CORRESPONDENCE

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Combined Spinal/Epidural Anesthesia for Outpatient Surgery

To the Editor.—Urmy et al. evaluated combined spinal/epidural (CSE) anesthesia for outpatient surgery and provided useful information regarding the dose–response characteristics of intrathecal isobaric lidocaine. Of interest, the authors report that the needle-through-needle technique was unsuccessful in 24.5% of patients in whom a 3.5-inch Weiss epidural needle was used and in 12.5% of patients in whom a 3-inch Weiss epidural needle was used. I wish to expand on this important point. Failure rate of the CSE technique may be decreased by using a spinal needle protruding at least 13 mm beyond the epidural needle. In addition to the length of the spinal needle, the design of the epidural needle may be responsible for the success of the CSE technique. The use of a modified epidural needle with an aperture in its curve (“back hole”) for the insertion of a spinal needle may be beneficial. The back hole was designed to reduce the likelihood of dural penetration by the epidural catheter by directing the catheter away from the dural puncture. Furthermore, it provides a better ability to “feel” the dural puncture.

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References

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In Reply.—We appreciate Joshi’s support of our data. Success with the combined spinal–epidural (CSE) needle–through-needle technique relates to some degree to the protrusion length of the spinal needle beyond the tip of the epidural needle. In our study and in clinical practice, we have found that some patients require up to a 15 mm clearance. In this same study, when a 3-inch Weiss epidural needle was used, there was adequate needle length in all patients. However, the technical price that one pays with an exceedingly long protrusion length is diminished stability of the needle–through-needle setup. We prefer a spinal needle that protrudes at least 12 mm but no longer than 15 mm.

However, we disagree with Joshi regarding the use of an epidural needle with a separate “back hole.” No data support the use of a back hole in terms of CSE success. To the contrary, in the study by Joshi and McCarroll, the needle combination using an epidural needle with a separate back hole had a 15% failure rate, whereas the normal Tuohy needle was associated with a 100% success rate in 13 patients. Further, there are no data from a prospective, blinded study supporting the better “feel” of dural penetration with an epidural needle that has a separate back hole. I have been able to perceive dural penetration using a 27-G Whitacre spinal through a Weiss epidural needle in the vast majority of patients. However, this also is anecdotal, unblinded, and operator-dependent. This claim must be evaluated with the investigators properly blinded and dural “feel” graded and confirmed (e.g., by the presence of cerebrospinal fluid) if meaningful conclusions are to be drawn.

In a study of fresh cadavers using epiduroscopy, Holmström et al. concluded that “it was impossible to force 16 or 18 gauge epidural catheters through the dural hole made by a single dural puncture with a 25 gauge spinal needle.” No data show a reduction in dural penetration with the catheter when a needle with a separate back hole is used. We agree that it is important to select a matched spinal and epidural needle combination of proper length to achieve acceptable success. Similar to Joshi, we recommend a protrusion length of the spinal needle of at least 12 mm. However, beyond this, there are inadequate data to conclude what constitutes the “optimal” needle combination.

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Mechanism of Hyperchloremic Metabolic Acidosis

To the Editor—Several points in the case report “Transient Perioperative Metabolic Acidosis in a Patient with Ileal Bladder Augmentation” merit further discussion.

We do not believe that the transient perioperative hyperchloremic metabolic acidosis in this patient required the presence of the ileal bladder augmentation. We accept that prolongation of contact of urine with bowel mucosa will allow for water reabsorption, passive chloride reabsorption, and active HCO₃⁻ secretion, leading to a net HCO₃⁻ loss and metabolic acidosis. In this patient, an indwelling urinary catheter was placed preoperatively, and although the catheter was transiently obstructed at the initiation of surgery, the decreased time of contact between the urine and bowel mucosa inherent with bladder drainage mitigates the importance of the ileal augmented bladder.

In our opinion, the principal reason for the acidosis was the large chloride load infused into this 13.5 kg patient. Although the authors admit that administration of a large amount of normal saline has been reported to result in a dilutional hyperchloremic acidosis, they also believed that this occurrence is an unusual clinical event and ‘‘that other factors were involved.’’ We acknowledge that the literature concerning hyperchloremic metabolic acidosis after normal saline infusion is sparse. However, there are data associated with the use of hypertonic saline that describe the development of hyperchloremic acidosis.1,2 These data suggest that less concentrated normal saline may cause hyperchloremic acidosis if administered in large quantities, as was done in this case.

The importance of hyperchloremia is emphasized by Stewart’s mathematically based approach to acid-base balance.3,4 According to Stewart, the major determinant of H⁺ concentration is the strong ion difference (SID) in the body. In a solution containing all collection of strong electrolytes, the H⁺ concentration is determined by the difference between the positively charged and negatively charged strong ions (molecules that completely dissociate in water). In the body, the SID is equal to the difference between the sum of the Na⁺ and K⁺ concentration and the Cl⁻ concentration (SID = Na⁺ + K⁺ - Cl⁻). A decrease in SID is associated with a metabolic acidosis, and an increase in SID is associated with a metabolic alkalosis. Change in Cl⁻ concentration is the major anionic contributor to change in H⁺ homoeostasis. The development of significant hyperchloremia with large volumes of saline for fluid resuscitation would result in a decrease in SID and metabolic acidosis. Furthermore, the Stewart model is able to quantify the effect that electrolyte change has on base deficit.5 Using this methodology, the expected base deficit effect from hyperchloremia in this patient would have sufficiently explained the observed base deficit.

We urge anesthesiologists to consider the importance of hyperchloremic acidosis during prolonged surgical procedures in which large volumes of saline are used for volume replacement. Because many still believe that the cause of metabolic acidosis in the surgical patient is the result of tissue hypoperfusion and cellular hypoxemia, the clinician attempts to maximize tissue perfusion with liberal volume infusions to support cardiac output. The common practice of treating acidemia with fluid may be worsening the acidemia rather than correcting it. We commend the authors in recognizing this acid-base problem and encourage anesthesiologists to consider the role of fluids in acid-base change.

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References


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