In a fifth group of six dogs we tested the hypothesis that ether may stimulate ventilation by producing cerebrospinal fluid (CSF) acidosis. These dogs were anesthetized first with halothane to 1.5 MAC and then with ether to the same MAC. Arterial P_{CO_2} and base deficit during ether anesthesia were held at those levels found with halothane by rebreathing and addition of intravenous $NaHCO_3$. Results obtained were: during halothane: Pa_{CO_2} 43 mm. of mercury, arterial *p*H 7.33, CSF HCO_3 24.7 mEq./liter and minute volume 3.5 liters/minute. Values during ether were: Pa_{CO_2} 43 mm. of mercury, arterial *p*H 7.34, CSF *p*H 7.31, CSF HCO_3 25.8 mEq./liter and minute volume 10.0 liters/minute. In three dogs still at 1.5 MAC with ether, the inspired CO_2 was subsequently eliminated. In these dogs, Pa_{CO_2} fell to 33 mm. of mercury, arterial *p*H rose to 7.39, CSF *p*H rose to 7.36, while CSF HCO_3 and minute volume decreased to 24.1 mEq./liter and 8.5 liter/minute, respectively. *Conclusion:* We conclude that vagotomy, carotid sinus denervation and peripheral and sympathetic blockade by spinal anesthesia do not depress ventilation during ether anesthesia. Further, arterial or CSF acidosis does not explain the ventilatory stimulation associated with ether because ventilation was three times greater in the ether dogs than in the halothane dogs at nearly identical and peripheral acid base status. That Pa_{CO_2} is normal at a time that the CO_2 response is depressed suggests that ether probably produces central respiratory depression, the effect of which is antagonized by a central respiratory stimulation. The specific sites of these conflicting central effects of ether are unknown. (This work was supported in part by USPHS Grant 5 RO1 HE 07946.)

Effects of Urea Administered During Hypothermia and General Anesthesia on the Osmotic Fragility of Human Red Cells. MARK B. RAVIN, M.D., and RICHARD S. MATTEO, M.D., *Department of Anesthesiology, Columbia University, College of Physicians and Surgeons, and the Anesthesiology Services, The Presbyterian Hospital, and the Jewish Memorial Hospital, New York City.* Following a report of the occurrence of intravascular hemolysis after the intravenous administration of urea during hypothermia (Ravin, Garber, and Gibson: ANESTHESIOLOGY 25: 576, 1964), a study was undertaken in anesthetized neurosurgical patients undergoing hypothermia to determine the effects of the intravenous administration of urea on the osmotic fragility of their red cells (Matteo and Ravin: ANESTHESIOLOGY 27: 318, 1966). This study has been extended to include the effects of cyclopropane and halothane on red cell osmotic fragility. *Method:* For the urea study, a Teflon catheter was inserted percutaneously in a brachial artery prior to the induction of anesthesia. Arterial blood samples were collected in heparinized Luer-Lok syringes before the induction of anesthesia, 30 minutes after anesthesia was established, immediately before and after the intravenous infusion of urea. The usual dose of urea was 0.5 g./kg. (40 per cent solution in 5 per cent invert sugar) administered intravenously over a 15 to 20 minute period. The blood samples were immediately analyzed for serum osmolarity, *p*H, P_{CO_2}, oxygen saturation, microhematocrit and osmotic fragility. Esophageal temperature was monitored. Following induction of anesthesia with intravenous thiopental, endotracheal intubation was facilitated with succinylcholine. Anesthesia was maintained with nitrous oxide 70 per cent–oxygen 30 per cent, supplemented by intravenous d-tubocurarine and chlorpromazine. Ventilation was mechanically controlled at a minute volume 110 to 125 per cent of that estimated from the uncorrected Radford Nomogram. Similar induction technique was employed in the cyclopropane and halothane studies. However, the patients were allowed to breathe spontaneously mixtures of either cyclopropane 20 per cent–oxygen 80 per cent or halothane 0.7–1.2 per cent in oxygen. Osmotic fragility for each sample was determined by a modification of the quantitative method described by Ham (Syllabus of Laboratory Examinations, Harvard University Press, 1951, p. 162). The per cent hemolysis of the red cells at varying concentrations of hypotonic saline was determined with a Beckman Model 8 Spectrophotometer. These values were used to construct four curves of osmotic fragility for each patient. *Results:* Neither hypothermia ranging