In Reply.—We agree in general with the authors' findings, but have some additional comments to help clarify the situation.

1. The primary purpose of the pacemaker enhancement circuit is to clearly identify and consistently display pacemaker spikes. Without this circuit, pacemaker spikes would be displayed with varying height. Some may not be displayed at all. This is because the frequency content of the spikes is well above that of the monitor, which is artificially limited to obtain electrocardiogram noise rejection and display performance. Missed and varying sized pacemaker spikes on the ECG display could be misunderstood as erratic pacemaker performance.

2. The impact of other sources of electrical spikes on the pacemaker circuit, such as those from neuromuscular blockade stimulators and somatosensory evoked potentials (SEPs), may be reduced and even eliminated by selecting another ECG lead on the 2000, where the amplitude of the spikes is below the activation threshold of the pacemaker detection circuit.

3. If a 2000R or 2000RS strip chart recorder is available for use with a 2000 or 2000A monitor, the unprocessed ECG can be recorded by switching to “Diagnostic ECG.” This mode records the amplified input ECG to AAMI diagnostic ECG recommendations (0.05 Hz to 100 Hz), without pacer enhancement. When switched to “Delayed ECG,” the screen ECG is recorded, including pacemaker enhancement and reduced frequency response (AAMI monitor bandwidth 0.5 Hz to 40 Hz).

4. A special modification to 2000 and 2000A series monitors can be made to disable the pacemaker enhancement circuit if required.

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High Spinal Anesthesia in an Infant

To the Editor:—We recently observed an unusual complication of a spinal anesthetic in an infant.

A 5-kg, 20-week-old former premature infant who was now 50 weeks postconceptual age was admitted for bilateral inguinal repair. He was otherwise healthy. An intravenous catheter was inserted, and a small dose of ketamine was given for sedation prior to positioning for a spinal anesthetic. Hyperbaric, tetracaine, 1.5 mg, with an epinephrine wash was injected intrathecally at the L4-5 interspace with the patient in the lateral decubitus position. Within the first minute after the infant was turned supine, the legs and buttocks were lifted to place a grounding pad on the lower back. Shortly after, arterial desaturation to 85% was detected by pulse oximetry. Evaluation of the infant revealed labored breathing with marked retractions. There was no response to pinprick along the entire chest wall and abdomen. Heart rate and blood pressure were unchanged from initial values. Oxygen was given and the trachea was intubated following ketamine, atropine, and succinylcholine. The remainder of the case was uneventful. The trachea was extubated 90 min later with a slight residual lower extremity blockade.

Spinal anesthetics are being used with increasing frequency in “high-risk” infants to avoid the potential complications of a general anesthetic. To our knowledge, high spinal levels resulting in respiratory distress have not been previously reported in this patient population, in spite of doses as large as 0.65 mg/kg of tetracaine. Our dose of 0.3 mg/kg with epinephrine was moderate and should not have caused an extremely high level. We suspect that the administration of epinephrine to the baby might have increased the uptake of the tetracaine to the hyperbaric solution to spread cephalad. We now place the grounding pad prior to turning the baby supine and have experienced no further problems.

The other factors deserve mention. First, in spite of a high spinal level and without a preceding volume load, there were no significant hemodynamic changes. This is in accord with the lack of hemodynamic change following spinal anesthesia reported in infants.4 Second, the onset of respiratory distress was heralded by the alarm of the pulse oximeter. Pulse oximetry is as invaluable for regional anesthesia in children as it is for general anesthesia.5

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