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## Total Spinal Anesthesia Following Early Prophylactic Epidural Blood Patch

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The use of an epidural blood patch (EBP) to treat post-lumbar puncture headache (PLPH) when conservative measures have failed is well established.<sup>1,2</sup> Complications are infrequent and usually transient.<sup>1-3</sup> The occurrence of headache is most frequent when the dura has been accidentally punctured during attempts to establish epidural anesthesia due to the large size and shape of the needle employed. In such cases, it has been suggested that a blood patch be used prophylactically<sup>4,5</sup> via a successfully placed epidural catheter before the catheter is withdrawn at the end of the operation. This practice, however, remains controversial.<sup>3,6-8</sup>

This is a report of an unpredicted and previously unreported response to the use of a prophylactic EBP administered at the end of surgery but before the block had receded.

### CASE REPORT

A 27-yr-old, 84-kg, 170-cm man was scheduled for the repair of a torn anterior cruciate ligament. He had previously had uneventful epidural anesthesia for arthroscopy of the knee. He agreed to epidural anesthesia again but requested that he be well sedated throughout the procedure. He was in otherwise excellent health.

After preanesthetic medication with 10 mg im morphine and 50 mg hydroxyzine, he was alert but calm. With the patient in the lateral position, the first attempt at placing an 18-G epidural needle resulted in accidental penetration of the dura. The epidural space was then successfully identified one space lower at L4-5. An epidural catheter was inserted and threaded 4 cm with the needle tip pointing cephalad. After a negative test dose, satisfactory anesthesia was established with

a total of 20 ml lidocaine 1.5% with 1:200,000 epinephrine. Three supplemental doses of 10 ml each were given during the procedure to maintain a level at T4-6. To assist with postoperative analgesia, 5 mg morphine was added to the final dose. The patient also received iv sedation with 2 mg midazolam and 7.5 mg nalbuphine intraoperatively. He was rousable but would fall asleep readily. Pulse and blood pressure remained in the normal range, and oxyhemoglobin saturation ( $SpO_2$ ) was 100% with oxygen 4 l/min via nasal prongs.

The surgery was completed sooner than expected after the last dose of lidocaine. The analgesic level was T4 at this time, which was 250 min after the initial dose and 35 min after the final dose that contained the morphine. Before withdrawing the epidural catheter, it had been decided to give an epidural blood patch. Fifteen milliliters of blood were drawn from an arm vein and injected through the epidural catheter while the surgical dressing was being applied. Two minutes later, spontaneous respiration abruptly ceased, and in response to commands the patient could move only his facial muscles. The lungs were ventilated via mask with 100% oxygen as the block rapidly ascended. The trachea was intubated without neuromuscular relaxants, but 125 mg thiopental was given to provide amnesia. His blood pressure decreased transiently to 100/60 mmHg before returning spontaneously to 120/70 mmHg. Naloxone (0.1 mg  $\times$  2) was given without any response. His  $SpO_2$  remained normal throughout, but his pupils became widely dilated and unresponsive to light.

On arrival in the postanesthesia care unit, the patient was deeply unconscious and required controlled ventilation of his lungs. Initially, his blood pressure increased to 167/107 mmHg and his pulse to 106 beats per min, but they returned to normal levels after he was given 5 mg labetalol and 5 mg hydralazine. The patient regained consciousness and cranial nerve function 70 min after the EBP (115 min after the last dose of lidocaine). The level of block receded steadily over the next 90 min, and the trachea was extubated when satisfactory spontaneous respiration resumed. He required one dose of im morphine for surgical pain after discharge to the ward. Subsequently, he developed no signs of meningeal irritation, back pain, or headache. He was totally amnesic for the whole perioperative period.

### DISCUSSION

The sequence of events in this case strongly suggest that the occurrence of acute total spinal anesthesia was temporally and directly related to the injection of 15 ml autologous blood via the epidural catheter. The timing of the event makes it highly unlikely that the previous dose of lidocaine, on its own, coincidentally resulted in

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the total spinal anesthesia either from dural or subdural penetration by the epidural catheter. Although the level of block had been somewhat higher than required for the surgery, it was stable. The rise in level was sudden and dramatic. It progressed rapidly from the upper thoracic segments through the cervical dermatomes to become total, with fixed dilated pupils. The time course of his recovery further supports the diagnosis of total spinal anesthesia.

The cardiovascular responses were unexpected. Frequent measurements showed only a brief minor decrease in arterial pressure at the time of onset of total spinal anesthesia. The subsequent hypertension and tachycardia caused some doubt about the diagnosis. Total spinal anesthesia is typically associated with profound hypotension<sup>9,10</sup> although in the phenomenon described by Dawkins<sup>11</sup> as "massive epidural" the blood pressure remained normal. Epidural anesthesia with epinephrine-containing solutions produce tachycardia but with hypotension that is more pronounced than with plain local anesthetic solutions.<sup>12</sup> After the epidural injection of epinephrine and lidocaine, Bonica *et al.*<sup>13</sup> found a 49% increase in cardiac output, and more recently, Kerkkamp and Gielen<sup>14</sup> reported up to a 60% increase. In an animal model with total spinal anesthesia, Butterworth *et al.*<sup>15</sup> demonstrated altered peripheral vascular responses to infused catecholamines. Dopamine produced a greater increase in mean arterial pressure in dogs with total spinal anesthesia than in those anesthetized with pentobarbital alone. The patient reported here was young, physically fit, and had received iv fluids throughout the operation with negligible blood loss. Epidural anesthesia with lidocaine and epinephrine had not produced hypotension. With the onset of total spinal anesthesia, the loss of dominant vagal tone along with the systemic absorption of exogenous epinephrine might, therefore, account for the observed atypical tachycardia and hypertension in a volume-replete patient.

Alternative diagnoses, investigations, and treatments were considered at the time of onset of the signs. The naloxone had been given in case the epidural morphine was contributing to the condition but was without response. Epidural morphine has been used after previous dural puncture and before EBP without ill effect.<sup>5</sup> A major cerebrovascular event was a possible diagnosis<sup>16</sup>; however, specific investigation of this was delayed to see whether the original diagnosis was confirmed by recovery in the expected time period. Lumbar puncture at this time could have shown whether blood was present in the cerebrospinal fluid (CSF), but its origin would have been unclear. Cerebrospinal fluid exchange with saline has been used to dilute local anesthetic or to remove toxic compounds accidentally introduced,<sup>17</sup> but it was believed advisable to limit any further invasive procedures while the patient's condition was stable. Fortunately, expectant therapy

proved correct, and the recovery pattern supported the original diagnosis. Furthermore, the patient made a complete recovery without headache or any signs of spinal irritation, suggesting that the blood was correctly placed and effective.

The mechanism of the total spinal anesthesia must remain speculative. The forcing of residual lidocaine through the dural puncture needs to be considered. This has long been a concern when continuing with epidural anesthesia after an initial dural puncture, but the fears of producing total spinal anesthesia are largely unfounded in the light of experience.<sup>11</sup> Epidurally injected solutions spread rapidly within the canal and escape to a varying degree through the intervertebral foramina.<sup>18,19</sup> This spread of fluid is most likely in a cephalad direction. Uptake and equilibration with blood and tissues are progressive. Therefore, it is unlikely that sufficient lidocaine would be present 35 min after injection, or be positioned to pass into the CSF through the tear, to cause these effects.

Concentrations of local anesthetics in CSF after epidural injection have been shown to reach analgesic levels after 20 min and be maintained for 40–60 min.<sup>20</sup> Lidocaine attained anesthetic concentrations in CSF after epidural injection in the dog.<sup>21</sup> Bromage *et al.*<sup>22</sup> demonstrated additional penetration into the neuraxis and later postulated that cord block and intradural root block were the principal sites of action of epidurally injected local anesthetics. In a discussion of the mechanisms of differential conduction block, Fink<sup>23</sup> largely discounted the role of CSF spread in epidural block. However, continuous epidural anesthesia with repeated injections of local anesthetic might result in a build-up of CSF concentrations of the agent, thereby altering the character of the block. On this basis, it is proposed that in this case, CSF concentrations would be at a high level 35 min after the fourth and last dose. The normal volume of CSF below the foramen magnum is 25–30 ml, and an analgesic level of T4 would indicate a significant reservoir of lidocaine-containing fluid.

It is postulated that the injection of blood into the epidural space below this level resulted in the upward displacement of CSF. Blood injected epidurally would be expected to have a more "space-occupying" effect than crystalloid. The intact dural sac would be resistant to compression, but a leak through a tear could result in a more deformable state. The backache, radicular pain, or rare root damage, in response to epidural blood injection,<sup>24–26</sup> may represent traction on the roots as the dural sac is compressed. High pressures of injectate can be produced by injecting through the epidural needle directly.<sup>18</sup> However, in a low-pressure system, even injection through the catheter might compress the sac sufficiently to displace CSF cephalad. The critical factors in this case that may

have led to the total spinal anesthesia include 1) the high level of block at the time of the EBP, 2) high CSF concentrations of lidocaine due to regular top-ups *via* the epidural catheter, 3) the delivery of a large volume of colloid epidurally as a bolus, and 4) a compressible dural sac as a result of dural puncture by an epidural needle.

Prophylactic EBP was chosen in this patient to avoid the high likelihood of PLPH<sup>27</sup> and associated rare complications.<sup>28-31</sup> Although the wisdom and effectiveness of early EBP have been questioned,<sup>3,7</sup> other reports have demonstrated a high success rate.<sup>4,5</sup> This case suggests that prophylactic EBP should be performed only in the late recovery period when the block has worn off.

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