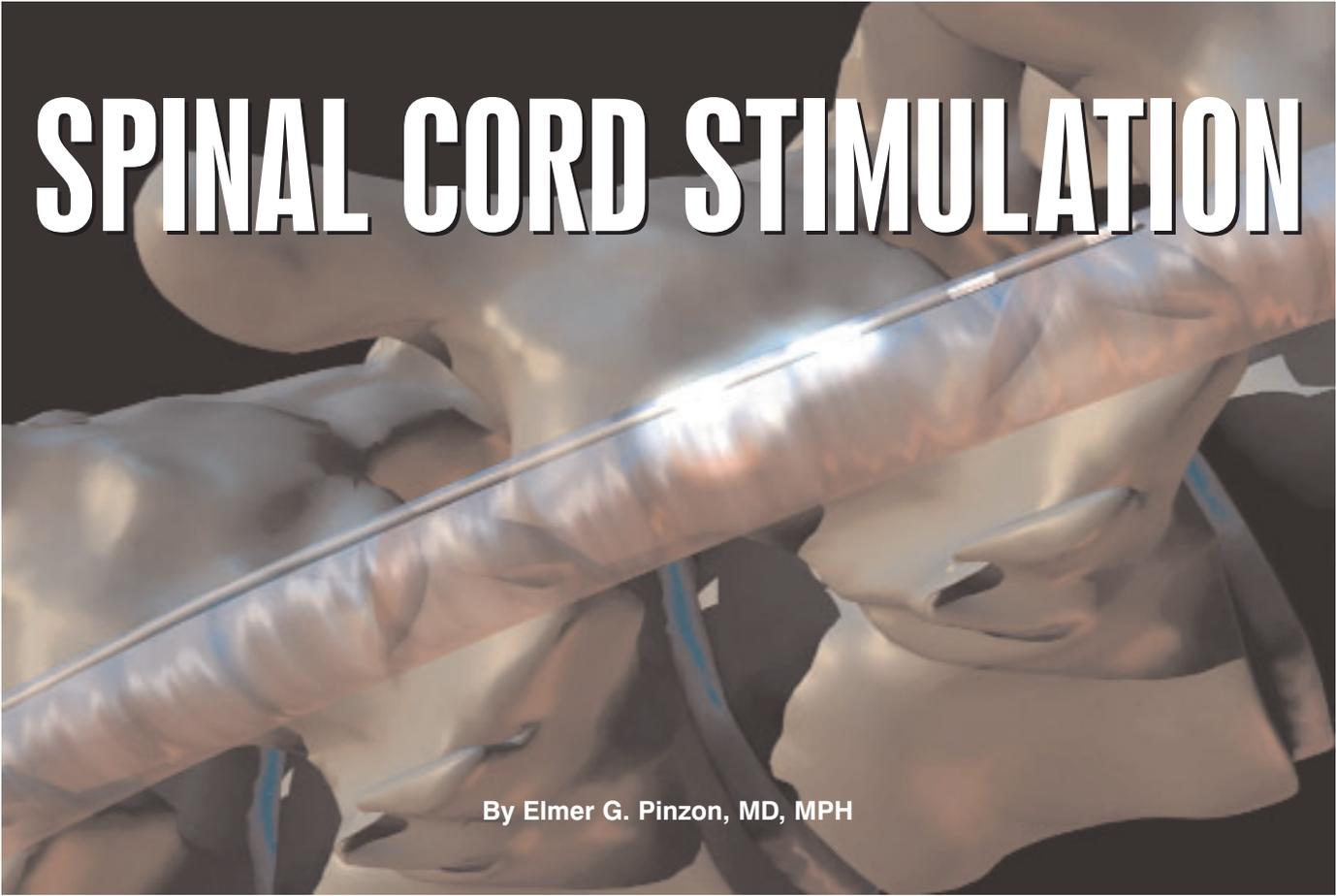# SPINAL CORD STIMULATION

By Elmer G. Pinzon, MD, MPH

An overview and case study of spinal cord (dorsal column) stimulation in a spine-centered/orthopaedic clinical practice setting.

Since the first published paper on spinal cord stimulation (SCS) (or dorsal column neuromodulation) by Dr. Shealy in 1967, there have been a cumulative total of well over 2500 articles, presentations, symposia, and abstracts on the topic of neuroaugmentation.[1,2] The long-term results of SCS published in the 1970's were disappointing, yet still provided promising outcomes.[3-6] Most of the studies published in the 1970's and early 1980's demonstrated success rates of approximately 40%.[7] As with many novel instrumentation devices, initial problems included poorly designed hardware, inadequate patient selection criteria, and suboptimal surgical technique. The hardware typically consisted of a single or dual electrode system that were implanted epidurally. They provided a small electrical field and thus were unable to consistently stimulate the spinal cord. In addition, these systems were implanted via laminectomy or laminotomy with the patient under general anesthesia, thus eliminating the possibility of surgeon-patient interaction. The electrodes were commonly implanted in the high thoracic or lower cervical region for lumbar pain syndromes and patients were not consistently screened for psychological dysfunction, drug habituation, secondary-gain issues, pain topography, and quality of pain. All of these factors have considerable impact on the overall efficacy of SCS, as we have seen the advancements of this technology over the years.[6,8-10]

Significant advances in SCS have been made in recent years. These results and postoperative outcomes of the procedures have shifted to more positive outcomes in the field of neuroaugmentation; especially with respect to more pertinent and practical factors, such as, return to work, reduction in medication use, reduction in visual analog pain scores (VAS), and improvement activities in daily living (ADL). The hardware is more durable, more effective, more maneuverable, and provides more range of coverage for the affected area for which it is aimed. The devices can be implanted percutaneously under fluoroscopic guidance (especially for the trial leads placement), which allows operator-patient verbal interaction and more accurate positioning of SCS leads, for trial and eventual permanent placement. Still the vast majority of SCS placements have

been for failed back surgery syndrome (FBSS), although this varies according to regional preference. Over three decades of experience have provided improved patient selection criteria; which is paramount in affecting a positive eventual outcome. The net result is an improved capability to control various chronic pain conditions, especially peripherally-referred more so than centrally-referred pain conditions.[7] This article will discuss the pathophysiology and mechanism of action and clinical applications of SCS, procedural indications/contraindications/ potential complications of SCS, SCS patient selection criteria, and current clinical results and potential future trends in dorsal column neuromodulation.[6,8-10]

Pathophysiology of Pain

Pain is an uncomfortable sensation associated with an emotional response.[11,12] The International Association for the Study of Pain (IASP) defined pain as "an unpleasant sensory and emotional experience associated with actual and potential tissue damage, or described in terms of such damage" (IASP, 1986).[11] It may originate from stimulation of chemical, mechanical, or thermal receptors found in free nerve endings within injured tissue. This is known as afferent pain, and can occur in ligamentous or muscular injuries of the spine.[13-16] Pain can also occur from direct injury to the peripheral nerve, which results in burning or shooting pain in the distribution of the affected nerve. This is called peripheral deafferentation (neuropathic) pain and is demonstrated in conditions such as complex regional pain syndrome, peripheral neuropathy, or radiculopathy.[5,17,18] Central deafferent pain appears after injury to the central nervous system structures, such as the thalamus, that are responsible for the transmission of pain. Peripheral pain signals are transmitted by either thinly myelinated A-delta or unmyelinated C fibers. The A-delta fibers convey discrete, sharp, fast pain at approximately 15 m/sec, whereas the C fibers transmit vague, chronic, burning, slow pain at less than 1 m/sec.[19,20]

In 1965, Melzack and Wall published their "gate control" theory in which they hypothesized that a "gate" system existed for pain modulation located in the dorsal gray horn within the substantia gelatinosa (laminae 2 and 3).[21] They proposed that excess tactile signals traveling along the large myelinated A-delta fibers closed the gate, which then inhibited the propagation of pain impulses along the poorly myelinated C fibers. Although the pain pathway is still not completely understood, researchers have uncovered important parts of the neuronal system. This includes descending inhibitory influences from the brain, which have been shown to suppress transmission of pain.[9,22-24] There is also evidence of an endogenous system of opioids that modulate sensory input.[25-27] Today, there is a better awareness that the pain experience is not just physiologic but is also influenced by culture, religion, and psychologial makeup.[28-31] In order to provide appropriate treatment all of these factors must be taken into consideration when evaluating patients.[6,8,9,10]

SCS Device Components
Pulse Generator
Leads
Transmitter

Mechanism of Action of Spinal Cord Stimulation

Although the exact mechanism for pain control from SCS is not entirely understood, it is believed to result from direct or facilitated inhibition of pain transmission.[2,4-6,21,32] There exist five mechanistic theories for SCS which should be noted: 1) Gate control theory— segmental, antidromic activation of A-beta efferents; 2) SCS blocks transmission in the spinothalamic tract; 3) SCS produces supraspinal pain inhibition; 4) SCS produces activation of central inhibitory mechanisms influencing sympathetic efferent neurons; 5) SCS activates putative neurotransmitters or neuromodulators.[32]

The gate control theory motivated Shealy, et al. in 1967 to apply SCS as a means to antidromically activate the tactile A-beta fibers through dorsal column stimulation.[2] Shealy reasoned that sustained stimulation of the dorsal columns would keep the gate closed and provide continuous pain relief. While the theoret-

ical model put forth by Melzack and Wall has been shown not to be precisely correct, pain gating or pain control has been shown to exist.[4-6,21]

Others believe that pain relief from SCS results from direct inhibition of pain pathways in the spinothalamic tracts and not secondary to selective large fiber stimulation.[33] This theory has been supported by Hoppenstein, who showed that the posterolateral stimulation of the spinal cord provided effective contralateral pain relief with substantially less current than posterior stimulation.[34]

Some investigators think that the changes in blood flow and skin temperature from spinal cord stimulation may affect nociception at the peripheral level.[15,16,35-37] This postulate is further supported in part by data from Marchand, et al. who investigated the effects of SCS on chronic pain using noxious thermal stimuli.[8,38-42] Since it was discovered that SCS causes vasodilation in animal studies, clinicians have used this modality for the treatment of chronic pain due to peripheral vascular disease and is the leading indication for SCS in Europe today.[32,39-42] The precise action of pain modulation by SCS is still in debate. A better understanding of the pain system may lead to more effective stimulators and allow for even greater success.

Indications and Contraindications for Spinal Cord Stimulation

Commonly-accepted indications currently used are labeled as per the ICD-9 CM codes:

- Post-herpetic neuralgia (053.19)
- Intercostal neuralgia (353.8)
- Post-laminectomy (thoracic region) syndrome (722.82) (i.e., failed back surgery syndrome)
- Post-laminectomy (lumbar region) syndrome (722.83) (i.e., failed back surgery syndrome)
- Cauda equina (chronic) injury syndrome (952.4)
- Chronic arachnoiditis (322.2)
- Complex regional pain syndrome (CRPS/RSD) of the upper limb (337.21)
- Complex regional pain syndrome (CRPS/RSD) of the lower limb (337.22)
- Complex regional pain syndrome (CRPS/RSD) of other specified site (337.29)
- Phantom limb pain syndrome (353.6)
- Cardiovascular angina/ischemic pain

(413.9)

- Atherosclerosis of the extremities with resting pain (i.e., PVD) (440.22)
- Brachial neuritis or chronic cervical radiculopathy (723.4)
- Thoracic or lumbosacral neuritis or chronic radiculopathy (724.4)
- Cervical nerve root injury (953.0)
- Thoracic nerve root injury (953.1)
- Lumbar nerve root injury (953.2)
- Other potential indications: chronic occipital neuralgia/cervicalgia, chronic pelvic pain, deafferentation pain, axial pain, thoracoabdominal aortic aneurysm, cerebral palsy, multiple sclerosis, and spinal cord injury, among others.

Commonly-accepted contraindications are as follows:

Absolute: sepsis, coagulopathy, previous surgery or trauma that obliterates the spinal canal, localized infection at the implantation site, spinal bifida.

Relative: physical and/or cognitive/psychological disability that interferes with proper usage of and understanding of the device, significant somatization/somatoform disorders, unmanaged substance abuse or cognitive disorders, lack of social support.

Patient Selection Criteria for Spinal Cord Stimulation

Proper patient selection is essential to the long-term success of a SCS system.[6,8,9,10]

Improper selection criteria was one of the principal reasons for suboptimal results reported in the 1970s. During the 1970's and early 1980's, most studies evaluating the long-term efficacy of dorsal column stimulation quoted success rates of ~40%. Technical advances leading to improved hardware coupled with improved patient selection, have improved the rate of long-term efficacy to ~70%.[2,3,7]

A SCS neuromodulation system should be considered for patients who have failed all reasonable conservative care including appropriate diagnostic, therapeutic and rehabilitative techniques, and have been given a reasonable period of time to recover from the condition.[7] An ideal patient should be motivated, compliant, and free of drug dependence.[44] Psychological screening is recommended but not mandatory to exclude conditions that presdispose to failure of the procedure. Diagnoses that are typical indications for this procedure include chronic radiculopathy, perineural fibrosis, neuropathic

pain, and complex regional pain syndrome.[19,34,45-47] In Europe, SCS is also used for peripheral vascular disease that is not amenable to medical therapy with excellent results have been reported.[39-42,48,49] In the United States, peripheral vascular disease is not an FDA-approved indication.

When considering pain topography, extremity pain responds better than axial pain, and the more distal the extremity pain the greater the clinical response.[50,51] Middle and upper lumbar pain as well as thoracic, cervical and chest wall pain are difficult to adequately control and maintain long term. Pain due to severe nerve damage that is superimposed on cutaneous numbness (i.e. anesthesia dolorosa) is also difficult to treat with SCS. Central pain syndromes do not respond to SCS and are best treated by other modalities.

The use of an outpatient percutaneous trial of between 3-7 days with a SCS system has been proven helpful in determining which patients will respond well enough to warrant a permanent SCS implantation and determine the future permanent implantation levels.[50-53] Absolute criteria that must be present for a patient to have a positive trial include tolerance of paresthesia, greater than 50-75% pain relief, and overall patient satisfaction. Relative requirements for a positive trial include improved functional level, reduced usage of pain medication, and reduced reliance on the healthcare system.

Postprocedure Care and Follow-Up Protocol

The patient undergoing a percutaneous trial SCS placement is routinely recovered after 30-60 minutes, in post-operative recovery setting.[6,8-10] Once in the recovery area and the patient is awake and alert, time should be spent to optimize the patient's SCS settings. The adjustable parameters of electrical stimulation in SCS are frequency (Hz), pulse width (stimulus duration), and amplitude (volts). A typical frequency of is 50-80 Hz although

higher frequency may be used as a stronger counter-stimulus. Increasing the pulse width increases the density of the stimulus, which provides for deeper penetration into the spinal cord. Clinically this usually means a broader disbursement of paresthesia. This may be beneficial when, for example, the stimulation pattern needs to cover the back but is only covering the hip. The pulse width can be increased and the paresthesia pattern may then incorporate the low back. The amplitude represents the electrical force of the stimulus. Clinically this usually means that the patient experiences a more dense stimulation pattern thus making it harder for the pain to "break through" the stimulation pattern. When the amplitude is adjusted too high, the patient may experience it as being noxious.

As long as the recovery period is uneventful then the patient is discharged home with post-operative instructions. During the recovery period, the SCS programming is fine-tuned, the patient and/or patient's family is educated on how to use the device, and any questions are answered. The patient is told to keep the SCS area clean and dry and specifically told not to bathe or shower but to take sponge baths during the trial period. Prophylactic oral antibiotics are provided. They are instructed to avoid excessive bending or twisting as this may dislodge the SCS lead. In addition, they are told not to alter medication consumption and to maintain their routine activity level. They are to alert the physician in case of any alteration in stimulation pattern, signs of infection, or any other unusual occurrences. Follow up is usually within 7-10 days following implantation and the lead is removed. The efficacy of the SCS is assessed, and the physician should then determine whether to proceed with a permanent SCS. Pain relief of greater than 50-75% is usually considered a positive response.

> "A SCS neuromodulation system should be considered for patients who have failed all reasonable conservative care including appropriate diagnostic, therapeutic and rehabilitative techniques, and have been given a reasonable period of time to recover from the condition."[7]

TABLE 1. SCS: REDUCTION IN PAIN STATISTICS			
Reference	**No. of Patients**	**Follow Up**	**Results**
North. Pain. 1993 (77)	171	7 years	52%: >50% relief; 60% would repeat
Turner. Neurosurgery. 1995 (20)	39	16 months	59%: >50% relief
De LaPorte. Pain. 1993 (19)	64	4 years	55%: good to excellent results
Kupers. Pain. 1994. (55)	70	3.5 years	52%: good to very good
Kumar. Neurosurgery. 1991 (65)	94	3+ years	66%: good to excellent
Burchiel. Spine. 1996. (53)	70	1 year	55%: >50% relief

Reprinted with permission, Advanced Neuromodulation Systems (ANS).

The patient undergoing a permanent SCS implantation is brought into the ambulatory surgery center or hospital the morning of the procedure. A urinalysis, complete blood count with differential and sedimentation rate should be obtained within 72 hours prior to the implantation. A chest X-ray should and EKG should be obtained in all patients over the age of forty-five, a history of cardiac or pulmonary disease, or ongoing signs or symptoms of cardiac or pulmonary difficulty. Preoperative and postoperative intravenous antibiotics are administered and the patient is discharged following recovery with 7-10 days of oral antibiotics, or kept for a 23-hour hospital observation (physician/surgical preference).

Upon discharge, the patient is given verbal and written instructions to avoid excessive lifting, twisting, or bending, and to sponge bathe only for 2 weeks. The first postoperative visit is 1 week following the permanent insertion. The surgical site is checked and any skin staples or sutures are removed. At that time there may be slight swelling noted in the pocket. This is probably a normal finding and represents a seroma although the clinician should have appropriate suspicion for infection. A seroma may last for 3-4 weeks and may interfere with transmission with the radiofrequency controlled devices (ANS RF or Medtronic Extrel device). Also during this visit, the SCS is reprogrammed as needed. The patient should be seen in follow-up 2 weeks later and then again in one month. After that, the patient should be seen as indicated. If a goal of returning the patient to work exists then aggressive rehabilitative should be performed.

Potential Complications of SCS

There are rarely any serious complications from the temporary percutaneous trial or permanent procedure for SCS implantation.[54] In one study, one nonfatal pulmonary embolism and one case of paraplegia lasting 3 months occurred.[55] The latter resulted from a laminectomy that was used to place the stimulating lead. Other rare reported complications include sphincter disturbance and gait abnormality.[56]

Most complications from the temporary or permanent devices include formation of scar tissue, poor localization of paresthesias, lead migration, lead fracture, pain at the pocket site or connection site, infection, nerve injury, and epidural hematoma.[19,42,48,54,57-62] In a comprehensive summary of different publications, lead migration or displacement varied from 3.7% to 69% although most studies reported migration between 16% and 25%.[54] Rates of lead fractures were reported in various series from less than 1% to more than 20% and superficial infections occurred in 2% to 12% of cases. Serious surgical infections were rare as were clinically apparent epidural hematomas. Cerebrospinal fluid leakage were found in one series in 2% of patients. Avoiding complications in spinal cord stimulation should follow an analytical, step-wise approach.

In a clinical setting experience with over 300 lead implants, there were experienced three in situ infections with permanent devices.[10] One infection resulted from an occult bone stimulator infection from a previous fusion and presented greater than 6 months following implantation; the second infection occurred 2 ½ months after implantation from an unknown source; and the third infection occurred 18 months following implantation. It appeared to have come from hematogenous seeding when the patient broke an abscessed tooth when he bit down on an apple the week before the infection presented. In the first two cases the SCS's were removed and the patients placed on intravenous antibiotics without further sequelae. In the third case the SCS was not removed and the patient was adequately treated with oral antibiotics and dental care. We have had no complications with any of the trial lead placements.

Spine Clinic Case Study

Mr. P. is a 68 year-old white male with > 40-year history of lower back pain and primarily bilateral lower extremity referred pain. His past medical history includes a total of four lumbar surgeries, including anterior/posterior fusions with laminectomy and pedicle/hardware placement. EMG study confirmed chronic polyradiculopathy and CT scan confirmed no recurrent herniation or significant stenosis, but reveals postsurgical epidural/perineural scar formation, with stable hardware alignment/placement at L3-L5 levels. Patient underwent multiple physical therapy sessions, including McKenzie/manipulation bias treatment from a certified instructor as well as aquatic therapy. Being a fairly stoic individual, with a military and athletic background and currently enrolled in a health fitness club; he chose to minimize his dependence on narcotic pain medications, but still would use this on a daily basis for pain management. He underwent multiple spinal injection trials including transforaminal epidural steroid injections, facet joint/medial branch nerve injections, and diagnostic discography; with only minimal, short-term improvements, mostly effectively with the transforaminal ESIs. Given the short-term results with these previous treatments, I chose to discuss in detail with Mr. P concerning the options available with spinal cord (dorsal column) stimulation or neuromodulation treatment.[9]

After choosing to proceed with the spinal cord stimulator percutaneous trial

TABLE 2. SCS: MEDICATION REDUCTION STATISTICS

Reference	No. of Patients	Follow Up	Results
Ohnmeiss. Spine. 1996 (56)	40	2 years	84%: decreased or eliminated narcotic use
North. Neurosurgery. 1993. (78)	171	7 years	58%: reduced or eliminated analgesics
De LaPorte. Pain. 1993. (19)	64	4 years	90%: reduced medications
Kumar. Neurosurgery. 1991. (65)	94	3+ years	40%: no longer need analgesics
Racz. Spine. 1989. (40)	26	1.8 years	81%: reduced or eliminated narcotic use

Reprinted with permission, Advanced Neuromodulation Systems (ANS).

TABLE 3. SCS: ACTIVITIES OF DAILY LIVING STATISTICS

Reference	No. of Patients	Follow Up	Results
De LaPorte. Pain. 1993 (19)	64	4 years	61%: improved ADL
Racz. Spine. 1989. (40)	26	1.8 years	66%: improved ADL
Ohnmeiss. Spine. 1996 (56)	40	2 years	Statistically significant improvement in ADL

Reprinted with permission, Advanced Neuromodulation Systems (ANS).

TABLE 4. SCS: RETURN TO WORK STATISTICS

Reference	No. Patients	Follow Up	Return to Work
De LaPorte. Pain. 1993 (19)	64	4 years	22%
North. Neurosurgery. 1993. (78)	171	7 years	24%
Burchiel. Spine. 1996. (53)	70	1 year	20%

Reprinted with permission, Advanced Neuromodulation Systems (ANS).

as a diagnostic means of determining if primarily his bilateral lower extremity neuropathic pain (and less so his lumbar axial pain) would be improved with this treatment method, we sent him for this evaluation. After a successful one-week percutaneous SCS trial by the interventionalist physiatrist, and noting that his lower extremity symptoms were improved by 75-80% as reviewed on his post-operative pain diary; we discussed permanent SCS placement. He was sent to the spine-trained, orthopaedic surgeon for permanent SCS placement, using the ANS leads and radiofrequency receiver implantation. After an appropriate amount of post-surgical healing and deemed surgically cleared, he was sent to the interventionalist physiatrist who coordinated a brief trial of aquatic-/land-based, McKenzie physical therapy (per their protocol), with the goal of improving his mobility and overall function. After subsequent periodic 3-6-12 month scheduled follow-up visits, it was determined by both the interventional physiatrist and the patient that he had regained improved mobility, overall function, with decreased narcotic dependence. Mr. P was so pleased with his results that he has resumed his private health fitness program, which includes weight-lifting and some non-impact, aerobic conditioning with elliptical cross-training and stationary bicycle training. He also notes that his marital relationship and social interactions have improved given his reduced symptomatic pain.[9]

Clinical Results

Original long-term results of pain control from spinal cord stimulation in the late 1960s and 1970s were disappointing.[3-6,61] This led to widespread disenchantment with SCS in general. Poor patient selection, inadequate equipment, and failure to perform implantations with the patient awake accounted for the dismal results. The advent of new technology, careful patient selection, trial implantation, percutaneous placement, and active physician-patient interaction during the procedure have all contributed to the success of spinal cord stimulation over the past 15 years.[6,8-10]

The most common SCS application in North America today is in the treatment of chronic low back and lower extremity pain due to chronic radiculopathy or postlaminectomy lumbar pain syndrome despite adequate surgical intervention.[51,53,61,63-65] This population represents the primary indication for SCS in our practice and has provided us with an effective treatment option. The largest SCS study incorporates 320 consecutive patients who underwent either temporary or permanent implants at the Johns Hopkins Hospital between 1971 and 1990.[32] This series includes follow-up on 205 patients, the majority of whom had the diagnosis of failed back surgery syndrome (FBSS). Permanent SCS implants were placed in 171 of these patients. At follow-up (mean interval 7.1 ± 4.5 yrs), 52% of patients had at least 50% continued pain relief, and 58% had a reduction or elimination of analgesic intake. About 54% of patients younger than 65 were working at the time of follow-up; 41% had been working preoperatively.

The percentage of patients having long-term pain relief is similar in the majority of large published SCS series of implants for FBSS. The success rate in most of these studies, which is generally reported as 50% or more pain relief, is approximately 50-60%.[46,66-70] Some studies report success rates as high as 88% and

others as low as 37%.[71,72] Although these latter studies differ in implantation technique and screening protocols, the success rate for pain reduction generally remains the same.

More recently published reviews have specifically looked at the efficacy of SCS in FBSS for reduction in pain, reduction in narcotic medication consumption, improvements of activities of daily living function, and return to work status.[42,73-76] Tables 1 thru 4 present summaries of studies of SCS patients relative to reduction in pain, medication reduction, activities of daily living, and return to work statistics, respectively.

According to these studies, long-term pain reduction (at least 2 years after implantation) can be expected to range from 50-70% in approximately 60% of SCS patients. In 50-90% of individuals, there will be an elimination or reduction in the use of opioids. The return to full employment rate after SCS reported by two studies is 25-59% which is very significant when

having a positive impact on their pain. The vast majority of our patients who have been permanently implanted would have it done again. In general, we see a reduction in narcotic medication consumption and an improvement in overall daily living function. In fact, we have a majority of workmen compensation population and have been able to successfully return the majority of the SCS-implanted patients to some form of employment (varying from light to medium duty level work), with a noticeable reduction in medication usage.

Most of our injured patients have a low educational level were injured at work doing relatively strenuous jobs. Even though there is a general tendency for function to improve with appropriate SCS implantation, few patients return to work. This is consistent with the observation of others and appears to relate more to the chronicity of their disability, attendant psychosocial changes, and a relatively low sophistication level as opposed to any fail-

facilitate successful long-term outcomes. Further neuromodulation devices and technology should assist in providing further options to be available for this select, but growing population of chronic pain patients and what is available for the future is still unknown.[6,8-10] ■

Elmer G. Pinzon, MD, MPH is an Interventional Spine & Physical Medicine/Musculoskeletal Specialist at SpineKnoxville/Tennessee Orthopaedic Specialists in Knoxville, TN. Dr. Pinzon is Fellowship trained in non-surgical spinal procedures, musculoskeletal medicine, and electrodiagnostics, and is Board Certified ABPMR, ABPM. Please direct any correspondence to: Elmer Pinzon, MD, MPH; Interventional Spine/Physical Medicine Staff Physician; SpineKnoxville; 10321 Kingston Pike; Knoxville, TN 37922

> "Approximately 75% of the patients the author has implanted report 50-80% pain relief and are satisfied with their devices. The majority of those patients who have received less than 50% pain relief still feel positively about their device as they perceive it as having a positive impact on their pain."

comparing it to the usual return to work rate in this population of 1-5%.[42,74]

Reasons for the disparity between pain reduction and return-to-work rates appear to reflect the high percentage of unskilled laborers among this population, the prolonged periods of disability and the attendant socio-behavioral changes that take place. Despite this disparity, there is a general increase in function and activities of daily living.

We have been implanting spinal cord stimulation for the treatment of chronic pain conditions in our orthopaedic clinical practice for three years. The majority of patients have had FBSS with the second leading indication being chronic lumbar radiculopathy. Approximately 75% of the patients the author has implanted report 50-80% pain relief and are satisfied with their devices. The majority of those patients who have received less than 50% pain relief still feel positively about their device as they perceive it as

ings of SCS. These characteristics make it unrealistic to expect a high return-to-work rate regardless of the intervention. Perhaps earlier intervention with stringent patient selection will help improve return to work rates in the future.

The Future

The future of SCS neuromodulation looks promising with the planned technological advances in these devices.[46,50,78-80] Both ANS and Medtronic have implanted pulse generators and lead devices that allow an adequate power supply for dual lead systems, which extends the life of the pulse generator. In addition, ANS has developed a pulse generator that employs a capacitor instead of a battery that is rechargeable by an external radiofrequency controlled device. With a coordinated program of multivaried treatment protocols, as outlined in this spine-centered, orthopaedic clinic setting model; further coordinated improvements may

References

1. Ray CD, Burton C, and Lifson A. Neurostimulation as used in a large clinical practice. *Appl Neurophysiol.* 1982. 45:160-206.

2. Ruda MA. Opiates and pain pathways: Demonstration of enkephalin synapses on dorsal horn projection neurons. *Science*. 1982. 215:1523-1524.

3. Saade NE, et al. Supraspinal modulation of nociception in awake rats by stimulation of the dorsal column nuclei. *Brain Res*. 1986. 369:307-310.

4. Fedorcsak I, et al. *Peripheral vasodilation due to sympathetic inhibition induced by spinal cord stimulation.* In Proceedings of the IBRO World Congress of Neurosciences. Paris, France. 1991. p. 126.

5. Fields H. Depression and pain: A neurobiological model. *Neuropsychiatry Neuropsychol Behav Neurol*. 1991. 4:83-92.

6. Gatchel RJ, Miller B, and Lou L. Failed Back Surgery Syndrome. *Practical Pain Management*. May/June 2004. 4(3);20-31.

7. Bedder MD. Spinal Cord Stimulation and Intractable Pain: Patient Selection. In Waldman & Winnie (ed): *Interventional Pain Management, 1st ed*. W.B. Saunders Company. Philadelphia. 1996. pp. 412-418.

8. Cameron T. Elliott S. Spinal Cord Stimulation. *Practical Pain Management*. May/June 2002. 2(3);13-15.

9. Pinzon EG. Persistent Spine-Centered Chronic Pain Scenarios and Treatment Options. *Practical Pain Management*. March/April 2004. 4(2);17-22.

10. Windsor RE, Falco FJE, and Pinzon EG. Spinal Cord Stimulation in Chronic Pain. In Lennard TE (ed): *Pain Procedures in Clinical Practice. 2nd Ed*. Hanley & Belfus, Inc. 2000. pp. 377-394.

11. Handwerker HO, Iggo A, and Zimmerman M. Segmental and supraspinal actions on dorsal horn neurons responding to noxious and non-noxious skin stimuli. *Pain*. 1975. 1:147-165.

12. Tasker RR. Deafferentation in central pain. In Wall PZ, Melzack R (eds.): *Textbook of Pain, 2nd ed*.

Churchhill-Livingstone. Edinburgh. 1989. pp. 154-180.

13. Kumar K, et al. Epidural spinal cord stimulation for treatment of chronic pain- some predictors of success: a 15-year experience. *Surg Neurol*. 1998. 50:110-121.

14. Robaina FJ. Spinal cord stimulation for relief of chronic pain in vasospastic disorders of the upper limbs. *Neurosurgery*. 1989.24:179-183.

15. LeDoux MS and Langford KH. Spinal cord stimulation for the failed back syndrome. *Spine* 1993. 18:191-194.

16. Bayliss WM. On the origin from the spinal cord of the vasodilator fibers of the hind-limb and on the nature of these fibers. *J Physiol*. 1987. 382:173-209.

17. Melzack R and Wall PD. Pain mechanisms: A new theory. *Science*. 1965. 150:971-979.

18. Melzack R. Anatomy and Physiology of Pain: Clinical Correlates. In Waldman & Winnie (Eds): *Interventional Pain Management, 1st ed.* W.B.Saunders Company. Philadelphia. 1996. pp. 1-9.

19. De La Porte C and Van de Kelft E. Spinal cord stimulation in failed back surgery syndrome. *Pain*. 1993. 52:55-61.

20. Turner J, et al. Spinal cord stimulation for chronic low back pain: a systematic literature synthesis. *Neurosurg*. 1995. 37:1088-1096.

21. Melzack R. Psychological aspects of pain: Implications for neural blockade. In Cousins MJ, Bridenbaugh PO (eds): *Neural Blockade in Clinical Anesthesia and Management of Pain, 2nd ed*. J.B. Lippincott. Philadelphia. 1989. pp 845-860.

22. Basbaum AL and Fields HL. Endogenous pain control systems: Brain-stem spinal pathways and endorphin circuitry. *Ann Rev Neurosci*. 1984. 7:309-315.

23. Meyerson BA. Electric stimulation of the spinal cord and brain. In Bonica JJ (ed): *The Management of Pain, 2nd Ed*. Lea & Febiger. Philadelphia. 1990. pp. 1862-1877.

24. North RB, et al. Prognostic value of psychological testing in patients undergoing spinal cord stimulation: a prospective study. *Neurosurgery*. 1996. 39:301-311.

25. Kalin MT and Winkelmuller W. Chronic pain after multiple lumbar discectomies-significance of intermittent spinal cord stimulation. *Pain*. 1990. 40:S241.

26. Reynolds DV. Surgery in the rat during electrical analgesia induced by focal brain stimulation. *Science*. 1969. 164:444-449.

27. Shealy CN, Mortimer JT, and Reswick JB. Electrical inhibition of pain by dorsal column stimulation. *Anesth Analg*. 1967. 46:489-491.

28. Feldman RA. Patterned response of lamina V cells: Cutaneous and dorsal funicular stimulation. *Physiol Behav*. 1975. 15:79-84.

29. International Association for the Study of Pain: Classification of chronic pain: Descriptions of chronic pain syndromes and definitions of pain terms. *Pain Suppl 3*. 1986. S1-S225.

30. Marchand S, Bushnell MC, Molina-Negro P, et al. The effects of dorsal column stimulation on measures of clinical and experimental pain in man. *Pain*. 1991. 45:249-257.

31. Meglio M, Cioni B, and Rossi GF. Spinal cord stimulation in management of chronic pain: A 9-year experience. *J Neurosurg*. 1989. 70:519-524.

32. Jensen MP, Turner JA, Romano JM, et al. Coping with chronic pain: A critical review of the literature. *Pain*. 1991. 47:249283.

33. Campbell JN. Examination of possible mechanisms by which stimulation of the spinal cord in man relieves pain. *Appl Neurophysiol*. 1981. 44:181-186.

34. Broseta J, Roldan P, Gonzalez-Darder V, et al. Chronic epidural dorsal column stimulation in the treatment of causalgic pain. *Appl Neurophysiol*. 1982. 45:190-194.

35. Broseta J, et al. Spinal cord stimulation in peripheral arterial disease. *J Neurosurg*. 1986. 64:71-80.

36. LeRoy PL. Stimulation of the spinal cord biocompatible electrical current in the human. *Appl Neurophysiol*. 1981. 44:187193.

37. Erickson DL and Long DM. Ten-year follow-up of dorsal column stimulation. *Advances in Pain Research and Therapy*. 1983. 6:583-589.

38. Linderoth B. *Dorsal column stimulation and pain: Experimental studies of putative neurochemical and neurophysiological mechanisms* (thesis). Karolinska Institute. Stockholm. 1992.

39. Hoppenstein R. Percutaneous implantation of chronic spinal cord electrodes for control of intractable pain. *Surg Neurol*. 1975. 4:195-198.

40. Racz GB, McCarron R, and Talboys P. Percutaneous dorsal column stimulator for chronic pain control. *Spine*. 1989. 14:1-4.

41. Foreman RD, et al. Effects of dorsal column stimulation on primate spinothalmatic tract neurons. J *Neurophysiol*. 1976. 39:534-546.

42. Nielson KD, Adams JE, and Hosobuchi Y. Experience with dorsal column stimulation for relief of chronic intractable pain. *Surg Neurol*. 1975. 4:148-152.

43. Spiegelmann R, et al. Spinal cord stimulation: a contemporary series. *Neurosurg*. 1991. 28:65-71.

44. Linderoth B, et al. Is vasodilation following dorsal column stimulation mediated by antidromic activation of small diameter fibers? *Acta Neurochir Suppl* (Wein). 1989. 46:99-101.

45. Barolat G, Schwartzman R, and Woo R. Epidural spinal cord stimulation in the management of reflex sympathetic dystrophy. *Stereotact Funct Neurosurg*. 1989. 53:29-39.

46. Krieger DT and Martin JB. Brain peptides. *N Engl J Med*. 1981. 304:876-885.

47. Loeser JD and Parker G. Assessment and investigation of the patient with chronic pain at the University of Washington Multidisciplinary Pain Center. In Loeser JD, Egan KJ (eds): *Pain Management*. Raven Press. New York. 1989. pp. 21-34.

48. Augustinsson LE, Carlsson CA, Holm J, et al. Epidural electrical stimulation in severe limb ischemia. Pain relief, increased blood flow, and possible limb-saving effect. *Ann Surg*. 1985.. 202:104-110.

49. Siegfried J and Lazorthes Y. Long-term follow-up of dorsal column stimulation for chronic pain syndrome after multiple lumbar operations. *Appl Neurophysiol*. 1982. 45:201-204.

50. North RB, Fowler K, Nigrin D, et al. Patient-interactive, computer-controlled neurological stimulation system: Clinical efficacy in spinal cord stimulator adjustment. *J Neurosurg*. 1992. 76:967-972.

51. Tallis RC, et al. Spinal cord stimulation in peripheral vascular disease. *J Neurol Neurosurg Psych*. 1983. 6:478-484.

52. Burchiel KJ, et al. Prognostic Factors of Spinal Cord Stimulation for Chronic Back and Leg Pain. *Neurosurgery*. 1995. 36(6):1101-1111.

53. Burchiel KJ, et al. Prospective, Multicenter Study of Spinal Cord Stimulation for Relief of Chronic Back and Extremity Pain. *Spine*. 1996. 21(23):2786-2794.

54. Augustinsson LE. Avoiding Difficulties in Spinal Cord Stimulation. In Waldman SD, Winnie AP (eds): *Interventional Pain Management*, 1st Ed. W.B. Saunders Company. Philadelphia. 1996. pp. 427-430.

55. Kupers R, et al. Spinal cord stimulation in Belgium: a nation-wide survey on the incidence, indications and therapeutic efficacy by the health insurer. *Pain*. 1994. 56:211-216.

56. Ohnmeiss D, et al. Prospective outcome evaluation of spinal cord stimulation in patients with intractable leg pain. *Spine*. 1996. 21(11):1344-1350.

57. Snyder SH. Opiate receptors in the brain. *N Engl J Med*. 1977. 296:266-271

58. Devulder J, De Colvenaer L, and Rolly G. Spinal cord stimulation in chronic pain therapy. *Clin J Pain*. 1990. 6:51-56.

59. Krames ES. Mechanisms of Action of Spinal Cord Stimulation. In Waldman & Winnie (ed): *Interventional Pain Management, 1st ed*. W.B.Saunders Company. Philadelphia. 1996. pp. 407-411.

60. Meyerson BA, et al. Spinal cord stimulation in chronic neuropathic pain. *Lakartidningen*. 1991. 88:727-732.

61. Shealy CN, Mortimer JT, and Hagfors NR. Dorsal column electroanalgesia. *J Neurosurg*. 1970. 32: 560-564.

62. Wall PD and Melzack R. Introduction. In Wall PD, Melzack R (eds): *Textbook of Pain, 2nd ed*., Churchhill-Livingstone. Edinburgh. 1989. pp. 1-18.

63. Robb LG, Spector G, and Robb MP. Spinal cord stimulation: Neuroaugmentation of the dorsal columns for pain relief. In Weiner RS(ed.): *Pain Management: a practical guide for clinicians, 5th edition*. St. Lucie Press. Boca Raton. Florida. 1998. pp 271-293.

64. North RB, et al. A prospective, randomized study of spinal cord stimulation vs. reoperation for Failed Back Surgery Syndrome: Initial results. *Stereotact Funct Neurosurg*. 1994. 62:267-272.

65. Kumar K, Nath R, and Wyant GM. Treatment of chronic pain by epidural spinal cord stimulation: 10-year experience. *J Neurosurg*. 1991. 75:402-407.

66. Burton CV. Session on spinal cord stimulation: Safety and clinical efficacy. *Neurosurgery*. 1977. 1: 164-165.

67. Law JD and Kirkpatrick AF. Update: Spinal cord stimulation. *Am J Pain Manage*. 1992. 2(l):34-42.

68. Miletic V, Hoffert MJ, Ruda MA, et al. Serotonergic axonal contacts on identified cat dorsal horn neurons and their correlation with nucleus raphe magnus stimulation. *J Comp Neurol*. 1984. 228:129-134.

69. Oliveras JL, Redjemi G, and Besson J. Analgesia induced by electrical stimulation of the inferior centralis of the raphe in the cat. *Pain*. 1975. 1:139-143.

70. Pineda A. Dorsal column stimulation and its prospects. *Surg Neurol*. 1975. 4:157-163.

71. Jacobs MJ, Jorning PH, Joshi SR, et al. Epidural spinal cord electrical stimulation improves microvascular blood flow in severe limb ischemia. *Ann Surg*. 1988. 207:179-183.

72. Segal R, et al. Spinal cord stimulation revisited. *Neurol Research*. 1998. 20:391-396.

73. Crue BL. The neurophysiology and taxonomy of pain. In Brena SF, Chapman SL (eds): *Management of patients with chronic pain*. Spectrum Publications. Jamaica, NY. 1983. pp. 21-31.

74. Law JD. Percutaneous spinal cord stimulation for the "failed back surgery syndrome." *Pain Management Update* 1990. l(l):12.

75. Mullet KR, et al. Design and function of spinal cord stimulators- theoretical and developmental considerations. *Pain Digest*. 1992. 1:281-287.

76. Pineda A. Complications of dorsal column stimulation. *J Neurosurg*. 1978. 48:64-68.

77. North RB and Wetzel TF. Spinal cord stimulation for chronic pain of spinal origin. *Spine*. 1993. 18:191-4.

78. North RB, Kidd D, and Zahurak M. Spinal cord stimulation for chronic intractable pain: Experience over two decades. *Neurosurgery*. 1993. 32:384-394.

79. Bell GK, et al. Cost-effectiveness Analysis of Spinal Cord Stimulation in Treatment of Failed Back Surgery Syndrome. *J Pain and Symptom Management*. 1997. 13(5):286-295.

80. Kumar K, et al. Epidural spinal cord stimulation for relief of chronic pain. *Pain Clinic*. 1986. 1:91-99.