To the Editor.—In their recent report entitled “Sevoflurane Inhibits Human Platelet Aggregation and Thromboxane A2 Formation, Possibly by Suppression of Cyclooxygenase Activity” (Hirakata H et al., Anesthesiology 1996;85:147-53), the authors demonstrate deterioration in platelet functions by a low concentration of sevoflurane; 0.5%. Such a strong inhibitory effect on platelet aggregation is inconsistent with the fact that clinically serious hemorrhagic complications have never been observed in sevoflurane anesthesia. Although their study is well done and informative, we have one concern about their methodology. They used ethanol in the platelet samples to dissolve inhalation anesthetics. Ethanol has been generally recognized to inhibit platelet aggregability. Moreover, the reporter findings that similar concentration of ethanol did not affect platelets were obtained using rabbits and rats. The platelet reaction to the aggregating agents differs with the species. For example, rabbit and rat platelets show essentially the primary aggregation alone in an adenosine diphosphate (ADP)-induced aggregation study (4). In a human in vitro study using ADP, platelet aggregation was inhibited even with ethanol at less than 100 mM (5) (they used 0.5% v/v of ethanol, which corresponds to approximately 100 mM). Thus, the inhibitory effect they demonstrated might be attributed to ethanol. We found that platelet aggregation in healthy volunteers (n = 5) is inhibited by ethanol at the same concentration as they used on platelet aggregation. We conclude that the inhibitory effect of sevoflurane on platelet aggregation they reported is attributed to the presence of sevoflurane and ethanol.

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References

(Accepted for publication June 19, 1997.)

In Reply:—We appreciate the interest shown by Drs. Aoki and Mizobe in our manuscript. However, we believe that the effect of ethanol used to dilute the anesthetics cannot explain our finding with sevoflurane. We used the same range of ethanol to dilute all the concentrated anesthetics (including halothane, sevoflurane and isoflurane) and isoflurane with ethanol did not affect platelet aggregation (fig. 1). The concentration of ethanol stated in the manuscript (less than 0.5%) was the possible maximal concentration, and was calculated basing on the total volume of ethanol divided by the volume of platelet-containing solution in the test tubes. Because of the presence of a gas space in the parafilm-sealed tube, the concentration of ethanol in the liquid phase could have been lowered as that in the gas phase increased during incubation at 37°C. In contrast, the concentration of volatile anesthetics mentioned was, of course, that in the liquid phase, confirmed by gas chromatography. This may explain why ethanol did not affect platelet aggregation during our experimental conditions.

As Drs. Aoki and Mizobe pointed out, no clinical report has ever suggested increased blood loss or blood transfusion during general anesthesia with sevoflurane. This situation can also be extended to halothane anesthesia, although many investigators have reported suppressive effects of halothane on platelet aggregation. The fact that the amount of hemorrhage during surgery depends on the surgical technique or skill of the surgeon probably makes clinical studies in this field difficult.

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Anesthesiology, V 87, No 4, Oct 1997
References


(Accepted for publication June 19, 1997)

Postdural Puncture Headache and Epidural Blood Patch

To the Editor.—The case report by Borum et al.1 illustrates the difficulty in the differential diagnosis of headache in a parturient. Postpartum headache may be due to dural puncture, eclampsia, menin gitis, or cortical vein thrombosis. Epidural blood patch is a recognized treatment for postdural puncture headache (PDPH). However, in the other conditions, injection of blood in the epidural space may cause complications.

Prolonged leakage of cerebrospinal fluid (CSF) may occur after a dural puncture. Before performing a blood patch, it is advisable to demonstrate this leakage. Cerebrospinal fluid rarely emerges from the needle when an epidural is performed in a patient with PDPH. In a series of 50 patients with PDPH, the CSF emerged from the epidural needle in only 8 patients (16%).2 However, injection of an epidural catheter and gentle aspiration revealed CSF in the epidural space of all these patients.3

The compliance of the dura depends on the volume and pressure of CSF contained within it. The CSF pressure is low in patients with PDPH.1 The compliance of the dura decreases when the CSF pressure is low. Reduction in the compliance of the dura lowers the epidural pressure.4 The epidural pressure gives a rough estimate of the prevailing CSF pressure.5 The mean epidural pressure of parturients is approximately 15 cm H2O in the lateral posture.6 However, it decreases in patients with PDPH. In 50 patients with PDPH the mean epidural pressure was 6.4 cm H2O.7

Demonstration of free CSF in the epidural space and a low epidural pressure assists in the differential diagnosis of PDPH in the parturient. Before performing a blood patch, it is advisable to demonstrate free CSF in the epidural space and a low epidural pressure. Epidural pressure is easily measured using a 16-gauge (1.1 mm OD) epidural catheter as a simple manometer.8,9 Injection of blood through a 16-gauge epidural catheter is easy and safe.10

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References


Anesthesiology, V 87, No 4, Oct 1997

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