PARESTHESIA-FREE SPINAL CORD STIMULATION OF THE S1 SPINAL NERVE ROOT FOR REFRACTORY SURAL NEURALGIA: A CASE REPORT

Viet L. Cai, MD and Thomas Simopoulos, MD

In clinical practice, the treatment of neuropathic pain can be particularly challenging. Because up to 1.5% of the general population afflicted with this condition may not respond adequately to conventional treatments, spinal cord stimulation (SCS) gained popularity as a way to enhance management (1). However, in patients with focal painful regions of the feet, SCS has been noted to have unpredictable outcomes mainly because of suboptimal pain-paresthesia overlap and side effects such as extraneous stimulation and variations in paresthesia intensity with changes in patient position (2-4). Lumbar and sacral nerve root stimulation were proposed years ago, utilizing spinal cord stimulator leads placed in a rostral-caudal direction (retrograde), but were often noted for technical challenge upon insertion (5). More recent advances in neurostimulation technology and techniques have permitted an anterograde approach to the nerve root and even the dorsal root ganglion (DRG). Indeed, DRG stimulation has been recently shown to be effective for multiple diagnoses including complex regional pain syndrome, causalgia, failed back surgery syndrome, and chronic postsurgical pain (6-8). It can be a great alternative to traditional SCS as a targeted therapy. Advantages of DRG stimulation include reduced stimulation amplitude secondary to less adjacent cerebrospinal fluid (CSF), diminished chances for unwanted stimulation of nonpainful areas, and appreciable reduction in positional effects on treatment efficacy (9). Similar to SCS, there are limitations to stimulating amplitudes of the DRG that can lead to overstimulation and compromise patient comfort (6). As first reported in 2017 (10), we present the long-term feasibility of paresthesia-free spinal nerve root stimulation in the management of pain related to sural nerve injury.

CASE PRESENTATION

A 63-year-old man with a remote history of left ankle surgeries following traumatic injury, with resultant neuropathic pain secondary to sural nerve injury, was treated successfully with a left L5 DRG stimulator. He re-presents several years after a right Achilles tendon repair with resulting severe right posterolateral ankle pain. Based on the distribution of pain, a right sural nerve block was performed with > 75% relief of pain for 2 months. Cryoablations of the right sural nerve provided him with excellent pain relief for 5 months’ duration. For unclear reasons, the degree and duration of relief decreased with successive cryoablations. We then transitioned to right radiofrequency lesioning (pulsed RFL) L5 and S1 DRG with 90% relief of pain. During sensory stimulation, it was noted that the L5 DRG was discordant with his pain at the time of lesioning. For several years, the patient received serial S1 DRG RFLs, each with near complete relief lasting 4 to 8 months. Over this time period, the patient developed burning at the site of his implantable pulse generator (IPG) when the device was in the “on” mode only. The impression was that there was an electrical leak at the site where the lead inserts into the IPG. The decision was made to replace the existing IPG and add a lead over the S1 nerve root on the right, as close as possible to the DRG.

The patient provided consent and was taken to the operating room and placed in the prone position. His back was prepped and draped in typical sterile fashion and local anesthetic was injected into the surgical site. Using anteroposterior (AP) and 45-degree con-
tralateral oblique imaging, a 14-gauge Coudé needle (Epimed, Dallas, TX) was advanced from the left L4 pedicle to the L4/5 epidural space in a retrograde fashion. The epidural space was confirmed by the saline loss-of-resistance technique. An 8-contact lead was then advanced to the right L5-S1 level terminating medial to the right S1 foramen. Stimulation was then checked with adequate coverage of the right ankle. In the back, one 1.5-inch vertical incision was then created. The lead was then secured in place. The previous battery and anchor sutures were removed. A new battery was inserted and connected to the new SCS right-sided lead as well as the previously functioning left-sided lead. The left lead closest to the pedicle that provided the best paresthesia coverage was connected to the Precision Plus (Boston Scientific, Marlboro, MA) IPG. AP and lateral fluoroscopy images in Fig. 1 show the right S1 nerve root lead, a superior nonfunctional L5 DRG lead, and an inferior functional L5 DRG lead.

At his 2-week postoperative follow-up, the patient had excellent coverage his bilateral ankle pain as well as resolution of the burning at the IPG site. He continued to do well for 2 years but noted periodic calf muscle spasm at times, only with active stimulation. He was unable to leave the stimulator on at night due to paresthesias and noted a recurrence of pain at night with disrupted sleep. He was reprogrammed to the settings shown in Table I, with a frequency of 469 Hz and cycled over a minute as 15 seconds on, 45 seconds off. Contacts were programmed in a fractional manner with 47% of anode on contact 1 and the rest on contact 7, while cathodic energy was split with 64% on contact 3 and 36% on contact 4. The stimulating contacts are in close proximity to the pedicle of S1 on AP view or just above the ventral S1 foramen on lateral view (Fig. 1). An axial computed tomography (CT) axial cut seen in Fig. 2 shows the S1 nerve root lead in the posterior aspect of the right S1 foramen. With this lead placement and settings, he continues to have excellent > 75% relief that is paresthesia-free. He is able to leave the stimulator on for 24 hours of the day. These results continue at follow-up 2 years out. The patient recharges the device consistently at one week intervals. Of note, at 2 years out he no longer uses the left L5 DRG lead on account of spontaneous pain resolution.

DISCUSSION

Nerve root and DRG SCS can be an effective long-
term paresthesia-free therapy for lower extremity neuropathies such as sural neuralgia, given the appropriate settings. For patients who experience motor side effects or who do not tolerate paresthesias while sleeping, the programming settings can potentially be modified to create subthreshold stimulation based on low amplitude, a higher frequency, and a cycling-off time. Motor stimulation still occurred in this patient despite retroneural positioning confirmed by CT scanning (Fig. 2) as well as adequate paresthesia coverage both intraoperatively and postoperatively. Fractionalization of the stimulating cathode over contacts 3 and 4 was not sufficient to preferentially target sensory neurons and alleviate motor activation. Both of these side effects were able to be overcome with higher frequency stimulation and may further enhance therapeutic options in patients with nerve root and DRG stimulation. Due to the common development of spinal stenosis with advancing age, however, the ability of patients to tolerate these leads at the L4/5 and L5/S1 levels in the long run remains uncertain. In our case report, it is questionable whether the S1 DRG was reached given the more caudal position of the DRG in most patients. We use the term spinal nerve root stimulation instead of DRG for this reason.

Table I outlines the stimulation parameters as well as charge delivery used in traditional SCS, paresthesia-free nerve root stimulation, and the 12-month average values from the ACCURATE study (6). Of particular note, in the ACCURATE trial, only 1 of 61 DRG leads were placed at the S1 level; therefore, most of the programming data reflects leads placed at the L4 and L5 levels. High-frequency DRG stimulation delivers significantly more charge per second to the DRG because of more “on” time, but the charge per minute is twice that of low frequency DRG stimulation on account of the cycling-off time. The charge delivery is within one order of magnitude less than traditional SCS, thus with a low recharging burden for the patient. As with

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Paresthesia-Free Nerve Root Stimulation</th>
<th>DRG Stimulation</th>
<th>Traditional SCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amplitude (mA)</td>
<td>0.500</td>
<td>0.8274</td>
<td>2.9297</td>
</tr>
<tr>
<td>Frequency (Hz)</td>
<td>649</td>
<td>19.0</td>
<td>63.6</td>
</tr>
<tr>
<td>Pulse Width (μs)</td>
<td>110</td>
<td>289.9</td>
<td>417.1</td>
</tr>
<tr>
<td>Charge/pulse (μC)</td>
<td>0.055</td>
<td>0.2399</td>
<td>1.222</td>
</tr>
<tr>
<td>Duty Time (% on time)</td>
<td>1.8</td>
<td>0.55</td>
<td>2.66</td>
</tr>
<tr>
<td>Charge/s (μC/s)</td>
<td>35.7</td>
<td>4.56</td>
<td>77.7</td>
</tr>
<tr>
<td>Charge/min (μC/min)</td>
<td>536</td>
<td>274</td>
<td>4660</td>
</tr>
</tbody>
</table>

Table 1. Stimulation parameters and estimated charge delivery for paresthesia-free nerve root stimulation, conventional dorsal root ganglion (DRG) stimulation, and traditional spinal cord stimulation (SCS). The values for the DRG stimulation were derived from the 12-month average parameters from the ACCURATE DRG study (6). The parameters for lower-frequency DRG stimulation and traditional SCS have been described previously.
SCS, it is now unclear what is the best mode/wave form for DRG stimulation.

The management of chronic pain related to nerve injuries supplying the foot at the level of the ankle remains empiric in clinical practice. The standard approach of multimodal analgesic management, rehabilitation, and neuro-blockade form the initial basis of management. Ablative techniques such as cyroanalgesia and/or radiofrequency are usually offered as next-line therapies. Neuromodulatory therapies are kept as last-resort options but it remains uncertain if this conservative approach is best for patient outcomes. It is important to keep in mind that often, injury to any of the 5 nerves at the level of the ankle that supply the foot can cause significant pain on ambulation and as a result be very disruptive to a patient’s quality of life.

The sural nerve provides sensory innervation to the lateral ankle, heel, and dorsal lateral foot. The nerve is derived from the tibial nerve medially and common peroneal nerve laterally, with contributing axons from the L5 and S1 DRGs. In this case, the S1 contributed more on the right and the L5 on the left. Mapping the neural targets via the sensory stimulatory mode with radiofrequency needles can help guide the placement of leads over the necessary spinal nerve roots and DRGs.

Although this case study has the limitations of a single patient and site, as well as the lack of randomization and control, the continued pain control 3 years out, coupled with the return of pain soon after powering off of the device, suggest that higher frequencies of stimulation can be applied to the spinal nerve root with profound paresthesia-free pain relief.

CONCLUSION

Spinal nerve root stimulation in the form of a cycling program and increased frequencies can bring excellent relief without paresthesia on a long-term basis. In a similar manner to SCS, additional programming options at the level of the spinal nerve roots can help overcome stimulating side effects and thereby enhance analgesia and patient satisfaction.

REFERENCES