Impact of Spinal Cord Stimulation on Sensory Characteristics in Complex Regional Pain Syndrome Type I

A Randomized Trial

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Background: A randomized trial was performed to assess the effect of spinal cord stimulation (SCS) on detection and pain thresholds for pressure, warmth, and cold and on the extent of mechanical hyperalgesia in patients with chronic complex regional pain syndrome type I.

Methods: Fifty-four chronic complex regional pain syndrome type I patients were randomized to receive both SCS and physical therapy (SCS+PT; n = 36), or to receive only physical therapy (PT; n = 18). Twenty-four SCS+PT patients responded positively to trial stimulation and underwent SCS implantation. During a 12-month follow-up period, six quantitative sensory testing sessions were performed. The main analysis compared 24 SCS patients with 29 nonimplanted patients—one PT patient was excluded.

Results: SCS showed no effect on detection thresholds for warmth and cold or on pain thresholds for any sensation. The pressure detection threshold initially increased by SCS, but after 3 months, pressure detection thresholds returned to normal. Mechanical hyperalgesia, both dynamic and static, was reduced slightly with SCS.

Conclusions: Although SCS has previously been shown to cause a significant pain reduction in complex regional pain syndrome type I, the treatment has no long-term effect on detection and pain thresholds for pressure, warmth, or cold. The treatment seems to have only minimal influence on mechanical hyperalgesia.

We conducted a randomized controlled trial of spinal cord stimulation (SCS) for chronic complex regional pain syndrome type I (CRPS I). The results showed that SCS reduces the intensity of pain caused by this disorder in patients in whom all conventional treatments have failed.† The current paper is about the identical group of patients and the study but discusses the effect of SCS on detection and pain thresholds for pressure, warmth, and cold and on the extent of mechanical hyperalgesia in patients with chronic CRPS I.

Spinal cord stimulation originated as a direct consequence of the Gate Control Theory for pain.² The “gate” in the dorsal horn was thought to control the central transmission of neural activity signaling pain. Excess of large (Aβ) fiber activity over small (C) fiber activity resulted in a closed gate and consequently in pain relief. In a mixed population of nerve fibers, the large fibers are more susceptible than the small to recruitment by an externally applied electrical field. This suggests that at a critical stimulation amplitude, large fibers might be recruited selectively, thereby closing the spinal gate. However, it has been demonstrated experimentally that hyperalgesia can also be signaled by large fibers, a finding directly in conflict with the gate theory.³ As a result, the actual working mechanism of SCS still remains to be explained.

Spinal cord stimulation has clearly been shown to relieve pain in chronic conditions, such as angina pectoris,⁴ failed back surgery syndrome,⁵ and the neuropathic pain syndrome of CRPS I.⁶ The pain-relieving effect of SCS has also been demonstrated objectively in chronic neuropathic pain patients. The amplitude of somatosensory evoked potentials (SSEP) on peripheral nerve stimulation was found to be decreased as a result of SCS; directly at the time of change in SSEP amplitude, patients reported being pain-free.⁶

Neuropathic pain is often accompanied by sensory symptoms, such as allodynia (pain caused by a stimulus that does not normally provoke pain) and hyperalgesia (increased response to a stimulus that is normally painful). It may also involve sensory symptoms, such as hypoalgesia (decreased pain in response to a normally painful stimulus) and hypoesthesia (decreased sensitivity to stimulation).⁷ Whether there is any influence of SCS on these sensory abnormalities has been little studied. Relief of neuropathic pain by SCS might be caused partly by a reduction of the aforementioned painful sensory symptoms⁸⁹; conversely, the paresthesiae that accompany SCS might disturb normal sensibility and produce a decreased sensitivity.¹⁰ Because these studies investigated patients in fairly small groups, often without control subjects, and ultimately produced equivocal results, the true effect of SCS on sensory characteristics is still unknown.

In the current study, the effects of SCS on sensory symptoms were investigated by means of a randomized controlled trial in patients with chronic CRPS I. Patients were allocated either to a group receiving both SCS and...
physical therapy (PT) or to a group treated with PT alone. Detection thresholds and pain thresholds for pressure, warmth, and cold and the extent of dynamic and static hyperalgesia were determined at baseline and at 1, 3, 6, and 12 months after implantation of the SCS system. The findings will be used to increase our understanding of the mechanism of action of SCS. To rule out initial adaptation effects, this paper will focus mainly on the 12-month results.

Patients and Methods

Patients

Patients meeting the following criteria were considered for participation in the study: aged 18–65 yr; CRPS I according to diagnostic criteria of the International Association for the Study of Pain; CRPS I clinically restricted to one extremity but affecting the whole of the hand or foot; duration of CRPS I of at least 6 months; no lasting success with standard therapy, which had to include 6 months of PT, sympathetic blocks, transcutaneous electrical nerve stimulation, and medication; and a mean pain intensity of at least 5, measured on a visual analog scale ranging from 0 (no pain) to 10 (very severe pain) according to Jensen. Exclusion criteria were as follows: a previous diagnosis of Raynaud disease; a history of neurologic abnormalities not related to CRPS I; conditions affecting function of the diseased or the contralateral extremity other than CRPS I itself; blood clotting disturbances or anticoagulant drug therapy; or an implanted cardiac pacemaker. All patients completed a standardized psychologic test, the Symptom Check List-90. Patients scoring 200 or more on this scale underwent a full examination by a psychologist to rule out significant drug habituation problems, major psychiatric diagnoses, or significant personality disorders and to address issues of secondary gain. Patients who were considered on the basis of the examination to have substantial psychopathology were to be excluded. The study complied with the Declaration of Helsinki regarding investigations in humans and was approved by the Medical Ethics Committee of Maastricht University Hospital, Maastricht, The Netherlands. All patients gave written informed consent.

Study Protocol

The study protocol is illustrated in figure 1. After completion of the baseline assessment (T₀), a concealed randomization procedure was used and patients were assigned either to a group with SCS plus a standardized PT program (SCS+PT group) or to a group with the standardized PT program alone (PT group). The randomization used a 2:1 ratio in favor of the SCS+PT group; 36 patients were allocated to SCS+PT and 18 were allocated to PT. All SCS+PT patients underwent 1 week of test stimulation. The decision to implant the SCS system permanently was made if during the past 4 testing days a 50% decrease in original (T₀) visual analog scale score was measured, or if the patient reported “much improved” or “best ever” on a global perceived effect scale (a seven-point scale, indicating “worst ever,” “much worse,” “worse,” “not improved/not worse,” “improved,” “much improved,” and “best ever”). Patients who did not meet these criteria continued to participate in the study with PT alone. Twenty-four SCS+PT patients responded successfully to test stimulation and then underwent SCS implantation. Therefore, 24 patients received an SCS system (with implant; SCS group), whereas this was not true for the remaining 30 patients (without implant; control group). The surgical procedures of test stimulation and SCS implantation as well as details about power calculation and randomization have been described previously.

Data Collection and Follow-up

Outcome measures were assessed before randomization (T₀) and on the day before implantation for patients in the group assigned to SCS+PT and before the start of PT for the patients in the PT group (T₁). Additional assessments were performed 1 month (T₂), 3 months...
(T1), 6 months (T4), and 12 months (T5) after the initiation of treatment.

Randomization was employed for blocks of six patients—patients from the same block were always examined within 1 week. In all cases, the unaffected side was tested first, followed by the affected side. Subjects were tested in a quiet room after the procedure had been explained. Room temperature was maintained at 21–23°C. Examination of SCS patients were performed while stimulation intensity of the system was at a constant level resulting in pain relief. The full examination was completed within 2 h.

**Pressure Sensibility.** The Semmes-Weinstein Pressure Aesthesiometer (Smith & Nephew Rolyan Inc., Germantown, WI) was used to measure pressure detection and pain thresholds. The instrument includes a kit of 20 probes, each probe consisting of a nylon monofilament attached to a rod. The probes are all marked with a number ranging from 1.65 to 6.65, representing in tenths of a milligram the logarithm of the force required to bend the monofilament. This force is reproducible as long as the monofilament is pressed into a C shape. Therefore, the finest filament, labeled 1.65, will bend when applied at an angle of 90° to the skin with a force of 0.0045 g, and the thickest filament, labeled 6.65, will bend at 447 g.

To evaluate all nerves supplying the hand or foot, nine stimulation sites representative of various peripheral nerves and dermatomes were tested (fig. 2); the mean of nine pressure scores was used in further calculations. Ascending levels of filament forces were applied up to the pressure detection threshold—a level that the subject is able to detect in at least 2 of 3 trials. The pressure pain threshold—the minimum force at which the subject reports pain—was then determined in a similar manner. The filament was applied perpendicularly to the skin, with care being taken to avoid contact with hairs or skidding. Stimulus timing was as follows: 1 s for placement, 1 s for bending, and 1 s for removal of the filament. From the thresholds obtained at the aforementioned nine sites, the mean detection and pain thresholds for the hand and foot were calculated in grams. The hands were examined while the subject was sitting; examination of the feet was performed with the subject in a supine position. During testing, subjects were required to keep their eyes closed and were thus unable to observe either their hands and feet or the probes. On the unaffected side, pressure pain cannot be measured with monofilaments; here, the maximal filament load (447 g) is not perceived as painful.

**Warmth and Cold Sensibility.** Thresholds for warmth, cold, heat-induced pain, and cold-induced pain were measured using a Thermal Sensory Analyser (TSA2001; Medoc Ltd., Ramat Yishai, Israel). The TSA2001 operates on the Peltier principle: passing an electrical current through two dissimilar semiconductors displaces heat in the direction of the current. If a good heat conductor, such as a metallic plate, is juxtaposed to one side of the semiconductor system, it is heated or cooled depending on the direction of the current. The opposite side of the system is buffered by water at a temperature of 20°C, this acting as a heat sink or heat source, depending on the direction of current. The temperature at the surface of the stimulator probe (thermode) is measured through a built-in thermocouple made of dissimilar wires (copper and constantan), the voltage difference of which varies in response to change in temperature. A rectangular thermode with a surface of 5 × 2.5 cm was used for cutaneous stimulation and was attached to the skin by means of an elastic Velcro (Velcro USA Inc., Manchester, NH) tape, with care being taken to minimize variation of thermode application pressure. The thermode was applied at a standard baseline temperature of 32°C, and to prevent thermal injury and protect the Peltier instrument, the temperature limits were set at 0 and 50°C. When these temperatures were reached, current direction was automatically reversed, thus returning the temperature to the baseline value.

Warmth and cold detection thresholds were assessed using the Method of Levels; at each temperature step, subjects responded with “yes” if a thermal sensation was
perceived or “no” if this was not the case. For warmth, there was an initial temperature step of 2°C, with the temperature returning to adaptation immediately at the time of stimulus termination. The extent of increase or decrease of the subsequent stimulus was dependent on the subject’s reaction—it remained constant as long as the patient continued to provide a sequence of wholly positive or wholly negative responses but was halved if either sequence was broken. The procedure was continued until interval size reached 0.1°C. For cold, an initial temperature step of 1°C was used, this being the only difference compared with the procedure to assess the warmth perception threshold. The time interval between subject response and the subsequent stimulus was 6 s, the final result being the mean of two measures. Warmth and cold pain thresholds were assessed with the Method of Limits (a linear change in temperature of 1.5°C/s, an interstimulus interval of 10 s, and a mean derived from four stimuli). Thresholds for the foot were assessed at the dorsal aspects of both feet, immediately proximal to the bases of the second and third toes. Thresholds for the hand were assessed at the volar aspects of both wrists, immediately proximal to the base of the hand.

Mechanical Hyperalgesia. Dynamic hyperalgesia was assessed by transiently stroking the skin with a soft brush, and static hyperalgesia was evaluated by applying gentle mechanical force by manual pressure. The tests were performed on the aforementioned nine stimulation sites. Patients were asked to rank on a scale from 0 to 10 the degree of pain evoked when the mechanical stimuli were applied. The tests were performed only on affected extremities—the described stimuli being in general not painful for unaffected extremities.

Statistical Analysis
In this explanatory trial, patients with an implanted SCS system were compared with those without: the implant effect analysis. The decision to combine data from patients in the SCS+PT group with failed test stimulation and those from patients in the PT group was based on clinical grounds (both groups eventually received the same treatment). A secondary analysis was performed according to the intention-to-treat principle: all patients remained in the group to which they had been assigned by randomization.

Differences between T₁ (just before the start of treatment) and T₅ (12 month) values for each individual were calculated and compared between the two groups using means, SDs, and t tests for normal distributed parameters (warmth and cold thresholds) or medians, interquartile ranges, and nonparametric tests if the results were not normally distributed (pressure thresholds and mechanical hyperalgesia). The decision to use T₁ instead of T₀ values to measure the effect of treatment was made to prevent bias; improvements before the actual start of treatment (between T₀ and T₁) have been demonstrated by this study (a project that could not possibly be blinded because of the paresthesiae that accompany stimulation).19 Change of detection and pain thresholds, and mechanical hyperalgesia for the complete group of patients (n = 53) was evaluated through paired samples tests. Two-tailed P values of less than 0.05 were considered to indicate statistical significance.

Results
Fifty-four patients, aged 21–65 yr (37 women and 17 men) with chronic CRPS I of one extremity who had been referred to our department during the period from March 1997 to July 1998, took part in the study. In 33 patients, an arm was the affected limb; in 21, a leg was the affected limb. Of these 54 patients, one (assigned to PT) refused to undergo any physical test after T₀, and this subject was not included in the analysis. Randomization was performed successfully, and both the intention-to-treat analysis groups and the implant effect analysis groups proved largely similar at baseline regarding outcome measures (table 1). Four patients in the PT group and one patient in the SCS+PT group were lost to follow-up after the 6-month assessment (T₄). To prevent loss of information, their T₄ scores were also used in the T₅ analysis.

Table 2 presents changes in quantitative sensory test parameters between 12-month and start-of-treatment results (T₅ to T₁) for the implant effect analysis and for the complete group of patients (n = 53). On the unaffected side, the changes in SCS patients and controls between T₁ and T₅ were not significantly different. Of the complete group of patients (n = 53), only the cold pain threshold changed on the unaffected side (P < 0.001). The findings obtained on the affected side are described below.

Detection Thresholds
Figure 3 shows the absolute detection thresholds for pressure, warmth, and cold during the study period. Warmth and cold detection thresholds were not influenced by SCS. At T₅, the pressure detection threshold did not differ significantly from the T₁ threshold. However, at 1 month (T₂, P = 0.04) and 3 months (T₃, P = 0.03) after implantation, SCS patients demonstrated mechanical hypoaesthesia. In patients with an SCS system (n = 24), correlation coefficients between pain change and change in detection thresholds were −0.06 for pressure, 0.21 for warmth, and −0.28 for cold. Detection thresholds of the complete group of patients (n = 53) did not change between T₁ and T₅ (table 2).

Pain Thresholds
Figure 4 shows the absolute pain thresholds for pressure, warmth, and cold during the 12-month follow-up.
period. Although the graphs seem to show an increase in the pain threshold of SCS patients (especially for pressure), this increase was found not to differ significantly from the increase in the control group (table 2). Therefore, SCS showed no effect on pain thresholds for pressure, warmth, or cold. In patients with an SCS system (n = 24), correlation coefficients between pain change and change in experimental pain thresholds were 0.05 for pressure, −0.42 for warmth, and 0.08 for cold. The visible increase of pain thresholds in figure 4 could be measured by testing the affected side of the complete group of patients (n = 53); pain thresholds were significantly warmer and colder at T1, T2, and T3 (P < 0.005) as compared with T1. In the case of pressure pain threshold.

Table 2. Change of Quantitative Sensory Analysis Results after Twelve Months (T0 to T1) in Patients with and without SCS and in All Patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>With Implant (n = 24)</th>
<th>Without Implant (n = 29)</th>
<th>P Value</th>
<th>Complete Group (n = 53)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pressure (g; median difference [IQR])</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>DT-A (n = 20)</td>
<td>0 (−0.1/0.2)</td>
<td>0 (−0.2/0)</td>
<td>0.16</td>
<td>0 (0.1/0.1)</td>
<td>0.09</td>
</tr>
<tr>
<td>DT-U (n = 20)</td>
<td>0 (−0.1/0.1)</td>
<td>0 (−0.2/0)</td>
<td>0.39</td>
<td>0 (−0.1/0)</td>
<td>0.17</td>
</tr>
<tr>
<td>PT-A (n = 20)</td>
<td>2.2 (0/192)</td>
<td>0.2 (−0.9/6.6)</td>
<td>0.18</td>
<td>0.7 (−0.2/8)</td>
<td>0.007</td>
</tr>
<tr>
<td><strong>Mechanical hyperalgesia (0−10 scale; median difference [IQR])</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>DH-A (n = 29)</td>
<td>−0.4 (−1.6/0)</td>
<td>0 (−0.5/0.1)</td>
<td>0.06</td>
<td>−0.1 (−1.2/0)</td>
<td>0.007</td>
</tr>
<tr>
<td>SH-A (n = 29)</td>
<td>−0.3 (−2.7/0)</td>
<td>0 (−0.6/0.3)</td>
<td>0.07</td>
<td>−0.2 (−0.2/8)</td>
<td>0.005</td>
</tr>
<tr>
<td><strong>Warmth (°C, mean difference ± SD)</strong></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DT-A (n = 29)</td>
<td>0.1 ± 1.6</td>
<td>0.4 ± 1.5</td>
<td>0.44</td>
<td>0.3 ± 1.5</td>
<td>0.02</td>
</tr>
<tr>
<td>DT-U (n = 29)</td>
<td>−0.2 ± 1.8</td>
<td>0.5 ± 1.5</td>
<td>0.19</td>
<td>0.2 ± 1.7</td>
<td>0.43</td>
</tr>
<tr>
<td>PT-A (n = 29)</td>
<td>1.3 ± 2.4</td>
<td>2.1 ± 3.2</td>
<td>0.34</td>
<td>1.8 ± 2.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PT-U (n = 29)</td>
<td>1.3 ± 2.5</td>
<td>−0.1 ± 3.4</td>
<td>0.12</td>
<td>0.5 ± 3.1</td>
<td>0.21</td>
</tr>
<tr>
<td><strong>Cold (°C; mean difference ± SD)</strong></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>DT-A (n = 29)</td>
<td>0.3 ± 1.8</td>
<td>−0.4 ± 1.3</td>
<td>0.11</td>
<td>−0.1 ± 1.6</td>
<td>0.71</td>
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<tr>
<td>DT-U (n = 29)</td>
<td>0.1 ± 1.3</td>
<td>−0.4 ± 1.3</td>
<td>0.16</td>
<td>−0.2 ± 1.3</td>
<td>0.29</td>
</tr>
<tr>
<td>PT-A (n = 29)</td>
<td>−3.3 ± 6.7</td>
<td>−5.0 ± 7.7</td>
<td>0.38</td>
<td>−4.2 ± 7.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PT-U (n = 29)</td>
<td>−4.1 ± 5.6</td>
<td>−3.1 ± 7.8</td>
<td>0.59</td>
<td>−3.6 ± 6.9</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*P values for the difference between T2 to T0 change score of the group with implant (n = 24) and the group without implant (n = 29). †P values for the difference between T1 and T0 scores of the complete group of patients (n = 53).

SCS = spinal cord stimulation; DT = detection threshold; PT = pain threshold; DH = dynamic hyperalgesia; SH = static hyperalgesia; A = affected side; U = unaffected side; IQR = interquartile range.
Mechanical Hyperalgesia

The extent of both dynamic and static hyperalgesia was reduced in the SCS group, but not in the control group (fig. 5). In the SCS group, the median value of dynamic hyperalgesia was reduced by 0.4 ($P = 0.06$), and the median value of static hyperalgesia was reduced by 0.3 ($P = 0.07$) on a scale from 0 to 10. In patients with an SCS system ($n = 24$), correlation coefficients between pain change and change in hyperalgesia were 0.31 for dynamic and 0.39 for static hyperalgesia.

Although the decrease of mechanical hyperalgesia be-
between T1 and T5 was not statistically different for SCS patients and controls, the complete group of patients showed a significant reduction of both dynamic (P < 0.007) and static hyperalgesia (P < 0.005).

**Discussion**

Our study, the first randomized controlled trial on SCS in CRPS I, provides evidence that in chronic CRPS I patients, SCS neither reduces painful sensory symptoms (it caused no increase in pain thresholds or reduction of hyperalgesia) nor produces a decreased sensitivity (it generated no long-term increase in detection thresholds). Another report from this study has demonstrated that with strict selection procedures and successful test stimulation, SCS reduces pain and improves health-related quality of life but does not change functional status.1 Pain caused by normally painless stimuli (allodynia) is frequently reported and disabling in CRPS I.7 The fact that SCS does not relieve allodynia should be clearly communicated to potential candidates for this treatment.

Because SCS stimulates in the main Aβ fibers, it is conceivable that the treatment affects the normal functioning of such sensory fibers. Indeed, previous studies have reported diminished sensations of vibration and touch during SCS treatment10 and demonstrated that during peripheral nerve stimulation, the evoked SSEP is longer in latency and lower in amplitude and is also accompanied clinically by a rise in the touch threshold.8 However, these investigations involved small groups (five subjects in one case and eight in the other) and focussed purely on short-term effects. Nevertheless, the current study has confirmed the findings in these small patient groups. Interestingly, however, we found that the initial increase of the pressure detection threshold in the SCS group faded after 3 months and that at 6 and 12 months, SCS patients again demonstrated detection thresholds similar to those of nonimplanted patients. This observation indicates that SCS may stimulate Aβ fibers, initially interfering with the normal conduction of these fibers. In time, however, the Aβ fibers seemingly adapt to the distracting electricity from the stimulator, at which point the original function returns. The fact that SCS did not influence warmth and cold detection thresholds can be considered indirect evidence that electrical stimulation does not affect Aβ or C fiber function.

Although SCS in all cases in our study was set to treat one extremity, additional stimulation of the contralateral extremity often could not be prevented. This side effect seemed not to affect normal sensibility because detection thresholds of the unaffected extremity did not change significantly during 12 months of follow-up.

Pain thresholds for pressure, warmth, and cold increased significantly in the complete group of patients, but the changes were not significantly different for SCS and control patients. Apparently, the application of quantitative sensory tests in itself would seem to have some desensitizing effect on thresholds of all patients. Apart from habituation, this effect may be explained by the fact that psychophysical tests are influenced by the attention and motivation of test subjects. Simply performing the tests can cause adaptation of subjects to extreme pressure, heat, or cold; on successive assessments, subjects may accept increasingly higher stimuli. This finding stresses that treatment evaluation by quantitative sensory tests cannot reliably be conducted in the absence of a control group.

Several studies in patients with chronic painful neuropathies and in normal subjects with acute experimental chemogenic pain have indicated that the severity of background pain correlates with the intensity of experimental pain.20,21 Therefore, it seems remarkable that this correlation could not be demonstrated in the current study (0.42 being the highest correlation coefficient). There may be some explanations for the apparent
differences between findings in our study and others. First, previous studies involved small sample sizes without control groups and even then focused on subgroups. The current study assessed the effect of SCS on sensory function of the complete group of CRPS I patients. In our opinion, only effects that generate a difference between the SCS group and the control group are of clinical relevance. Second, whereas previous experiments were terminated within 1 day, the current study had a 12-month follow-up. Possibly, the influences of attention and motivation on psychophysical testing weigh stronger in short experiments.

Mechanical hyperalgesia seemed to be the only sensory characteristic that remained reduced in SCS patients, even after 12 months. An effect of SCS has also been found for heat hyperalgesia by Marchand et al. However, the reduction of both dynamic and static hyperalgesia in our study was less than 0.5 on a scale from 0 to 10 and not statistically significant. Therefore, this finding might be considered of limited value for the outcome of individual patients. Studies on neuropathic pain patients have indicated that dynamic hyperalgesia is mediated by myelinated A fibers, whereas static hyperalgesia depends on unmyelinated afferents. Therefore, our finding of reduced mechanical hyperalgesia is also of limited value because other pain measures of Aβ (mechanical allodynia) and C fibers (warmth allodynia) were not influenced by SCS. A possible explanation for the changes in mechanical hyperalgesia might be that this measure is dependent on reported pain scores. It is arguable that such measures can easily be influenced because patients are likely to remember their previous scores.

The effect of clinical use of SCS, considered as a management trial, was studied using an intention-to-treat analysis. In that study, one third of patients in the intention-to-treat SCS+PT group did not respond to test stimulation positively and consequently did not receive an implant. Because the current study conversely was aimed at assessing the effect of SCS treatment on sensory characteristics, the nonimplanted patients in the SCS+PT group would have confused the issue. Therefore, this study has focused mainly on the implant effect analysis, comparing patients with an SCS system to those without.

Most work assessing the effect of SCS on experimental detection and pain thresholds is now somewhat dated. The main aim of these studies had been to find a measure that could objectively evaluate the effect of SCS. The investigators attempted to extrapolate quantitative sensory test results into some kind of measure of subjectively perceived pain; for example, attempts to express pain relief in degrees centigrade. Now, it is commonly accepted that pain is not a homogeneous sensation but a constellation of different sensitivities in normal and diseased states. The same symptom may be produced by different mechanisms, and, conversely, a single mechanism may elicit different symptoms. This implies that subjectively perceived pain is not necessarily paralleled by an increase in experimental pain from various stimuli and, likewise, relief of subjectively perceived pain does not have to be correlated with relief of experimental pain. Therefore, the original aim of applying quantitative sensory analyses as objective pain measurements is theoretically impossible. However, presence of important disabling sensory symptoms, such as allodynia and hypoesthesia, can be measured exactly only by such sensory analyses. In addition, the acquired information is of value for the classification of pain, and may help to reveal why SCS results in pain relief.

Although SCS has been shown to produce significant relief of pain in chronic CRPS I, the current study has demonstrated that the treatment has no effect on experimental pain thresholds for pressure, warmth, and cold and only a slight influence on the extent of dynamic and static hyperalgesia. In addition, SCS seemed not to produce a decreased sensitivity, such as an increase of detection thresholds, either on treated CRPS I limbs or on contralateral extremities.

References


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