Is Spinal Anesthesia After Failed Epidural Anesthesia Contraindicated for Cesarean Section?

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In a recent review article on spinal anesthesia for cesarean section (1), Kestin recommended the use of subarachnoid anesthesia if epidural block was inadequate after the administration of the maximum allowable dose of local anesthetic (2). The use of spinal anesthesia in obstetrics has also been advocated for patients with potentially difficult airways, as this approach usually would obviate the need for general anesthesia and endotracheal intubation (3,4). From these statements, it might be construed that spinal anesthesia, rather than general anesthesia, would be safer under circumstances in which epidural block for cesarean section has failed in a patient with a potentially difficult airway. Two recent reports (2,5), and the following case report, suggest that subarachnoid anesthesia may be specifically contraindicated under these circumstances.

Case Report

A 24-yr-old patient (G3P0, gestation 37 wk, weight 98 kg, height 155 cm) with chronic hypertension and superimposed preeclampsia, who had been treated chronically with oral a-methyldopa (500 mg four times a day) and with a magnesium sulphate infusion (2 g/h) during active labor, required epidural analgesia after having received a single (and only) dose of meperidine (50 mg) and promethazine HCl (25 mg) intravenously (IV) at 3:00 PM. Her renal function and electrolyte status were within normal limits. A coagulopathy was not present, and arterial blood pressure was 140/80 mm Hg and heart rate 73 beats/min. Hemoglobin was 10.7 g%, and the serum magnesium was within the therapeutic range. Lactated Ringer’s solution was administered and the epidural catheter was inserted 4 cm into the space. Aspiration through the catheter was negative for cerebrospinal fluid or blood. A 3-mL test dose of 0.25% bupivacaine mixed with 100 µg of fentanyl (2 mL) was then administered, and a continuous infusion of a mixture of bupivacaine (0.125%) and fentanyl (1 µg/mL) was started at 4:45 PM. This mixture was infused at a rate of 16 mL/h, providing adequate analgesia for labor. This infusion was stopped at 8:30 PM in an attempt to improve the ability of the patient, whose cervix was now fully dilated, to bear down. At 10:45 PM, the patient was scheduled for cesarean section for failure to progress. Sodium citrate (Bicitra, 15 mL) was administered by mouth and a further 750 mL of lactated Ringer’s solution infused IV. After a 3-mL epidural test dose of 0.5% bupivacaine, this local anesthetic solution was administered incrementally via the epidural catheter over 40 min to a total dose of 30 mL. This resulted in a patchy block to a level of T10 on the left and T6 on the right, which was inadequate for surgery. In view of her preeclampsia and habitus, we decided to proceed with a spinal anesthetic. The patient was returned to the sitting position and her blood pressure (120/80 mm Hg) and arterial oxygen saturation (SaO2) (100%) were monitored continuously. A further 500 mL of lactated Ringer’s solution was administered and the epidural catheter removed. Twenty minutes after the last injection of epidural local anesthetic, the L3–L4 interspace was used to inject 11.25 mg of bupivacaine (0.75% + dextrose 8.5%) plus 25 µg of fentanyl mixed to 3 mL with cerebrospinal fluid via a 25-gauge Whitacre needle into the subarachnoid space. Immediately after this injection, the patient was placed in the supine position with left lateral tilt and head slightly up. Thirty seconds later, she complained of dyspnea, and the SaO2 decreased to 90%. Oxygen (100%) was administered via a facemask attached to the anesthesia circle system. Despite this, the SaO2 decreased further to 80%, and the patient was unable to breathe effectively and showed evidence of weakness of both arms. Manual ventilation of the lungs via the facemask and cricoid pressure were instituted. Anesthesia was induced within 2–3 min of placement of the subarachnoid block by means of IV thiopental (250 mg). In addition, succinylcholine (140 mg IV) and ephedrine (15 mg IV) were administered and the patient’s trachea was rapidly intubated without further incident. The arterial saturation returned promptly to 97% (significant arterial desaturation was of ±25 s duration) and, immediately postintubation, the heart rate was 105 beats/min and the arterial blood pressure 162/100 mm Hg. The systolic blood pressure never decreased below 104 mm Hg throughout the surgical procedure. Immediately after intubation, the pupils were found to be widely, eccentrically dilated and unresponsive to light. Pupillary dilatation persisted

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for approximately 30 min and then, gradually, the pupils became concentric and smaller. Ventilation was controlled to normocapnia, and anesthesia was maintained with 50% nitrous oxide (N₂O) and 0.5% isoflurane in oxygen until delivery of the baby, after which the isoflurane was stopped and 70% N₂O in oxygen was administered. No further muscle relaxation or analgesia was required for the remainder of the operation. A nerve stimulator attached along the course of the left ulnar nerve indicated normal neuromuscular transmission 15 min after induction of anesthesia. A female infant was delivered soon after induction, with Apgar scores at 1, 2, and 5 min after delivery of 7, 9, and 9, respectively. Surgery lasted 45 min, and at its conclusion, after discontinuation of the administered N₂O, the patient awakened, could maintain a sustained headlift, and had adequate grip strength. She was subsequently tracheally extubated on her side. Immediately postextubation, the patient had no pain, but the block was now patchy, with a sensory level of T2 on the left and T6 on the right with little or no residual paresis of the legs. Supplemental analgesia (morphine) was first given 45 min after extubation. The following day, the patient was comfortable with no sequelae.

Discussion

We present a patient who, after inadequate epidural anesthesia for cesarean section, rapidly developed apnea and ascending paralysis requiring endotracheal intubation after a mixture of hyperbaric bupivacaine (11.25 mg) + 25 µg of fentanyl was injected in the subarachnoid space. Kestin (1) has recommended the use of subarachnoid anesthesia under circumstances such as these. It is clear that if this complication had occurred in a patient with a difficult airway, the outcome might have been disastrous.

Classically, total central neurologic block (total spinal analgesia) occurs within 3 min of injecting an analgesic drug and is associated with apnea, fixed dilated pupils, loss of consciousness, and hypotension (6,7). In our patient, apnea and ascending muscle weakness, resulting in significant arterial oxygen desaturation, occurred soon after the placement of the block. The need for rapid induction of anesthesia, using IV thiopental, to secure the airway, as well as the prophylactic administration of ephedrine, did not allow us to ascertain whether unconsciousness or hypotension supervened. However, the patient's pupils were found to be unequally dilated, eccentric, and unreactive immediately after intubation and for a period of approximately 30 min, suggesting that total spinal analgesia had indeed occurred.

The overall safety of combined spinal and epidural anesthesia first described by Brownridge (8) has been established (9-11). Although techniques vary as to the site of spinal puncture and epidural catheter placement (8,12), they all have in common initial subarachnoid and subsequent epidural local anesthetic administration. Of further importance in using this technique is the observation that there is a decreased need for epidural (13), spinal (10), or total (11) local anesthetic administration to achieve a required level of sensory block. The mechanisms postulated to explain this are (a) leakage of epidural local anesthetic into the subarachnoid space (14), (b) the presence of "subclinical analgesia" (13) as a result of earlier local anesthetic in the cerebral spinal fluid, or alternatively (c), it is postulated that the epidural pressure becomes atmospheric due to epidural catheter placement and that this results in better spread of local anesthetic through an effect on circulation and volume of cerebral spinal fluid (10).

In contrast to conventional combined spinal and epidural anesthesia, in the present case report, spinal local anesthetic was administered after prior epidural administration of 30 mL of bupivacaine, resulting in an excessively high block with probable total spinal anesthesia.

It is possible that in this case, the observed high block was solely due to the administration of spinal anesthesia in a short patient, and independent of the prior epidural administration of local anesthetic. However, Norris showed, in 50 patients, that there was no relationship between the extent of block and the height or weight of the patient when 15 mg of hyperbaric spinal bupivacaine were administered for cesarean section. In addition, no high spinal block requiring intubation occurred (15). High spinal anesthesia after prior epidural anesthesia has been reported by two other authors, suggesting that there may be a common mechanism for this phenomenon (2,5).

This case report differs from the only two others (2,5) published to date, in that there was an extremely rapid onset of the high block (less than 1 min) accompanied by significant, and potentially dangerous, arterial oxygen desaturation. In contrast, in the quoted case reports, the authors administered 1.6–2.0 mL of 0.5% bupivacaine in 8% dextrose spinally under circumstances similar to the present report, but onset of the high spinal was gradual, requiring conversion to general anesthesia and intubation 10–15 min after institution of subarachnoid block while significant oxygen desaturation apparently did not occur. The reason for this discrepancy with our patient is not apparent. Perhaps the addition of 25 µg of fentanyl to the local anesthetic mixture (the standard protocol in our institution) may have played a role. Although this practice does not affect the onset or extent of subarachnoid block (16), it could be postulated that rapid rostral spread of this lipophilic opioid may have contributed to the rapidity of the onset of apnea (17), but this explanation cannot be invoked to explain the observed muscular weakness.

The mechanism for the extensive block common to these case reports is not clear. Although all the mechanisms quoted (10,13,14) for combined spinal epidural
anesthesia above might be invoked, it has also been postulated that prior epidural administration of local anesthetic may collapse the subarachnoid space below the termination of the cord (18); hence, a given volume of subarachnoid anesthetic may spread more extensively (2). That this may be a physical effect independent of the nature of fluid administered is suggested by one of the case reports (2), in which only saline had been placed epidurally before subarachnoid block. This mechanism could also explain the postextubation (±50 min after subarachnoid block) finding in our patient of minimal residual leg paresis if it is postulated that the local anesthetic mixture was widely (and thinly) spread, predominantly away from the lumbosacral segments, as a result of the mass effect of the epidurally administered local anesthetic.

In conclusion, the technique of combined spinal and epidural block, in which epidural anesthesia is administered after prior subarachnoid block, has been shown to be safe. This is because the decreased dose of epidural local anesthetic required when using this technique can be carefully titrated. In contrast, this case report suggests that the administration of a subarachnoid block after prior epidural anesthesia is not safe. This is because the appropriate dose for spinal anesthesia is not predictable and cannot be titrated readily. This case report clearly demonstrates that the use of the latter technique cannot be recommended and may have grave consequences for the patient, especially if intubation proves difficult or impossible.

References