

shortened the absolute refractory period slightly but increased the diastolic threshold by 30 per cent. When administered during epinephrine infusion, lidocaine returned the duration of absolute refractoriness and the diastolic threshold to normal. Acute digitalization of the non-failing heart produced a shortening of the refractory period, the absolute and relative periods changing in about the same proportion. The threshold during diastole was diminished by 30 per cent. The institution of cardiopulmonary bypass did not alter excitability. *Conclusions:* Current studies are centered on the effects of various depths of halothane anesthesia on ventricular excitability. Preliminary observations indicate that the diastolic threshold is markedly reduced (excitability increased) in lightly anesthetized patients.

Prolonged Apnea Following Succinylcholine Administration in Cancer Patients Receiving AB-132. RICHARD I. H. WANG, M.D., and CHARLES A. ROSS, M.D., *Roswell Park Memorial Institute, Buffalo, New York, and New York State Department of Health.* One of the postanesthetic complications that confronts the anesthesiologist is the failure of restoration of spontaneous breathing. The differential diagnosis of the causes of apnea rests on the anesthesiologist. In this report two cases of prolonged apnea will be presented. *Case Reports:* One patient with carcinoma of the lung was given intravenous thiopental anesthesia, 80 mg. of succinylcholine and hyperventilation. Bronchoscopy was performed under an apneic state. He failed to have spontaneous breathing for seven hours. Within 30 hours following bronchoscopy he had numerous episodes of hypoventilation, bradycardia and hypertension. He expired on the second postoperative day in spite of extensive supportive measures. Another patient with carcinoma of the urinary bladder was given intravenous thiopental and N₂O + O₂ anesthesia for cystoscopy. During bimanual abdominorectal examination muscle relaxation was required and he was given 20 mg. of succinylcholine intravenously. Spontaneous breathing did not return for an hour. Fortunately, subsequent postoperative recovery was uneventful. *Discussion:* In both cases the probable cause for the prolonged apnea was

believed to be low blood cholinesterase levels. Since both patients had liver function tests within normal limits, the reduced cholinesterase activities were likely the result of medication that they received prior to surgery. In reviewing their records it was found that they received a course of a cancer chemotherapeutic agent, AB-132 or ethyl-N-(bis(2,2-dimethylethylenimido)phosphoro) carbamate. In order to evaluate the effect of AB-132 on blood cholinesterase levels, the cholinesterase levels in plasma and red blood cells in four other cancer patients were measured before, during, and after a course of AB-132 therapy. It was found that following the intravenous administration of a single dose 0.5 g. AB-132, the plasma pseudo-cholinesterase level dropped markedly without significant decrease in the true cholinesterase activities. On repeated daily administration of the drug both the true and pseudo-cholinesterase activities decreased to as little as 20 per cent of their initial levels. AB-132 is a potent inhibitor of cholinesterase and resembles diisopropylpyrophosphate in that the inhibition is irreversible and long acting. The true cholinesterase activities failed to return to normal until 30 to 40 days after the cessation of the AB-132 therapy. *Conclusion:* From these studies, it is concluded that succinylcholine should not be given during anesthesia to patients receiving this cancer chemotherapeutic agent, AB-132. Caution should be exercised in administering muscle relaxants during anesthesia to any patient receiving new drugs which may potentiate and prolong the period of apnea. The determination of blood cholinesterase levels in these patients before surgery should be encouraged.

Effects of General Anesthetics on Tissue Oxygen "Tensions." HOWARD L. ZAUDER, M.D., PH.D., LOUIS S. MASSA, M.D., and LOUIS R. ORKIN, M.D., *Department of Anesthesiology, Albert Einstein College of Medicine, New York, New York.* The influence of some anesthetics on oxygen "tensions" of superficial tissues in man has been determined by Greene *et al.* (*Anesthesiology* 20: 830, 1959). No information is available, however, on the effects of these drugs on the deeper, less accessible tissues. It was thought of interest, therefore, to observe the effects of com-

monly employed anesthetics on oxygen "tensions" in representative tissues. *Methods:* Utilizing a polarographic technique, oxygen tensions in cerebral cortex, right lobe of the liver, right renal cortex and skeletal muscle were measured in dogs. Platinum electrodes, 0.01 inch in diameter, insulated within 2 mm. of the tip were used. Body temperature and end expiratory P_{CO_2} were maintained constant. The electrocardiogram, electroencephalogram, arterial P_{O_2} , P_{CO_2} and pH were monitored during the course of the experiment. Upon completion of the preparation, the animals, previously anesthetized with sodium pentobarbital (25 mg./kg.), were permitted to emerge into a very light plane of anesthesia before being anesthetized with N_2O , halothane, diethyl ether, cyclopropane or methoxyflurane. Inspired concentration of anesthesia was determined by gas chromatography. Depth was determined electroencephalographically. *Results:* Nitrous oxide in clinically employed concentrations was without significant effect on available tissue oxygen "tensions." Halo-

thane produced an increase in skeletal muscle and renal oxygen tensions independent of changes in blood pressure until marked hypotension is induced. At that time, tension in these tissues decreased. Available oxygen in the liver is decreased over the entire range of concentrations (0.5-3.0 per cent) of halothane employed. Diethyl ether induced a marked decrease in muscle oxygen "tension" which was independent of blood pressure. The effects of this anesthetic on "tensions" in the liver and kidney appeared to be dependent upon pressure. Cyclopropane, on the other hand, caused an increase in muscle oxygen "tensions." There was a moderate decrease in liver and a marked decrease in renal "tensions." These changes were relatively independent of changes in blood pressure. Methoxyflurane caused a decrease in renal and muscle oxygen "tensions" and a moderate increase in hepatic oxygen "tensions." Changes in cerebral oxygen tension throughout these experiments were negligible if end-expiratory P_{CO_2} was maintained constant.

AEROSOL ANESTHESIA The present method of obtaining oropharyngeal and tracheobronchial anesthesia, although satisfactory, has many disadvantages. This consists of three-stage technique which requires: first, spraying the oropharynx with the anesthetic material; second, swabbing of the anesthetic in the pyriform fossae; and, finally, directly instilling the anesthetic agent into the tracheobronchial tree via curved cannula or through a needle-puncture of the cricothyroid membrane. An alternate method is proposed which utilizes a simple aerosol anesthesia-dispensing device with a metering valve. Ten per cent lidocaine in Freon propellant is used. Short-term topical anesthesia of the pharynx, larynx, and tracheobronchial tree was obtained so that examination of the oropharynx, intubation, laryngoscopy, bronchography, and bronchoscopy could be satisfactorily performed. Administration was rapid, simple and highly effective. By employing a mean particle size of 5 micron, using a hygroscopic agent, and adjusting the characteristics of the nebulizer, satisfactory short-term oropharyngeal and tracheobronchial anesthesia was obtained. (Tomashefski, J. F., Nelson, S. W., and Christoforidas, A. J.: *Oropharyngeal and Tracheobronchial Aerosol Anesthesia*, *Dis. Chest* 42: 181 (Aug.) 1962.)