Tight adhesions after spinal cord stimulation observed during dorsal root entry zone lesioning for pain after spinal root avulsion: illustrative cases

Yuki Kimoto, MD,1 Koichi Hosomi, MD, PhD,1,2 Yuichiro Ohnishi, MD, PhD,1,3 Takuto Emura, MD,1 Nobuhiko Mori, RPT, MHSc,1,2 Asaya Nishi, MD,1 Takufumi Yanagisawa, MD, PhD,1,4 Naoki Tani, MD, PhD,1 Satoru Oshino, MD, PhD,1,2 Youichi Saitoh, MD, PhD,5,6 and Haruhiko Kishima, MD, PhD1,2

1Department of Neurosurgery, Osaka University Graduate School of Medicine, Suita, Osaka, Japan; 2Center for Pain Management, Osaka University Hospital, Suita, Osaka, Japan; 3Department of Neurosurgery, Osaka Gyoumeikan Hospital, Osaka, Japan; 4Institute for Advanced Co-Creation Studies, Osaka University, Suita, Osaka, Japan; 5Osaka University Graduate School of Engineering Science, Toyonaka, Osaka, Japan; and 6Tokuyukai Rehabilitation Clinic, Toyonaka, Osaka, Japan

BACKGROUND Patients often experience strong shooting pains after spinal root avulsion. The efficacy of spinal cord stimulation (SCS) for this type of pain is inconsistent; however, dorsal root entry zone (DREZ) lesioning (DREZ-lesion) has often proven to be an effective treatment modality. The authors report two cases in which DREZ-lesion was performed to treat pain after spinal root avulsion after implantation of SCS, but the operations were challenging due to strong adhesions.

OBSERVATIONS The authors present two cases of patients with pain after spinal root avulsion in whom SCS implantation was only temporarily effective. Patients complained of persistent and paroxysmal shooting pains in the upper extremities. SCS removal and DREZ-lesion were performed, but adhesions in the epidural and subdural space contacting the leads were strong, making it difficult to expose the DREZ.

LESSONS Although adhesions around the spinal cord can be caused by trauma, the authors believe that in these cases, the adhesions could have been caused by the SCS leads. There are few previous reports confirming the efficacy of SCS in treating pain after spinal root avulsion; therefore, caution is required when considering SCS implantation.

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KEYWORDS spinal root avulsion pain; dorsal root entry zone lesioning; spinal cord stimulation

Pain after spinal root avulsion is often caused by strong traction on the upper limbs or shoulders, as occurs in motorcycle accidents, and is characterized by strong shooting pain (paroxysmal pain). Pain occurs in ~70–90% of patients after spinal root avulsion, and 20% of these cases are intractable.1,2 Various pain treatments have been attempted, including dorsal root entry zone lesioning (DREZ-lesion), spinal cord stimulation (SCS), motor cortex stimulation, intrathecal analgesic pump implantation, stellate ganglion block, and thalamic deep brain stimulation.3 DREZ-lesion is effective in treating root avulsion pain, but the efficacy of SCS remains unclear.4–10 The British Pain Society indicated in 2009 that SCS for root avulsion pain is not responsive.11 Recent reports suggest that burst or high-frequency SCS may reduce pain, but the long-term effects are still uncertain.12,13 SCS is often considered an attractive option because it is minimally invasive; however, recent reviews suggest that DREZ-lesion should be performed for pain after spinal root avulsion because of its efficacy in pain relief.12,14 We report two cases in which DREZ-lesion was performed in patients with pain after spinal root avulsion who had SCS devices implanted but there was difficulty in exposure of DREZ due to strong spinal cord adhesions.

Illustrative Cases

Case 1
A 40-year-old male who was involved in a motorcycle accident 8 years before his visit to our hospital, presented with spinal root...
avulsion pain in his left upper limb. He had an SCS device implanted at another hospital 3 years before the visit to our hospital (Fig. 1A and B). The persistent pain subsequently improved, but the paroxysmal pain persisted; strong opioids and stellate ganglion blocks did not improve the pain. The prescription at the time of his visit was fentanyl patch (2 mg), tramadol (50 mg), and acetaminophen (3,600 mg). The SCS was not used at the time of the visit because it was no longer effective. The pain presented in the left hand and the ulnar side of the forearm (C6–T1 region) as paroxysmal pain of numerical rating scale (NRS) 5–9 and persistent pain of NRS 4 (Fig. 2A). The patient also had sensory loss in the left forearm, hyperalgesia in the left C5 and T1–2 regions, and severe motor paralysis in the left upper limb. Magnetic resonance imaging (MRI) revealed a pseudomeningoele on the left side of the C5–6, C6–7, and C7–T1 spinal levels. The nerve roots of the left C5–8 were not visualized. Magnetic resonance neurography showed poor visualization of the left spinal nerves of C5–8 (Fig. 3A). A diagnosis of C5–8 avulsion injury was made based on these symptoms and radiological findings. Computed tomography (CT) myelography indicated that the spinal cord was deviated dorsally in the dural canal and contacted the dura mater just beneath the SCS leads (Fig. 4A–D).

Surgery was performed simultaneously with SCS removal and DREZ-lesion. We performed a hemilaminectomy from C5–7 with a caudal partial laminectomy of C4 and a cranial partial laminectomy of T1 and incised the dura; the ligamentum flavum was tightly adherent to the dura and the dura was thickened. When the dura was incised, the arachnoid membrane immediately beneath the lead was muddy, and the spinal cord at the C7–T1 level was strongly adherent to the dura, making it difficult to expose the DREZ (Fig. 5A–C). Only the DREZ that could be exposed (C5–7) was radiofrequency (RF) coagulated (70°C for 30 s, 51 points at 1 mm intervals), using an RF lesion generator (RFG-3C, Radionics Inc), and an RF lesion needle electrode with a 2 mm tip. Dural reconstruction was performed using muscle flaps and fat. Postoperatively, pain in the area corresponding to the coagulated DREZ (~80% of the total) disappeared, but pain on the ulnar side of the forearm corresponding to the area that could not be coagulated persisted. The persistent pain improved 1.5 years after surgery; however, the patient still experienced NSR 9 paroxysmal pain in the same area (Fig. 2B). His medication regimen remained unchanged (fentanyl patch [3 mg], tramadol [75 mg], and acetaminophen [650 mg]).

Case 2

The second case was a 50-year-old male who had a motorcycle accident 2 years before his visit to our hospital and presented with spinal root avulsion pain in the right upper limb. He received SCS at another institution 1 year before his visit to our hospital.
After SCS implantation, the persistent pain was partially reduced, but the paroxysmal pain did not improve. He was referred to the pain clinic and then to the Department of Neurosurgery. When he was referred to our hospital, his medication comprised tramadol (150 mg), acetaminophen (1,300 mg), and pregabalin (300 mg); SCS was no longer effective at the time of his first visit, and there was no change in pain when it was turned off. The painful area was the entire right upper limb (C5–T1 region), with paroxysmal pain of NRS 9 at 30-minute intervals and persistent pain of NRS 4 (Fig. 2C). There was sensory loss in the right C6–7 region, hyperalgesia in the right C5 and T1–2 regions, paresthesia in the left C6 and below, due to Brown–Séquard syndrome, severe motor paralysis in the right upper and lower limbs and left half-blindness due to right occipital lobe infarction caused by traumatic right vertebral artery injury. MRI revealed high-intensity lesions in the right spinal cord at the C6 spinal level in T2-weighted images and a pseudomeningocele on the right side of the C6–7 and C7–T1 spinal level, and the nerve roots of the right C6–8 were not visualized. Magnetic resonance neurography showed poor visualization of the right spinal nerves of C6–8, and a diagnosis of C6–8 avulsion injury was made (Fig. 3B). Similar to case 1, CT myelography revealed that the spinal cord was deflected dorsally within the dural canal and contacted the dura just beneath the lead (Fig. 4E–H).

Surgery was performed as in case 1, with SCS extraction and DREZ-lesion. When we performed a hemilaminectomy from C4 to T1 and incised the dura, the ligamentum flavum was adherent to the dura mater. When the dural incision was made, the arachnoid was cloudy and partially adherent to the spinal cord (Fig. 5D–F). Although it was time-consuming to carefully detach the ligamentum flavum and arachnoid, we were able to expose the entire DREZ, and RF coagulation (70°C for 30 seconds, 49 points at 1 mm intervals, using an RF lesion generator (RFG-3C) and an RF lesion needle electrode with a 2 mm tip was performed as planned. After surgery, paroxysmal and persistent pain in the right upper extremity disappeared. Paroxysmal pain recurred 8 months after surgery; at 18 months following surgery, persistent NRS 4 pain and paroxysmal NRS 6 pain remained on the outer side of the upper arm (Fig. 2D). However, the extent and degree of pain improved from preoperative levels, and the patient was extremely satisfied with the surgical outcome. Medication was reduced to tramadol (112.5 mg), acetaminophen (975 mg), and pregabalin (150 mg).

**Discussion**

**Observations**

We encountered two cases of DREZ-lesion after SCS implantation. In both cases, the SCS is ineffective, and the DREZ-lesion was difficult to complete because of strong adhesions around the SCS lead. It is known that SCS causes epidural adhesions, and
there are reports that the previous implantation affects the degree of epidural adhesions and prolongation of operative time for reimplantation. In this case, the SCS was suspected to cause the adhesions between the dura mater and the ligamentum flavum.

There are many reports examining the pain-relieving effects of spinal root avulsion suggesting that DREZ-lesion is more effective than SCS. In the previous study, the 5-year postoperative analgesic efficacy of DREZ-lesion for pain after root avulsion (>50% improvement in visual analogue scale [VAS]) was 84.6% for paroxysmal pain and 73.1% for persistent pain, with a higher analgesic efficacy for paroxysmal pain. In our report, 2.5 years after DREZ-lesion surgery, VAS exhibited >50% improvement for paroxysmal pain in 7 of 10 patients, whereas paroxysmal pain disappeared in 5 patients, and persistent pain disappeared in 2 patients.

On the other hand, in studies investigating the effect of SCS on pain after spinal root avulsion, some have reported that it is effective in all cases, whereas others have indicated that it is not effective. There are no reproducible reports. In our experience, SCS was ineffective.

The difference in the pain relief effect could be due to the nature of the pain after spinal root avulsion. Spinal root avulsion causes intractable pain due to afferent pathway blockage and damage to the spinal dorsal horn neurons, which can be divided into persistent and paroxysmal pain according to the underlying mechanism. Paroxysmal pain is believed to be caused by overactivity in layer V of the dorsal horn of the spinal cord due to disruption of inhibitory pathways, whereas persistent pain is believed to result from degeneration of the neurons damaged by avulsion, central sensitization (increased release of glutamate and substance P in presynaptic A-delta and C fibers and altered sensitivity of postsynaptic N-methyl-D-aspartate receptors), and involvement of the limbic system and cerebral cortex, suggesting involvement of both the brain and spinal cord.

DREZ-lesion is a treatment that prevents overactivity by destroying the superficial layers I–V of the dorsal horn of the spinal cord. Based on the underlying mechanism, it is understandable that DREZ-lesion is effective for paroxysmal pain, but its effectiveness may be limited in persistent pain which includes a supra-spinal component.

SCS is based on the gate control theory published by Melzack and Wall in 1965, whereby electrically stimulated dorsal funiculus retrogradely stimulate Aβ fibers that transmit tactile information and inhibit Aδ fibers that transmit pain information. However, in the case of root avulsion, the dorsal horn neurons are damaged and contact between the dorsal funiculus and Aβ fibers is physically broken; therefore, the stimulation provided by SCS may not be conducted retrogradely to the Aδ fibers, and the pain-relieving effect may not be exerted.

For these reasons, DREZ-lesion should be performed preferentially for pain after spinal root avulsion, particularly for paroxysmal pain.

The first limitation of this report is that the epidural adhesions may be traumatic. The adhesions between the arachnoid and dura mater and between the dura mater and the ligamentum flavum were strong and difficult to dislodge during surgery (Fig. 5A–H).

DREZ-lesion resulted in marked improvement of pain in the cases with traumatic adhesions, whereas in cases without traumatic adhesions, the pain relief was minimal.

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Most reports examining traumatic changes after avulsion injury describe pseudomeningocele and none describe adhesions inside or outside the dura. The present cases had pseudomeningocele, but the adhesion sites were different from that of a pseudomeningocele, suggesting that SCS leads may have caused the adhesions. We performed DREZ-lesion in other patients, but none possessed such strong intradural and extradural adhesions as in these cases.
Another limitation is that there are no reports of subdural adhesions after SCS implantation. However, the dorsal deviation of the spinal cord on CT myelography suggests adhesion between the spinal cord and the dura mater beneath the SCS leads, and the dorsal deviation of the spinal cord on axial section imaging is associated with intradural adhesions in tethered cord syndrome.33 There is a possibility that irritation owing to SCS will affect and cause the inflammation to spill over into the subdural area. If the dorsal deviation of the spinal cord is observed on preoperative imaging, it would be important to pay attention to subdural adhesions.

Lessons
In spinal root avulsion, SCS is not very effective for paroxysmal pain. In many patients, paroxysmal pain is more severe and is more problematic than persistent pain. Because previous SCS can fail dorsal root entry zone surgery for brachial plexus avulsion pain. In many patients, paroxysmal pain is more severe and is more problematic than persistent pain. Because previous SCS can make subsequent DREZ-lesion difficult due to adhesions within and outside the dura, caution is required when considering SCS.

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Author Contributions
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Supplemental Information
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Correspondence
Koichi Hosomi: Osaka University Graduate School of Medicine, Suita, Osaka, Japan. k-hosomi@nsurg.med.osaka-u.ac.jp.