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*