anesthesia for major surgery. Our anesthetic technique has been presented previously (*J. A. M. A. 161: 1534, 1956*). The following is a representative polarographic study of a patient with pulmonary tuberculosis undergoing right upper lobectomy under N_2O-O_2 anesthesia: Control: 73 mm. of mercury; after rapid intravenous premedication: 60 mm. of mercury; after induction with 75 per cent N_2O–25 per cent O_2: 99 mm. of mercury; ventilated with 33 per cent O_2 prior to intubation: 145 mm. of mercury; during hilar surgery, on 50 per cent O_2–50 per cent N_2O: 258 mm. of mercury; operation completed–100 per cent O_2 for 4 mins.: 430 mm. of mercury. General observations made include: (1) control values in bed patients were found to be considerably lower than normal values reported for healthy subjects by other investigators; (2) the pO_2 falls rapidly after intravenous premedication; (3) denitrogenation and induction of anesthesia with N_2O-O_2 (20–25 per cent O_2) results in pO_2 values considerably higher than control values; (4) ventilation with 30–35 per cent O_2 (70–65 per cent N_2O) is adequate to maintain high pO_2 levels during the period of laryngoscopy and tracheal intubation where apnea may persist for several seconds; (5) during N_2O-O_2 anesthesia we often administer O_2 in concentration of 50 per cent or even higher once the patient is completely denitrogenated. Although high pO_2 tensions are the rule with 50 per cent O_2, in some surgical situations this concentration of O_2 is necessary to provide an adequate tension (intrathoracic surgery with compression of the lung by retraction and other similar surgical problems); (6) ventilation with 100 per cent O_2 for approximately three to five minutes will increase the pO_2 to the 400–500 mm. of mercury range; (7) once the 100 per cent O_2 is discontinued it is not unusual for the pO_2 to fall to low levels after five minutes breathing of room air. This corroborates our clinical impression and rule that all patients are observed very closely for several minutes in the Operating Room at the conclusion of surgery. (*This work was supported in part by the New Hampshire Heart Association.*)

Bronchoscopy and Laryngoscopy with the Wrap-around Chest Respirator. SIDNEY W. HELPERIN, M.D., AND WALTER H. WASKOW, M.D. *Department of Anesthesiology, Mt. Sinai Hospital, Cleveland, Ohio.* This study shows that the Emerson wrap-around chest respirator provides adequate respiration for bronchoscopy or laryngoscopy in patients who are anesthetized and paralyzed. Previous cuirass respirators did not always accomplish adequate respiration, because the shell rested on the body, thus limiting expansion. So far we have used the wrap-around chest respirator in 39 cases; 20 bronchoscopies and 19 laryngoscopies, on patients ranging in age from 7 years to 76. The duration of artificial respiration varied from 1½ minutes to 40 minutes. Clinically all cases did well. In 31 patients we measured the tidal volume produced by the respirator. To do this we used a Bennett ventilation meter. In each of these cases we also counted the number of breaths per minute. Using this number, we consulted a Radford nomogram to determine what the tidal volume should have been for that patient at that rate. In 24 of the 31 cases measured, the respirator produced a tidal volume equal to, or better than, the calculated amount. In 4 cases the actual tidal volume, although less than the calculated amount, was more than 80 per cent of that amount. Eighty per cent of the Radford nomogram can probably be safely classified as indicating adequate respiration. In only 3 cases was the actual tidal volume below 80 per cent of the calculated volume. The first two such cases can probably be ascribed to the fact that we were still learning how to use the respirator. There are many different factors to be taken into consideration in order to get the maximum or optimum effect from the respirator. The third case with a tidal volume less than 80 per cent of the calculated volume was a lady who had a carcinoma of the lung with diffuse pulmonary involvement. Her lungs just couldn't expand. Clinically these cases with the relatively low tidal volumes seemed to do just as well as the other patients. They had good color throughout. The oximeter was used in 12 of the patients. These patients were all breathing room air while the respirator was working. In one patient undergoing bronchoscopy, the oximeter reading went down to 81 per cent at a time when there was bleeding

in the bronchus. At this time the patient appeared slightly cyanotic. After the bleeding stopped and clots were sucked out, his color and oximeter reading improved. Another patient had a similar experience, but the oximeter was not attached. Two other patients had temporary bouts of cyanosis. One was a patient for laryngoscopy, who was obstructed while the laryngoscopist was trying to locate her glottis. The other became cyanotic when a biopsy was taken of a tumor of the pharynx and bleeding was produced. If obstruction is avoided or corrected, the Emerson wrap-around chest respirator, properly used, provides adequate respiration.

Effect of Ether Anesthesia on Human Skeletal Muscle Metabolism. DOROTHY H. HENNEMAN, M.D., AND LEROY D. VANDAM, M.D. *Division of Anesthesia, Peter Bent Brigham Hospital, Boston, Mass.* Phosphocreatine, total creatine, and lactic acid were measured in triplicate on paired biopsies of nontraumatized *rectus-abdominis* muscle taken at the time of initial abdominal incision and of final abdominal closure, 25 to 150 minutes after induction of ether anesthesia in fifteen women. Surgery included ovarian biopsy. Simultaneous measurements of blood *p*H and serum inorganic phosphorus were made. Standard error of technique for phosphocreatine was 0.43 micromoles per gram of wet tissue. In the first sample obtained prior to surgery or trauma by abdominal retraction, mean concentrations of phosphocreatine varied inversely with the time between induction and biopsy: 20–30 minutes after induction total creatine was 37.2 and phosphocreatine 21.3 micromoles per gram of wet tissue; after 30–60 minutes total creatine was 24.4 and phosphocreatine 12.9 micromoles per gram. Between the first and second biopsy in the same patient, during the course of surgery, and with minimal retraction of the muscle biopsied, concentrations remained low or fell further: after 60 minutes of surgery (120 minutes of anesthesia) total creatine was 25.5 and phosphocreatine 11.5 micromoles per gram of wet tissue. Since a decrease in phosphocreatine occurred prior to surgery or abdominal retraction, trauma was not responsible. Concentrations of free creatine did not increase as phosphocreatine fell, hence our technique of tissue handling was not responsible. Skeletal muscle lactic acid did not change significantly as phosphocreatine decreased. No change in blood *p*H occurred, but serum total inorganic phosphorus increased as expected. When hypotension occurred prior to the time of initial biopsy concentrations of phosphocreatine were abnormally low (7.0, 8.0, 9.0, 10.1, 10.4 micromoles phosphocreatine per gram) even though duration of anesthesia was relatively short. In addition, if hypotension occurred, with or without the subsequent administration of ephedrine, between the first and second biopsy the fall in phosphocreatine was more pronounced (23.0 to 16.0, 24.5 to 10.6, and 13.6 to 4.3 micromoles phosphocreatine per gram). Muscular contraction produces a fall in skeletal muscle phosphocreatine; anesthesia, however, produces relaxation. Normally, contraction increases muscle lactic acid; none was observed by us in muscle although blood lactic acid regularly increases during ether anesthesia. Earlier studies from this laboratory demonstrated that ether produces abnormal elevations in serum inorganic phosphorus and blood glucose following the administration of glucose or epinephrine. In addition, ether produces resistance to the glucose and phosphorus lowering effects of insulin. Abnormalities in lactic, pyruvic, and citric acids were not present under the same conditions. In view of this, it was suggested that ether alters the entrance of glucose into the cell due perhaps to changes in cellular permeability, glucose phosphorylation, or insulin activity. The present data are in keeping with this suggestion and indicate further that ether in some manner decreases the availability of high-energy phosphate compounds. Does such a fall in phosphocreatine occur also in cardiac muscle? Is this in part responsible for myocardial depression during general anesthesia?

The Effect of Cyclopropane and Cyclopropane Plus Hypercarbia on Blood Clotting. WILLIAM S. HOWLAND, M.D., M. B. ZUCKER, M.D., E. E. CLIFFTON, M.D., AND C. P. BOYAN, M.D. *Department of Anesthesiology and Enzyme Research Section, Sloan-Kettering Institute, Memorial Center for Cancer and Allied*