Regression of Sensory Analgesia with Lumbar Epidural Catheters

To the Editor—I read with interest the study by Boylan et al. comparing epidural bupivacaine-morphine analgesia with patient-controlled analgesia after abdominal aortic surgery. However, I have a major concern.

After a loading dose of 10 ml of 2% lidocaine followed by 15 ml of 0.25% bupivacaine via a lumbar epidural catheter, sensory block in the epidural group was probably at an adequate thoracic level for upper abdominal surgery. Forty-eight-hour postoperative analgesia was provided by epidural infusion of morphine combined with 0.125% bupivacaine at a mean infusion rate of 4.3 ml/h and a few 5-ml bolus injections of 0.25% bupivacaine in some patients. This dose of bupivacaine is too low to prevent regression of sensory analgesia below the level of surgery. This would have been noticed if not only visual analog pain scores but also levels of sensory analgesia had been obtained, as in any other study investigating the analgesic effects of neuraxial local anesthetics.

Nonlipophilic opioids such as morphine slow the regression of sensory analgesia, as was demonstrated by Hjortso et al. Using a lumbar epidural catheter, they maintained a thoracic level of sensory analgesia for 16 h by adding 0.5 mg/h of morphine to 8 ml/h of 0.5% bupivacaine (nearly eight times more bupivacaine than that used by Boylan et al.).

Ferrante et al. also used a lumbar epidural catheter to infuse 4 ml/h of 0.25% bupivacaine combined with the nonlipophilic meperidine after they had established sensory anesthesia to T6. The dose of bupivacaine in their study (which was approximately twice as high as the dose used by Boylan et al.) was chosen to allow regression of sensory block. They found that the mean time for regression of five dermatomes was 6 h.

In conclusion, Boylan et al. infused bupivacaine via a lumbar epidural catheter after upper abdominal surgery without reporting levels of sensory analgesia and using lower doses than those that caused early regression of sensory block in another study. Therefore, in my opinion, bupivacaine did not contribute to postoperative analgesia, and their study, in fact, compared intravenous morphine to epidural morphine.

Robert Jan Quist, M.D.
Chef de Clinique
Department of Anesthesiology
Medical Center Alkmaar
Alkmaar, The Netherlands
r.quist@inter.nl.net

References


(Accepted for publication February 1, 1999.)

In Reply—We thank Dr. Quist for his comments. However, the deliberate maintenance of high levels of epidural blockade with bupivacaine was not at all the aim of our study. Our aim was to use the potentially synergistic effect of bupivacaine and morphine to minimize the side effects (hemodynamic, motor blockade, pruritus) associated with either analgesia alone. In addition, as we pointed out in our article, epidural bupivacaine-morphine is known to provide superior analgesia on movement relative to epidural morphine alone after abdominal surgery. Our use of 0.125% bupivacaine reflects our concerns about hemodynamic stability associated with higher concentrations; this is borne out by the experience of Liu et al. who compared analgesia after colonic surgery using, among other modalities, epidural bupivacaine and epidural bupivacaine-morphine. These investigators reported an incidence of 67% and 30% for supine and absolute hypotension, respectively, during analgesia with 0.15% bupivacaine at a rate of 10 ml/h. During pilot data collection, we established that sensory levels using 0.125% bupivacaine could not be predictably elicited, and our protocol provided for deliberate decreases in infusion rate to a preset minimum of 4 ml/h, so as to minimize the chance of significant autonomic blockade. If we had elicited significant sensory blockade, it would have caused us concern.

To establish the relative contributions of bupivacaine and morphine to the analgesia obtained, we would have needed at least a third group that received epidural morphine only and maybe a fourth that received epidural bupivacaine only. For logistic reasons, this was impossible. However, the movement visual analog scale scores observed in our epidural group were very similar to those observed by Liu et al. using a broadly similar bupivacaine-morphine regimen; these latter scores were superior to those during epidural morphine alone, and mobilization VAS scores were similar in patients who received either epidural morphine or patient-controlled intravenous morphine. These findings strongly suggest a contributory role for bupivacaine. It might have been interesting to examine LEVELS OF SENSORY ANALGESIA WITH LUMBAR EPIDURAL CATHETERS.
CORRESPONDENCE

this issue as part of our study, but we felt that the work of Liu et al. had already answered this question definitively.

John F. Boylan, M.B., F.R.C.P.C.
Staff Anesthetist
St. Vincent's University Hospital
Dublin 4, Ireland

Alan N. Sandler, M.B., F.R.C.P.C.
Anaesthetist-in-Chief
University Health Network and Mount Sinai Hospital
Professor of Anaesthesia
University of Toronto
Toronto, Canada M5G 2C4

References


(Accepted for publication February 1, 1999.)

Pretreatment Intravenous Lidocaine for Intubation of the Asthmatic Patient: More Data Are Needed

To the Editor:—Important issues regarding the use of lidocaine as a pretreatment agent before intubation in patients with reactive airway disease remain unresolved. In the October 1998 issue of Anesthesiology, the use of intravenous lidocaine was recommended to attenuate airway reflexes in patients with bronchial hyperreactivity. This recommendation is based on the finding that lidocaine attenuates reflex bronchoconstriction caused by inhaled histamine. Furthermore, the authors reported no significant change in mean values of forced expiratory volume in 1 s at baseline and after lidocaine administration.

I remain unconvinced that intravenous lidocaine is a safe and effective agent in patients with reactive airway disease. Multiple studies have demonstrated an acute bronchoconstrictor effect by inhaled lidocaine. The ability of lidocaine to attenuate histamine-induced bronchoconstriction does not refute the concern that lidocaine may precipitate small airway bronchoconstriction in patients with reactive airway disease. McAlpine and Thomson found no correlation between asthmatic subjects with histamine airway hyperresponsiveness and those who suffered bronchoconstriction produced by inhaled lidocaine. In addition, changes in expiratory flow rates after inhaled lidocaine demonstrate much intersubject variability; use of mean number values can overlook this important intersubject variability. Prakash et al. reported that 3 of 15 stable asthmatic subjects experienced a 10% decrease in maximum midexpiratory flow, and 1 of 15 subjects experienced a 10% increase in maximum midexpiratory flow. McAlpine and Thomson reported that maximum percent change in forced expiratory volume in one s among stable asthmatic subjects after inhaled lidocaine varied from a -21% to +28%. Thus, I am skeptical that attenuation of a marker of bronchial reactivity, such as a histamine challenge, is adequate data to support a recommendation of intravenous lidocaine use in patients with reactive airway disease. Intravenous lidocaine efficacy should be demonstrated in a general population of asthmatic patients, not just a subgroup that demonstrates hyperreactive responsiveness to histamine, and data presentation should address possible intersubject variability.

R. Ben Zemenick, D.O.
Department of Emergency Medicine
St. Paul Medical Center
Dallas, Texas 75235
bzemenick@pol.net

References


(Accepted for publication February 18, 1999.)

Anesthesiology, V 91, No 1, Jul 1999