patients (halothane 8, cyclopropane 7) were studied in the supine position before and during elective herniorrhaphy. *Method:* Forearm blood flow was determined by the electrocapacitance plethysmograph (Hyman, C., Burnap, D., and Figar, S.: J. Appl. Physiol. *18:* 997, 1963), and heart rate was recorded on an electrocardiograph. Depth of anesthesia was estimated with an electroencephalograph. The above three measurements were recorded on a Grass polygraph. Mean arterial blood pressure was calculated (map = (systolic + 2X diastolic)/3 from blood pressure observed by the method of Riva-Rocci. $P_{CO_2}$ and *p*H were analyzed by the Astrup technique and were maintained at normal levels by either assisting or controlling ventilation. Halothane or cyclopropane was administered until there was a burst suppression pattern with silent intervals of 3–10 seconds on the encephalogram. *Results:* In both groups, forearm blood decreased 0.6 ml. of blood/100 cc. of tissue/minute following five minutes of oxygenation, with little or no change in mean arterial blood pressure or heart rate. In the halothane study, the forearm blood flow increased during induction (2.5 to 3.2), fell (3.2 to 2.5 ml. of blood/100 ml. of tissue/minute during deep anesthesia, and then linearly increased to 5.2 and remained at this level until and during emergence. Mean arterial pressure was unchanged during induction (84 mm. of mercury), decreased to 64 mm. of mercury during deep anesthesia and returned to 72 mm. of mercury within ten minutes after discontinuing the halothane. Heart rate increased during induction from 76 to 85 beats per minute, decreased during deep anesthesia (85 to 68) and then rose linearly to 96 where it remained through emergence. With cyclopropane, forearm blood flow rose during induction (3.9 to 8.8 ml. of blood/100 ml. of tissue/minute) and remained elevated during deep anesthesia (6.7 to 8.6). At five minutes post-cyclopropane, there was a decrease (7.1 to 6.2) and at ten minutes, an increase (6.2 to 7.1 ml. of blood/100 ml. tissue/minute). Mean arterial pressure increased during induction from 96 to 112 mm. of mercury, remained elevated in deep anesthesia and also after emergence. The heart rate increased during induction from 75 to 94, decreased during deep anesthesia (94 to 58) and increased on emergence (58 to 92). Cyclopropane anesthesia in four instances was associated with transient periods of bigeminy during induction and emergence. These arrhythmias did not alter the forearm blood flow. Ventricular tachycardia occurred in one patient who was too deeply anesthetized. The forearm blood flow dropped to baseline until the ventricular tachycardia spontaneously reverted—whereupon forearm blood flow rose to previous levels. Mean arterial blood pressure was decreased slightly (10 mm. of mercury) during the ventricular tachycardia. *p*H and $P_{CO_2}$ at this time were 7.38 and 26.5. *Summary:* In this study with these methods, cyclopropane elevated forearm blood flow, and blood pressure, and caused an increase in heart rate followed by a decrease; whereas with halothane all three parameters were decreased initially. However, during deep halothane anesthesia, the forearm blood flow and heart rate increased while blood pressure remained depressed. Ventricular tachycardia during cyclopropane anesthesia was associated with a decrease in forearm blood flow.

**Effect of Lidocaine on Digitalis Intoxication.** JOSE E. USUBIAGA, M.D., JAIME A. WIKINSKI, M.D., BETTY VESTAL, M.S., and FRANK MOYA, M.D., *Department of Anesthesiology, University of Miami School of Medicine and Jackson Memorial Hospital, Miami, Florida.* Cardiac glycosides are still the drugs of choice for the enhancement of the contractile force of the failing heart. Since a large number of digitalized patients undergo operation and anesthesia, considerable experimental work has been devoted to the study of alterations in the cardiovascular effects of digitalis induced by general anesthetic agents (Morrow, D. H., and Townley, N. T.: Anesth. Analg. *43:* 608, 1964), muscle relaxant drugs (Dowdy, E., Duggar, P. N., and Fabian, L. W.: Anesth. Analg. *44:* 608, 1965; Akdikmen, S. A., Boba, A., and Landmesser, C. H. M.: New York J. Med. *65:* 2902, 1965), hypothermia (Hernandez, A., Goldring, D., and Sleater, W.: J. Lab. Clin. Med. *62:* 884, 1963), and sympathetic blockade (Wilson, D., Paradise, R. P., and Stoelting, V. K.: Anesth. Analg. *43:* 729, 1964). Surgery in patients

receiving digitalis is also performed with the aid of regional anesthesia and since local anesthetic agents are known to affect the circulation, it is important to investigate the interaction between these drugs. We have studied the effect of the commonly used local anesthetic lidocaine on the development of digitalis toxicity in the dog. *Method:* The toxic dose of the short-acting glycoside, ouabain, was determined in a group of 15 normal dogs and, following complete recovery, compared to the dose required to produce toxicity in the same animals pretreated with lidocaine. The dogs, therefore, served as their own controls. In the control experiments, under pentobarbital anesthesia, the fifteen dogs received a steady intravenous infusion of ouabain at a rate of 6 μg./kg. per minute up to the ECG appearance of a ventricular ectopic pattern. The dogs were allowed to recover and, a week later, those that survived were pretreated with 10 or 20 mg./kg. lidocaine intramuscularly followed immediately by the intravenous ouabain up to the appearance of ventricular ectopic beats. Arterial blood pressure, central venous pressure and two leads of the ECG were recorded. The temperature was maintained with radiant heat. Serial samples of arterial blood were withdrawn at frequent intervals for the measurement of $P_{O_2}$, *p*H, $P_{CO_2}$ and plasma potassium. Heart rate was measured on the ECG and respiratory rate on the CVP recording. The total dose of ouabain necessary to develop ventricular arrhythmias was calculated in both groups and compared statistically with the aid of Student's *t* test. *Results:* In the control experiments, the infusion of ouabain produced a significant increase in arterial blood and pulse pressure, tachycardia followed by bradycardia, lengthening of the PR interval, ventricular extrasystoles followed by ventricular tachycardia and, in two out of fifteen dogs, ventricular fibrillation and death. Postmortem examination showed a firm, tonic myocardium. Plasma levels of potassium and arterial *p*H increased steadily during ouabain administration. Pretreatment with lidocaine, on the other hand, attenuated the hypertension and significantly increased the dose of ouabain necessary to develop arrhythmias from 72 μg./kg. to 138 μg./kg. ($P = < 0.01$). Auriculo-ventricular block was the most common ECG finding and, in three dogs, death was produced by ventricular standstill. The rise in plasma potassium induced by the ouabain was not significantly modified by the lidocaine administration. *Conclusion and Summary:* It appears that lidocaine prevents the appearance of characteristic ventricular arrhythmias associated with digitalis toxicity such as ventricular extrasystole and ventricular tachycardia. However, death still occurs and is associated with ventricular standstill rather than ventricular fibrillation. It was concluded that lidocaine masked the cardiac arrhythmias induced by digitalis without truly protecting the heart muscle itself against the toxic effect of digitalis.

**Breath-by-Breath Analysis of Respiratory Work During Anesthesia.** DANIEL C. WEAVER, M.D., *Division of Anesthesiology, Yale University, School of Medicine, Department of Anesthesia Yale-New Haven Hospital, New Haven, Connecticut.* Respiratory patterns clinically vary from light to deep anesthesia without obvious respiratory obstruction or intercostal paralysis. It is therefore of interest to consider changes in lung mechanism with spontaneous respiration and with assisted and controlled ventilation. Mechanical work of the lung has been electronically calculated during clinical conditions of light and deep planes of anesthesia in healthy patients prior to elective surgery. Differential esophageal-airway pressure and gas flow are measured, multiplied by an analog computer and the product integrated. Pulmonary work (kg.-mg.) (or work done on lung) = transpulmonary pressure × volume (Comroe, and others: The Lung, ed. 2. Yearbook Medical Publishers, 1963, p. 166). Work = S pressure × Flow. This work in progress presents a model of on-line computation of pulmonary work. There are, in such a model, inherent and potential errors beyond the scope of this report, of which the investigator must be aware. *Methods:* An esophageal balloon is inserted into patients premedicated with pentobarbital and scopolamine. Differential pressure is measured by a Statham differential pressure transducer from catheters passing through an anesthesia mask. EEG electrodes are applied. No endotracheal tube was inserted, since light