LETTER TO THE EDITOR

Contact Dissociation From Paddle Leads:
A Report of Two Cases

To the Editor:

INTRODUCTION

The most common forms of lead failure are migration of the implant and fracture of the electrodes (1,2). This is a report of two cases in which stimulation was lost due to dissociation of the contacts from the ventral surface of the paddle. The implications for diagnosis and treatment and a possible cause will be discussed.

Case Reports

Case 1
A 60-year-old white male developed neck and bilateral shoulder pain two and a half years ago. There was no history of trauma. He had numbness and paresthesias along the ulnar border of his left forearm and in his left ring and little fingers. Pinprick sensitivity was decreased along the medial and lateral borders of his left forearm and in his left middle finger. Reflexes and motor testing were normal. A CT myelogram of his cervical spine showed small disc bulges at C3-4, C4-5, and C5-6 without neural impingement. He had been treated with pain medications and epidural blocks without improvement. He had a spinal cord stimulator trial with greater than 50% improvement in his pain. He underwent implantation of a Boston Scientific permanent percutaneous spinal cord stimulator (Boston, MA, USA) and placement of a rechargeable battery above the left hip on August 3, 2010, and was kept in a soft cervical collar for four weeks. Figure 4 shows the AP X-rays pre- and post-revision. Eight months post-op, she reported stimulation in her left upper extremity but no stimulation in her right upper extremity. X-rays revealed a windshield wiper type of displacement of the upper end of the paddle into the left lateral gutter. She underwent repositioning of the permanent cervical spinal cord stimulator on August 3, 2010, and was kept in a soft cervical collar for four weeks. Figure 4 shows the AP X-rays pre- and post-revision. Eight months later she lost coverage. Her IPG was functioning normally, and the impedances were normal. The patient requested removal of the implant and refused to consider any further revision surgery. Therefore, no new X-rays were obtained.

Case 2
A 37-year-old black female developed neck pain with radiation to the entire right upper extremity following a motor vehicle injury on September 21, 2008. She had numbness and paresthesias in all fingers of her right hand and weakness of her entire right upper extremity. Deep tendon reflexes were normal. Pinprick sensitivity was decreased in the entire right upper extremity. Manual motor testing showed the right biceps to be 4/5. An MRI of her cervical spine showed a small, right-sided disc bulge at C4-5 without neural impingement. She had been treated with anti-inflammatories, pain medications, and epidural blocks without improvement. She had a cervical spinal cord stimulator trial with greater than 50% improvement in her pain. She underwent implantation of a Boston Scientific permanent spinal cord stimulator through laminectomies at C3 and C4 and placement of a rechargeable battery above the left hip on October 1, 2010. Eight months post-op, she reported stimulation in her left upper extremity but no stimulation in her right upper extremity. X-rays revealed a windshield wiper type of displacement of the upper end of the paddle into the left lateral gutter. She underwent repositioning of the permanent cervical spinal cord stimulator on August 3, 2010, and was kept in a soft cervical collar for four weeks. Figure 4 shows the AP X-rays pre- and post-revision. Eight months later she lost coverage. Her IPG was functioning normally, and the impedances were normal. The patient requested removal of the implant and refused to consider any further revision surgery. Therefore, no new X-rays were obtained.

Address correspondence to: Jacob Amrani, MD, Deer Valley Spine Center, Phoenix, AZ 85027, USA. Email: dramrani@yahoo.com

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Conflict of Interest: Dr. Amrani has a licensing agreement with Boston Scientific, Inc. Dr. Amrani has no other conflicts of interest to disclose.
On July 18, 2012, the patient underwent removal of the Boston Scientific paddle and IPG. Since this was another second revision, great care was again taken during the dissection. The tails of the implant were again traced to the paddle. The paddle was noted to be in the same position as seen on the X-ray made four weeks post-repositioning. After opening the fibrous sheath over the dorsum of the lead, the bottom half of the lead again popped into the surgical field. Half the contacts were noted to have separated from the ventral surface of the paddle. As noted in the first case, the wires remained in the silastic paddle and, again, did not appear to be attached to the contacts. The paddle and all but one of the disengaged contacts were removed. The most cephalad contact could not be removed without completing the laminectomy at C3 and was left in place (Figure 5). (Although clinically innocuous, this sole contact prevents her from having any further MRI studies. The other option was to leave her with a three-level laminectomy from C3 to C5 and risk a postlaminectomy kyphosis to remove the last contact. This is a judgment call that the surgeon will have to make individually, as there is no literature supporting one approach over the other.)

**DISCUSSION**

In a 2008 paper published in this journal, Akmal and Eljamel (3) stated that SCS “has earned its rightful place in modern therapy against pain because ... the dedication of SCS implanters and the detailed reporting of SCS successes and failures. However, for SCS to retain its current therapeutic and cost-effective edge in the future, long-term hardware failures need to be studied and reported to improve lead design and implantation techniques.”

Note should be taken of the technical difficulties encountered with cervical stimulation, as seen in these two patients. Both patients required revisions of their original implants, and both patients required laminectomies in order to insert the paddle leads. The current author has submitted a separate manuscript documenting the rate of mechanical failure in this area.

When the stimulation from a permanent spinal cord stimulator changes in the first six weeks after implantation, the most likely cause is lead migration. There may or may not be a history of trauma, such as a fall. If the IPG has been inserted correctly, it can be charged and interrogated. The impedances should be normal. If the lead has displaced out of the epidural space, the patient will have no coverage, or the coverage will be limited to the area around the incision, as seen with a peripheral nerve field stimulator. If the area of coverage peripherally changes, the lead has probably displaced within the spinal canal. X-rays are diagnostic. When there is a delayed loss of stimulation, a fracture of the electrode is usually suspected. The X-rays will appear normal, but the impedances will be high.

Akmal and Eljamel (3) reported the incidence of electrical lead failure (i.e., not related to migration) to be 15% in primary cases and 56% in revision cases. The failure rate was 13.5% in the thoracic spine and 22% in the cervical spine. The average time to failure was 39 months. Heidecke et al. (1) reported electrode fractures in 19% of patients treated for failed back surgery syndrome at an average of 15 months after implantation. All fractures occurred in permanent percutaneous leads. Kumar et al. (2) and Cameron (4) reported on mechanical lead failures with lead breakage listed at 9%. Simpson et al. (5) reported his results for cervical paddle lead insertion in 41 patients. He noted lead migration in 7% of patients and lead fracturing in 15%. However, the current author was unable to find any reference to the type of lead failure described in this paper.
Although the development of scar around the paddle might be thought intuitively to cause a loss of stimulation, there are no reports in the literature studying this phenomenon specifically. One would think that the loss of stimulation in such cases would be gradual and would take a significant amount of time to become complete. In both cases reported here, there was a delayed, acute onset of loss of stimulation (8 and 14 months after implantation). IPG function and impedances were normal. In both cases, half the contacts had dissociated from the ventral aspect of the paddle. The fibrous sheath that had formed around the implants prevented the contacts from migrating. This explains why the separation of the contacts was not evident on the X-ray of the first patient.

We do not always know why impedances change. Farris and Giroux (6) published a report in this journal in 2010 of a patient who had normal impedances but lost stimulation on the left side. X-rays showed a clear fracture of the lead. Clarke et al. (7) also reported in this journal in 2011 a case of a patient who lost stimulation in the left occipital lead. Impedances were normal, but the anchoring sutures had cut through the insulation, necessitating replacement of the lead. In Neural Blockade in Clinical Anesthesia and the Management of Pain, Cousins and Bridenbaugh (8) recommends interrogating the IPG and checking the impedances when there has been a loss of previously successful stimulation. He states that the impedances will be elevated if there is a fluid leak or a lead fracture. In Complications in Regional Anesthesia and Pain Management, Neal and Rathmell also state that loss of stimulation and high impedances are signs of fluid leak or lead fracture (9). The impedances may have appeared normal in the cases reported here because the electrodes were still intact. It may be an oversimplification to assume that a disengaged contact would act like a lead fracture, as there are no reports in the literature dealing with electrical testing of this particular situation.

The X-rays in the first case showed a buckling deformity of the paddle. In the patients who received a Boston Scientific Artisan lead, the paddle appeared smooth with the neck in flexion, but assumed a “Z” shape with the neck in extension. Since CT scans were not obtained with the neck in lateral rotation, the type of displacement, if any, of the paddle in the axial plane is unknown. However, the documented motion in the sagittal plane and the delayed onset of the loss of stimulation suggest to the current author that the separation of the contacts from the paddle was the result of fatigue failure. The author has seen only one example of the contacts dissociating from a Boston Scientific lead implanted in the thoracic spine. That followed a fall in the shower four weeks post-op that resulted in the catastrophic failure of the implant. The author believes that the relative immobility of the thoracic spine protects

Figure 3. AP and lateral X-rays after revision. Note the retained contact at C3-4.

Figure 4. AP X-rays pre- and postrepositioning of the paddle.
the implant from this type of fatigue failure. Alternatively, since the paddles were inserted into areas of prior surgery, there is the possibility that the paddle leads were under tension at initial insertion due to scarring and could not settle into a place that tolerated movement.

Dissociation of the contacts from the paddle should be suspected in the patient who presents with a delayed, acute onset of loss of previously adequate coverage when the IPG is functioning normally and the impedances and X-rays appear normal. Caution should be used when implanting a paddle that deforms in a mobile area of the spine. Testing the implants in flexion and extension in animals and cadavers should be considered by the manufacturers.

Authorship Statement
Dr. Amrani was the sole author.

Jacob Amrani, MD
Deer Valley Spine Center, Phoenix, AZ

REFERENCES

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