

anesthetic management from regional (continuous spinal) anesthesia, previously administered in 88 per cent of the cases, to general anesthesia. Serious cardiac arrhythmias (bradycardia, frequent premature ventricular contractions, ventricular tachycardia or fibrillation) were observed in five patients during general anesthesia—three of the five episodes were associated with tracheal intubation. *Summary:* The hazards of general anesthesia in anephric patients are minimized by preoperative preparation.

Tracheostomy Cuffs: Physical Characteristics and Physiologic Sequelae. R. CARROLL, M.D., M. HEDDEN, F.E.A.R.C.S., and P. SAFAR, M.D., *Department of Anesthesiology, University of Pittsburgh School of Medicine, Pittsburgh, Penna.* Recent reports have implicated tracheostomy cuffs as the primary etiologic factor in the production of subglottic stricture and tracheoesophageal fistula. Departmental experience suggested that the potentials of the cuffs to cause injury varied with residual volume. *Methods:* To test this hypothesis, cuffs were evaluated with a special strain gauge directly implanted in the anterior wall of tracheas. *In vivo* measurements during intermittent positive-pressure ventilation (IPPV) were conducted, monitoring cuff sealing area, intracuff pressure, airway pressure, and lateral tracheal wall pressure. Pressures were measured dynamically *in vivo* in the intact dog, as well as in excised canine and human tracheas. All measurements were made after the cuff had been expanded just to the point of abolishing leak, and again after overinflation by one ml. *Results:* Cuffs could be grouped into four categories, depending on residual cuff volume and sealing surface area. Group A cuffs had high residual volumes so that only a small amount of air was necessary to nudge the cuff walls against the tracheal mucosa. The large Sanders and Auchincloss cuffs are seal-inflating, in that airway pressure equilibrates across the soft cuff wall, thereby cyclically raising the intracuff and tracheal-wall pressures to equal airway pressure. Group B₁ included orotracheal tubes with built-in rubber cuffs. Tracheal wall pressures averaged 37.5 mm Hg. Group B₂ cuffs were

built into plastic tracheostomy tubes and had tracheal pressures ranging from 55 to 160 mm Hg. Group C included narrow, very-high pressure, unevenly-inflating, slip-on tracheostomy cuffs. Tracheal wall pressures always exceeded arterial pressure. *Summary:* Large-residual-volume cuffs allow IPPV at tracheal wall pressures never exceeding airway pressure, *i.e.*, usually of the order of capillary blood pressure. Small-residual-volume cuffs commonly produce tracheal-wall pressures in excess of arterial pressure. Double-cuff tubes offer no advantage, because they simply incorporate two narrow, low-volume, high-pressure cuffs.

Influence of Methoxyflurane on Myocardial Mechanics of the Digitalized Heart. P.-Y. CHEN, M.D., C. SHANKS, B.A., C. GAMBLE, B.S., and S. SHIMOSATO, M.D., *Department of Anesthesiology, Tufts University School of Medicine and New England Medical Center Hospitals, Boston, Mass.* Recently, we reported that methoxyflurane exerts a negative inotropic effect upon the contractile state of the intact canine left ventricle, as determined by the force-velocity relations (ANESTHESIOLOGY 29: 538, 1968). It has been shown that digitalis exerts positive inotropism in normal and diseased hearts, and that it may be used as a prophylactic measure to combat hypotension during anesthesia and surgery (J. Clin. Invest. 40: 52, 1961). This study was undertaken to ascertain whether the positive inotropic effects of digitalis counteract the negative inotropic effect of methoxyflurane on the performance of the intact left ventricle. *Methods:* Studies were performed in dogs anesthetized with chloralose-urethane given intravenously. Measurements of force-velocity relations were made before and following the administration of ouabain (0.05 mg/kg) and during methoxyflurane anesthesia at a mean arterial concentration of 17 mg/100 ml. *Results:* Methoxyflurane caused significantly less decreases in peak force in seven predigitalized dogs (-28 ± 4 per cent) than in eight nondigitalized dogs (-45 ± 4 per cent). Average changes in mean aortic pressure in predigitalized and nondigitalized dogs during anesthesia were -22 ± 3 per cent and -19 ± 2

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per cent, respectively. *Summary:* These findings suggest that prophylactic digitalization improves the inotropic state of the normal heart, especially the ability to develop the maximum force, during anesthesia. (Supported by USPHS Grant HE-01711 from the National Heart Institute.)

Effects of Halothane on Mitochondrial Oxygen Uptake: Site of Action. P. J. COHEN, M.D., B. E. MARSHALL, M.D., and J. LECKY, B.A., *Department of Anesthesia, University of Pennsylvania School of Medicine, Philadelphia, Penna.* *Methods:* Rat liver mitochondria were exposed for 20 minutes to halothane vaporized in air. Control mitochondria were treated similarly but exposed to air alone. In order to study reversibility, a portion of the exposed suspension was then equilibrated with air for an additional 20 minutes. Oxygen uptake was measured polarographically. Substrate (glutamate, 10 mM; succinate, 10 mM; and dihydronicotinamide adenine dinucleotide [NADH], 280 μ M), inorganic phosphate, 10 mM; oxygen, air-saturated reaction medium; and adenosinediphosphate, 250 μ M, were not rate-limiting. Since NADH does not penetrate intact mitochondria, the mitochondrial suspension was further treated by aging and resuspension in distilled water when NADH was to be substrate. *Results:* When glutamate was substrate, halothane produced a dose-related decrease in oxygen uptake. Halothane (0-10 per cent) had no effect on oxygen uptake when succinate was substrate. The effect of halothane on NADH oxidation was similar to that observed when glutamate was oxidized. In both cases inhibition was dose-related, was observed when less than 1 per cent halothane was administered, and was completely reversible provided that less than 3 per cent halothane had been used. Maximum inhibition (oxygen uptake 25 per cent of normal) was seen following exposure to 4 per cent halothane; concentrations greater than this had no additional effect. The addition of 5 mM amytal permitted the evaluation of amytal-sensitive oxygen uptake during NADH oxidation (Exper. Cell. Res., Suppl. 3, 124, 1955). Addition of amytal to control mitochondria reduced oxygen uptake to 25 per cent of nor-

mal; this represents amytal-insensitive respiration. In mitochondria exposed to less than 4 per cent halothane, amytal resulted in a further diminution of respiration to 25 per cent of control. In mitochondria whose oxygen uptake had already been reduced to 25 per cent of normal by concentrations of halothane greater than 4 per cent, amytal produced no further changes. Similar findings were made when amytal was added to a suspension oxidizing glutamate. *Summary:* The action of halothane upon the mitochondrial respiration chain is to inhibit NADH oxidation reversibly. Furthermore, since halothane inhibits only amytal-sensitive respiration, total oxygen uptake is not reduced below 25 per cent of normal by even high concentrations of halothane. (Supported in part by USPHS Grants GM-5-P01-09970-05, 5-T1-GM-215-01, 1-P01-GM-15430-01, and a grant from the Wellcome Trust.)

A Graphic Analysis of Cardiopulmonary Changes Following Major Surgery. F. J. COLGAN, M.D., and P. D. MAHONEY, M.D., *University of Rochester School of Medicine and Dentistry, Rochester, N. Y.* *Methods:* Twelve patients were studied before and after major upper abdominal surgery to determine the significance of any changes in cardiac output and FRC on intrapulmonary shunting. From this study, a method for the sequential plotting of changes in shunt in the critically ill patients was developed. Shunting was determined from simultaneously drawn samples of arterial and mixed venous blood and end-expired air. Cardiac output was determined by the Fick principle and FRC by closed-circuit helium dilution. *Results:* Prior to surgery, the mean total shunt breathing air was 22 per cent and the true shunt while breathing oxygen was 12 per cent. No change in mean total shunt, true shunt, or FRC occurred following surgery. Mean cardiac output for the group, however, was significantly reduced from 6 l/min to 4.8 l/min following surgery, and marked individual variation in both cardiac output and $\text{CaO}_2\text{-C}\bar{\text{v}}\text{O}_2$ was noted. If these changes had not been taken into account in computing shunt, a significant underestimation of the mean total shunt would have