Migrating Disc Complicating Spinal Decompression in an Achondroplastic Dwarf: Intraoperative Demonstration of Spinal Cord Compression by Somatosensory Evoked Potentials

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Achondroplasia, a dysplasia of endochondral bone formation, involves both the vertebral column and long bones. Approximately 50% of achondroplastic dwarfs have symptomatic spinal stenosis.1 Although stenosis of the vertebral canal is most common in the thoracolumbar region, cervical and generalized stenosis also can occur.2 In achondroplastic dwarfs, the intervertebral discs are relatively hyperplastic and have a tendency to bulge laterally and posteriorly.3 Although routine extradural

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exploration has been recommended during spinal decompression in achondroplastic dwarfs, such exploration involves the risk of injury to the cauda equina.

Symptomatic disc herniation in achondroplastic dwarfs is uncommon and usually is preceded by exertion or trauma. An achondroplastic patient is presented in whom an abrupt increase in spinal cord compression due to an extruded intervertebral disc occurred during spinal decompression for thoracolumbar stenosis. Intraoperative monitoring of somatosensory evoked potentials (SEP) detected this event and allowed measures to be taken to quickly restore the previous recorded SEPs. Intraoperative SEP monitoring in such cases is suggested, since apparently simple surgical maneuvers may result in neurologic deterioration.

REPORT OF CASE

A 24-year-old achondroplastic dwarf was evaluated for spinal stenosis. Exercise tolerance was less than two blocks. The patient was without bowel or bladder symptoms, and motor strength was normal in all major muscle groups. Light touch sensation was normal, except for a decrease over the right anterior thigh. Deep tendon reflexes were normal except for slightly decreased right patellar and right ankle jerk. No pathologic reflexes were present. Electromyography suggested partial denervation of L4–S2 nerve roots bilaterally. A myelogram performed by C1–C2 puncture demonstrated spinal stenosis beginning at T11 and progressing to complete block at L1–L2, and those findings were confirmed by computerized tomography (CT) scan.

Thoracolumbar decompression was performed with the patient positioned prone on chest rolls. Arterial pressure, central venous pressure, ECG, heart rate, and end-tidal CO2 (Hewlett Packard Capnograph) were recorded continuously. Anesthesia was induced with fentanyl (25 µg/kg, iv) plus thiopental (1 mg/kg, iv), and pancuronium (0.1 mg/kg) was given for skeletal muscle relaxation. Anesthesia was maintained by inhalation of enflurane (0.5–1.0% in oxygen). Controlled ventilation maintained end-tidal CO2 between 30–35 mmHg throughout the procedure.

Monitoring of the somatosensory evoked potential (SEP) was begun prior to induction of anesthesia. Gold-plated silver-cup electrodes were placed 2 cm posterior to C3, C4, and C2 (international 10–20 system). These electrodes were designated C3, C4, and C2, respectively. An electrode also was placed over the second cervical vertebra. All electrodes were referenced to a frontal electrode (FPz). Electrode impedance was maintained less than 2 k ohms. Sterile needle stimulating electrodes were placed near each median and posterior tibial nerve in a location to produce a digital twitch. A four-channel signal averager was used to generate the SEP (Nicolet Med 80°, Nicolet Biomedical, Madison, Wisconsin). For both upper and lower SEP, a band pass of 5–1,500 HZ was used. One hundred twenty-eight stimuli were averaged. Stimulus rate was 5.9/s for upper-extremity and 3.9/s for lower-extremity SEP. An observation time of 80 ms after stimulus were used. Waveforms were evaluated immediately for amplitude and latency and then stored on magnetic disk for later detailed analysis. For lower extremity SEP the initial positive wave (P1), which is considered the initial cortical wave, and the following negative wave (N1) were evaluated. A complete set of data, consisting of bilateral upper and lower extremity SEPs, including analysis and disk storage, required 5–6 min. Upper-extremity SEP were monitored to assess effects of anesthesia and other systemic factors. Therefore, during spinal decompression lower extremity SEPs were obtained routinely, with occasional determination of upper extremity SEP.

As decompression proceeded from T10 downward, a tight stenosis was appreciated at T11 and the dura was noted to bulge and become pulsatile shortly after decompression. During decompression of the T12 vertebra, the latency of both lower extremity SEPs increased above post induction value without alterations of the upper extremity SEPs (fig. 1; 0920 and 1049). This change initially was attributed to the use of room temperature irrigation solution, since latency was increased in both lower-extremity SEPs. However, the unexplained change in amplitude (decrease on left; increase on right; Table 1) suggested the possibility of neurologic injury, and SEPs for each lower extremity were determined as rapidly as possible. The SEP to stimulation of each lower extremity decreased progressively over an 8-min period with the response to stimulation of left posterior tibial nerve becoming unrecordable (fig. 1; 1057). The SEP changes occurred despite a constant anesthetic level and absence of surgical maneuvers (such as pressure on the spinal cord or cold irrigation solution) known to alter the SEP. The surgeon was apprised of the

![Fig. 1. Scalp averaged responses (C3–C4) to stimulation of each posterior tibial nerve are shown. Each pair (left and right posterior tibial nerve) SEP determinations (n = 128) required approximately 2 min. Note the rapid return of waves following fragment removal is shown.]

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Amplitude (µV)</th>
<th>Latency (ms)</th>
<th>Amplitude (µV)</th>
<th>Latency (ms)</th>
<th>Amplitude Ratio (L/R)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0920</td>
<td>1.2</td>
<td>45.1</td>
<td>1.8</td>
<td>41.3</td>
<td>0.66</td>
</tr>
<tr>
<td>1049</td>
<td>0.9</td>
<td>50.4</td>
<td>2.5</td>
<td>44.1</td>
<td>0.36</td>
</tr>
<tr>
<td>1057</td>
<td>—</td>
<td>—</td>
<td>0.5</td>
<td>49.7</td>
<td>—</td>
</tr>
<tr>
<td>1102</td>
<td>0.9</td>
<td>49.7</td>
<td>5.5</td>
<td>42.6</td>
<td>0.26</td>
</tr>
<tr>
<td>1130</td>
<td>2.2</td>
<td>43.5</td>
<td>3.4</td>
<td>42.0</td>
<td>0.65</td>
</tr>
</tbody>
</table>

Amplitude is voltage difference between maximum positive voltage at P1 (initial positive wave) and maximum negative voltage at N1 (initial negative wave). Latency is time from stimulus to maximum negative deflection (N1).
SEP change, and decompression was carried out quickly to relieve the obvious distortion of the dura, where it bulged out of the caudal end of the laminectomy. As the lamina of L1 was removed, a 1-cm extradural free fragment was found indenting the dura posteriorly at the L1–L2 level. The fragment was removed and SEP waves in response to stimulation of each lower extremity were recordable 2 min later (fig. 1; 1102). The amplitude of the waves progressively approached baseline values (fig. 1; 1130). An additional free fragment was found at the L4–L5 level, and a defect in the annulus of the L4–L5 disc was appreciated. The decompression proceeded without difficulty, and the patient awakened without change from preoperative status. Pathologic examination showed the fragment to be fibrocartilaginous material consistent with intervertebral nucleus pulposus. The amplitude of the initial wave complex (P1N1) and the latency of the initial negative peak (N1) are summarized in table 1.

**DISCUSSION**

Reversible SEP deterioration is presumptive evidence of prevention of intraoperative neurologic deficit. Permanent neurologic deficit may have occurred, had rapid completion of decompression not occurred. The accuracy of changes of SEP reflecting either changes in neurologic function or oxygen delivery to neural tissue has been demonstrated in both humans and animals. The acute deterioration and return of SEP described reflects only events occurring in posterior columns of the spinal cord. We have no direct evidence that function of the remainder of the spinal cord (specifically motor function) would have deteriorated, had not rapid decompression indicated by SEP changes been carried out. However, lower extremity SEP is altered by symptomatic intervertebral disc herniation and returns to normal after disc removal. In our case presented, SEP was monitored because of anticipated technical difficulties in this achondroplastic patient, but the source of neurologic change, a migrating intervertebral disc, was totally unexpected.

Although disc herniation in the achondroplastic dwarf is usually posterior and lateral, and symptomatic posterior disc fragments have been reported. Disc herniation usually is associated with exertion or back trauma. This patient had no history of back trauma. The adverse effect of the free fragment was manifest intraoperatively, when only minimal vertebral movement and minimal mechanical stress from instruments encompassing the laminae might have caused herniation. Decompression of the more superior vertebrae may have caused the free fragment to shift due to a hydrostatic gradient between the decompressed and yet undecompressed area, or there may have been distortion of the spinal cord as the dura bulged at the junction of the decompressed and undecompressed segments. Indeed, bulging of the dura has been reported following decompression in achondroplastic dwarfs. In the lumbar region, the cross-sectional area of the canal in the achondroplastic dwarf is smaller than normal by as much as 50%, and the area is further decreased by degenerated inferior facets. With such compromise of the intrathecal space, a small mass can produce significant neurologic symptoms by spinal cord or nerve compression.

The neuroradiologic techniques used preoperatively (C1–C2 puncture and CT imaging) can delineate spinal stenosis well in the achondroplastic dwarf and are likely to define both anticipated and unanticipated stenotic areas of the spinal cord. A limitation of this technique (as well as previous myelographic techniques) is in opacifying those regions distal to a high-grade stenosis. Disc herniations or extrusions in high-grade spinal stenosis are also “blind” to plain (noncontrast) CT examination because of the minimization of epidural fat that otherwise would afford an “endogenous” contrast medium. Intraoperative spinal cord compression could have occurred even if the presence of the fragment had been known. However, had the posterior fragment been appreciated preoperatively, modification of surgical technique by initially performing a narrow, full-length laminectomy and then progressively widening it might have minimized dural bulging and distortion.

The SEP is transmitted mainly in the posterior columns of the spinal cord. When a mass presses on the posterior aspect of the spinal cord, the posterior columns are affected first and changes in SEP may occur without change in function of the remainder of the cord. The observation at surgery was that the entire spinal cord was chronically compressed in the anterio-posterior diameter and that the disc fragment further indented the dura overlying the cord.

We have observed an alteration in neurologic function (failure of SEP) during spinal decompression in an achondroplastic dwarf. This neurologic alteration appears due to sudden pressure on the spinal cord by an extruded disc fragment. Rapid return of SEP following removal of the fragment strongly suggests prevention of permanent neurologic injury. We believe that SEP monitoring should be used during spinal decompression in achondroplasia and other causes of spinal cord compression, since sudden cord compression can occur as a result of surgical manipulation distant from the site of compression.

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**REFERENCES**

Sensitivity to Pain Predicts CNS Sensitivity to Lidocaine

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Local anesthetics apparently provide analgesia when given systematically in substantial doses.1,2 In an attempt to better define the relationship between systemic concentrations of local anesthetics and sensitivity to pain, Rowlingson et al.3 and Friedman et al.4 infused lidocaine and bupivacaine, respectively, into volunteers and assessed their effects on experimentally induced tourniquet pain. Lidocaine was shown to provide sedation but not analgesia at blood levels of 2–3 μg/ml. Bupivacaine, however, did provide analgesia, but only in subjects who experienced mild central nervous system (CNS) side effects with infusion of the drug. Interestingly, this subgroup of individuals who manifested CNS side effects from bupivacaine also had decreased pain threshold (P < 0.025) and decreased tolerance to tourniquet pain during predrug-infusion (control) tourniquet tests compared with the group that did not exhibit CNS side effects.4 To assess whether this was a generalized phenomenon associated with systemic infusion of amide local anesthetics, the data of Rowlingson et al. were reexamined to determine whether subjects sensitive to CNS side effects from lidocaine demonstrated an increased sensitivity to pain, as was observed in the bupivacaine subjects.

METHODS

Data previously collected by Rowlingson et al.5 were reexamined to extract previously unreported information. In this study, 14 healthy male volunteers received, in a double-blind manner, on two separate days, either lidocaine 0.5% or normal saline solution as a 10-ml intravenous bolus, followed by increasingly rapid infusions of the test solution. Subjects underwent the tourniquet ischemia test of Smith6 to determine their threshold (time to onset of pain) and tolerance (time to unbearable pain) several times prior to each test infusion (at the beginning of each day before any drug was given) and then intermittently during the infusion of the test solutions. The data of Rowlingson then were analyzed with respect to two sets of variables: the presence or absence of CNS symptoms after the first lidocaine 50 mg bolus, and threshold and tolerance to tourniquet pain during the preinfusion control tourniquet tests performed at the beginning of each test day.

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