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

other similar pharmacologic responses in the perinatal period, for instance ketamine requirement is much greater in infants under 6 months of age, and Lockhart and Nelson[4] suggested changes in neuronal density, incomplete myelination, or impaired axonal transmission as possible explanations. One may speculate that a common factor relating to neural maturity could explain both the larger local anesthetic dose requirement and the subsequent absence of sympathetic blockade. Dr. Dohi and his group have shed some welcome light on this puzzling feature of spinal anesthesia in the very young.

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Clinical Significance of Perioperative T-wave Inversion

To the Editor:—Breslow *et al.*[1] showed that patients with perioperative T-wave changes did not have an increased incidence of adverse cardiac events in the immediate postoperative period. They did not present a long-term follow-up of these patients.

In a 1–2-yr follow-up of patients with unstable angina and new T-wave inversions of > 2 mm, a higher incidence of adverse cardiac events was observed by Haines *et al.*[2] Granborg *et al.*[3] found in a 3-yr follow-up that in patients suspected of acute myocardial infarctions, the number of leads with transient T-wave inversions as well as the sum of negative T-wave amplitudes significantly correlated with the rates of acute myocardial infarctions and death.

In the immediate postoperative period, most cardiac events are not associated with anginal pain. The patient with known or suspected coronary artery disease may have a significant intraoperative ischemic event leading to no externally detectable manifestations other than T-wave inversion.

In the study of Breslow *et al.*,[1] 40 out of 394 patients had known or suspected coronary artery disease. Although not stated in the report, it is likely that few or none of these patients had unstable angina. The incidence of significant ischemic events in the perioperative period under these circumstances is not known, but it is suspected to be quite low. Nine of 40 patients who had new T-wave abnormalities did not have adverse cardiac events in the perioperative period. If they were to be followed for 2–3 yr, it is possible that they would have higher morbidity and mortality than the remaining 31 patients. In addition, if the sample size had been significantly greater than nine, perioperative ischemic events may also have been detected.

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In reply:—Dr. Jain has questioned whether perioperative T-wave abnormalities are associated with late cardiac complications (1–3 yr postoperative). This suggestion is based on data demonstrating a relationship between late cardiac complications and T-wave abnormalities in patients presenting with acute ischemic events (unstable angina, acute myocardial infarction).

Dr. Jain's comments are correct but not relevant to our