent implants, SCS was beneficial in 50%. This result is similar to North et al. results for a large series ($n = 320$), where 52% of patients reported at least 50% pain relief (13).

Although these and other studies support the use of SCS for certain chronic pain syndromes, methodological problems preclude drawing final conclusions. Its retrospective nature, lack of controls, and heterogeneous patient populations flaw much of the research on SCS. More prospective studies, perhaps looking at SCS for individual pain syndromes, will be needed before this expensive technology becomes widely accepted.

FUTURE USES OF SCS

Greater understanding of both peripheral and central pain mechanisms combined with evolving technology have expanded the potential uses of SCS. Multiple-electrode configurations have allowed coverage of diffuse pain generators and made reprogramming simple when coverage is lost or new pain symptoms arise. Lumbosacral placement of SCS leads may allow treatment of refractory pelvic neuropathic conditions including sacral neuralgia, vulvodynia, or coccydynia. Urinary incontinence may also be treatable with this technique (14). Peripheral placement of SCS leads has been used for a number of conditions, including occipital neuralgia (i.e., spinally transformed migraine) and trigeminal neuralgia (15,16).

Other current and evolving uses for SCS include chronic regional pain syndromes (RSD and causalgia), postherpetic neuralgia, and postamputation pain. Patients with peripheral vascular disease suffering from rest and night pain seem to benefit from SCS; this indication is used more commonly in Europe than the United States. Spinal cord stimulation has also been shown to be effective in refractory angina (17). Other conditions treated with SCS include severe Raynauds phenomena, Buerger's disease, and diabetic neuropathy.

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See also BIOELECTRODES; BLADDER DYSFUNCTION, NEUROSTIMULATION OF; ELECTRONEUROGRAPHY; FUNCTIONAL ELECTRICAL STIMULATION; PERIPHERAL VASCULAR NONINVASIVE MEASUREMENTS; TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION (TENS).