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Time of Peak Hypotension during Rapid Induction Approximates Time of Peak Brain Halothane Tension in the Dog

To the Editor:—Behnia and Koushanpour[1] conclude that halothane suppresses the baroreceptor reflex by action on the vasomotor center, or perhaps on an inhibitory efferent pathway, rather than by a direct effect on the baroreceptors themselves. Workers in this field may be interested in our data, arising as an aside to an unrelated study,[2] which lend support to this conclusion.

During the testing in dogs of an automatic control system for induction and maintenance of halothane anesthesia, a precipitous decrease in blood pressure invariably was observed during induction. We were not especially surprised at this serious hypotension because, since one of the performance criteria of the study was to see how rapidly the system could increase the animals' brain tension of halothane to the desired level, inspired tension was taken very rapidly to 4% and the arterial tension (computed from a mathematical model) intentionally was driven above the desired brain tension transiently. However, we were interested to note that the peak arterial halothane tension in the model occurred at 85 s (SD 30 s) from start of induction and peak brain tension occurred at 145 s (SD 31 s), while peak depression of blood pressure (to 54% (SD 16%) of the preinduction value) occurred at 129 s (SD 41 s).[2] This peak blood pressure effect is much nearer the time of peak brain tension than to the time of peak arterial tension, which is the opposite of what would be expected if halothane acted directly on the baroreceptors.

Our published conclusions were specific to the aims of our own study. However, a more general conclusion would be similar to that of Behnia and Koushanpour: that suppression of baroreceptor reflex by halothane is less likely to be a direct effect on the baroreceptors than it is to be a central effect.

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Postoperative Methemoglobinemia in a Neonate

To the Editor:—We feel that the following case report is worthy of your readers' attention.

REPORT OF A CASE

The second twin, birth weight 640 g, gestational age 26 weeks, required urgent ligation of a patent ductus arteriosus. When presented for surgery, the baby was 7 days of age and weighed 580 g. He had been on continuous mechanical ventilation for severe respiratory distress syndrome since birth.

The anesthesia was accomplished without incident, and vital signs (ECG, rectal temperature, intraarterial blood pressure) remained stable during an anesthetic based on fentanyl, pancuronium, oxygen, and nitrogen. During surgery, the baby received 0.01 mg of atropine, 10 μg fentanyl, and 100 μg pancuronium. At the end of the procedure, following transfer to the Neonatal Intensive Care Unit, the patient's color was peculiarly dusky, in spite of adequate ventilation with an $F_{I_{O_2}}$ of 1.

The arterial blood gas analysis at that time revealed the *p*H to be 7.38, P_{CO_2}—38 mmHg, P_{O_2}—112 mmHg, and O_2 Sat—79%.

The discrepancy between the apparent satisfactory P_{O_2}, the O_2