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.

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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-for-weight basis, should be used for spinal anesthesia in premature infants[2–4] (table 1). Similarly, Blaise and Roy[4] 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.[5] 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 premature infants.

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 *et al.*[5] (1979)	8 months–15 yr	0.3 mg·kg^{-1} *	<5 yr, no change; >6 yr, variable decreases in BP
Abajian *et al.*[6] (1984)	<1 yr	0.22–0.32 mg·kg^{-1} †	No hypotension, no bradycardia
	0–3 months	0.4–0.5 mg·kg^{-1}	
Blaise, Roy,[4] (1986)	4–24 months	0.3–0.4 mg·kg^{-1}	No hypotension, no bradycardia
	24 months	0.2–0.3 mg·kg^{-1}	
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).

the results of cardiovascular variables such as blood pressure and heart rate following spinal anesthesia, which usually blocks only the sympathetic nervous system. This information will provide us with important information for the anesthetic management of small infants whose cardiovascular homeostatic mechanisms could be vulnerable to inhaled anesthetics.[10]

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In reply:—The excellent letter of Drs. Dohi and Seino refers to some of the most fascinating aspects of spinal anesthesia in the premature infant. Each factor mentioned in their discussion must contribute to the higher-dose requirement and shorter duration of tetracaine anesthesia. In addition, delayed myelination of the spinal cord and its surface area may cause the extremely rapid onset of this block. Blood levels of tetracaine have not been measured in infants, and the correlation between peak levels and the duration of analgesia is unknown.

Publications on spinal anesthesia in children emphasize the absence of adverse cardiovascular effects. In spite of this, Berkowitz and Green[1] consider preanesthetic vasopressor administration "always desirable although usually unnecessary." They inject neosynephrine to prevent retching from transient falls of blood pressure (BP). Their report on 350 cases includes only one child under 2 yr of age.

Singler[2] recommends hydration with 6 ml/kg Ringer's lactate prior to performing the block and uses urinary specific gravity as an indicator of hydration. Our fluid administration policy is similar, but more conservative in those infants who still retain fluid in their lungs.

In our report on 21 spinal anesthetics, we referred to the absence of cardiovascular instability. Currently, we have data on 30 patients. There were no instances of bradycardia. We see transient rises of 20 beats/min and attribute this to excessive physical stimulation caused by positioning, prepping, draping, *etc.*

BP changes did not exceed 10 mmHg, with the exception of one 4,860-g infant whose systolic pressure dropped from 90 to 75 mmHg. This was not considered deleterious, and it self-corrected in 10 min without intervention. We did not observe any retching. Atropine was only given if general anesthesia supplement was required.

Abajian *et al.*[3] administered 29 spinal anesthetics to premature infants and in 16 of these, 0.01 ng epinephrine was added. Although they did not report specifically on pulse rate and BP change differences between these two groups, they report no bradycardia or hypotension. Thus, the possible systemic effect of absorbing epinephrine is unlikely to play any role in supporting cardiovascular stability.

Dr. Dohi reminds us that the sympathetic nervous system is incompletely developed at birth and the parasympathetic is dominant. This well-known feature of the infant's circulation is further accentuated in the premature infant.

Cardiac output is rate-dependent in early infancy. Atropine was not administered routinely in our series. With this in mind, had sympathetic blockade occurred, bradycardia and hypotension would have featured as Dr. Dohi suggests. It may well be that the immature sympathetic nerves are resistant to local anesthetics. There are