Cauda equina syndrome has been recognized as a rare, devastating complication of spinal anesthesia since Ferguson et al.1 focused attention on the syndrome in 1937. The syndrome results when diffuse injury occurs across the lumbosacral nerve roots, producing varying degrees of three specific symptoms: saddle anesthesia, sphincter (bowel and bladder) dysfunction, and paraplegia.2

Cauda equina syndrome has complicated spinal anesthesia administered with a single injection of dibucaine, pipercaine, procaine, mepivacaine, and tetracaine.3,4 Previously, lidocaine spinal anesthesia has been associated with cauda equina syndrome only when repeated, large doses of the drug were administered through a continuous spinal catheter.5–10 We now report a case of cauda equina syndrome after single spinal injection of 100 mg lidocaine, 5%.

**Case Report**

A healthy 74-year-old man was scheduled for elective inguinal hernia repair during spinal anesthesia. With the patient in sitting position, clear cerebrospinal fluid (CSF) was obtained at the L3–L4 interspace with a single insertion of a 25-gauge Whitacre spinal needle using an unexpired, custom-supplied spinal tray (B. Braun Medical, Bethlehem, PA). Approximately 0.2 ml of CSF were aspirated before the injection of 100 mg of 5% lidocaine in 7.5% dextrose (Astra USA Inc., Westborough, MA), with 0.2 mg of epinephrine (Elkins-Sinn Inc., Cherry Hill, NJ) added. The direction of the spinal needle side-port and the speed of injection were not noted, and CSF was not aspirated after injection. No blood or paresthesia was encountered during placement. A T8 upper sensory level was obtained to testing by pinprick, and adequate surgical anesthesia was present. The patient received 1000 cc of lactated Ringer’s solution, 2 mg of midazolam (Roche Laboratories, Nutley, NJ) intravenously for sedation, experienced no hypotension, and required no pharmacologic blood pressure support.

The patient’s upper sensory spinal level receded to T10 in the postanesthesia care unit, and he was able to move his legs on arrival to his day surgery room 1 h after the completion of surgery. Approximately 12 h after initiation of spinal anesthesia, the patient was ambulating but complaining of a numb perineum and the inability to void. He was then easily catheterized, spent 24 h in the hospital, and was discharged with an indwelling urinary catheter and an initial diagnosis of mechanical bladder outlet obstruction. 3 days, at follow-up examination with his surgeon, he had no improvement in his symptoms and complained of persistent constipation.

Subsequent urologic evaluation 6 days after surgery revealed a saddle distribution of sensory deficit, a decreased bulbocavernous reflex, an intact anal reflex, and a small prostate and normal bladder without evidence of mechanical obstruction on cystoscopic examination. Cauda equina syndrome was suspected, and neurologic consultation was obtained at 3 weeks. Neurologic assessment was consistent with a diagnosis of cauda equina syndrome, revealing hypesthesia in the S2–S4 dermatomes. Results of a magnetic resonance image (MRI) without contrast were normal, and those of a cystometrogram after 4 months were consistent with a diagnosis of cauda equina syndrome demonstrating normal sensation, normal capacity, and detrusor hyporeflexia. Five months after surgery, the patient is able to have spontaneous bowel movements on a high fiber diet but still has perineal hypesthesia and remains unable to void spontaneously. The manufacturers of the custom spinal trays and of the spinal medications have been contacted.

**Discussion**

This patient’s symptoms of perineal anesthesia, bowel and bladder dysfunction, and decreased reflexes are consistent with cauda equina syndrome and are similar to all the previously reported cases (table I). Just how
### Table 1. Cauda Equina Syndrome and Lidocaine Spinal Anesthesia: An Accumulation of 10 Reported Cases to Date

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Anesthetic</th>
<th>Additive</th>
<th>Needle/Catheter</th>
<th>Patient Position</th>
<th>Sensory Deficit</th>
<th>Sphincter Dysfunction</th>
<th>Paraplegia</th>
<th>Months Followed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rigler et al., 1991</td>
<td>175 mg, 5% hyperbaric lidocaine in four doses</td>
<td>0.25 mg of morphine</td>
<td>28-g catheter</td>
<td>Supine</td>
<td>S1–S4</td>
<td>Bowel (constipation), bladder (hesitancy)</td>
<td>None</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>300 mg, 5% hyperbaric lidocaine in six doses</td>
<td>Epinephrine</td>
<td>28-g catheter</td>
<td>Decubitus</td>
<td>S3–S5</td>
<td>Bowel (constipation), bladder (catheterized)</td>
<td>None</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>190 mg, 5% hyperbaric lidocaine in seven doses</td>
<td>None</td>
<td>28-g catheter</td>
<td>Supine</td>
<td>Perineal</td>
<td>Bowel (constipation), bladder (catheterized)</td>
<td>None</td>
<td>10</td>
</tr>
<tr>
<td>Schell et al., 1991</td>
<td>285 mg, 5% hyperbaric lidocaine in eight doses + general anesthesia</td>
<td>None</td>
<td>28-g catheter</td>
<td>Supine/lithotomy</td>
<td>S3–S4</td>
<td>Bowel (constipation), bladder (transient catheter-dependent)</td>
<td>Transient bilateral lower extremity weakness</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>215 mg, 5% hyperbaric lidocaine in nine doses</td>
<td>None</td>
<td>28-g catheter</td>
<td>Supine</td>
<td>Perineal</td>
<td>Bowel (constipation), bladder (transient catheter-dependent)</td>
<td>None</td>
<td>9</td>
</tr>
<tr>
<td>Ross et al., 1991</td>
<td>125 mg, 5% hyperbaric lidocaine in two doses</td>
<td>Unknown</td>
<td>28-g catheter</td>
<td>Unknown</td>
<td>Perineal</td>
<td>&quot;Loss of sphincter tone&quot;</td>
<td>None</td>
<td>Unknown</td>
</tr>
<tr>
<td>Drasner et al., 1992</td>
<td>540 mg, 2% dextrose-free lidocaine + 50% nitrous oxide</td>
<td>Sodium bicarbonate</td>
<td>20-g catheter</td>
<td>Decubitus</td>
<td>Saddle</td>
<td>Bowel (constipation), bladder (catheterized)</td>
<td>Transient? left foot weakness</td>
<td>16</td>
</tr>
<tr>
<td>Mangar et al., 1993</td>
<td>150 mg, 5% hyperbaric lidocaine in 2 doses + general anesthesia</td>
<td>1.5 mg of morphine</td>
<td>20-g catheter</td>
<td>Supine</td>
<td>Perineal</td>
<td>Bowel (incontinence), bladder (catheterized)</td>
<td>None</td>
<td>Died 2 months postoperatively</td>
</tr>
<tr>
<td>Cheng, 1994</td>
<td>600 mg, 2% hyperbaric lidocaine in three doses + general anesthesia</td>
<td>1,200,000 Epinephrine</td>
<td>19-g catheter</td>
<td>Lateral/supine</td>
<td>Perineal</td>
<td>Bowel (incontinence), bladder (catheterized)</td>
<td>Transient bilateral lower extremity weakness</td>
<td>12</td>
</tr>
<tr>
<td>Gerancher, 1997</td>
<td>100 mg, 5% hyperbaric lidocaine in one dose</td>
<td>0.2 cc Epinephrine</td>
<td>25-g Whitacre</td>
<td>Sitting</td>
<td>S2–S4</td>
<td>Bowel (transient constipation), bladder (catheterized)</td>
<td>None</td>
<td>5</td>
</tr>
</tbody>
</table>

A modern spinal anesthetic might be associated with cauda equina syndrome has been the focus of considerable investigation and debate. Maldistribution of hyperbaric local anesthetics has been implicated because many of the patients with cauda equina syndrome had inadequate continuous spinal anesthesia despite repeated and relatively large local anesthetic doses. Evidence generated in spinal models in vivo suggests that neurotoxic concentrations of local anesthetics are attainable when repeated doses of hyperbaric local anesthetic are administered through a continuous spinal catheter. Single doses of hyperbaric lidocaine, 5%, administered in spinal models through 25-gauge and 27-gauge Whitacre needles also can produce maldistribution and potentially neurotoxic concentrations. The neurotoxic potential of lidocaine has been most recently characterized in vivo in a rat model with the spinal administration of lidocaine, 5%, wherein toxicity is not altered by the presence of 7.5% glucose but is enhanced by the presence of epinephrine.

When hyperbaric lidocaine, 5%, has been used clinically in the absence of a spinal catheter, neurologic deficits consistent with maldistribution or neurotoxicity have been rare. Two instances of isolated sensory deficits have been reported. One report was of a prolonged, but transient perineal hypesthesia in a volunteer who received 100 mg of hyperbaric lidocaine, 5%, via

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a 25-gauge Whitacre needle; the other was a report of permanent perineal hypesthesia in a volunteer who received 200 mg of hyperbaric lidocaine, 5%, in two administrations via separate 27-g Whitacre needles. Cauda equina syndrome was not described before the current report. Despite this record of clinical safety over the past 40 yr, Astra USA has revised the prescribing information for hyperbaric lidocaine, 5%, as discussed in a recent “Dear doctor” letter (table 2), referring to the reported cases of cauda equina syndrome after repeated injection, animal and in vitro studies, and recent reports of transient postoperative radicular pain associated with 5% lidocaine.

This new prescribing information was not present in the package insert used in this case report; these new guidelines were not followed, and there still remains no definite clinical evidence that following them would have prevented cauda equina syndrome in this patient. This report serves to document that spinal anesthesia administered in a single injection through a small gauge pencil tip needle, epinephrine, and as little as 100 mg of hyperbaric lidocaine, 5%, have now been associated with cauda equina syndrome.

References


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