

problem, even in patients who had recent cardiac surgery, and reversion was not refused to any suitable candidate on the basis of anesthetic risk. The safety of the procedure under the proper circumstances justifies an attempt at the correction of ectopic cardiac arrhythmias in all patients who will benefit from normal sinus rhythm. (Miller, H. S.: *Synchronized Precordial Electroshock for Control of Cardiac Arrhythmias*, J.A.M.A. 189: 549 (Aug. 17) 1964.)

**CARDIOVERSION** Studies have convincingly demonstrated the safety of direct current electroshock; nevertheless, sequential changes in the electrocardiogram invite a modicum of caution in the application of this promising means of terminating cardiac arrhythmias. One patient with chronic atrial fibrillation has been studied in whom, although direct current electroshock successfully restored regular sinus-dominated rhythm, the conversion coincided with the appearance of a striking elevation of the ST segment of the electrocardiogram. Similar ST segment changes endure for many hours or days following acute myocardial infarction, and a variant form of angina pectoris may be associated with strikingly similar ST elevation which disappears with relief of pain. Whether, in the case of cardioversion, such deviations reflect alterations in the distribution of myocardial cellular electrolyte, protein, enzymes, or actual necrosis is unknown. The question as to whether repeated shocks of this nature could be additive and result in permanent injury must await further experience. (Sussman, R. M., and others: *Myocardial Changes After Direct Current Electroshock*, J.A.M.A. 189: 739 (Sept. 7) 1964.)

**CEREBRAL BLOOD FLOW** Cerebral blood flow increased linearly with arterial carbon dioxide tension over a range of tensions from 20 to 60 mm. of mercury during halothane anesthesia in human subjects. Mean cerebral blood flow and cerebral vascular resistance at arterial carbon dioxide tension of

40 mm. of mercury were 49.7 ml./100 g./minute and 1.0 mm. of mercury/ml./100 g./minute, respectively, in anesthetized subjects. These values are different from corresponding values in conscious subjects which are 44.4 ml./100 mg./minute and 1.9 mm. of mercury/ml./100 mg./minute. The shape of the cerebral blood flow-arterial carbon dioxide tension response curve was altered because of the cerebral vasodilatation caused by halothane. (Alexander, S. C., and others: *Cerebrovascular Response to PaCO<sub>2</sub> During Halothane Anesthesia in Man*, J. Appl. Physiol. 19: 561 (July) 1964.)

**PULMONARY BLOOD FLOW** When excised dog lungs which had been almost maximally inflated with air were perfused, pulmonary artery pressure was less than alveolar pressure with flows up to 50 ml./minute. Pulmonary artery pressure exceeded alveolar pressure at all flows when lungs were inflated to comparable volumes with water. The presence of an air-liquid interface facilitates perfusion in lungs due to lowering of pericapillary pressure caused by effects of surface tension of the fluid film which lines alveoli. (Bruderman, I., and others: *Effect of Surface Tension on Circulation in the Excised Lungs of Dogs*, J. Appl. Physiol. 19: 707 (July) 1964.)

**THORACIC MECHANICS** A plethysmograph was used to measure relative contribution of chest wall expansion and diaphragmatic descent to inspiration. In 15 normal subjects, one-third of the tidal volume was effected by diaphragmatic descent and two-thirds by rib cage expansion. This partitioning of tidal volume was not altered by changes in breathing pattern or inhalation of various special gas mixtures. Elastic recoil alone was sufficient to move the rib cage at low tidal volumes, so that no work was required for chest expansion until tidal volumes exceeded one liter. (Bergofsky, E. H.: *Relative Contributions of the Rib Cage and the Diaphragm to Ventilation in Man*, J. Appl. Physiol. 19: 698 (July) 1964.)