beating heart for the same interval. The energy from the arrested heart comes chiefly from residual aerobic carbohydrate metabolism that is completed within the first 10 minutes of arrest. The fall in pH in the circulatory bed of the arrested heart is gradual. (Winterscheid, L. C., and others: Myocardial Carbohydrate Metabolism During Induced Cardiac Arrest and Post-Arrest Perfusion, Ann. Surg. 148: 481 (Sept.) 1958.)

ARRHYTHMIAS Mephentermine (Wyamine) has an anti-arrhythmic effect upon the diseased human heart, especially on ventricular ectopic systoles. It causes a decrease in the refractory period, an increased velocity of conduction and has a positive inotropic effect. Doses up to 3 mg./kg., given intravenously, have no significant ECG effects in patients with chronic atrial fibrillation. Mephentermine is contraindicated in patients with supraventricular tachycardia with second degree A-V (Wilson, M., Perez-Arzola, M., and block. Oppenheimer, M. J.: Mephentermine and the Arrhythmias, Am. J. Med. Sc. 236: 300 (Sept.) 1958.)

CARDIAC ARRHYTHMIA Cyclopropane in oxygen was administered to 15 normal adults using a semiclosed system containing a soda lime cannister. pCO_2 in the respired gas was elevated by partially bypassing the can-Cyclopropane increased the concentration of circulating norepinephrine, but not Hypercarbia elevated that of epinephrine. High spinal anesthesia prevented the arrhythmias during hypercarbia and virtually abolished the increase in plasma catechol Intravenous infusions of epiconcentration. nephrine and norepinephrine duplicating the plasma catechol concentrations found during hypercarbia did not produce arrhythmias. Blockade of the stellate ganglia bilaterally prevented the occurrence of arrhythmias during hypercarbia, but did not prevent increases in plasma catechol concentration. Arrhythmias produced by hypercarbia during cyclopropane anesthesia in man are therefore more likely caused by an increase in the activity of the cardiac sympathetic nerves than by an increase in circulating catechols. (Price, H. L.: Role of Catecholamines in the Initiation of Ar-

rhythmic Cardiac Contraction by Carbon Dioxide Inhalation in Anesthetized man, J. Pharmacol. & Exper. Therap. 122: 63A (Jan.) 1958.)

HYPOCALCEMIA Digitalis is known to cause egress of potassium from the myocardial cell and this relative intracellular potassium deficit may be in part responsible for the electrocardiographic effects of digitalis. Calcium ions exert a stabilizing effect on the myocardial cell membrane to inhibit re-entry of potassium into the cell. Reduction of calcium ion concentration at the cell membrane might facilitate re-entry of potassium into the myocardial cell. Induced hypocalcemia was carried out by administering ethylenediaminetetraacetate (Na₂EDTA) to patients receiving digitalis who showed ECG evidence of increased myocardial irritability. Na₂EDTA abolished ectopic arrhythmia in 11 of 26 patients studied. This is thought to be due to binding of serum calcium by Na₂EDTA facilitating potassium reentry into the myocardial cell through a membrane of decreased stability. Induction of transient hypocalcemia in patients receiving digitalis who show evidence of hyperirritability would be of value in evaluating the role of potassium depletion in production of ectopic rhythms; in safely abolishing ectopic arrhythmias during repair of systemic hypokalemia; and in evaluation of ECG contour changes observed in patients receiving digi-(Kabakow, B., and Brothers, M.: Effects of Induced Hypocalcemia on Myocardial Irritability and Conductivity, A. M. A. Arch. Int. Med. 101: 1029 (June) 1958.)

PERFORATION OF THE HEART During cardiac catheterization a National Institutes of Health catheter was imbedded into the wall of the right ventricle with subsequent penetration of the ventricular wall during the high pressure injection. The resulting cardiac tamponade necessitated surgical relief. When the intra-pericardial pressure was relieved, the patient's condition immediately improved and she made an uneventful recovery. (Escher, D. J., and others: Perforation of the Heart During Cardiac Catheterization and Selective Angiocardiography, Circulation 8: 418 (Sept.) 1958.)