CORRESPONDENCE

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Simultaneous Recording of Maternal and Fetal Heart Rate

To the Editor:—I read with interest the letter to the editor, "Monitoring Maternal Heart Rate during Epidural Injection of a Test Dose Containing Epinephrine," by Chestnut and Weiner. We tried to duplicate the technique using the same machine. It was difficult to do so for the following reasons: The electrodes are connected originally to a hook that is supposed to be attached to the fetal scalp; therefore, the wires have to be cut and their terminal ends exposed before being attached to the regular electrodes placed on the maternal chest. Subsequently, conductive gel has to be applied to the cable block, which is strapped to the patient's arm. Also, by using the fetal scalp electrodes, one may deny the fetus from being monitored during the procedure. The absence of fetal heart rate monitoring is evident in the figure in their correspondence.

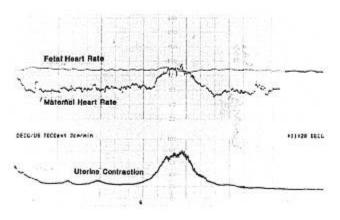


FIG. 1. Simultaneous recording of fetal heart rate by ECG (light tracing), maternal heart rate using Doppler (dark tracing), and uterine contractions. The heavy vertical lines are 1 min apart.

We hereby describe an easier way. Using the same machine (Hewlett-Packard Model 8040A), leave the internal monitor to record the fetal heart rate by ECG. At the same time place the Doppler component of the machine over major maternal arterial blood flow, e.g., the aorta at the substernal angle. Thus, you can simultaneously record § the fetal heart rate, maternal heart rate, and uterine contractions, as shown in figure 1. Also, we would like to 2 draw attention to the fact that during a uterine contraction, the maternal heart rate can increase by 50% (approximately 40-50 beats/min), as shown in figure 1. This maternal heart rate acceleration in response to uterine contractions was one of the reasons we objected to the use of epinephrine as a test dose because of the possibility of a false positive result.2 If you still prefer to use epinephrine, inject the test dose immediately after a uterine contraction.

EZZAT ABOULEISH, M.D.

Professor of Anesthesiology, Professor of Obstetrics and Gynecology

KERRY JOHNSON, M.D. Resident of Obstetrics and Gynecology

Department of Anesthesiology The University of Texas Houston, Texas 77030

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In reply:—We appreciate the interest of Drs. Abouleish and Johnson in our recent correspondence. We regret the misunderstanding regarding the technique that we described. We have not used a fetal scalp electrode to monitor the maternal heart rate. Rather, we have used pregelled electrodes and electrocardioscope lead wires, such as those routinely used in the operating room. The

lead wires may be easily inserted into the cable block. Further, we have not found it necessary to apply conductive gel to the cable block.

Drs. Abouleish and Johnson have described use of the dual heart rate monitoring capability, an option that was not standard equipment on earlier editions of the Hewlett-Packard Model 8040A. This "twin option" is now a stan-

dard feature of this machine. Since submitting our letter, we too have obtained machines with the capability of dual heart rate monitoring, and we agree that it is preferable to record maternal and fetal heart rates simultaneously.

The technique described by Drs. Abouleish and Johnson allows for simultaneous recording of maternal and fetal heart rates only in patients with ruptured membranes. Hewlett-Packard has introduced an electrode cable (15241A) specifically designed for monitoring the maternal heart rate. The maternal heart rate may be monitored using the direct ECG mode of the monitor while the fetal heart rate is monitored using the Doppler ultrasound mode. In this way there may be dual monitoring of maternal and fetal heart rates in a patient with intact fetal membranes.

> DAVID H. CHESTNUT, M.D. Assistant Professor Departments of Anesthesia and Obstetrics and Gynecology

CARL P. WEINER, M.D. Assistant Professor Department of Obstetrics and Gynecology

University of Iowa College of Medicine Iowa City, Iowa 52242

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Spinal Anesthesia in Premature Infants: Dosage and Effects of Sympathectomy

To the Editor:—Recently, Harnik et al. 1 reported their experience with spinal anesthesia in premature infants recovering from respiratory distress syndrome. Their work raised two important issues: 1) dosage of the agents used to produce spinal anesthesia; and 2) cardiovascular effects of sympathetic block in neonates.

The report by Harnik et al. 1 confirmed the impression that a larger dose of local anesthetic, on a weight-forweight basis, should be used for spinal anesthesia in premature infants²⁻⁴ (table 1). Similarly, Blaise and Roy⁴ recommended that younger infants and children receive more local anesthetic for spinal anesthesia (table 1). Possible reasons for this may relate to age-related physical and physiologic differences among infants, children, and adults, including the amount of cerebrospinal fluid, diameter and surface area of the spinal cord and nerve roots, and rate of absorption of local anesthetics from the subarachnoid space. These factors should also contribute to the relatively short duration of tetracaine spinal anesthesia seen in infants.5,6

Preganglionic sympathetic block produces cardiovascular changes in spinal anesthesia. Children are said to tolerate vasomotor imbalance secondary to sympathetic block particularly well. Dohi et al.5 demonstrated that children less than 5 yr of age had little or no change in blood pressure and heart rate following T3-4 level of spinal anesthesia, but children more than 6 yr old had widely variable decreases in blood pressure. They attribute these age-related differences partly to less development of the sympathetic nervous system in small children. Also, no episodes of hypotention or bradycardia have been observed in small infants (table 1). In experimental animals, there is increasing evidence that development of the sympathetic nervous system is incomplete at birth.^{7,8} Unfortunately, Harnik et al.1 did not report any data of cardiovascular effects of spinal anesthesia in their pre-

Because the sympathetic nervous system is inadequately developed but the parasympathetic nervous system, on the other hand, is quite active in infants, 9 we wish to know

TABLE 1. Dosage of Tetracaine for Pediatric Spinal Anesthesia and Changes in Blood Pressure (BP) and Heart Rate (HR)

Investigator(s) (yr)	Age Ranges	Dose of Tetracaine	Changes in BP and HR after Spinal Anesthesia
Berkowitz, Green ¹¹ (1951)	1–13 yr	0.1 mg•pound ⁻¹ or 1 mg•yr ⁻¹ of age	No change, but recommend use of prophylactic vasopressor
Dohi <i>et al.</i> ⁵ (1979)	8 months-15 yr	0.3 mg·kg ⁻¹ *	<5 yr, no change; >6 yr, variable decreases in BP
Abajian <i>et al.</i> ⁶ (1984)	<1 yr 0-3 months	0.22-0.32 mg·kg ⁻¹ † 0.4-0.5 mg·kg ⁻¹	No hypotension, no bradycardia
Blaise, Roy, ⁴ (1986)	4–24 months 24 months	0.3-0.4 mg·kg ⁻¹ 0.2-0.3 mg·kg ⁻¹	No hypotension, no bradycardia
Harnik et al. 1 (1986)	35-78 weeks (1.7-5.9 kg)	0.24-0.65 mg·kg ⁻¹	

Inclusion of phenylephrine (0.075 mg·kg⁻¹).

[†] Inclusion of epinephrine (0.02 mg).