

standard embolus was administered at fifteen minute intervals for four administrations. Multiples of the standard embolus were then administered at fifteen minute intervals until a fatal dose was reached. In other dogs this procedure was modified by administration of certain drugs given immediately after each air embolus. Drugs tested were atropine, mephentermine, isoproterenol, trimethaphan, benzalkonium, papaverine. **Results:** An immediate and marked increase in right ventricular pressure was a consistent effect of venous air embolus. This increase persisted 5 to 15 minutes with gradual return to normal. After a massive embolus, increase in right ventricle pressure occurred briefly, before collapse of both pulmonary and systemic circulations. The first and second injections of standard embolus caused minimal brief arterial hypotension, third and fourth injections caused systemic hypotension of increasing severity. The ECG failed to reveal obvious changes early, only occasional PVC was seen. S-T depression and marked arrhythmia occurred late and coincided with marked systemic hypotension. Changes in the breathing pattern occurred with the smallest embolus, namely tachypnea or transient apnea followed by tachypnea. All animals died in apnea when not on a mechanical ventilator. When ventilation was maintained mechanically, alveolar P_{CO_2} decreased and P_{N_2} increased following embolus. Arterial hypotension was not present when these gas changes occurred. Cardiac murmurs were always heard and were obvious after a small volume embolus. There was great variability in the volume of the lethal dose of air. None of the drugs tested had any demonstrable effect on the course or outcome of these experiments. The aspiration of embolized air from the right ventricle or superior vena cava was disappointing and could not be regularly accomplished. **Summary:** Distinctive cardiac murmurs were always heard and were a most reliable sign of venous air embolus. Gross irregularity of the breathing pattern occurred after the smallest volume embolus. With mechanical ventilation, a fall in alveolar P_{CO_2} and increase in P_{N_2} occurred after venous air embolus. Systemic hypotension and obvious ECG changes were not seen after small volume embolus. Aspiration of

embolized air from the heart has been unreliable. Treatment of air embolus directed at pulmonary vascular dilatation has been unsuccessful.

Rebreathing in Pediatric Anesthesia Systems. ELWYN S. BROWN, M.D., and ROBERT F. HUSTEAD, M.D., *The Children's Mercy Hospital, Kansas City, Missouri.* **Method:** Rebreathing was determined in equipment used for infant anesthesia. External dead space was measured using an infra-red carbon-dioxide analyzer and flowmeter (Elam, J. O., and Brown, E. S.: *ANESTHESIOLOGY* 16: 886, 1955). Conventional screen or orifice type flowmeters modified the aerodynamic characteristics of the systems. Therefore, external dead space was studied by comparing curves from a model incorporating a double-pitot flowmeter in the trachea with curves obtained from patients, omitting flowmeters. The model consisted of a silicone rubber impression of the face and pharynx of a 3.45 kg. infant, trachea, and bellows for lungs. The model had an anatomical dead space of 9.5 ml. of which 4.5 ml. were in the trachea and bronchi. The physiologic dead space varied from 8 to 11 cc. and FRC was maintained at 50 ml. To simulate spontaneous respiration, a motor and crank moved the bellows 30 times per minute at constant tidal volume. CO_2 was introduced into the lung bellows and sampled at the lips or glottis. The rate of denitrogenation of the model and attached anesthesia system was measured using a nitrogen meter (Hamilton, W. K., and Eastwood, D. W.: *ANESTHESIOLOGY* 16: 861, 1955). The system was initially filled with air and was washed out with oxygen from the anesthesia machine. **Results:** The external dead space added by the Bennett no. 3, the Rendell-Baker Soucek No. 1, and the Ohio Newborn masks was 13, 8.7, and 7.4 ml., respectively. Using a Bennett no. 3 mask the Stephen-Slater, Digby Leigh and Sierra valves with a 90-degree elbow and the Ohio Swivel-Y valve increased the external dead space to more than 19 ml. The Foregger mask elbow with nipple, the Norman elbow, the NRPR Elbo, the Ohio Infant Circle, and the Stephen non-rebreathing mask reduced the dead air space under the mask to less than 5 ml. in the model

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 CO_2 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. CERVENKO, 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 1.854 ± 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