Spinal cord mapping with evoked responses for accurate localization of the dorsal root entry zone

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Direct spinal cord stimulation and recording techniques were used intraoperatively to localize the dorsal root entry zone (DREZ) in four patients with brachial plexus avulsion and severe intractable pain. The spinal cord was stimulated by a cordotomy needle placed on the pia-arachnoid at the DREZ or the dorsal or dorsolateral aspect of the spinal cord. Recordings were obtained from a subdural silver ball electrode placed rostral or caudal to the stimulation site. Spinal cord conduction velocity was significantly faster following dorsolateral stimulation than dorsal stimulation (mean = 66 and 45 m/sec respectively). The spinal cord evoked potential was significantly larger in amplitude following dorsolateral stimulation than dorsal stimulation at a specific stimulus intensity. Stimulation at the DREZ failed to evoke a response. These neurophysiological phenomena helped to accurately localize the DREZ before DREZ lesioning was undertaken. There were no untoward neurological deficits related to the DREZ lesions and all patients had satisfactory pain relief following the procedure. Intraoperative spinal cord mapping facilitates accurate DREZ localization when the DREZ cannot be visually identified.

Clinical Material and Methods

Four patients, ranging from 22 to 40 years of age, with brachial plexus avulsion resulting in severe intractable pain underwent electrophysiological spinal cord mapping to localize the DREZ prior to undergoing DREZ coagulation. The operation was performed with the patient in a prone position and the spinal cord was exposed from C-5 to T-1 inclusive. The first three patients had complete laminectomies; the last patient underwent laminotomy followed by laminoplasty to prevent swan-neck deformity. Each patient received pancuronium intraoperatively to obliterate electromyographic responses during evoked
potential recording. In spite of the use of warming blankets, mere exposure of the spinal cord as well as the use of irrigation probably resulted in a subnormal spinal cord temperature by the end of the procedure.

Electrophysiological Recording Techniques

The recording electrode (a silver ball electrode 1 mm in diameter, insulated except for the ball) was placed over the dorsolateral or dorsal aspect of the spinal cord at the rostral or caudal end of the exposed cord. Occasionally the recording electrode was slipped under the dura but remained visible. A needle electrode was placed lateral to the silver ball electrode in the paraspinal muscles to serve as the reference electrode. The stimulating electrode was a cordotomy needle with a diameter of 200 Å (insulated except for the 2.5-mm tip) connected to a constant current unit and a stimulator. A metal plate attached to the patient’s thigh served as the reference electrode. A 100-μsec square-wave pulse and a stimulation rate of one stimulus per second were used. Responses were amplified by amplifiers with a gain of 100,000 and a passband of 3 to 3000 Hz.* Responses were averaged using a computer with an analog-to-digital converter† and a sampling rate of 51.2 kHz. Typically, five to 10 responses were included in each average. Response conduction velocity was calculated by measuring the distance between the stimulating and recording electrodes and dividing by the latency to the initial negative deflection of the response. Response amplitude was measured as the peak-to-peak voltage of the response.

Initially the stimulating electrode was placed where the spinal cord anatomy appeared normal (the level at which the dorsal root filaments were intact and could be seen). The stimulating needle was placed over the pia-arachnoid, between the denticulate ligament and the posterior lateral sulcus (the DREZ), which should be directly over the lateral corticospinal tract and dorsal spinocerebellar tract.

This was termed the dorsolateral stimulating electrode position. The distance between stimulating and recording electrodes varied between 5 and 8 cm. The intensity of the stimulation was gradually increased until a spinal cord evoked potential with an amplitude of approximately 20 μV was elicited. The stimulus intensity necessary to evoke this response was termed the baseline stimulus intensity. Then the stimulating electrode was gradually moved dorsally toward the posterior lateral sulcus in approximately 1-mm steps (Fig. 1). Stimulation and recording were repeated at each step using the same baseline stimulus intensity. This procedure was performed under an operating microscope so that placement of the stimulating needle could be well visualized.

This itinerant stimulating technique was repeated along the long axis of the spinal cord at 1-cm intervals in the area of the avulsed rootlets. The dorsal spinal cord was not stimulated at every interval. Radiocoagulation lesions (25 to 30 mA for 25 to 30 seconds) were made at sites where stimulation failed to evoke a response. The size of the coagulation lesion was observed under the microscope. After making these multiple lesions at 1-cm intervals, the electrophysiological spinal cord mapping techniques were abandoned and the lesion sites were connected in a straight line by additional lesions made at 1- to 1.5-mm intervals using the same intensity and duration (Fig. 2).

Results

Four patients with brachial plexus avulsion were included in the study. Three patients underwent direct stimulation of the DREZ and the dorsolateral and dorsal spinal cord, and one patient underwent stimulation of the

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* Needle electrode, constant current unit, model S44 stimulator, and model 12A amplifiers manufactured by Grass Instruments, Quincy, Massachusetts.
† Datacon A-D board obtained from Clark-Davis Medical, London, Ontario, Canada.

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**TABLE 1**

Reported complication rates in DREZ lesioning

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Patients</th>
<th>Complication Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nashold, et al., 1976</td>
<td>4</td>
<td>25%</td>
</tr>
<tr>
<td>Nashold &amp; Osidahl, 1979</td>
<td>18</td>
<td>50%</td>
</tr>
<tr>
<td>Samii &amp; Moringlane, 1984</td>
<td>35</td>
<td>25%</td>
</tr>
<tr>
<td>Thomas &amp; Jones, 1984</td>
<td>34</td>
<td>47%</td>
</tr>
<tr>
<td>Wynn Parry, 1984</td>
<td>24</td>
<td>40%</td>
</tr>
<tr>
<td>Campbell, et al., 1988</td>
<td>10</td>
<td>0%</td>
</tr>
<tr>
<td>Friedman &amp; Bullitt, 1988</td>
<td>39</td>
<td>60%</td>
</tr>
<tr>
<td>Friedman, et al., 1988</td>
<td>18</td>
<td>50% (mild)</td>
</tr>
<tr>
<td>Ishijima, et al., 1988</td>
<td>19</td>
<td>57%</td>
</tr>
<tr>
<td>Powers, et al., 1988</td>
<td>40</td>
<td>7%</td>
</tr>
<tr>
<td>Saris, et al., 1988</td>
<td>22</td>
<td>44%</td>
</tr>
<tr>
<td>Prestor, et al., 1989</td>
<td>7</td>
<td>14%</td>
</tr>
</tbody>
</table>

*DREZ = dorsal root entry zone.*
dorsolateral spinal cord and the DREZ. The first site to be stimulated in all patients was the dorsolateral spinal cord. A constant-current stimulus intensity between 0.1 and 0.9 mA was required to evoke a spinal cord potential with an amplitude between 10 and 20 µV. This same stimulus intensity failed to evoke a response when the electrode was moved to the visible DREZ, suggesting no stimulation of long tracts (Fig. 3).

Three patients underwent dorsal, dorsolateral, and DREZ stimulation using the baseline stimulus intensity. The mean conduction velocity following dorsolateral spinal cord stimulation was 66 meters per second (m/sec) (standard deviation (SD) = 18, standard error (SE) = 4). Mean conduction velocity following dorsal spinal cord stimulation was 45 m/sec (SD = 4, SE = 1.7). The mean amplitude was 14.3 µV (SD = 4, SE = 1.7) following dorsolateral stimulation and 7 µV (SD = 6, SE = 2.7) following dorsal stimulation. Amplitude and conduction velocity of evoked responses following dorsolateral stimulation were significantly higher than those following dorsal stimulation (paired t-test; T = 2.7, p < 0.04 for conduction velocity and T = 3.1, p < 0.03 for amplitude).

It is interesting to note that one patient showed no response following dorsal spinal cord stimulation at one site when the stimulus intensity that evoked a 20-µV response was used.

Fig. 2. Diagram illustrating dorsal aspect of the spinal cord at the level of dorsal root avulsion. After the dorsal root entry zone was electrophysiologically localized and coagulated at 1-cm intervals, the radiofrequency (RF) lesion sites were connected by additional RF lesions at 1- to 1.5-mm intervals without the aid of electrophysiological spinal cord mapping. Note the position of the stimulating electrode (a cordotomy needle) and silver ball recording electrode placed at the level of the ipsilateral dorsal root fibers.

Fig. 3. Diagram illustrating cross-section of the cervical spinal cord and three different stimulation sites, resulting in three different evoked potentials in one patient. The evoked response was absent when the dorsal root entry zone (DREZ) was stimulated. Dorsolateral spinal cord stimulation (Lateral Column) evoked a larger response with a higher conduction velocity than dorsal spinal cord stimulation (Dorsal Column).
response from dorsolateral stimulation was used. Notwithstanding this fact, when the dorsal spinal cord was stimulated at a different cervical level in this patient, a response was elicited.

**Use of High-Intensity Stimulus**

In one patient, stimulation of the dorsolateral spinal cord using a stimulus intensity of 0.81 mA evoked a relatively large response, with an amplitude of 28 μV and a conduction velocity of 61 m/sec. We prefer to use a stimulus intensity low enough to evoke only a 10 to 20 μV response, but in this patient we went on to stimulate the adjacent DREZ (dorsal roots present and served as landmarks at this level) and dorsal spinal cord using the same stimulus intensity that evoked the 28-μV response. Dorsal spinal cord stimulation evoked a response with an amplitude of 3.8 μV and a conduction velocity of 50 m/sec. Paradoxically, DREZ stimulation also evoked a response (5.3 μV, 57 m/sec), suggesting stimulation of adjacent fast-conducting fibers via stimulus current spread (Fig. 4). Consequently, we reduced the dorsolateral stimulus intensity so that it evoked a 10- to 20-μV response. The lower stimulus intensity failed to evoke a response at the DREZ.

**Neurological Outcome**

Each patient was assessed 1 day, 2 weeks, 6 months, and 1 year following surgery. There was 100% pain relief in all patients immediately after surgery and 6 months later. All patients continued to remain pain free except one patient who had a recurrence of pain localized in his thumb, which was successfully managed by nonnarcotic analgesic medications. No motor deficits in the lower extremities were revealed on neurological examinations conducted immediately after surgery and at the postoperative assessment times described above. There was no evidence of increased muscle tone or hyperreflexia of the ipsilateral leg. All patients had normal proprioception and vibration sense. There was no evidence of clumsiness in the lower extremities and no bladder or sexual dysfunction.

**Discussion**

Stimulation of the dorsal or dorsolateral spinal cord and recording of evoked responses cephalad or caudal to the stimulation site has been studied in both animals and humans. Levy and York used these direct spinal cord stimulation and recording techniques in humans to monitor sensory and motor pathways during spinal surgery. In their study the mean conduction velocity in the fastest conducting fibers following dorsal and dorsolateral spinal cord stimulation in humans was 64 and 101 m/sec respectively. They concluded that direct motor tract recording, in addition to large-fiber sensory system monitoring with somatosensory evoked potentials, should provide information about spinal cord function during surgery.

Recording evoked potentials directly from the spinal cord following low-intensity stimulation is a simple technique that is reproducible and easily applied during DREZ operations. A spinal cord potential is easily evoked by applying the needle stimulating electrode to the pia-arachnoid between the dentate ligaments and the dorsolateral sulcus. For spinal cord mapping it is useful to gradually increase the stimulus intensity until an evoked potential with an amplitude of 10 to 20 μV is elicited.

The evoked potential following direct stimulation of the dorsolateral aspect of the spinal cord (between the dentate ligaments and dorsal roots) reflects activity in fast-conducting spinal cord pathways. The fast-conducting pathways closest to the dorsolateral stimulator position are most likely to contribute to the evoked potential. These are the dorsal spinocerebellar, lateral corticospinal, and rubrospinal tracts.

In our study the spinal cord evoked potential disappeared when the stimulating electrode was moved more dorsally over the DREZ and generally reappeared when the stimulating electrode was placed over the dorsal column. The wave form recorded following dorsolateral spinal cord stimulation had a significantly larger amplitude than that following dorsal spinal cord stimulation. The conduction velocity was significantly faster following dorsolateral stimulation.

We directly stimulated and recorded from the spinal cords of four patients with brachial plexus avulsion undergoing DREZ lesioning. The best location for DREZ lesioning was identified electrophysiologically when stimulation of the spinal cord with baseline stimulus intensity failed to evoke a response. No complications were detected immediately after surgery, and all patients experienced immediate pain relief in the affected limb. On follow-up examination, only one patient experienced recurrence of pain, which was limited to his thumb and is currently controlled by nonnarcotic analgesic agents. Two patients developed a swan-neck deformity as a result of the multiple bilateral laminectomies used for spinal cord exposure. Consequently, laminotomies followed by laminoplasty at the end of the procedure were used to prevent this complication in subsequent patients.
Spinal cord mapping for DREZ localization

Table 1 shows the incidence of complications associated with DREZ lesioning, which can be as low as 0% (Campbell, et al.) and as high as 60% (Friedman and Bullitt). Neurological deficits may be caused by lesions that are too large or misplaced. Controlled thermocoagulation and the use of lasers have been effective techniques to control the size of the coagulated area, thereby preventing unwanted central or lateral extension of the lesion into structures surrounding the DREZ. Misplaced lesions may be related to an inappropriate angle of penetration of the coagulation needle and/or lack of anatomical landmarks in the spinal cord with avulsed roots. For example, loss of the posterior lateral sulcus and distortion of the DREZ by gliosis are common following brachial plexus avulsion. The electrophysiological mapping technique described above may be helpful in localizing the DREZ in the presence of gliosis and loss of the posterior lateral sulcus.

It appears that this technique will work only when a low-intensity stimulus current is employed. The use of a low-intensity stimulus was effective in stimulating only those structures in the vicinity of the stimulating electrode, which may be the reason for the disappearance of the evoked potential when the needle electrode was placed over the DREZ, an area that is devoid of any pyramidal or extrapyramidal fiber tracts. It is interesting to note that the amplitude of the evoked potential is generally higher following dorsolateral than dorsal spinal cord stimulation when the same baseline stimulus intensity is used. This may be related to a different threshold for activation in the two pathways and may account for our inability to evoke responses following dorsal spinal cord stimulation in one patient. Nevertheless, we have found this technique to be quite reproducible and accurate in localizing the DREZ in patients in whom the DREZ cannot be visually identified. In addition, this technique may have prevented complications that can result from inadvertent coagulation of spinal cord structures adjacent to the DREZ.

References