plasma transfusion, a raised pulse rate may still indicate a lowered red cell volume. (*Clarke, R.: On the Nature and Treatment of Wound Shock, Ann. Roy. Coll. Surgeons England 24: 239 (April) 1959.*)

TRANSFUSION REACTIONS A transfusion reaction exhibits three clinical phases: (1) acute reaction, (2) oliguric phase, (3) diuretic phase. Anesthesia masks most of the classical symptoms of acute transfusion reactions; during an operation, the most prominent sign may be bleeding from cut surfaces. Emergency measures of the acute reaction phase are: (1) Immediate discontinuance of the transfusion. The needle should be left in place for treatment of shock should it develop: intravenous fluids, dextran, or compatible blood. Arterenol may be required in addition. (2) Establishment of the diagnosis. The physician himself should draw blood from the patient, examine it for hemolysis, and have it cross-matched with blood from the incriminated bottle. He should refuse to accept any previously used samples. (3) Immediate intravenous administration of five per cent solution of dextrose in water (two liters) with 300 mEq. of sodium bicarbonate (to prevent blocking of renal tubules with acid heme). (4) Administration of Mannitol, 50 or 100 ml. of a 25 per cent solution, in the hope of increasing urinary flow by osmotic diuresis. Medical management of the oliguric phase includes restriction of fluid intake and prohibition of sodium intake. Protein catabolism is discouraged by a high-calorie, low-protein regimen. Prophylactic antibiotic treatment is indicated. Indications for the artificial kidney are chemical and clinical. Dialysis is an important adjunct to therapy and should be employed relatively early in the oliguric phase before the patient's clinical condition deteriorates. (*Barlas, G. M., and Kloff, W. J.: Transfusion Reactions and Their Treatment, Especially with the Artificial Kidney, J. A. M. A. 169: 1969 (April 25) 1959.*)

AMMONIA INTOXICATION Ammonia in stored blood increases at an average rate of 20 micrograms per 100 ml. per day. The most practical method at present for reducing or controlling ammonia intoxication that may result from massive transfusion in a cirrhotic patient is to administer intravenously arginine simultaneously with the blood as described. (*Britton, R. C.: Ammonia Intoxication from Bank Blood in Patients with Cirrhosis of the Liver, Cleveland Clinic Quarterly 26: 81 (April) 1959.*)

CARDIAC ARRHYTHMIAS Direct coronary artery pressure and flow, coronary sinus flow, and systemic blood pressure were measured in 264 dogs. After control studies, cardiac arrhythmias were induced. Auricular and ventricular premature contractions, paroxysmal auricular tachycardia, auricular fibrillation and flutter, ventricular tachycardia and fibrillation all were found to decrease coronary artery pressure and flow and systemic arterial pressure significantly, particularly when irregular and rapid rates (190 and above) were present. Direct brachial artery pressure measured in humans, showed significant decrease in blood pressure when heart rate was 180 or above. Vasopressor drugs may abolish arrhythmias plus correcting hypotension. On the basis of animal experimental evidence, therapy of cardiac arrhythmias should aim toward rapid correction with vasopressors and later permanent correction should be secured with quinidine and digitalis drugs. Patients with coronary artery disease should have arrhythmias treated to maintain adequate coronary perfusion since both clinical and electrocardiographic evidence of myocardial ischemia may become evident with even very small reduction of coronary artery pressure and flow. (*Corday, E., and others: Effect of Cardiac Arrhythmias on the Coronary Circulation, Annals. Int. Med. 50: 535 (March) 1959.*)

VENTRICULAR FIBRILLATION Fibrillation of the ventricle in the human is produced by an electric charge made within the heart itself. A checkerboard distribution of coronary artery blood produces these electric charges. They do not appear when the heart is uniformly deprived of oxygenated blood, but when only part of the muscle is deprived of oxygenated blood. Conversion of oxygen differentials into electric charges requires further investigation. Hearts with adequate inflow but with checkerboard distribution require even distribution for

correction. This problem yields to treatment by the creation of intercoronary channels. The physiology and treatment of this aspect of coronary artery disease deserve careful scientific analysis because positive steps in protection and treatment are in the realm of possibility. (*Editorial: Ventricular Fibrillation in Hearts Too Good to Die, J. A. M. A. 170: 471 (May 23) 1959.*)

EKG AT HIGH ALTITUDE Of 120 electrocardiograms of healthy adults living at 14,900 feet above sea level, 23 showed definite signs of right ventricular hypertrophy, 39 showed highly suggestive signs of the above, 37 showed right bundle branch block and 21 were normal. Chronic hypoxia due to altitude produces an increase in the transverse diameter of the heart, an increase in the volume of the right auricle and ventricle, and a small but significant increase in pulmonary artery pressure and total pulmonary resistance. (*Rotta, A., and Lopez, A.: Electrocardiogram in Patterns in Man at High Altitudes, Circulation 19: 719 (May) 1959.*)

INTRACARDIAC EKG Intracardial electrocardiograms with simultaneous intracardiac pressures and standard Lead I were recorded in 13 normal and 123 patients with cardiovascular abnormalities during routine cardiac catheterizations. Normal configuration and potentials are described for the various chambers and accessible vessels. These configurations and potentials of the normal intracardiac electrocardiogram are sufficiently characteristic to permit accurate localization of the catheter tip. An injury pattern appears when the catheter tip impinges upon the ventricular endocardium. Intracardiac electrocardiography aids in the detection of phenomena within the heart not readily apparent on surface leads. Two infant cases with severe congenital heart disease are discussed. (*Bertrand, C. A., and others: Intracardiac Electrocardiography in Man, Am. J. Med. 26: 534 (April) 1959.*)

HYPERVAGISM Case reports of two infants manifesting signs of sino-atrial block believed to be due to hypervagism are presented. One had localized areas of myxedema and the attacks were probably due to compression of the carotid sinus by the masses in the neck. Its attacks consisted of periods of asystole followed by slow A-V nodal rhythm and associated with apnea, cyanosis and convulsions. Atropine protected this patient until, under thyroid therapy, the masses regressed. The second infant had an esophagogastrostomy for repair of an esophageal atresia. Nipple feedings were followed by periods of apnea associated with sino-atrial block and the appearance of slow A-V nodal rhythm. It is suggested that the vagus nerve was compressed by the dilated stomach. During one such incident, the patient died. Atropine was not used in this patient until the last three days of life and then only in small dosage (0.1–0.2 mg. every 12 hours). (*Bauer, C. H., Engle, M. A., and Mellins, R.: Hypervagism and Cardiac Arrest, Bull. New York Acad. Med. 35: 260 (April) 1959.*)

EXTRACORPOREAL CIRCULATION Light cyclopropane and ether anesthesia is employed. Sodium bicarbonate is added to the donor blood to prevent metabolic acidosis. Arterial pressure should not fall below 70 mm. of mercury. Hypothermia is avoided by placing the patient on a mattress perfused with warm water and by keeping the blood in the oxygenator at a temperature of 37 to 38 C. (*Beer, R., and Loeschcke, G.: Problems with Operation Using Extracorporeal Circulation, Der Anaesthesist: 8: 70 (March) 1959.*)

CARDIOPLEGIA A solution of potassium citrate 0.81 per cent, and magnesium sulfate 2.46 per cent with 1 mg. of neostigmine per 100 cc. was injected into the ascending aorta proximal to the occlusion site. At the end of the intracardiac procedure the occluded aorta was released and extracorporeal perfusion washed the solution from the heart. The solution must be injected with enough pressure to close the aortic valve in order to prevent poor perfusion and incomplete standstill. This technic was used in 34 patients, and in 80 per cent cardiac arrest occurred within 30 seconds. In the remaining 20 per cent there was a delay of 60–120 seconds. There were 12 deaths in the series, but in none of these was the induced standstill thought to be a major factor. (*Sealy, W. C., and others: Potassium, Magnesium, and*