

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** Intracardiac 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 esophagostomy 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 Loeschke, G.: Problems with Operation Using Extracorporeal Circulation, Der Anaesthetist: 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*