

CORRESPONDENCE

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Bupivacaine Toxicity and Bier Block: The Drug, the Technique, or the Anesthetist

To the Editor:—Heath's letter states “. . . seven patients have died in the United Kingdom as a result of Bier blocks in which bupivacaine was used . . .” and “. . . it appears that the recommended drug dosage (1.5 ml/kg as a 0.2% solution) was used. . . .”¹ This together with the implication that 0.5% prilocaine is safer leaves the impression that bupivacaine was responsible.

Heath knows that bupivacaine was not the sole etiology of the deaths, but do those reading her letter? She previously reported five of those seven deaths, stating: “Three elements merit discussion: the equipment, the drugs, and the people who used them.”² Furthermore, a report from the United Kingdom indicated that “junior hospital doctors” (registrars in anesthesia) routinely were using questionable doses of lidocaine for regional anesthesia under less than ideal circumstances.³ This also was the situation with the previous five deaths where “. . . the doctor setting up the block was a senior house officer in accident and emergency and was due to perform the operation without help from another doctor.”²

In 1965, abandoning the technique of intravenous regional block in the United Kingdom was suggested because with lidocaine “seven patients were found to have arrhythmia or other changes in the ECG and one patient developed cardiac arrest in asystole that was treated successfully with external massage.”⁴ This recommendation was supported in the United States.⁵ Certainly abandoning the technique of intravenous regional anesthesia solves the problems. But is it the right solution with a valuable technique when neither the

technique nor the drug alone is at fault? Interestingly, lidocaine is the only local anesthetic drug indicated and approved for intravenous regional block by the FDA.⁶ Therefore, Heath's letter¹ seeking advice from this country regarding the use of prilocaine may never be answered conclusively.

To conclude, Heath already has made the critical point, namely, the most important requirement for avoiding untoward sequelae with any regional block is not the technique or the drug but by whom and under what circumstances they were used.²

DANIEL C. MOORE, M.D.
*Professor of Anesthesiology
Department of Anesthesiology
University of Washington
School of Medicine
Seattle, Washington 98195*

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Carbon Dioxide Detection to Verify Intratracheal Placement of a Breathing Tube

To the Editor:—In a recent letter, Berman and co-workers¹ reported on a device to aid in detecting esophageal intubation by bubbling expired gas through an indicator solution in a De Lee trap to confirm the presence (or absence) of carbon dioxide. We share the authors' concern about esophageal intubation, but we found their technique to be awkward, messy, and dependent on prior preparation. We much prefer the inexpensive, electronic carbon dioxide detector advertised in the very same issue of the journal. The instru-

ment (Tri Med® model 510, \$1,575) is small, lightweight, and can be powered by its own battery or alternating current. It aspirates gas through a fine plastic capillary that can be attached, in advance, to the elbow connector of the anesthesia breathing circuit, allowing it to sense carbon dioxide in the first exhalation after intubation without requiring further maneuvers by the operator. The cost of the disposable capillary tube (\$2.05) is comparable to the cost of a disposable De Lee mucus trap (\$1.03) plus the necessary reagents, and the elec-

tronic instrument has the further advantage that it will function as a breathing circuit disconnection alarm and as a respiratory rate meter throughout the operation. The instrument is also useful as an apnea alarm for nonintubated patients during sedation with regional block, for recovery from anesthesia, or when epidural or intrathecal narcotics are employed.

We keep one of these devices attached to the anesthesia machine in our cesarean section room and have found it entirely satisfactory for verifying proper intubation. We keep another unit in the operating suite to supplement our (more slowly responding) time-shared mass spectrometer system during difficult intubations, and we have a third unit to verify all intubations performed in the intensive care unit. The portability of

the instrument would make it appear to be useful in emergency rooms and ambulances as well.

G. BASHEIN, M.D., PH.D.
Assistant Professor of Anesthesiology

FREDERICK W. CHENEY, JR., M.D.
Professor of Anesthesiology

*Department of Anesthesiology
University of Washington
Seattle, Washington 98195*

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Concerning the Site of Action of Verapamil on Skeletal Muscle

To the Editor:—I read with great interest the report of Durant *et al.*¹ on the potentiation of the neuromuscular (NM) blocking effect of pancuronium and succinylcholine by verapamil in the rabbit. I would like to take issue, however, with their statement that “the action of verapamil is not centered on the muscle fiber itself.” They based their conclusion on two observations, both of which appear to be irrelevant: The first of these, the observation that under the experimental conditions described, verapamil alone had no effect on the indirectly elicited twitch in the rabbit does not give any information on the site of action of verapamil. It only indicates that, because the sensitivity of the cardiovascular system to verapamil is greater than that of skeletal muscles, it was impossible to use high enough doses to inhibit twitch development. In *in vitro* experiments, in the absence of cardiovascular effects, twitch could be inhibited completely by verapamil.² It was also possible to moderately inhibit tension development *in vivo* by the infusion of 0.4 mg/kg verapamil over a 10-min period in the tibialis anterior muscle of rats, indirectly stimulated by 0.1 s trains of 50 Hz supramaximal impulses of 0.2 ms duration, every 20 s.³

Their second observation, referring to good correlation between the electromechanogram and the electromyogram (EMG) is also irrelevant to the site of action of verapamil. Both the twitch and the EMG represent the end result of a long chain of events. Interruption of this chain at any one or more levels (*e.g.*, motor nerve terminal, cholinergic receptors or ionophores of the postjunctional membrane, sarcolemma) by verapamil would cause similar decrease of the twitch and the amplitude of the EMG. About the only situation when there can be a

significant difference between the EMG and the twitch is when the intracellular utilization of Ca^{2+} , essential for the formation of the contractile actomyosin complex, is prevented by a compound such as dantrolene.

In contradistinction to the assumption that the site of the inhibitory effect of verapamil is the NM junction there is considerable evidence indicating that verapamil acts primarily at the sarcolemma or the sarcoplasmic reticulum membrane. The finding that *in vitro* the ED_{50} of verapamil was lower ($26.3 \mu\text{M}$) during direct than during indirect ($37.7 \mu\text{M}$) stimulation² supports this alternative hypothesis. If the primary site of the inhibitory effect of verapamil would be the NM junction, twitch tension should be inhibited more during indirect than direct stimulation. Furthermore, during indirect stimulation, inhibitory concentrations of verapamil at first increase tension development. Clarification of the mechanism of the initial stimulating effect and the primary site of action of verapamil will have to await the outcome of neurophysiologic studies. At the present time, however, most of the evidence favors a postsynaptic site of action for verapamil.

FRANCIS F. FOLDES, M.D.
*Professor Emeritus of Anesthesiology
Albert Einstein College of Medicine
Consultant in Anesthesiology
Montefiore Medical Center
Bronx, New York 10467*

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