and start to decrease, as monitored by the cardiotocograph and electrocardiograph respectively, the test dose is injected. At the same time, the parturient, the nurses, and the family (when present) are instructed not to converse. Under these conditions, if as is usual 30 s elapse prior to the next contraction, 15 μg epinephrine intravascularly results in the following: 1) the pulse rate shows a marked sustained increase; 2) without exception and particularly if asked, the parturient states that her heart is pounding or that her "heart feels like it is about to jump out of her chest," which she does not notice during a contraction; and 3) her blood pressure is elevated.

Also, theoretically, based on bench investigation, uterine blood flow in humans should be reduced by an iv injection of epinephrine.[7] While these data cause concern, its magnitude is similar to that observed with a normal human uterine contraction.[7] Nonetheless, although its clinical significance is unknown, the use of epinephrine in a test dose has become controversial.[7] As yet, I am not aware of a single case report of *human* fetal morbidity or mortality resulting from the intravascular injection of 15 μg epinephrine. Have I missed something? If so, citation by Leighton *et al.* of such data would be helpful.

Lastly, although the injection of isoproterenol, other drugs, or even air into the epidural or subarachnoid space in animals is a step forward in determining their safety,[1] it is only a rough guideline to the situation in humans.[8–11] Epinephrine is safe when injected into these spaces in humans.[1] Whether other markers are safe is the real issue.

In conclusion, until the *theoretical* problems of epinephrine as a test[1–3] dose are confirmed in the pregnant human and until injection of other substances into the epidural, subdural, or subarachnoid spaces are proven safe, it is hoped that anesthesiologists have not begun to use them rather than epinephrine as a test dose in their clinical practice.

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In Reply:—In his letter, Dr. Moore raises several important questions. First, he questions our finding that epinephrine injection lacks specificity because of the inherent maternal heart rate variability of laboring women. Three independent research groups have found that in actively laboring women, maternal heart rate variability exceeds 20 beats per min in 24–90% of patients and exceeds 30 beats per min in 12–45% of patients.[1–3]

Unfortunately, Dr. Moore never specifies either in his current letter or in his original article[4] how much a patient's heart rate must increase to indicate an intravenous injection of epinephrine 15 μg. In the past, Dr. Moore has stated that epinephrine 15 μg should increase the heart rate by at least 25 beats per min, and that this increase should last at least 15 s.* Yet when this criterion was applied in a prospective, randomized, double-blind manner, it correctly identified only 50% of the patients who received epinephrine 15 μg iv.[5] Furthermore, 20% of patients receiving intravenous saline had false-positive results. In this study we followed Dr. Moore's recommendations for performance of the injections.[5] All injections were performed when patients were calm and alert, 30 s after a uterine contraction. Nonetheless, neither heart rate changes nor subjective symptoms occurred reliably. In fact, one patient's heart rate decreased after intravenous epinephrine, and six patients receiving intravenous epinephrine reported no or minimal symptoms upon direct questioning.[5]

We agree that intravenous epinephrine may decrease uterine blood flow.[5] Although this effect may be similar in magnitude to that observed during a uterine contraction, the combined effect of a uterine contractions and intravenous epinephrine is unknown. In fact, new, ominous fetal heart rate changes occurred in two of ten patients receiving epinephrine 15 μg iv in our study. (One patient exhibited persistent late decelerations for 10 min, and the other exhibited 4 min of mild fetal bradycardia followed by 7 min of decreased fetal heart rate variability.)[5] In both cases, cesarean section was considered until the fetal heart rate changes resolved.

We agree with Dr. Moore that only those substances proven to be safe should be injected into the epidural space. Therefore, we cautioned the readers of our isoproterenol article that isoproterenol should not be injected into the epidural space until appropriate neurotoxicity

* Moore DC: Personal communication. October, 1986

studies have been performed.[6] However, the safety of injecting air into the epidural space is well-established. Dr. Moore and others recommend the use of air as a test for entry of needles into the epidural space (as the air loss-of-resistance test).[7,8] When this test is performed, air is necessarily injected into the epidural space. Does Dr. Moore believe that maternal precordial Doppler monitoring alters the safety of the epidural injection of air?

Air is safe also when injected into the subarachnoid space during pneumoencephalography.[9] Additionally, the use of the air loss-of-resistance technique may lead to the subarachnoid injection of large volumes of air in the event of a dural puncture. Is Dr. Moore aware of any significant complications resulting from such an occurrance?

With regard to the Doppler test,[10,11] we believe that clinicians who try it will like it.

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Latent Myotonic Dystrophy: The Cause of Hydramnios and an Increase in Serum Creatine Kinase Concentration

To the Editor:—We recently reported on increased serum creatine kinase (CK) concentration related to the appearance of variant CK in a pregnant woman with hydramnios.[1] We have since found the cause to be latent myotonic dystrophy. Approximately 1 yr after her first pregnancy, hydramnios and an increase in serum CK level recurred. Also, her medical history revealed that her uncle had been suffering from myotonic dystrophy for 5 yr. We examined her again carefully, and found a slight cataract and an EMG characteristic of latent myotonic dystrophy.

Pregnancy unmasks latent myotonic dystrophy, but may not cause any apparent clinical symptoms.[2] In addition, increased serum CK is only moderate in myotonic dystrophy, and variant CK has no diagnostic value.[3] Hydramnios, which is caused by impaired swallowing of amniotic fluid by the affected fetus, may be the first sign of the disease.[4] Thus, diagnosis may be made only after occurrence of hydramnios and neonatal death.[4]

Even latent myotonic dystrophy can cause many complications and problems during anesthesia,[5] and requires a precise family history and careful examination of the patient.[6]

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