Spinal cord stimulation paresthesia and activity of primary afferents

Case report

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A patient with failed back surgery syndrome reported paresthesia in his hands and arms during a spinal cord stimulation (SCS) screening trial with a low thoracic electrode. The patient’s severe thoracic stenosis necessitated general anesthesia for simultaneous decompressive laminectomy and SCS implantation for chronic use. Use of general anesthesia gave the authors the opportunity to characterize the patient’s unusual distribution of paresthesia. During SCS implantation, they recorded SCS-evoked antidromic potentials at physiologically relevant amplitudes in the legs to guide electrode placement and in the arms as controls. Stimulation of the dorsal columns at T-8 evoked potentials in the legs (common peroneal nerves) and at similar thresholds, consistent with the sensation of paresthesia in the arms, in the right ulnar nerve. The authors’ electrophysiological observations support observations by neuroanatomical specialists that primary afferents can descend several (in this case, at least 8) vertebral segments in the spinal cord before synapsing or ascending. This report thus confirms a physiological basis for unusual paresthesia distribution associated with thoracic SCS.

Key words • spinal cord stimulation • stimulation paresthesia • evoked potential • neuroanatomy • side effect • case study

For nearly the entire history of SCS, clinicians have considered that concordance of stimulation-induced paresthesia with a patient’s topography of pain is a necessary (but not sufficient) condition for successful treatment of pain.9 This paresthesia, however, often exceeds the area of pain, and if this side effect is excessive or uncomfortable, it can compromise treatment.8

Upper-extremity paresthesia is an uncommon side effect of the common practice of placing low thoracic SCS electrodes for the treatment of low-back and leg pain. Sweet and Wepsic33 reported a case in 1974; to our knowledge this phenomenon has not been described since. During the past 3 decades, R.B.N. has observed this phenomenon occasionally, in no more than 1% of patients. We present the case of a patient with failed back surgery syndrome who reported paresthesia in his hands and arms during a successful SCS screening trial with a low thoracic electrode. He was the first patient to report this unusual paresthesia who also required general anesthesia at the time of SCS electrode implantation for chronic use. (We routinely implant thoracic laminectomy electrodes under local anesthesia.) Thus, we used this opportunity to investigate this unusual phenomenon.

Case Report

History and Examination. This 64-year-old man with chronic pain refractory to medical therapy after 5 lumbosacral spine surgeries was referred for SCS. He complained of low-back pain (which he estimated accounted for 60% of his pain) and right-leg pain in a sciatic distribution. Examination revealed concordant neurological abnormalities (absent ankle reflex, weakness and sensory loss in an L-5 distribution) and no functional signs. Routine psychological testing revealed no comorbidity that would contraindicate the intervention.

Routine pre-SCS thoracic spine MRI (Fig. 1) revealed stenosis spanning T9–12 that was sufficient to explain some of the patient’s symptoms and warranted decompression, as it posed a risk for progressive neurological deficit. It was unlikely, however, that decompression would relieve the patient’s pain to the point where he would no longer be a candidate for SCS.
Accordingly, we (R.B.N. and K.S.) first performed a 12-day percutaneous SCS screening trial with a small-diameter 4-contact electrode (Axxess, St. Jude Medical). During the trial, the patient reported paresthesia concordant with his right sciatica, with resulting 100% relief of this component of his pain. He reported no paresthesia coverage and no relief of his low-back pain. He also reported extraneous bilateral paresthesia in his arms and hands.

Because the patient required a decompressive T9–12 thoracic laminectomy and because a plate/paddle electrode (which requires a mini-laminectomy for implantation) has been shown to improve low-back paresthesia coverage,10 we offered the patient implantation of such an electrode at the time of the laminectomy. He proceeded to uneventful laminectomy and implantation at T-8 of a plate/paddle electrode (Exclaim, St. Jude Medical Neuromodulation) (Fig. 2). Unlike our usual thoracic electrode implantation via mini-laminectomy, easily done in most cases under local anesthesia, this multilevel procedure required the use of general anesthesia.

During the procedure, the neurologist (P.J.F.) and his assistant (L.R.) monitored SSEPs with transdermal electrical stimulation of the median and peroneal nerves and made SSEP recordings from multiple scalp channels (C3, C4) with screw electrodes according to standard procedures.5 We used the Nicolet Endeavor CR for intraoperative monitoring.

The anesthetic agents used during the 4-hour procedure included fentanyl (total of 500 mcg), midazolam (2 mg), lidocaine (total of 170 mg), succinylcholine (160 mg), rocuronium (total of 20 mg), metoclopramide (10 mg), odansetron (4 mg), propofol (total of 335 mg), and cefazolin (1 g). The estimated blood loss was 200 ml, the urine output was 160 ml, and the total volume of lactated Ringer solution infused was 2700 ml.

As it has been our longstanding practice to assess the symmetry of electrode placement when implanting cervical plate/paddle electrodes under general anesthesia, we placed electrodes over the ulnar nerves at the wrist and the posterior tibial nerves at the ankle to record antidromically conducted nerve evoked potentials at physiologically relevant amplitudes. In this case, we delivered stimulation to the T-8 level with longitudinal bipolar con-

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**Fig. 1.** A: Sagittal T2-weighted MR image of the spine showing thoracic stenosis spanning T9–12, with obliteration of the CSF space around the spinal cord due to a combination of bulging discs and osteophytes anteriorly and ligamentum flavum and facet joint hypertrophy posteriorly.  
B and C: Axial T2-weighted images obtained at levels corresponding to the arrows in panel A showing that the caliber of the CSF space is normal (B) half a segment from one of the levels of maximum stenosis (C), where the CSF space is obliterated and the spinal canal is slightly deformed.
Unusual neuroanatomy of primary afferents

We obtained SNAP thresholds from the left and/or right anterior tibial nerves over a range of stimulation intensities (0.5–5 mA), with the right-left position of the SCS electrode adjusted until we recorded symmetrical SNAPs in the left and right posterior tibial nerves. We also recorded upper-extremity SNAPs and examined them for any objective correlate for the unusual paresthesia reported by the patient.

Findings. Stimulation of the spinal cord in the midline created reproducible (an average of 268 trials) SNAPs recorded from the left and right posterior tibial nerves with a threshold of 2.7–3.75 mA with latencies of 22.6 msec in the left and 22.1 msec in the right. Consistent with the patient’s report of arm and hand paresthesia, we also observed a reproducible SNAP in the right ulnar nerve at the same threshold and slightly decreased latency (19.3 msec) (Fig. 3). Control stimulation of adjacent non–spinal cord tissue did not cause this result.

Postoperative Course. Postoperative SCS programming on multiple occasions revealed that bilateral upper-extremity paresthesia could be elicited consistently, using the same contact combinations and pulse parameters that afforded the best coverage of the patient’s low back and leg pain. The most detailed programming session, 4.5 months postoperatively, showed that this occurred consistently at amplitudes just above the threshold for low-back coverage and well below the discomfort threshold. As of the last follow-up, nearly a year postoperatively, the patient reported ongoing use of SCS up to 14 hours per day, with 90% coverage and 90% relief of his otherwise intractable low-back and leg pain.

Discussion

Electrophysiological testing and monitoring have been used for many years to investigate the mechanism of action of SCS and to optimize patient and electrode placement. Here, we used these techniques to provide objective physiological confirmation of what had until this point been merely a subjective SCS side effect.

In his more than 3 decades of experience with SCS, R.B.N. has noted an occasional patient report of upper-extremity paresthesia with low thoracic to midthoracic electrode placement. This unusual paresthesia seemed at first to be of questionable physiological significance. Similar to other motor and sensory SCS-evoked phenomena, however, the paresthesia seemed to occur in an amplitude-dependent fashion and to be reproducible in affected patients with specific electrode contact positions and pulse parameters. Typically, the abnormal sensations followed a T1–2 distribution (axillae, inner arms, ulnar border of hands). Thus, an anatomical and physiological substrate seemed likely.

Neuroanatomists teach us that after entering the dorsal column, sensory fibers may descend for 1 to 2 levels before synapsing, but this would not explain our clinical observations. In contrast, the observation that branches of sensory afferents may descend up to 10 levels in the fasciculus interfascicularis offers an anatomical explanation for the physiological response that patients have reported and we have observed.

To our knowledge, this is the first report of a physiological demonstration of an intact sensory afferent pathway in a human that spans 8 levels from the cervical to the midthoracic cord to the peripheral ulnar nerve. Sweet and Wepsic reported a similar clinical observation in 2 patients in whom stimulation of the posterior column at C-2 provoked ipsilateral face and arm sensations. These investigators ascribed their observation to “presently undescribed anatomicallyphysiological connections” that they believed were “the most easily explained of the rostral references because of the extension of the spinal trigeminal tract into the uppermost cervical segments lateral to the fasciculus cuneatus.” Clinicians have since taken

Fig. 2. Intraoperative fluoroscopic image showing the electrode array in place, beneath intact laminae at T-8; the decompressive laminectomy extends below.

Fig. 3. The averaged action potential is recorded over the left (A) and right (C) ulnar nerves ~ 2 cm proximal to the wrist crease and over the left (B) and right (D) common peroneal nerves ~ 2 cm anterior to the head of the fibula. The arrows show the peak along with the stimulus locked peak time latencies. Stimulation parameters: 5.1 mA current, 4.7 Hz rate, and 300 µsec duration. Display settings: time base = 100 msec, sensitivity = 1 mV.
advantage of this neural pathway to treat facial pain with high-cervical SCS.3

Potentials evoked by SCS can be recorded not only from the scalp, like orthodromic somatosensory potentials evoked by peripheral nerve stimulation, but also antidromically from peripheral nerves. Conversely, SCS electrodes can be used to record potentials evoked by peripheral stimulation.2 Yingling and Hosobuchi described the use of antidromically evoked potentials to guide SCS electrode placement by laminectomy under general anesthesia.4 Recording over major peripheral nerves (for example, the posterior tibial or inguinal nerves) while stimulating the spinal cord reveals evoked potentials reliably enough to determine symmetry with respect to the midline for electrode placement.

Electrophysiological study in patients under general anesthesia might show evidence of this pathway in more patients than report its effects clinically. In the conscious patient, stimulation amplitude is generally limited to what is clinically useful and relevant, and the discomfort threshold is seldom exceeded. In our practice, thoracic laminectomy electrodes are routinely placed under local anesthesia, which allows us to use the “gold standard” of paresthesia mapping in the awake patient but limits opportunities to study this phenomenon. Other centers routinely implant SCS electrodes under general anesthesia.6 It might be possible, therefore, to study a series of patients at such centers and determine the frequency of the phenomenon. Furthermore, the occurrence of upper-extremity evoked SNAPs might help to guide appropriate electrode placement in those patients most likely to experience extraneous, potentially uncomfortable paresthesia.

Conclusions

Our electrophysiological observations provide physiological evidence of a clinical side effect of SCS and support previous neuroanatomical observations2,13 that primary afferents can descend several (in this case, at least 8) vertebral segments in the spinal cord before synapsing or ascending. This confirms the physiological basis for this unusual side effect of thoracic SCS.

Disclosure

Dr. North’s current (Sinai Hospital of Baltimore) and former employers (Johns Hopkins University) received funding from industry (Boston Scientific, Inc., Medtronic, Inc., and St. Jude Medical, Inc.) as does the non-profit Neuromodulation Foundation, of which he is an unpaid officer. He has consulting/Equity interest in Algostim LLC. Ms. Streelman is a consultant for Algostim LLC. Mr. Rowland and Dr. Foreman report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: North, Foreman. Acquisition of data: all authors. Analysis and interpretation of data: all authors. Drafting the article: North, Foreman. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: North. Administrative/technical/material support: Streelman.

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