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/m^2., 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, 7.8 per cent, $P < .01$); increased systolic (10 mm. Hg, 9.4 per cent, $P < .01$) and diastolic (8 mm. Hg, 10.3 per cent, $P <$ [illegible]) 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

and stabilized. Sympathetic blockade was considered adequate if the secondary pressor response and "overshoot" were absent following a Valsalva maneuver (Price, H. L., and Conner, E. H.: J. Appl. Physiol. *5:* 499, 1953). The subjects then breathed compressed air from a Tissot spirometer for 20–30 minutes. Cardiac output, direct systemic arterial pressure, electrically integrated arterial mean pressure, arterial blood *p*H, arterial blood oxygen and carbon dioxide content, and hematocrit were determined. The presence of sympathetic blockade was again determined and the Tissot spirometer flushed and filled with oxygen. After inhaling oxygen for at least twenty minutes, the same determinations were obtained. A third test for sympathetic blockade was then performed. *Results:* The hemodynamic responses to oxygen breathing during sympathetic blockade were as follows: decreased cardiac index (0.14 l./minute/m^2., 4.4 per cent $P <$ .10); decreased heart rate (6 beats/minute, 6.1 per cent, $P <$ .01); increased mean systemic arterial pressure (11 mm. Hg, 17.5 per cent, $P <$ .20); increased systolic (14 mm. Hg, 16.1 per cent, $P <$ .10) and diastolic (10 mm. Hg, 19.6 per cent, $P <$ .20) arterial pressure; increased systemic resistance (218 units, 25.2 per cent, $P <$ .05); and no change in stroke volume. Arterial *p*H and P_{CO_2} were essentially unchanged during oxygen breathing. *Comment:* With such a small number of subjects, the occurrence of a statistically significant rise in systemic vascular resistance is interesting. The similarity of the hemodynamic response of oxygen breathing during sympathetic blockade to the response in intact subjects is remarkable. During sympathetic blockade with loss of arteriolar constriction, the peripheral vascular resistance is maintained and altered by the capillary bed (Griffiths, H. W. C., and Gillies, J.: Anaesthesia *3:* 134, 1948). Therefore, we concluded that the pressor effect of oxygen breathing was due to a vascular constriction, specifically in the capillary bed, rather than due to an increase in sympathetic tone. [Supported by the Texas Heart Association.]

Clinical Experiences with Electronarcosis for Surgical Patients. L. W. FABIAN, M.D., J. D. HARDY, M.D., M. D. TURNER, PH.D., F. J. MOORE, M.D., and technical assistance of C. D. MCNEIL, *Departments of Anesthesiology, Surgery, and Psychiatry, University of Mississippi Medical Center, Jackson, Mississippi.* Following an extensive laboratory investigation using electrical current to produce anesthesia for surgical procedures in dogs, a clinical study of this method was instituted early this year. *Method:* The equipment employed for this project includes (1) a regulated power supply which is a full-wave transformer with a 6.3 volt AC winding which supplies filament voltage for the circuit, (2) an amplifier system and (3) an oscillator, described in detail by Hardy *et al.* (J. Surg. Res. *1:* 152, (July) 1961). The electrodes are circular metal plates approximately the size of a half-dollar and were placed bitemporally on the patients. The electrical parameters utilized for the present clinical investigation include a 700 cycle frequency, 35 to 40 volts and up to 100 milliamperes of current applied continuously throughout the procedures. The 19 patients studied to date in this series have all been adults, ranging in age from 40 to 65 years, who were scheduled for short surgical procedures such as cholecysto-jejunostomy or simple mastectomy. Each patient has received secobarbital 75 to 100 mg, and atropine 0.8 to 1.0 mg. for premedication. Endotracheal intubation has been performed in all patients with the aid of small doses of thiopental (100 to 200 mg.) and succinylcholine (60 mg.) intravenously. Topical anesthesia of the larynx using 5 per cent Cyclaine has also been employed in all patients. Throughout each procedure a constant drip of 0.2 per cent succinylcholine has been administered intravenously to control muscle spasms which are produced by the current and to allow more adequate control of ventilation since respiratory depression and decreased compliance are produced also by the current. During the period in which the electrical current is applied, manual control of respiration is maintained using 100 per cent oxygen at flow rates of 4 to 6 liters per minute and at a respiratory rate of 16 to 18 per minute. During the procedure blood samples are drawn from a brachial or radial artery for determinations of *p*H, P_{CO_2}, oxygen saturation, blood sugar, adrenocortical steroids, catechol amines and