ventilation rate is increased there is a slight increase in the mean size of the brain. A higher endotracheal pressure will cause wider pulsation and a simultaneous increase in mean size of the brain. Since hypercapnia and hypoxemia can both be present during obstructed breathing, one could assume that there would be an increased blood supply to the brain. Intravenous dextran was administered to a group of animals to quickly increase circulating blood volume. It was found that infusion of 150 cc. of dextran produced a dramatic increase in brain size. *Comment:* It is suggested that during respiratory obstruction blood is shifted in each cycle to the pulmonary and intrathoracic vessels on inspiration and to the peripheral circulation, particularly to the brain, on exhalation. The amount of blood going to the brain is enhanced in those situations which create hypercapnia.

Determination of Halothane-Ether Ratios by Infrared Spectrometry. JOHN I. DAVIES, M.D., SEYMOUR BAKERMAN, PH.D., M.D., GARETH B. GISH, M.S., STEVEN N. ANGELL, A.B., and EVAN L. FREDERICKSON, M.D., *Division of Anesthesiology, University of Kansas Medical Center, Kansas City, Kansas.* Halothane-ether mixtures form an azeotrope in proportions of halothane 68.3 per cent and diethyl ether 31.7 per cent by volume (Boivin, P. A., Hudon, F., and Jacques, A.: Canad. Anaesth. Soc. J. *5:* 409, 1959; Hall, K. D., Norris, F., and Downs, S.: Anesthesiology *21:* 522, 1960), and gas chromatography will separate the two components. Biological membranes may also differentiate between the two components. The purpose of this study was to ascertain whether a difference could be demonstrated in the proportions of halothane and ether in respired gases during induction of, and recovery from, anesthesia using the azeotrope. *Method:* Samples for analysis were collected as follows: (1) Directly from a Fluotec vaporizer containing the azeotrope and vaporized with oxygen. (2) In a closed system following induction to a moderately deep surgical plane using the azeotrope in oxygen (after minimal thiopental, succinylcholine, topical cocaine and intubation). (3) Following surgery, with oxygen being inhaled, exhaled gases were collected using a Rudolph Anesthesia Valve. Oxygen, nitrogen and carbon dioxide in the samples were removed in a vacuum system by distillation using liquid nitrogen (−195° C.), acetone and dry ice (−79° C.), and ice water mixtures (0° C.). The samples were transferred in the vacuum system to a sample cell path length of 10 cm. and a volume of 200 ml. at known pressures and at room temperature for infrared analysis. The Perkin-Elmer Model 21 double-beam infrared spectrometer with sodium chloride optics was used to analyze the gases in the sample cell. The instrument was calibrated with water, carbon dioxide and polystyrene between 2 and 15 microns. The spectra of ether and of halothane were determined at various pressures. There was at least one prominent band for each compound between 2 and 15 microns wave length that was not overlapped by bands of the other component—3.5 micron band of ether and the 14 micron band of halothane. Optical density concentration curves were constructed for each compound and for the azeotrope using the base-line optical density technique (Heigl, J. J., Bell, M. F., and White, J. V.: Anal. Chem. *19:* 293, 1947). The calibration curves for halothane and for halothane in the azeotrope were superimposed, while those curves for ether and for ether in the azeotrope showed a pressure broadening-like effect. Therefore, the calibration curves for each component in the azeotrope were used as standard references. *Results:* Satisfactory samples were obtained from 6 patients. Results attained by the base-line method of analysis of these samples for determining optical density and interpreted by the Beer-Lambert law show that during induction of anesthesia the ratio of halothane to ether increased from a mean (± S.D.) of 2.39 (± 0.15) to 3.03 (± 0.37). This shows that ether is preferentially absorbed by the body from the azeotrope. During the immediate postanesthetic period, the ratio of halothane to ether is decreased to a mean of 1.68 (± 0.25) and shows that ether is eliminated in a greater proportion than in the original azeotrope. These differences are found to be significant ($P \leqq 0.001$) according to the *t* test. These results demonstrate that bio-

logical mechanisms exist that are able to break the "bonds" that are resistant to distillation in the halothane-ether azeotrope.

Uptake and Distribution of Intravenous Ether. EDMOND I. EGER, II, M.D., and EDWARD A. JOHNSON, M.D., *Anesthesia Department, University of California Medical Center, San Francisco, California.* We have measured the rate of administration of 5 per cent intravenous ether necessary to maintain a constant level of anesthesia. *Method:* Ether solution was infused into subjects at a rate sufficient to achieve and maintain electroencephalographic level 4. The minute by minute volume of 5 per cent ether thus required was noted and later converted to milliliters of pure ether vapor. *Results:* Uptake of ether vapor was found to average 900 ml. per minute during the first 5 minutes, rapidly falling to half this in the second 5 minutes. Uptake in succeeding 5 minute intervals gradually decreased. At 60 minutes, uptake averaged 180 ml. per minute. *Comment:* This rate of uptake also represented the rate at which ether was taken up by the tissues since the conditions of the study made loss to other areas negligible. Since a given EEG level may be equated to a constant arterial level of ether, then this study describes body uptake of ether at a constant arterial tension. A mathematical model incorporating the effect of differential tissue perfusions, tissue-blood concentration gradients, and tissue-blood partition coefficients was used to describe the data obtained. Good correlation was shown between the data obtained experimentally and theoretically. The model adequately described similar data for nitrous oxide and for cyclopropane. All such uptake curves were found to follow a pattern of rapidly decreasing uptake during the first few minutes of anesthesia with a subsequent gradual decline. A breakdown of the distribution of agents in the model indicated that well-perfused tissues account for the first few minutes of anesthesia, that muscle is the main depot for loss for the succeeding 1–2 hours, and that fat and poorly perfused tissues form a base line of uptake which continues during and beyond the two periods noted above. Variations in the basal state were shown to affect the uptake predicted by the model. Shock decreased uptake throughout anesthesia while excitement caused an initial high uptake which rapidly fell to below the basal state.

Hemodynamic Effects of Oxygen Inhalation. G. W. N. EGGERS, JR., M.D., W. J. DE GROOT, M.D., AND J. J. LEONARD, M.D., *Section of Anesthesiology, University of Missouri School of Medicine, Columbia, Missouri. In conjunction with the Department of Internal Medicine, University of Texas Medical Branch, Galveston, Texas.* The hemodynamic effects of oxygen breathing on awake, healthy volunteers have been studied in our laboratory (Eggers, G. W. N., Jr., and others: J. Appl. Physiol., in press). These studies revealed that oxygen inhalation resulted in: decreased cardiac index (0.37 l./minute/m², 12 per cent, $P < .02$); decreased heart rate (4 beats/minute, 6.5 per cent, $P < .05$); increased mean systemic arterial pressure (6.1 mm. Hg, 7.5 per cent, $P < .01$); increased systolic (10 mm. Hg, 9.4 per cent, $P < .01$) and diastolic (6 mm. Hg, 10.3 per cent, $P < .01$) arterial pressures; increased systemic vascular resistance (242 units, 22 per cent, $P < .01$); and no change in either central blood volume or stroke volume. Arterial *p*H and P_{CO_2} were unchanged during oxygen breathing. Surprisingly, the pressor effects of oxygen inhalation persisted for at least forty minutes after oxygen was discontinued and the arterial blood oxygen content had returned to the control values. This pressor phenomenon was believed to be due to one of two mechanisms: (1) vascular constriction as a result of increased tissue oxygen tension, or (2) increased sympathetic tone due to an increase of carbon dioxide in the central nervous system which occurs during oxygen breathing (Lambertsen, C. J., and others: J. Appl. Physiol. *5:* 803, 1953). To ascertain which of these mechanisms was the true one, we repeated the previous study in the presence of an autonomic blocking drug. *Method:* Four young, healthy, male volunteers were studied as in the previous report except that cardiac output determinations were performed using centrally injected indocyanine green dye rather than RISA. After control values were obtained for each subject, an intravenous infusion of trimethaphan was begun