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*

Neostigmine for Controlled Cardioplegia, J. Thoracic Surg. 37: 655 (May) 1959.)

EXTRACORPOREAL PROBLEMS Many problems occur in extracorporeal circulation relating to venous cannulation and drainage, many of which have been satisfactorily resolved. Siphon caval drainage is preferred to fixed venous pumping. Hepatic venous drainage should not be interfered with. Venous catheters should be accurately and securely placed with attached metal tips. The vena cavae should not be intubated until just before the start of the perfusion to avoid interference with cardiac output. Lethal air embolism in the right atrium may occur. Its mechanism and prevention is outlined. Oxygen consumption in experimental studies is increased slightly by elevation of the venous pressure at flow rates of 50–100 cc./kgm. Modifications in venous cannulation necessitated by anomalies of the cavae system are described. *(Bosher, L. H., Jr.: Problems in Extracorporeal Circulation Relating to Venous Cannulation and Drainage, Ann. Surg., 149: 652 (May) 1959.)*

GAS ANALYSIS By combining a vacuum extraction method with gas chromotography it is possible to make rapid, accurate, and reproducible determinations of gases in biological fluids (*e.g.*, plasma). Determination of oxygen tension in one milliliter of human plasma is possible. *(Ramsey, L. H.: Analysis of Gas in Biological Fluids by Gas Chromotography, Science, 129: 900 (April 3) 1959.)*

CARBON DIOXIDE ANALYZER An apparatus consisting of a bridge-type continuous-flow carbon dioxide analyzer is described which repeatedly samples end expiratory air for use in patients receiving artificial respiration, either by intermittent positive pressure or in a tank respirator. The results of the analysis are rapidly obtainable. A comparison of results obtained with this and other methods is presented. *(Smith, A. C., Schuster, E., and Spalding, J. M. K.: An End-Tidal Air Sampler for Use During Artificial Respiration, Lancet 1: 277 (Feb. 7) 1959.)*

CARBON DIOXIDE STUDIES Under certain conditions, the difference between arterial and venous *p*H and pCO_2 is negligible in the arm. This occurs when patients are at rest in bed with the skin warm, and the temperature of the skin over the dorsum of the hand is at least 35 C. It also occurs in patients under general anesthesia, or upon heating the hand and arm for 15 minutes with electric pads. Observations made from venous blood under these conditions simplified the assessment of alveolar ventilation by blood studies. *(Brooks, D., and Wynn, V.: Use of Venous Blood for pH and Carbon-Dioxide Studies, Lancet 1: 227 (Jan. 31) 1959.)*

PULMONARY FUNCTION After irradiation to the chests of dogs, pulmonary diffusing capacity, lung compliance and functional residual volume decreased progressively, but pulmonary vascular resistance remained normal for a period of five months. A decrease in compliance before six months suggests that an increase in fibrous tissue was present even though it was not demonstrated by pulmonary vascular resistance studies. Little pathologic change was observed after a single dose of irradiation except for capillary dilatation. Four to five months after fractional irradiation, the histologic findings demonstrated focal atelectasis with some fibrosis and hyperemia of the interstitial areas. These changes were not evident on x-ray examination of the chest. In the animals studied after longer periods, there was obvious interstitial fibrosis with a paucity of cellular elements and capillaries. There was some evidence of narrowing due to endothelial proliferation, focal necrosis of the walls and a few areas of acute hemorrhage. *(Sweany, S. K., Moss, W. T., and Haddy, F. J.: The Effects of Chest Irradiation on Pulmonary Function, J. Clin. Invest. 38: 587 (March) 1959.)*

HYPOVENTILATION SYNDROME The fifth of a series of cases of "primary hypoventilation syndrome" is described along special studies which have served to clarify the etiology concerned. It is believed that in "primary hypoventilation syndrome," the essential defect is in the respiratory regulatory mechanism. There is no underlying disturbance in the lungs or musculoskeletal apparatus of the chest. In the special studies performed, there was a totally absent response to hypoxia and