evidenced by decreased mean arterial pressure (from 120 to 64 torr) left ventricular dp/dt (from 16 to 7), cardiac output (from 2.63 to 1.71 l/min) and left ventricular stroke work (from 37.33 to 10.02 gm meters) with increased right atrial (from 2.6 to 6.7 torr) and left ventricular end-diastolic (from 4 to 11.4 torr) pressures with no change in heart rate or peripheral vascular resistance. While MBF (from 42 to 23 ml/100 gm/min) and myocardial O_2 consumption (from 5.5 to 3.1 ml/100 gm/min) decreased, myocardial excess lactate also decreased. NEFA and pyruvate uptake decreased, lactate uptake did not change, and an elevated threshold for glucose uptake was seen. *Summary:* The results indicate that there was no myocardial hypoxia in the halothane-depressed heart and that the depression may be related to an interference in glucose transport.

The Effects of Inhalation Anesthetics on Pulmonary Surfactant. E. K. MOTOYAMA, M.D., L. GLUCK, M.D., Y. KIKKAWA, M.D., M. V. KULOVICH, M.D., and Y. O. SUZUKI, M.D., *Section of Anesthesiology and Department of Pediatrics, Yale School of Medicine, New Haven, Conn., and Department of Pathology, Albert Einstein College of Medicine, New York, N. Y.* The effects of halothane and cyclopropane anesthesia on pulmonary surfactant were investigated in 25 rabbits. *Methods:* Animals were anesthetized and artificially ventilated for four to seven hours and their lungs were compared with those of control animals sedated with pentobarbital and ventilated for equivalent lengths of time. *Results:* Successive measurements for eight to 24 hours of the minimum surface tension values of lung–saline extracts showed that animals anesthetized with halothane had significantly higher surface tension values than control animals. These results indicate that halothane in some way decreases the surface activity of the lung. This effect was not observed with cyclopropane anesthesia. Biochemical analysis showed that halothane anesthesia was associated with a significant decrease in the alveolar lining of the surface-active lecithin fraction containing myristic acid on the beta carbon. This finding indicates: 1) depression of the lecithin biosynthesis by the transmethylation of phosphatidyl ethanolamine, a major lecithin synthetic pathway which takes place within the alveolar lining (Pediat. Res. 1: 247, 1967); or 2) reduced transport of these lecithin molecules from the intracellular store onto the alveolar surface. This reduction of the lecithin containing myristic acid was not significant with cyclopropane anesthesia. Lung tissues were studied with electron microscopy, but no apparent abnormalities of the Type II alveolar cells with osmiophilic inclusions or of the alveolar-lining layer were found. (Supported by grants: USPHS HD-00989, HD-01299, HD-02459, and Josiah Macy, Jr., Foundation.)

Acid–Base Changes during Lidocaine-induced Seizures in *M. Mulatta*. E. S. MUNSON, M.D., and I. H. WAGMAN, PH.D., *Departments of Anesthesiology and Physiology, School of Medicine and the National Center for Primate Biology, University of California, Davis, Calif.* Lidocaine-induced seizure threshold, acid–base, equilibrium and behavioral and electrical changes were studied during intravenous infusion of lidocaine into unanesthetized rhesus monkeys at a constant rate (4 mg/kg/min). *Method:* Sixty-six experiments were performed on 19 male *M. mulatta* (4–7 kg). Nine animals were prepared with chronically-implanted electrodes in various depth locations. Arterial plasma lidocaine concentration was measured using the methyl orange method. *Results:* Mean (± SD) lidocaine seizure dosage was 13.8 ± 3.0 mg/kg. Arterial plasma lidocaine concentration at the onset of seizure activity was 24.5 ± 4.5 μg/ml. Animals that ventilated spontaneously developed a significant ($P < 0.01$) metabolic acidosis (15.9 ± 6.8 Meq/l base deficit). Duration of seizures in these animals was longer than that in artificially-ventilated (paralyzed) animals. No correlation between plasma lidocaine levels at the onset of seizures and base deficit of arterial blood was observed. Mild hypercarbia (Pa_{CO_2} 68 mm/Hg) also did not influence lidocaine seizure threshold. Alterations in behavior were similar to those observed in other animals. In addition to tonic–clonic seizure activity nystagmus and drowsiness were always present. The characteristic electrical responses previously