Rare Side-effects during Spinal Cord Stimulation: Gastrointestinal Symptoms

Marco La Grua, MD

Department of Anesthesiology and Critical Care, Pain Unit, “Misericordia e Dolce” Hospital, Prato, Italy

ABSTRACT

In current medical literature, most reported complications during spinal cord stimulation (SCS) concern technical problems, such as malfunction, migration or breakage of the lead, or internal pulse generator dysfunction, while reports about side-effects caused by SCS are rare. In this clinical report, we describe uncommon and unexplained gastrointestinal (GI) side-effects of constipation, abdominal pain, and distension during SCS in a patient suffering for chronic neuropathic pain caused by failed back surgery syndrome. These GI symptoms disappeared after suspension of SCS and were reduced if the stimulation settings were reduced below paresthesia threshold. The symptoms experienced by our patient could be related to a functional and reversible block of parasympathetic outflow in the GI system since SCS may involve not only dorsal horn structures but also somatic and visceral sensory afferents to these structures in an unpredictable way.

KEY WORDS: Chronic pain, gastrointestinal, implant, internal organ function, SCS, side-effects, spinal cord stimulation.

Introduction

Spinal cord stimulation (SCS) was first used by Shealey et al. in 1967 (1) and is currently a treatment option for chronic neuropathic pain. Despite the high number of patients treated worldwide with SCS, in current medical literature most of reported complication caused by SCS concerns technical problems, such as malfunction, migration or breakage of the implanted lead, or internal pulse generator dysfunction while only few clinical reports deal with side-effects caused by SCS. Gastrointestinal (GI) symptoms as they relate to SCS are rarely described in current medical literature (2–4).

In this clinical report, we described uncommon and unexplained GI side-effects due to SCS in a patient that underwent implantation of a neurostimulation system to control pain caused by failed back surgery syndrome who previously had never complained of GI symptoms.

Case Report

A 49-year-old woman was seen at our Pain Unit for evaluation and management of radicular neuropathic pain to her lower extremities caused by failed back surgery syndrome. Her pain score using a numerical rating scale of 0–10 (NRS) was 8. She had poor analgesic responses to pharmacological treatment with nonsteroidal anti-inflammatory drugs (NSAID), opioids, and antidepressive–anticonvulsant drugs, and a short and incomplete pain relief following four repeated epidural steroid administrations, performed using caudal approach under fluoroscopic guidance. Because of the intractable nature of her pain, a trial of SCS was offered to the patient.

After informed consent was obtained and a psychological assessment was performed (negative for psychologic barriers to success), a percutaneous 8-polar lead OCTAD® (Medtronic Inc., Minneapolis, MN, USA) was placed in the midline of

Submitted: December 23, 2007; accepted: August 25, 2008. Address correspondence and reprint requests to: Marco La Grua, MD, Via Rosso Fiorentino 118. 51100 Pistoia, ITALY. Email: lagruam@yahoo.it

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the posterior epidural space with the distal electrode at the T8 lower end-plate level. During intraoperative stimulation, the patient reported a 90% coverage of her pain areas with stimulation-induced paresthesias.

Throughout the 21-day trial period, the patient reported good pain relief, reporting a 3 on the NRS during stimulation. Her programmed settings during the trial were as follows: active electrodes 3(+), 6(−); pulse width, 300 μsec; frequency, 60 Hz; amplitude 3.2 V. After this 21-day successful trial, permanent implantation was performed positioning a Synergy Versitrel® implanted pulse generator (Medtronic Inc.) into a subcutaneous pouch surgically created in the lower abdomen, connecting a Medtronic bifurcated extension to the previously implanted 8-polar lead. The long duration of the trial period (21 days) is our standard practice, chosen to detect a possible rapid loss of efficacy to stimulation sometimes caused by a placebo effect in patients.

The patient did complain of GI symptoms during this trial period. These symptoms of constipation, abdominal pain, and distension were thought by us to be related to her pharmacological therapy with tramadol (100 mg t.i.d.), which was discontinued after permanent placement of her SCS system, since pain relief obtained with SCS was satisfactory, even in absence of pharmacological intervention. Surprisingly, her disabling GI symptoms of stypsis, constipation, and distension were not lessened after discontinuation of tramadol. Pharmacological treatment with laxatives, simethicone, and prokinetic drugs, such as metoclopramide and domperidone, for these symptoms were ineffective.

The patient reported that these symptoms disappeared after suspension of SCS and were reduced if the stimulation settings were reduced below paresthesia threshold. Unfortunately, however, using these subthreshold parameters of stimulation caused the patient’s pain to recur; even if lower than the preimplant level.

Multiple complex reprogramming, modifying electrode configurations, frequency, and pulse width did not prevent the recurrence of her GI symptoms. Returning stimulation to the settings reported as effective for pain relief during trial stimulation caused the recurrence of GI symptoms after several hours of stimulation.

Following this patient’s preference, we did not attempt to reposition or replace the epidural lead. Currently, after 12 months of stimulation, her side-effects still remain, appearing just when amplitude of stimulation is increased to a level just above paresthesia perceptual threshold. These compromised settings reduce her GI side-effects, but, unfortunately also reduce her pain control (NRS, 5–6).

### Discussion

To our knowledge, only two brief case reports focus on GI symptoms during SCS (2,4). Interestingly, one of these cited case reports deals with two patients experiencing symptoms related to increased parasympathetic tone, resulting in nausea, vomiting, diarrhea, and increased GI motility. These reported symptoms, however, were thought to be evidence of functional sympathectomy with SCS (5). The report by Krames and Mousad (4) dealt with one patient who suffered from abdominal pain and diarrhea caused by irradiated bowel syndrome, who, after SCS, reported a reduction of diarrheal episodes.

It is well known that disabling GI symptoms, including constipation and abdominal pain, may follow spinal cord injury (6) and a tendency toward increased colonic transfer time has been documented in paraplegic patients who have been implanted with sacral nerve root stimulators (7). Other authors report urethral spasm in spinal cord injury patients who had been treated with SCS. This urethral functional obstruction reportedly lasts approximately 3 hours after deactivation of the stimulator (8).

A mechanism that involves antidromic activation of sensory afferents may play a role in the inhibitory effect of SCS on visceromotor activity of spinal neurons (4). The effects of SCS on neural structures located in the dorsal aspect of the spinal cord are still not fully explained. Stimulation may involve not only dorsal horn structures but also somatic and visceral sensory afferents to these structures in an unpredictable way, generating antidromic activation of these fibers and modifying balance between sympathetic and parasympathetic outflow with a partially unexplained mechanism, producing unexpected clinical manifestations.

The GI symptoms experienced by our patient could be related to a functional and reversible block of parasympathetic outflow to the GI system. This block appears to be strictly related to stimulation on, causing the onset of symptoms (constipation, abdominal distension, and pain) several hours after the start of stimulation and disappearing during a 12-h interval after suspending or reducing the stimulation below threshold.

### Conclusion

We have clinically and consistently shown that the GI symptoms abdominal pain, constipation, and distension were directly related to above paresthesia perceptual threshold of SCS in our patient. A review of the literature revealed that the GI side-effects of stimulation are either rare or unreported. The mechanisms of SCS related GI symptoms in this patient is unknown, but might represent either functional sympathectomy resulting in increased GI parasympathetic tone or might be related to antidromic activation of sensory afferents by SCS on visceromotor activity of spinal neurons (4). A review of the literature does not reveal any large multicenter study of the effects of SCS on GI symptomology for us to conclude that this is an effect of the therapy.
Conflict of Interest
Financial resources were supplied by the Pain Unit, “Misericordia e Dolce” Hospital (Prato, Italy). This work has not been presented at any meeting.

References