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fresh Memory board, will clean up the visible display of the signal, but with substantial side effects. One of the most obvious symptoms is the elimination or attenuation of all signals in the noise source and the physiologic signal within the band width of the filter. In practical terms, the 50 cycle filter will reduce or remove the pacemaker pulses from the QRS complex display (we have provided a distinctly separate path for pacemaker detection and display). Due to inherent time constants in the filters, the QRS complex will tend to “wonder” on the screen, extending artifact recovery times.

We have standardized this filter because, on the whole, the benefits are justifiable. I believe that an understanding of the subject matter and prevention of the root causes will benefit this and other users.

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Radicular Irritation after Spinal Anesthesia

To the Editor.—In their recent article concerning transient radicular irritation, Pollock et al.1 incorrectly represented our earlier work. They wrote that our study of the neurotoxic potential of commercially available local anesthetics used for spinal anesthesia in sciatic nerves2 showed that 5% hyperbaric lidocaine, 0.5% tetracaine, and 0.75% bupivacaine caused nonreversible ablation of the stimulated compound action potential. Although it is true that 5% lidocaine and 0.5% tetracaine abolished the compound action potential, 0.75% bupivacaine did not.

This is important, because the reports of cauda equina syndrome after continuous spinal anesthesia implicate lidocaine and tetracaine, but not bupivacaine.3,4 In addition, lidocaine, but not bupivacaine, produces the so-called transient radicular irritation syndrome.1,5,4 In our study, 0.75% bupivacaine, the highest concentration of bupivacaine used clinically, did not cause nerve injury. It is our opinion that bupivacaine is probably the safest local anesthetic for intrathecal use because it is the only local anesthetic that, to our knowledge, has not caused neural injury in patients, and it shows the least toxicity from intrathecal injections in rats5,6 or exposure to isolated nerves in vitro.7 In addition, intrathecal infusions of clinical concentrations of bupivacaine are nontoxic in dogs.8,9

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In Reply—We appreciate the comments of Lambert et al. and apologize for incorrectly representing their work. We agree that clarification on this point is important because both laboratory and clinical data support the safety of intrathecal bupivacaine in comparison with other local anesthetic agents.

Although we are convinced of the safety of intrathecal bupivacaine, we believe that, currently, there is insufficient clinical data to warrant the total abandonment of 5% and 2% lidocaine for spinal anesthesia. Despite an incidence of transient radicular irritation of 16% in our study, all patients were recovered and completely asymptomatic at 2-week follow-up. In addition, it is difficult to ignore the long safety record of intrathecal lidocaine. One of the many questions remaining to be answered is, after years of clinical use of subarachnoid lidocaine, why are we only now beginning to see patients with postoperative radicular syndrome? We agree with Lambert et al. that ongoing investigation is essential to answer these questions and to identify other appropriate spinal agents for practitioners seeking an alternative to lidocaine.

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Airway Obstruction after Oral Midazolam

To the Editor—I report a case of a child with congenital airway abnormalities in whom marked airway obstruction developed after administration of oral midazolam.

A 5-year-old boy presented as an outpatient for direct laryngoscopy, rigid bronchoscopy, and possible removal of his tracheostomy. The child was born with multiple congenital cervicofacial anomalies, including subglotic stenosis, tracheomalacia, and chondal stenosis, and received a tracheostomy shortly after birth. Previous general anesthetics (without premedication) for tonsillectomy and undescended testicle were uneventful. The parents reported that he had been doing extremely well with the tracheostomy ‘capped’ during the previous 6 months and requested that it be removed. Nighttime pulse-oximetry readings (without supplemental oxygen) were consistently greater than 95%. Because it was noted on the patient’s records that the child had a great deal of anxiety at the time of his previous surgery, 0.5 mg/kg oral midazolam was given in the ambulatory surgery center, and the child was then sent to the preanesthetic holding area. When I arrived to see the patient (approximately 5–10 min after oral midazolam had been given), his parents were quite concerned and claimed to me that his heartbeat and respirations were double their usual rates. On my initial inspection, the patient appeared dazed and slightly cyanotic, with severe chest wall retraction that the parents acknowledged were also abnormal. Chest auscultation revealed very little respiratory air entry and apparent airway obstruction. Removal of the tracheostomy cap provided immediate relief. The respiratory and heart rates decreased and the cyanosis disappeared within 30 s. Oxygen saturation was not recorded during this episode. Surgery proceeded uneventfully after induction of general anesthesia,

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