at an inflow rate of 3 liters per minute. Rebreathing was reduced to 7.5 and 16 ml., respectively when an endotracheal tube was used with the Sierra and Ohio Swivel-Y valves. The Stephen-Slater and Digby Leigh valves and the Norman elbow, NRPR Elbo, Ayers "T," and Ohio Infant Circle show no rebreathing with an endotracheal tube if inflow gas exceeded 3 liters per minute; in fact, part of the dead space in the endotracheal tube was washed out. Denitrogenation was rapid in the open systems, slightly delayed in the valved systems, and prolonged in the circle systems. Hyperventilation in the open systems caused rebreathing but simultaneously reduced alveolar CO₂ tension. In the valved systems hyperventilation did not increase rebreathing. The valved systems improve in performance as tidal volume increases while the open systems require larger inflow. For the premature, or infant of less than 7 kg. with cardiopulmonary disability, the valved systems with a mask add too great a dead space to permit compensation by the infant. The adult circle with valve-in-chimney is marginal, even with an endotracheal tube in such infants, whereas, the open systems perform well if inflow is adequate (Lim, H. S., and others: Anesthesiology 26: 254, 1965). (This study was supported in part by funds provided under Public Health Service Research Grant HE-09010 from the National Heart Institute, Public Health Service.)

Effect of Cyclopropane on Glucose Assimilation Coefficient in Man. F. W. Chenvenko, M.D., and N. M. Greene, M.D. Department of Anesthesiology, Yale-New Haven Hospital, Section of Anesthesiology, Yale School of Medicine, New Haven, Connecticut. The effect of cyclopropane on glucose metabolism was evaluated under clinical conditions in patients undergoing elective surgery, aged 23-66, who had no evidence of metabolic disorders. Patients were unpremedicated (except for atropine in 4) and received no other anesthetics or drugs except for muscle relaxants. In one group (5 patients) venous blood samples showed blood glucose levels to rise significantly from preinduction control levels of 74.8 ± 2.6 mg./100 ml. to a peak of 94.2 ± 3.6 mg./100 ml. twenty minutes after start of anesthesia, following which blood glucose levels decreased to levels of 82.3 ± 2.1 mg./100 ml. over the next hour. In another group of patients, glucose utilization was evaluated by determination of the effect of cyclopropane on the glucose assimilation coefficient, K, as calculated 45-60 minutes following performance of rapid intravenous tolerance tests. Calculation of K as percentage per minute glucose disappearance from blood at the appropriate time avoids many of the problems otherwise associated with interpretation of absolute changes in blood glucose levels. In 5 of these patients who served as their own controls, preanesthetic values for K were 1.517 ± 0.137 in the absence of cyclopropane and 0.961 ± 0.087 in the same patients during cyclopropane anesthesia, the statistical comparison of which is significant. Additional studies in patients who did not serve as their own control showed K values of 1.85 ± 0.566 in three patients in the absence of anesthesia and 0.972 ± 0.311 in 3 patients during cyclopropane anesthesia. Summary: It is concluded that uncomplicated cyclopropane anesthesia in surgical patients is associated with only a mild hyperglycemia and that cyclopropane anesthesia is associated with a statistically significant decrease in glucose utilization. (Supported by a research grant from the Josiah Macy Jr., Foundation.)

Non-Adrenergic Vasoconstriction Produced by Halothane and Cyclopropane Anesthesia. Michael F. Cristoforo, M.D., and Michael J. Brody, Ph.D., Departments of Pharmacology and Anesthesia, College of Medicine, University of Iowa, Iowa City, Iowa. Controversy exists about effects of anesthetics on peripheral vasculature and central vasoregulatory mechanisms. While studying effects of halothane and cyclopropane (1.5 per cent and 35 per cent end-expired concentration) on baroreceptor reflexes produced in the cross-perfused dog gracilis muscle, it was noted that both anesthetics, administered to the donor dog, increased muscle perfusion pressure. Vasoconstriction was anticipated with cyclopropane, but was totally unexpected with halothane, which is considered to be a vascular smooth muscle depressant. It was the purpose of these experiments to elucidate the
mechanism of vasoconstriction observed with both anesthetics. *Methods and Results:* Dogs were anesthetized initially with sodium pentobarbital. A gracilis muscle of a recipient dog was vascularity isolated, denervated, and perfused at constant flow with arterial blood from a donor dog. Venous effluent from the muscle was returned to the donor. Administration of halothane and cyclopropane to the donor elicited vasoconstriction in the muscle before and after alpha-adrenergic blockade produced by intra-arterial injection of phentolamine. These experiments indicated that halothane and cyclopropane either liberated vasoconstrictor substances that are not catecholamines, or possess direct vasoconstrictor properties. To distinguish between these possibilities the completely isolated gracilis muscle was perfused with fresh oxygenated (95 per cent O₂, 5 per cent CO₂) blood from a reservoir utilizing a bubble oxygenator. Cyclopropane again produced vasoconstriction which was unaffected by alpha-adrenergic blockade. Halothane, however, produced vasodilatation before and after alpha-adrenergic blockade. Identical results were found when the intact dog, perfusing its own gracilis muscle with arterial blood, was converted to a heart-lung-gracilis preparation. These data indicated that the direct effect of cyclopropane is vasoconstriction while halothane is capable of releasing a non-adrenergic vasoconstrictor substance in the intact animal. The source of vasoconstrictor substance appeared to be the head, since in intact animals muscle vasoconstriction observed with inhalation of halothane was reversed to vasodilatation by elimination of the entire head from the circulation. Further evidence for this was obtained when one muscle was perfused with external jugular vein blood and a second muscle perfused with inferior vena cava blood. Considerably greater, and more immediate, vasoconstriction was observed in the muscle perfused with jugular blood. Experiments in which intracarotid hypertonic saline produced the same muscle vasoconstriction as did bilateral intracarotid halothane (0.01 ml.) and inhalation of halothane suggested that the substance released might be vasopressin. To test this hypothesis experiments were performed in dogs undergoing water diuresis. Intracarotid hypertonic saline, and halothane by intracarotid injection and inhalation, all significantly increased urine osmolality and decreased urine volume. Intracarotid halothane and hypertonic saline had no significant effect on the arterial pressure of the animal. Further evidence that halothane causes elaboration of vasopressin was obtained in experiments in which acute hypophysectomy of dogs completely abolished the vasoconstriction seen prior to hypophysectomy in the muscle perfused with jugular blood. *Summary:* Non-adrenergic vasoconstriction in skeletal muscle is produced by inhalation of halothane and cyclopropane. The vasoconstriction seen with cyclopropane is probably a direct drug effect. Halothane, by a mechanism other than hypotension, causes elaboration of the polypeptide vasopressin in sufficient concentration to cause vasoconstriction, increased urine osmolality, and decreased urine volume. (Supported by U.S.P.H.S. grants NB-04889 and 5T1-HE-5577.)

Use of Xenon and Xenon-Halothane in a Study of Basic Mechanisms of Anesthesia in Man. STUART C. CULLEN, M.D., EDMOND I. EGER, II, M.D., and PAUL GREGORY, Department of Anesthesia, University of California Medical Center, San Francisco. In 1951, Cullen and Gross (Science 113: 580) reported use of xenon anesthesia in man for operative procedures. This verified in human beings the observations of Lawrence et al. and Lazarov on the anesthetic properties of xenon in lower forms of life. A number of additional observations further demonstrated the anesthetic properties of xenon and indicated that xenon had little, if any, toxic properties. The observations suggested a potency to be somewhere between that of nitrous oxide and ethylene. Because xenon is too expensive for clinical use, the primary interest in this inert gas has been as a research tool to study the basic mechanisms of anesthesia. Our interest in xenon was stimulated recently by the reports of Pauling (Science 134: 15, 1961) and Miller (Proc. Nat. Acad. Sci. 47: 1513, 1961) on their closely related theories of anesthesia. Pauling stated that, "It is known that two anesthetic agents can cooperate to increase the stability of the hydrate framework." Increasing the stability presumably augments the an-