also has instituted a registry of "blood patch" treatment for postspinal headache, the results of which are published in this section.[5]

Annual meetings of SOAP have planned scientific sessions. Formal presentations on a specific topic are delivered by guest speakers. Reviews of "what is new in obstetric anesthesia," "what is new in obstetrics," and "what is new in neonatology" are given by members who are specialists in these fields. Presentations of "work-in-progress" are followed by discussions and constructive criticism. Next year's SOAP Meeting will be held in Philadelphia under the aegis of B. B. Gutsche. All interested physicians are welcome!

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Halothane MAC in the Rabbit

To the Editor:—The development of a reliable method for endotracheal intubation and halothane anesthesia in white New Zealand rabbits[1] has facilitated the use of these animals for major surgical and shock experiments in our laboratory. To monitor the rabbits a simple, reliable means for assessing anesthetic depth was needed. Therefore, we determined the minimum alveolar concentration (MAC)[2] of halothane in unpremedicated, spontaneously breathing rabbits under normothermic, normotensive conditions with blood gases maintained in the normal range. MAC was determined as the mean of the lowest alveolar concentration preventing and the highest permitting a response when the rabbit's tail was clamped with a bulldog arterial clamp. MAC was determined after an anesthetic equilibrium had been reached, assessed as inspired halothane concentration approximately 10 per cent higher than expired gas tension. Expired gas samples were obtained by aspirating serial (1 ml) samples at the end of each spontaneous expiration through an Intracath catheter located at the tip of the endotracheal tube. The mean halothane MAC for these rabbits was 0.82 ± 0.3 vol per cent. Arterial blood samples were simultaneously drawn with the end-tidal gas samples and also analyzed for halothane. The average arterial halothane value corresponding to MAC was 27.6 ± 8 mg/100 ml. Both arterial blood and end-tidal gas samples were analyzed for halothane by gas chromatography. Our studies are in agreement with findings of other investigators[3] who have shown that the anesthetic requirement for halothane cannot be accurately measured from inspired gas samples. The inspired-to-alveolar gradient in our rabbits showed the inspired halothane concentration to be approximately 10 per cent higher than the alveolar concentration.

These "MAC" values for both alveolar and arterial blood halothane concentrations are reported for the information of other investigators interested in the anesthetic requirement for halothane of the rabbit.

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Isoflurane Metabolism

To the Editor:—The paper "Metabolism of Isoflurane in Fischer 344 Rats and Man" by Hitt *et al.* (ANESTHESIOLOGY 40:62–67, 1974) shows the formation of fluoride ion and "non-ionic" fluoride, probably trifluoroacetic acid, from isoflurane *in vivo*. Two possible pathways for the metabolism are given, and while the equations are not balanced, they both predict, by stoichiometry, two moles of fluoride ion for each mole of trifluoroacetic acid. Furthermore, because of the known lack of biotransformation of trifluoromethyl groups[1,2] and the known ease of biotransformation of CF_2H groups,[2,3] for each mole of isoflurane metabolized two moles of fluoride ion and one mole of a trifluoromethyl-containing metabolite (*i.e.*, trifluoroacetic acid) are to be expected regardless of the pathway.

Summing the values for excretion given in figures 1 and 2 in the paper by Hitt *et al.* shows that the ratio of fluoride ion to trifluoroacetic acid in rats is approximately 1.6 to 1, close to stoichiometric. However, in man the ratio is 0.3 to 1. Thus, the evidence needing explanation in the comparison of rats and man is not the high fluoride excretion in rats but the low fluoride excretion in man.

The authors suggest three possible answers for the patterns of fluoride excretion. Two of the suggestions, more complete biotransformation of non-ionic intermediates in rats and binding of non-ionic fluoride to protein, would explain high fluoride ion-to-non-ionic fluoride ratios but not the observed low ratios. The third suggestion, differences in renal excretion, deserves more consideration, but the focus should not necessarily fall on the kidney. It is well known that not all the fluoride ion ingested is excreted in the urine.[4] The amount excreted is often 50 per cent or less. Therefore, the question of the fate of the missing fluoride in the metabolism of isoflurane in man remains unanswered.

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