REVIEW ARTICLE

Neural Modulation by Stimulation

José De Andrés, MD, PhD*; Jean-Pierre Van Buyten, MD†

*Valencia General University Hospital, Valencia, Spain; †AZ Maria Middelares Pain Clinic, St Niklaas, Belgium

■ **Abstract:** Spinal cord stimulation (SCS) for the treatment of neuropathic pain is supported by good-quality randomized controlled trials, prospective and retrospective case studies, and observational case series that confirm its efficacy and safety. SCS has been successfully used in various refractory neuropathic pain conditions, including failed back surgery syndrome (FBSS), neuropathic back and leg pain, and complex regional pain syndrome (CRPS) types I and II. According to the Harbour and Miller Scale (2001), the evidence for SCS in FBSS has been classified as grade B, while that for CRPS type I has been classified as grade A. Clinical evidence has shown that compared to conventional pain therapy, more than two-thirds of carefully selected patients treated with SCS achieved sustained pain relief of 50% or more, with minimal side effects. Many patients were able to reduce their analgesic consumption. Quality of life improved and the majority of patients were happy with their treatment; in some cases, patients were able to return to work. Trial stimulation, which is relatively inexpensive and completely reversible, provides predictive value for long-term efficacy and increases the cost-effectiveness of permanent implantation. Studies consistently report that over time, SCS is potentially cost saving to the healthcare system. At present, SCS is considered a "last resort" in the treatment of refractory neuropathic pain, yet evidence suggests that early intervention with SCS results in greater efficacy and, in the case of FBSS, should be considered before re-operation. ■

Key Words: spinal cord stimulation, Complex regional pain syndrome, health-related quality of life

Spinal cord stimulation (SCS) is an evidence-based therapy that has been used for many years in the treatment of refractory neuropathic pain, particularly failed back surgery syndrome (FBSS) and complex regional pain syndrome (CRPS) types I and II. While not all patients are suitable for treatment with SCS, careful patient selection and implantation after evaluation by a multidisciplinary pain management team can offer safe and effective treatment of refractory neuropathic pain. The search for objective criteria predicting an optimal result using implantable systems must include psychological criteria in the decision algorithm.[1–3] In order to implant systems for the treatment of chronic pain, the existence of an organic lesion for the cause of pain must be identified and the existence of organic pathology must be placed within the context of the psychological experience of the patient.[4] To date, in some countries the decision to implant has been based only on the clinical indication for the pain condition of the patient and not within the context of the psychological experience of the patient.

A multidimensional approach to the assessment of the patient is vital to promote interdisciplinary decision making and increase the quality of health care and, eventually, the quality of life of the individuals referred to pain departments. In addition, the assessment and control of sensory, emotional, and cognitive variables allows better adjustment of patients to the neuromodulation technique, limits the chances of the therapy being applied inappropriately, and enhances the benefit of the intervention in the short, middle, and long term.[5]

MECHANISM OF ACTION

The introduction of SCS[6] was inspired by Melzack and Wall's gate-control theory in 1965.[7] This proposed that painful "nociceptive" information in the periphery is transmitted to the spinal cord in small-diameter, unmyelinated C fibers and lightly myelinated A-delta fibers,

Address correspondence and reprint requests to: José De Andrés, MD, PhD, Valencia General University Hospital, Avda Tres Cruces s/n, 46014 Valencia, Spain. E-mail: deandres_jos@gva.es.

Pain Practice, Volume 6, Issue 1, 2006 39–45

which terminate in the superficial laminae of the dorsal horn—the gate—of the spinal cord. Other sensory information, such as touch or vibration, is carried in large, myelinated A-beta fibers that pass through this gate. As they do, they give off small branches that terminate in the dorsal horn, where they have an inhibitory effect on the nociceptive conduction. The basic premise of the gate-control theory was that stimulation of large, low threshold fibers would close the gate to the reception of small-fiber information (Figure 1[8]). The clinical result of gate closure was postulated to be analgesia.

However, the mechanism of action of SCS must involve more than a direct inhibition of pain transmission in the dorsal horn of the spinal cord. If this were the principal mode of action, then SCS would control nociceptive pain, and this is generally not the case. In a recent review of the mechanisms of action of SCS, it was concluded that a multiplicity of different mechanisms are activated by SCS, some essential for the desired effect but many irrelevant to it.[9]

At present, spinal segmental inhibition seems to be crucial. Animal studies show that second-order afferent nerves and interneurons can be activated by SCS,[10] and a proportion of these may manifest delayed inhibitory activity following brief stimulation.[11] SCS also selectively inhibits abnormal hypersensitivity in dorsal horn neurons.[12] SCS does not directly activate the neurons of the pars gelatinosa, but elicits inhibitory responses of these neurons via A fibers by reducing excitatory neurotransmitters.[13] Pain modulation by SCS also may involve supraspinal activity via the posterior columns of the spinal cord (ie, down-modulation involving loops or feedback mechanisms that influence rostral transmission of pain from higher centers to the spinal cord).

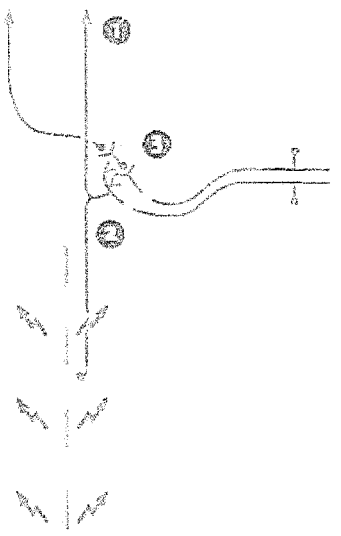

Figure 1. The "classic" theory for pain inhibition by SCS.[8] SCS, Spinal cord stimulation. ❶ Orthodromic transmission of impulses produces paresthesia experienced by patient; ❷ Antidromic transmission of impulses excites neurons in dorsal horns to activate; ❸ Gate mechanism producing inhibitory effect on transmission in small-diameter fibers subserving pain.

It is possible that long-term relief of neuropathic pain is mediated by a suppressive effect on the hyperexcitability of neurons in the dorsal horns as a consequence of neural injury. The biochemical basis for this includes a reduction in the levels of the excitatory amino acids glutamate and aspartate by gamma-aminobutyric acid (GABA) and adenosine-dependent mechanisms.[14] Recent SCS studies performed on rat models of mononeuropathy have demonstrated a preferential effect on A-beta fiber mediated functions, and the hyperexcitability of wide-dynamic-range dorsal horn neurons was attenuated. These effects were coupled to increased release of GABA and reduced glutamate and aspartate release in the dorsal horn. Intrathecal administration of GABA, baclofen, and adenosine enhanced the SCS effect on tactile allodynia even in previously nonresponsive rats. Preliminary results indicate that gabapentin may have a similar effect. GABAergic and adenosine-related mechanisms conceivably represent only examples of a number of putative receptor systems involved in SCS. Other biochemical indicators include an increase in beta-lipoproteins and beta-endorphins in the cerebrospinal fluid (CSF) following SCS[15] and the fact that analgesic action cannot be blocked with naloxone.[16]

REVIEW OF THE EVIDENCE IN NEUROPATHIC PAIN

SCS is still met with opposition by some physicians, who prefer to use conventional therapies such as pharmacotherapy to treat neuropathic pain, even if these therapies may not be appropriate for a particular patient. SCS is often perceived as an invasive and costly treatment whose efficacy does not warrant the complexity of the procedure and the potential for side effects. Confusion also exists regarding which patients are suitable for SCS and when treatment should be initiated.

Yet there is much evidence that addresses these concerns. Since it was first introduced, SCS has undergone numerous technical modifications and advances that make the procedure less complex and more effective, allowing it to be applied in a variety of pain conditions.

Table 1. Clinical studies assessing spinal cord stimulation[21–23]

Indication	RCTs		Case Series		Evidence Grading (Harbour and Miller 2001)[24]	Patients with ≥50% Pain Relief (%)	Cost-effectiveness Studies (*n*)
	n	Quality (Jadad Score*)	*n*	Quality† (Median [Range])			
Failed back surgery syndrome	1	4	72	1 (0–6)‡	B	62	4
Complex regional pain syndrome	1	3	25	2 (0–4)	A	67	1

*0 (weakest)—5 (strongest); †0 (poor)—7 (excellent); ‡increases with more recent studies.
RCT, randomized controlled trial.

SCS is used most often in patients with FBSS,[17] followed by refractory neuropathic back and leg pain and CRPS types I and II. There is a substantial evidence that supports its use in these indications: good-quality randomized controlled trials (RCTs) have been undertaken in FBSS and CRPS type I, and many case reports, retrospective and prospective case series, and observational comparative studies are available. Nevertheless, systematic reviews have highlighted the need for further methodologically rigorous studies to provide definitive data regarding improvement in pain, function, quality of life, etc. in the short and long term.[18–22]

The most recent systematic reviews/meta-analyses of SCS were undertaken to evaluate the evidence for the clinical and cost-effectiveness of SCS compared to conventional therapies in the treatment of refractory neuropathic back and leg pain/FBSS and CRPS types I and II.[21–23] Relevant studies were selected by examining bibliographic resources (eg, Medline, EMBASE, Cochrane Database of Systematic Reviews, etc.), hand searching reference lists of included studies, and contacting experts and companies. Outcomes included pain relief, return to work, functional disability, complications, quality of life, patient satisfaction/preference for intervention, health service utilization, and costs (excluding "technical outcomes"). Studies were organized according to the hierarchy of evidence, their quality assessed, and an overall level of evidence grading applied (Harbour and Miller Scale).[24] The quality of comparative studies was evaluated using a modified version of the Jadad scale (0 being the weakest and 5 being the strongest).[25] Studies also were examined for the presence of bias (selection, confounding, performance, detection, attrition). Given the lack of a recognized tool, the quality of case series studies was assessed qualitatively.

The systematic reviews found one RCT, one cohort study, 72 case series, and four cost studies for refractory neuropathic back and leg pain/FBSS (grade B evidence).[26] One RCT, 25 case series, and one economic evaluation were identified for CRPS type I (grade A evidence) or CRPS type II (grade D evidence)[22,26] (Table 1).

Refractory Neuropathic Back and Leg Pain/FBSS

Persistent or recurrent radicular pain after lumbosacral spine surgery (FBSS) is a challenge to multidisciplinary pain management strategies, including medical, surgical, rehabilitative, and behavioral therapy. FBSS patients, by definition, have failed to obtain lasting relief despite receiving a variety of therapies, including repeated operations, oral medications, nerve blocks, corticosteroid injections, physical therapy, and chiropractic care.

For more than 30 years, however, clinicians have successfully used SCS to treat selected patients with chronic pain, especially FBSS patients. Yet despite its apparent efficacy, SCS is generally reserved as a last resort in the treatment of FBSS. Therefore, a prospective RCT was carried out to explore the relative merits of SCS vs. re-operation (back surgery, the standard treatment in FBSS), and to determine whether it would be more appropriate to offer SCS as a late, rather than a last, treatment strategy.[27,28]

For the purposes of the systematic review, the full trial results published in 2005 were used.[28] The RCT was judged to be of high quality and scored 4 out of 5 on the Jadad scale (Table 1). A total of 50 surgically remedial FBSS patients with refractory, mainly radicular neuropathic pain (with or without low back pain), were randomized to treatment with SCS or re-operation. Primary outcome measures included patient preference for treatment (frequency of therapy crossover), greater than 50% pain relief and patient satisfaction, and opioid analgesic use at follow-up. Patients were followed for an average of three years.

The study found that in the long term, SCS was significantly more successful than re-operation, with 47% of SCS patients achieving greater than 50% pain relief and expressing satisfaction with treatment compared with only 12% of re-operation patients ($P < 0.01$). In addition, significantly fewer SCS patients (13%) required an increase in opioid medication than re-operation patients (42%) ($P = 0.025$), and it was noted that 87% of SCS patients had stable or decreased opioid use. Patient preference for treatment with SCS was indicated by the fact that SCS patients were significantly less likely to crossover than re-operation patients (21% vs. 54%, respectively) ($P = 0.02$). Overall, the complication rate for SCS was very low, with only 9% of patients experiencing hardware revisions.

The 65 case studies in refractory neuropathic back and leg pain/FBSS reflected follow-up of up to 10 years. A meta-analysis of pooled outcomes showed that 62% of SCS patients achieved greater than 50% pain relief (Table 1), and 53% of patients no longer required analgesics. Furthermore, 70% of SCS patients expressed satisfaction with their treatment. Functional capacity and HRQoL (health-related quality of life) measures were significantly improved by SCS, and 40% of patients were able to return to work. A SCS complication rate of 18% per year was observed, mainly due to electrode or lead problems. Most complications were reversible and no serious adverse events or neurological-related complications were reported.

Although no economic evaluation of SCS for refractory neuropathic back and leg pain/FBSS was identified, four studies have examined the costs of SCS and consistently report that over time SCS is potentially cost saving to the healthcare system. When the costs of SCS were compared to best medical treatment/conventional pain therapy for FBSS, it was found that the time to cost neutrality ("payback" period) for SCS was 2.5 years post implant,[29] although in some centers it was as low as 15 months.[23] The payback period is sensitive to a variety of factors, including the efficacy of SCS, battery/electrode life, and the level of SCS usage by patients.[23]

Complex Regional Pain Syndrome

Many methods have been used to reduce the pain intensity in CRPS, including conventional pain medication, physical therapy, sympathetic blocks, and transcutaneous electrical nerve stimulation. However, all produce mainly unfavorable results. Thus, the RCT of SCS in CRPS was carried out to determine whether greater efficacy could be achieved by adding SCS to a standard treatment protocol (physical therapy, PT).[30] The RCT was of high quality and scored 3 out of 5 on the Jadad scale (Table 1). Patients with therapy-resistant CRPS type I were randomized to treatment with SCS + PT ($n = 36$) or PT alone ($n = 18$). Parameters assessed included pain intensity (using a visual analog scale, VAS), the global perceived effect, functional status, HRQoL measures, and complications associated with SCS. Patients were followed up for 24 months.

Results at six months showed that pain intensity was reduced in the SCS + PT group (mean change in VAS −2.0), but was unchanged in the PT alone group ($P < 0.001$). In addition, a significantly higher proportion of patients in the SCS + PT group (58%) had a score of 6 ("much improved") for the global perceived effect compared with the PT group (6%) ($P = 0.01$). Although no significant improvement in functional status was noted, only treatment with SCS + PT led to an 11% increase in the HRQoL score; in this group, significant improvements were seen for patients with an affected hand ($P = 0.02$) or foot ($P = 0.008$). Four complications associated with SCS were observed: one infection, two complications related to the generator pocket, and one to the leads. The VAS, global pain, and HRQoL results were maintained at the 24-month follow-up, confirming the long-term efficacy of SCS + PT compared to standard treatment alone in the treatment of CRPS.[31]

The 25 case series identified for CRPS type I or II ($n = 500$) provided a median follow-up time of 33 months post implant. Results showed that 67% of implanted patients achieved pain relief of 50% or more with SCS (seven studies used VAS to assess pain relief, with a significant pooled mean reduction of 4.7) (Table 1). Functional capacity (Oswestry Questionnaire, McGill Pain Questionnaire) was reported in three studies and there was a trend toward improvement across all scales and subscales following SCS implantation. Two case series assessed HRQoL (Sickness Impact Profile, Nottingham Health Profile) and both studies reported significant improvements. No case series were identified that reported the level of patient satisfaction, level of analgesic medication, or work status following SCS. Data from eight studies indicated that overall, 33% of CRPS patients experienced one or more problems with SCS. However, the majority were reversible and due to electrode or lead problems. No serious adverse events were reported and no neurological complications were observed.

In 2005, a prospective clinical study was published that assessed the long-term effect of SCS on the improvement of functional status in patients with chronic, sympathetically maintained CRPS type I ($n = 29$).[32] The study found that after treatment with SCS, both deep pain and allodynia were significantly and permanently reduced from 10 to 0–2 on a 10-cm VAS ($P < 0.01$). During the tests where SCS was inactive, reoccurrence of pain up to eight VAS was recorded. Severe impairments and strong functional limitations in daily living activities of more than 60% to 90%, objectively documented by the pain disability index, were significantly reduced ($P < 0.01$). After a follow-up period of 35.6 ± 21 months, 75% of patients with an affected upper limb showed a significant increase in fist grip strength ($P < 0.01$). A total of 80% of patients with lower limb disability resumed walking without crutches. In addition, previous pain medication was significantly reduced ($P < 0.01$). It was concluded that as a result of the permanent pain relief following long-term treatment with SCS combined with physiotherapy, the functional status and quality of life could be significantly improved in sympathetically maintained CRPS type I.

A formal cost-effectiveness analysis based on the RCT showed that treatment with SCS + PT in CRPS resulted in a lifetime cost saving of €58,471 per patient compared to the standard treatment alone.[33] A cost-utility analysis of this trial indicated that the mean cost per quality-adjusted life-year (QALY) at the 12-month data follow-up was €22,500, a figure that is consistent with a level of cost-effectiveness that is commonly regarded as representing good value and appropriate use of the resources of society and the healthcare system.

CONCLUSIONS

The evidence obtained from recent systematic reviews/meta-analyses indicates that compared to standard pain treatment strategies, SCS is an effective method of treating refractory neuropathic pain, especially CRPS type I (grade A evidence) and FBSS (grade B evidence). The majority of patients achieved sustained, long-term pain relief, with a reduction in concomitant pain medication.[28,31,33–35] Quality of life was improved and the majority of patients expressed satisfaction with their treatment.[28,29,31,33–36] In some cases, functional status and activities of daily living were enhanced and certain patients were able to return to work.[29,34,35] SCS is a cost-effective alternative to conventional therapies[23] and resulted in minimal side effects, most of which were reversible.[21,22]

It has been observed that the results of SCS have improved over time, mainly because of better patient selection (eg, SCS is generally more effective for radicular pain than for axial pain), improvements in matching electrode (lead) placement to sites of pain and the advent of multipolar stimulation systems.[35,37] In addition, trial stimulation is a minimally invasive, inexpensive option that provides enhanced predictive value for long-term efficacy of SCS and increases the cost-effectiveness of permanent implantation.[35,38] If the trial fails, the therapy is completely reversible and does not hinder patients like more invasive options such as repeated surgery. Therefore, the authors claim that in the algorithm of treatment of patients with chronic back and leg pain due to FBSS, SCS should be tried before repetitive surgery. SCS may be delivered in parallel with other therapies and should be used as part of an overall rehabilitation strategy. Evidence suggests that early intervention with SCS results in greater efficacy.[35,39]

REFERENCES

1. Burchiel KJ, Anderson VC, Wilson BJ. Prognostic factors of spinal cord stimulation for chronic back and leg pain. *Neurosurgery.* 1995;36:1101–1111.

2. North RB. Psychological criteria are outcome measures as well as prognostic factors. *Pain Forum.* 1996;5:111–114.

3. North RB, Kidd DH, Wimberly RL, Edwin D. Prognostic value of psychological testing in patients undergoing spinal cord stimulation: a prospective study. *Neurosurgery.* 1996;39:301–311.

4. Dumoulin K, Devulder J, Castille F, et al. Psychoanalytic investigation to improve the success rate of spinal cord stimulation as a treatment for chronic failed back surgery syndrome. *Clin J Pain.* 1996;12:43–49.

5. Monsalve V, De Andres JA, Valia JC. Application of a psychological decision algorithm for the selection of patients susceptible of implantation of neuromodulation systems for the treatment of chronic pain. A proposal. *Neuromodulation.* 2000;3:191–200.

6. Shealy CN, Mortimer JT, Reswick JB, et al. Electrical inhibition of pain by stimulation of the dorsal columns: preliminary clinical report. *Anesth Analg.* 1967;46:489–491.

7. Melzack R, Wall PD. Pain mechanisms: a new theory. *Science.* 1965;150:971–979.

8. Linderoth B, Meyerson BA. Dorsal column stimulation: modulation of somatosensory and autonomic function. In: McMahon SB, Wall PD, eds. *The Neurobiology of Pain Seminars in the Neuroscience*, Vol. 7. London: Academic Press; 1995:263–277.

9. Linderoth B, Meyerson BA. Spinal cord stimulation: mechanisms of action. In: Burchiel KJ, ed. *Surgical Management of Pain*. New York: Thieme; 2002:505–526.

10. Dubuisson D. Effect of dorsal-column stimulation on gelatinosa and marginal neurons of cat spinal cord. *J Neurosurg*. 1989;70:257–265.

11. Lindblom U, Tapper DN, Wiesenfeld Z. The effect of dorsal column stimulation on the nociceptive response of dorsal horn cells and its relevance for pain suppression. *Pain*. 1977;4:133–144.

12. Yakhnitsa V, Linderoth B, Meyerson BA. Spinal cord stimulation attenuates dorsal horn neuronal hyperexcitability in a rat model of mononeuropathy. *Pain*. 1999;79: 223–233.

13. Baba H, Yoshimura M, Nishi S, Shimoji K. Synaptic responses of substantia gelatinosa neurones to dorsal column stimulation in rat spinal cord in vitro. *J Physiol*. 1994;478:87–99.

14. Meyerson BA, Linderoth B. Mechanisms of spinal cord stimulation in neuropathic pain. *Neurol Res*. 2000; 22:285–292.

15. Tonelli L, Setti T, Falasca A, et al. Investigation on cerebrospinal fluid opioids and neurotransmitters related to spinal cord stimulation. *Appl Neurophysiol*. 1988;51:324–332.

16. Boivie J, Meyerson BA. A correlative anatomical and clinical study of pain suppression by deep brain stimulation. *Pain*. 1982;13:113–126.

17. Kupers RC, Van den Oever R, Van Houdenhove B, 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.

18. Grabow TS, Tella PK, Raja SN. Spinal cord stimulation for complex regional pain syndrome: an evidence-based medicine review of the literature. *Clin J Pain*. 2003;19:371–383.

19. Cameron T. Safety and efficacy of spinal cord stimulation for the treatment of chronic pain: a 20-year literature review. *J Neurosurg (Spine 3)*. 2004;100:254–267.

20. Turner JA, Loeser JD, Deyo RA, Sanders SB. Spinal cord stimulation for patients with failed back surgery syndrome or complex regional pain syndrome: a systematic review of effectiveness and complications. *Pain*. 2004;108: 137–147.

21. Taylor RS, Van Buyten JP, Buchser E. Spinal cord stimulation for chronic back and leg pain and failed back surgery syndrome: a systematic review and analysis of prognostic factors. *Spine*. 2005;30:152–160.

22. Taylor RS, Van Buyten J-P, Buchser E. Spinal cord stimulation for complex regional pain syndrome: a systematic review of clinical effectiveness and cost effectiveness. *Eur J Pain* 2006. Manuscript accepted for publication.

23. Taylor RS, Taylor RJ, Van Buyten JP, et al. The cost effectiveness of spinal cord stimulation in the treatment of pain: a systematic review of the literature. *J Pain Symptom Manage*. 2004;27:370–378.

24. Harbour R, Miller J. A new system for grading recommendations in evidence based guidelines. *BMJ*. 2001; 323:334–336.

25. Jadad AR, Cook DJ, Jones A, et al. Methodology and reports of systematic reviews and meta-analyses: a comparison of Cochrane reviews with articles published in paper-based journals. *JAMA*. 1998;280:278–280.

26. Taylor RS. Spinal cord stimulation in complex regional pain syndrome and refractory neuropathic back and leg pain/failed back surgery syndrome: results of a systematic review and meta-analysis. *JPSM*. Manuscript submitted for publication.

27. North RB, Kidd DH, Lee MS, et al. A prospective, randomised study of spinal cord stimulation versus reoperation for failed back surgery syndrome: initial results. *Stereotact Funct Neurosurg*. 1994;62:267–272.

28. North RB, Kidd DH, Farrokhi F, Piantadosi SA. Spinal cord stimulation versus repeated lumbosacral spine surgery for chronic pain: a randomized, controlled trial. *Neurosurgery*. 2005;56:98–106.

29. Kumar K, Malik S, Demeria D. Treatment of chronic pain with spinal cord stimulation versus alternative therapies: cost-effectiveness analysis. *Neurosurgery*. 2002;51: 106–116.

30. Kemler MA, Barendse GAM, van Kleef M, et al. Spinal cord stimulation in patients with chronic reflex sympathetic dystrophy. *NEJM*. 2000;343:618–624.

31. Kemler MA, De Vet HCW, Barendse GAM, et al. The effect of spinal cord stimulation in patients with chronic reflex sympathetic dystrophy: two years' follow-up of the randomized controlled trial. *Ann Neurol*. 2004;55:13–18.

32. Harke H, Gretenkort P, Ladleif HU, Rahman S. Spinal cord stimulation in sympathetically maintained complex regional pain syndrome type I with severe disability. A prospective, clinical study. *Eur J Pain*. 2005;9:363–373.

33. Kemler A, Furnee CA. Economic evaluation of spinal cord stimulation for chronic reflex sympathetic dystrophy. *Neurology*. 2002;59:1203–1209.

34. Van Buyten J-P, Van Zundert J, Vueghs P, Vanduffel L. Efficacy of spinal cord stimulation: 10 years of experience in a pain centre in Belgium. *Eur J Pain*. 2001;5:1–10.

35. Kumar K, Toth C, Nath RK, Laing P. Epidural spinal cord stimulation for treatment of chronic pain—some predictors of success. A 15-year experience. *Surg Neurol*. 1998; 50:110–121.

36. Ohnmeiss DD, Rashbaum RF. Patient satisfaction with spinal cord stimulation for predominant complaints of chronic intractable low back pain. *Spine J*. 2001;1:358–363.

37. Burchiel KJ, Anderson VC, Wilson BJ, et al. Prognostic factors of spinal cord stimulation for chronic back and leg pain. *Neurosurgery*. 1995;36:1101–1111.

38. Bell GK, Kidd D, North RD. Cost-effectiveness analysis of spinal cord stimulation in treatment of failed back surgery syndrome. *J Pain Symptom Manage.* 1997;13:286–295.

39. Stanton-Hicks MD, Burton AW, Bruehl SP, et al. An updated interdisciplinary clinical pathway for CRPS: report of an expert panel. *Pain Pract.* 2002;2:1–16.