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Total Spinal Anesthesia following Epidural Saline Injection after Prolonged Epidural Anesthesia

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TOTAL spinal anesthesia is a complication that follows inadvertent introduction of local anesthetics into the intracranial subarachnoid space. It has been reported during attempted interscalene,[1] epidural,[2] and spinal[3] blocks. With some cases, the signs of total spinal anesthesia were produced during neuraxial block, but the mechanism of production was obscure.[3–9] We report an unusual case in which total spinal anesthesia developed after injection of epidural saline following a prolonged combined epidural–general anesthetic.

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Case Report

A 29-yr-old 56 kg woman underwent arthroscopic repair of the anterior cruciate ligament of the left knee. An epidural catheter was atraumatically placed on the first attempt at the L2-L3 interspace using a loss of resistance technique. After a test dose with negative results, 12 ml lidocaine, 2%, produced loss of sensation to T10 on the right and L1 on the left. General anesthesia was induced using a propofol bolus (140 mg) and was maintained by a continuous propofol infusion of 25–30 $\mu g \cdot kg^{-1} \cdot min^{-1}$ while the patient breathed a mixture of nitrous oxide and oxygen (60:40) *via* laryngeal mask. Epidural anesthesia was

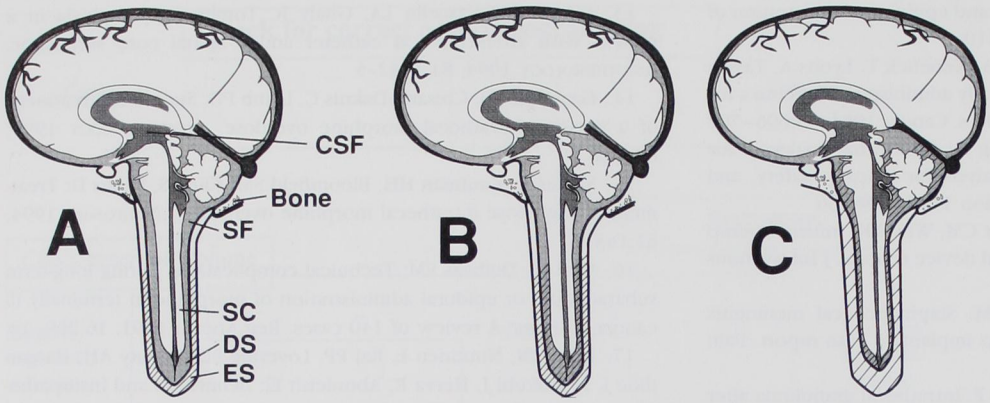

Fig. 1. Theoretical mechanism for production of total spinal anesthesia after epidural saline injection. (*A*) Immediately after epidural injection, the local anesthetic solution (slashed lines) is confined predominately within the epidural space, although diffusion into the dural cuff regions and the superficial layers of the spinal cord undoubtedly occurs during the initial onset of sensory blockade. (*B*) As the duration of the procedure increases, local anesthetic accumulates in the spinal subarachnoid space. (*C*) Bulk injection of saline into the epidural space then compresses the spinal subarachnoid compartment, pushing local anesthetic–containing subarachnoid fluid into the intracranial compartment. Venous sinus blood volume (black) is diminished. To account for persistent dilated nonreactive pupils in the absence of extreme hypoxia, the local anesthetic must reach as far as the interpeduncular cistern, where the oculomotor nerve exits the mesencephalon. Although we have postulated that dural compression forces the subarachnoid fluid cephalad, other factors such as diffusion and pulsatile oscillations may contribute to the movement of local anesthetic–containing subarachnoid fluid into the skull. CSF = cerebrospinal fluid within the ventricular system; DS = dural sac; ES = epidural space; SC = spinal cord; SF = subarachnoid fluid.

provided by 2% lidocaine for the first 3 h of the procedure, then 0.5% bupivacaine was administered when it became apparent that the procedure would be prolonged. The procedure lasted 8 hr 5 min, during which 70 ml lidocaine, 2%, 50 ml bupivacaine, 0.5%, and 2 mg morphine sulfate were administered epidurally. The last epidural dose of bupivacaine was administered 2 hr 15 min before the end of the procedure. After general anesthesia was discontinued, the patient awakened promptly, but complained of bilateral lower extremity motor weakness. A distinct sensory level on the trunk was not determined, but arm movements and sensation were intact. To accelerate regression of the motor block,[10] we administered 50 ml epidural saline at a rate of 2 ml/min. The patient was awake and comfortable but drowsy at completion of the injection. Approximately 6 min after the saline injection she suddenly became apneic, unresponsive, and totally flaccid, with bilateral dilated nonreactive pupils. Assessment of trigeminal sensory function was impossible, but the jaw was without tone. Phenylephrine, 50 μg, was administered to treat a systolic blood pressure of 80 mmHg; pulse oximetry oxygen saturation ($Sp_{O_2}2$) (Nellcor Puritan Bennet, Pleasanton, CA) was 100%. The trachea was intubated immediately without muscle relaxants, and the patient was transferred to the postanesthesia care unit, where she required mechanical ventilation. Systolic blood pressure remained above 100 mmHg (range, 110 to 140 mmHg) and $Sp_{O_2}2$ was 100%; no additional vasoactive agents were necessary. Pupillary light reflexes returned within 30 min, and motor function returned in a cephalad-to-caudal manner. Recovery was complete within 4 h. The patient had no recall of the events associated with tracheal intubation or extubation. There was no postural headache in the postoperative period.

Discussion

Epidural injection of saline has been promoted as a technique intended to speed the return of motor function after epidural anesthesia with local anesthetics.[10] This technique may be particularly useful after epidural anesthesia for orthopedic procedures when surgeons are concerned about motor function in the extremity that was operated on, as in this case. However, the development of total spinal anesthesia in our patient indicates that such large volume epidural injections may not be safe after prolonged epidural anesthesia.

The mechanism whereby epidural saline injection resulted in total spinal anesthesia cannot be proven with certainty. A common cause of total spinal anesthesia—accidental subdural or subarachnoid catheter placement—may be largely discounted in this case. Aspiration of fluid was not possible through the catheter, and the high block followed injection of a solution without local anesthetic activity. Subdural or subarachnoid catheter locations would be expected to require the administration of small doses of local anesthetics to produce adequate analgesia.[11] However, in this case, local anesthetic requirements for analgesia were large, perhaps because the catheter tip was preferentially directing solution to the side not operated on. The patient did not exhibit dilated nonreactive pupils or apnea at any time during the procedure. Similarly, it is unlikely that this sequence of events could be caused by rapid penetration of local anesthetic into the subarachnoid space *via* a dural tear[12,13] sustained during insertion of the epidural needle. This would also result in decreased, rather than increased, local anesthetic requirements. Furthermore, the cath-

eter was placed atraumatically on the first attempt, and postural headache did not develop, which would be expected after a dural tear.

Epidural saline injection might force local anesthetic solution, already within the epidural space, cephalad, perhaps as far as the foramen magnum (the rostral limit of the spinal epidural space). This could result in a sensory and motor block below the neck with paralysis of the diaphragm. Loss of consciousness might then occur from diffusion of local anesthetic into the fourth ventricle, which is situated partially within the foramen magnum. Although this mechanism theoretically is possible, the production of dilated nonreactive pupils and flaccid jaw muscles would require the motor branch of the trigeminal nerve and the oculomotor nerve to be blocked, and this would necessitate entry of the local anesthetic into the rostral subarachnoid cisterns. Of course, these cranial nerve findings could result from extreme hypotension, hypoxia, or both, but our patient was hypotensive only briefly, remained flaccid with dilated nonreactive pupils when normotensive, and was never hypoxic. Other reports have noted that hypotension is not necessarily a feature of total spinal anesthesia.[2]

We believe the most likely explanation in this case is that epidural saline injection in this patient produced total spinal anesthesia by shifting local anesthetic-containing spinal subarachnoid fluid into the intracranial space (fig. 1). Local anesthetics injected into the epidural space pass into the subarachnoid space in proportion to the total dose of local anesthetic administered, the physiochemical properties of the drug, the duration of local anesthetic exposure to the spinal meningeal surfaces, and the area of meningeal surface area exposed to the local anesthetic.[13-15] We postulate that because the procedure was prolonged, significant concentrations of local anesthetic may have accumulated in the subarachnoid space. Furthermore, progressively, the dural sac became compressed by subsequent epidural injections and, consequently, spinal subarachnoid fluid volume may have been diminished. The injection of additional fluid into the epidural space displaced this local anesthetic-containing subarachnoid fluid cephalad, producing a total spinal anesthetic. In this situation, a previous subarachnoid puncture or an intrathecal injection would not be necessary to produce total spinal anesthesia. Bolus injections of any solutions into the epidural space would have an increasing potential for producing intracranial effects as the duration of the epidural anesthetic increases.

The concept that the dural sac is an elastic structure in which liquid contents can be shifted rostrally by pressure gradients within the epidural space[16] is supported by several observations. For example, epidural saline injections raise the level of spinal anesthetics, and this is thought to occur simply by compression of the dural sac.[17-19] Leivers[4] reported the development of total spinal anesthesia after an epidural blood patch administered *via* the epidural catheter at the end of an epidural anesthetic that was complicated by a dural puncture. Although it was suggested that the dural puncture allowed the dural sac to become compressed by the epidural injection of blood, our case is remarkably similar and suggests that the volume of fluid within the dural sac can be altered even without a dural puncture.

The presence in the spinal subarachnoid fluid of significant concentrations of local anesthetic after prolonged epidural anesthesia has been shown experimentally[14] and is consistent with numerous clinical reports that emphasize the risk of inducing total spinal anesthesia by moderate doses of intrathecal local anesthetic injections after prolonged epidural anesthesia.[3,5-9] The mechanism for this complication is obscure. With any intrathecal injection, the creation of a hole in the dura might account for local anesthetic entry from the epidural space into the subarachnoid space. However, an alternative explanation[3] for this phenomenon is that significant concentrations of local anesthetics are present within a reduced spinal subarachnoid volume, and the addition of more local anesthetic directly into this smaller volume increases a previous subclinical concentration to a level high enough to produce total spinal anesthesia.

In summary, we report a case of total spinal anesthesia that followed epidural injection of saline after administration of a prolonged epidural-general anesthetic. The mechanisms involved cannot be determined with certainty, but we believe that a significant concentration of local anesthetic accumulated in the spinal subarachnoid space, which was then shifted cephalad into the intracranial cavity by the epidural saline injection. Until the mechanism is more fully understood, we advise caution when injecting bolus solutions into the epidural or intrathecal space after prolonged epidural anesthetics, when the concentration of local anesthetics in a contracted spinal subarachnoid space may be elevated. Clearly, the use of fractional injections[10] of small volumes into the epidural space is preferable to the bolus injection of a large volume, because fractional injections may avoid the creation of a sustained pressure gradient that causes cephalad movement of spinal subarachnoid fluid.

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Evoked Potential Monitoring and EKG: A Case of a Serious "Cardiac Arrhythmia"

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INTRAOPERATIVE neurophysiologic monitoring of spontaneous or evoked neural activity has become an important aspect of neuroanesthesia care. Evoked potentials are useful because they monitor the functional integrity of specific neural pathways that may be at risk for injury during surgery[1]; and, thus, are commonly used in many centers to monitor cerebral and spinal cord function during intracranial and spinal instrumentation procedures. Sensory evoked potentials are elicited using various stimulation techniques. Somatosensory evoked potentials (SSEP) can be obtained by electrical stimulation of a peripheral nerve; whereas, dermatomal sensory evoked potentials (DSEP) necessitate cutaneous stimulation of a specific dermatome. We report an occurrence of dermatomal evoked-potential monitoring causing what appeared

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