CORRESPONDENCE

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Concerning the Use and Abuse of Test Doses for Epidural Anesthesia

To the Editor:—We read with interest the correspondence by Batra and associates entitled, "Bupivacaine Cardiotoxicity in a Pregnant Patient with Mitral Valve Prolapse: An Example of Improperly Administered Epidural Block."[1] In discussing test doses they state, ". . . a test dose that can rule out an intravascular injection must contain a minimum of 15 μg of epinephrine and be monitored with an electrocardiogram," referencing an earlier article by Moore and Batra.[2] We cannot agree. We are surprised that the technique is offered as the standard and that the referenced article has escaped criticism thus far, since we believe it contains several methodologic flaws. First, the 175 patients who provide the bulk of the data upon which conclusions are drawn are not compared properly to a control group. Second, these patients were premedicated and were surgical patients, hardly a group suitable for drawing conclusions about obstetric patients. Thirdly, the statistical treatment could be improved. The before and after epinephrine heart rates that were compared by an unpaired *t* test should have been compared by a paired test, which could have provided confidence limits. As the results stand, we are told to use a test procedure whose specificity and sensitivity are unknown. The obstetric patient may produce false-positive results, since she is often apprehensive and seldom heavily sedated, as well has having a needle in her back and experiencing labor pains. Lastly, the data relating to the time until the heart rate increase and the duration of maximum increase very likely have a Poisson distribution but apparently have been treated as normally distributed, making interpretation very difficult. For example, when and for how long do we look for ECG signs of intravascular injection (*e.g.*, how easily can one clinically detect a tachycardia lasting 31 ± 17 s?) and what is our assurance of avoiding false-negative results? In short, we believe the paper contains information that, as presented, has dubious interpretation, especially for the obstetric patient.

Furthermore, epinephrine has its own drawbacks in obstetrics. Intravascular injection of epinephrine decreases the uterine blood flow in ewes.[3] In humans it does not decrease the intervillous blood flow in a normal situation[4]; however, this may not be the case in a compromised fetus. Moreover, the associated increase in blood pressure is not desirable in a preeclamptic patient. Despite these potential drawbacks, we feel there is merit in the suggestions of a test dose and monitoring; however, the data presented do not support raising them to the level of canon. This method has some dangers, complexity, and uncertainty.

A simple, reliable, and safe method is to use plain local anesthetic, *e.g.*, 0.5% bupivacaine. The test dose should be of such magnitude to produce prodromal symptoms in the majority of patients, *e.g.*, 5 ml 0.5% bupivacaine injected rapidly in 5 s. Then, in the absence of symptoms, the therapeutic dose is administered in fractions of 5 ml separated by 30 s. Thus, each fraction acts as an added test dose. This way, evidence of intravascular injection should appear before the total therapeutic dose has been injected; thus convulsions can be prevented, or if one occurs it can be managed easily and the chance of cardiac depression due to an excessive dose is prevented.

We feel that epidural analgesia, a potentially dangerous technique, has been overused. The substitution of spinal for epidural analgesia, especially in cesarean section, is not only preferable for the fetus[5] but safer for the mother. The same applies for most surgery whose duration is less than 2 h.

We agree with the authors and emphasize that immediate treatment of convulsions and maintenance of respiration are the keys to avoiding cardiac arrest. Trying to ventilate a convulsing patient with a face mask and bag is not only worthless, but dangerous, since it distends the stomach, thus predisposing to regurgitation and aspiration. Attempting laryngoscopy and intubation during a convulsion is often futile and dangerous, since it prolongs asphyxia, thus increasing the risk of cardiac arrest. Diazepam and thiopentone may not always stop convulsions, and the latter has specific cardiovascular depressant effects. Amnesia is not an indication for either drug, since it invariably occurs as a consequence of the convulsions. Therefore, when a convulsion does occur, the sequence should be succinylcholine and cricoid pressure. When relaxation occurs, the patient is ventilated with 100% oxygen, then intubated under controlled conditions, followed by hyperventilation.

In conclusion, it is only through anticipation, preparation, experience, vigilance, and dosage fractionation of local anesthetics that convulsions can be prevented, aborted, and adequately treated. There is ample evidence that patients have convulsed and some died despite the use of test doses, with or without epinephrine.[6,7]

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In reply: Abouleish and Bourke should provide data to support their contentions, rather than speculations, that: 1) our recommended test dose[1] lacks "specificity and sensitivity"; 2) the method itself has "dangers, complexity, and uncertainty"; and 3) it should not be raised "to the level of canon," which perhaps is correct but was not advocated. We suggest that they repeat our study[1] using methods and statistical analysis that they imply will either confirm or refute the necessary components of a test dose.

To substantiate our data,[1] in the past 4 years, using the described test dose and monitoring it as stated in our original article, during over 5,000 epidural blocks (in obstetric and surgical patients), no systemic toxic reactions or total spinal blocks have resulted. On the other hand, in prior years the incidence of convulsions was 1 to 2 in 1,000 patients and total spinal anesthesia 1 in 2,000 when attempting epidural block.

We, too, recognize the possible difficulties with the test dose in parturients but have had no difficulty in establishing a control for the maternal heart rate between contractions and then giving the test dose at such a time. It is also noteworthy that parturients scheduled for an abdominal delivery frequently are not in labor. We do not hesitate to use 15 to 20 μg epinephrine in obstetric patients[2] and neither do other obstetric anesthesiologists.[3] Furthermore, since the majority of obstetric patients are not "high risks," why deprive them of a test dose containing epinephrine when, unlike the study in five sheep, cited by Abouleish and Bourke,[4] evidence in humans indicates that it does not diminish intervillous blood flow nor does it compromise the normal fetus.[5] Also, we doubt that in the treated and controlled preeclamptic patient, 15 μg epinephrine would be dangerous, although we do not have concrete data.

In addition, fractional (incremental) dosing as described by Abouleish and Bourke has its drawbacks. It initially was recommended to prevent injecting a large dose of chloroprocaine solution into the subarachnoid space, thereby avoiding myelopathy from that solution.[6] It is not a "simple, reliable, and safe method" of avoiding a systemic toxic reaction when using amide local anesthetic drugs in an intermittent (continuous) injection technique. Unlike ester derivatives, which are hydrolized rapidly by the blood esterases[7] the amide derivatives cumulate.[8,9] Therefore, when establishing anesthesia or administering a reinforcing dose of them, the blood level can increase with each additional dose. Thus, while previous doses show no evidence of systemic toxicity, the next dose can trigger a convulsion that may not be of short duration or innocuous.[8]

We agree that for cesarean section, when plastic tubing is not in place in the epidural space, spinal anesthesia is perhaps a safer technique. Also, we take no exception to the described technique of treating a convulsion and in 1960 recommended the use of succinylcholine as the drug of choice to stop convulsions because of the depressing effects of thiopental[10] and more recently those of diazepam,* neither of which consistently stops convulsions. However, as yet, in 43 years, one of us (DCM) has not found it necessary to intubate the trachea, has used an oral airway to prevent the tongue from occluding the airway, and, as yet, not had a patient aspirate.

To conclude, regardless of the letter of Abouleish and Bourke, our article regarding the test dose accomplished at least the following: 1) it alerted anesthesiologists that most test doses as previously administered by one of us (DCM[11]) and others[12,13] were worthless; 2) it described an effective test dose, if not the only one, which, if properly monitored when attempting epidural block for surgery, obstetrics, diagnosis, or therapeusis, will within 2 min indicate a possible injection of the local anesthetic drug into either a blood vessel or the cerebrospinal fluid[1]; 3)

* Moore DC: Systemic toxicity of local anesthetic drugs, Seminars in Anesthesia. 2:62–74, 1983.