

and alterations in body-fluid volumes. (Guyton, A. C.: *Regulation of Cardiac Output*, New Engl. J. Med. 277: 805 (Oct.) 1967.)

MYOCARDIAL CIRCULATION Radioactive krypton may be used to measure regional myocardial blood flow, expressed as a clearance constant. Uniform clearance constants have been demonstrated in the normal canine and human myocardium. A distinct difference in the clearance constants in the normal canine myocardium and in areas of naturally-occurring disease has been demonstrated. Heterogeneous clearance constants have been found in a majority of human subjects with coronary artery disease, the lowest rates being in areas of fibrous aneurysm. It is a reasonable assumption that myocardial blood flow is distributed unevenly in the presence of sufficient coronary artery disease. Implantation of an extracardiac artery into relatively ischemic regions of the myocardium increases the development of communications with the coronary arteries by influencing the size, number, or extent of such communications. Implantation into areas of scar tissue lessens the chance for success of the implant. Although these areas are sometimes grossly visible at surgery, this method of charting regional myocardial perfusion, localizing areas of ischemia, and avoiding areas of scar tissue has proved useful in determining the site for mammary artery implantation. (Sullivan, J. M., and others: *Regional Myocardial Blood Flow*, J. Clin. Invest. 46: 1402. (Sept.) 1967.)

MITRAL STENOSIS The effects of artificially-induced tachycardia upon hemodynamic variables were studied in 11 patients with pure mitral stenosis. Ventricular rates were altered from a mean of 95 to a mean of 146 beats/min. Cardiac output fell significantly with the rise in ventricular rate, whereas oxygen consumption was unchanged. As a result, arteriovenous oxygen difference widened. Mean pulmonary arterial pressure increased in all patients when ventricular rate increased. Pulmonary artery wedge pressure increased linearly as ventricular rate increased, with dyspnea occurring in four patients. These findings of falling cardiac output and rising

wedge pressure demonstrate the deleterious effects of uncontrolled ventricular rates in patients with mitral stenosis. (Arani, D. T., and others: *The Deleterious Role of Tachycardia in Mitral Stenosis*, Circulation 36: 511 (Oct.) 1967.)

CARDIOPULMONARY BYPASS Anatomic changes in organs of dogs subjected to two hours of cardiopulmonary bypass were studied. Two types of pump oxygenators were compared: (1) A Clark-Selos bubble oxygenator and (2) a Mayo-Gibben screen oxygenator. In both groups, and in nonperfused control dogs, early ischemic cardiac lesions were noted. The causes of these lesions included altered coronary perfusion, manipulation of the heart, and trauma from simple sternotomy. The kidneys of dogs in the bubble oxygenator group contained massive silicone embolization. The bubble oxygenator uses a silicone antifoaming agent that is not baked on. The kidneys from the screen oxygenator group (silicone is baked onto the screens) had no emboli but were swollen and edematous. The lungs in all animals were grossly atelectatic and the livers in all groups were passively congested. No other abnormalities were found consistently. (Buettner, L. E., and others: *Pathological Findings in Experimental Extra Corporeal Circulation Utilizing Two Pump-Oxygenator Systems*, Amer. J. Med. Sci. 254: 438 (Oct.) 1967.)

HEART FAILURE Excess interstitial fluid is returned to the vascular compartment via the lymphatic system. Elevated systemic venous pressure in right heart failure will prevent or retard return of lymph to the circulation. This will protect the circulation but also will lead to the clinical manifestations of heart failure. By anastomosing the thoracic duct to the low-pressure pulmonary veins, lymph flow was increased substantially in dogs after right heart failure had been produced artificially. In addition, there was a fall in systemic venous pressure, an increase in renal salt and water excretion, and a reduction in ascites. These findings suggest that venous hypertension, salt and water retention and ascites seen in right heart failure are related as much to inadequate

return of excess interstitial fluid as to the cardiac lesion itself, which initially increased the production of lymph. Increased return of lymph to the left heart in patients with isolated right heart lesions might relieve consequences of venous hypertension and perhaps increase left heart output. (Cole, W. R., and others: *Thoracic Duct-to-Pulmonary Vein Shunt in the Treatment of Experimental Right Heart Failure, Circulation* 36: 539 (Oct.) 1967.)

PULMONARY CIRCULATION At moderately high lung volumes, the distribution of pulmonary blood flow in both the human and isolated dog lung can be explained by the relations between pulmonary arterial, alveolar and venous pressures. Employing a radioactive xenon and lung scanning technique in normal man in the upright position, it was found that blood flow increased over almost the whole vertical distance down the lung at total lung capacity. However, there was often a small area of higher vascular resistance in the most dependent zone. There was invariably a larger region of reduced blood flow near the base at functional residual capacity. At F.R.C., blood flow per alveolus usually decreased over the lower third of the lung, reaching two-thirds of its maximum value at the base. At residual volume, blood flow per alveolus commonly decreased from top to bottom of the lung. Similar patterns were found in the isolated dog lung where the effects of changing lung volume and of vasoconstrictor and vasodilator drugs showed that the increased vascular resistance was caused by pulmonary vessels outside the influence of alveolar pressure. These results suggest that any general increase in interstitial pressure in the human lung will affect the distribution of blood flow and pulmonary vascular resistance. (Glazier, J. B., and others: *Role of Interstitial Pressure in the Distribution of Pulmonary Blood Flow, J. Physiol.* 190: 23 (May) 1967.)

PULMONARY EDEMA The average rate of water accumulation in healthy dog lungs varied in a nonlinear way with the level of capillary hydrostatic-plasma colloid osmotic pressure difference, and was unaffected by the level of capillary hydrostatic pressure. At low levels of left atrial pressure minus plasma

colloid osmotic pressure, water accumulated in the lung at an average rate of 0.09 ml./gm. of dry lung per hour per mm. Hg pressure difference. At higher levels the average rate of accumulation was 0.22 ml./gm. per hour per mm. Hg pressure difference. In most experiments water accumulated in the lungs slowly during the first 30 minutes of the test period, and more rapidly as the period was extended. Obstruction of right lymphatic duct outflow did not alter the rate of water accumulation. In the present experiments, pericapillary pressures were estimated to be of the order of -9 mm. Hg and the filtration coefficient for the pulmonary capillaries is estimated to be of the order of one-tenth to one-twentieth that for canine muscle capillaries. These data indicate that edema formation in lung tissue cannot be defined solely in terms of intravascular forces, but may be governed to a significant degree by changes in pericapillary forces in the pulmonary interstitium. (Levine, O. R., and others: *The Application of Starling's Law of Capillary Exchange to the Lungs, J. Clin. Invest.* 46: 934 (June) 1967.)

PULMONARY EDEMA Using a double isotope dilution technique, pulmonary extravascular fluid volume (PEV) was measured in normal subjects and in patients with valvular heart disease. PEV, in normals, mitral valve disease, and aortic valve disease was 107, 193, and 154 ml./m.², respectively. PEV correlates closely with mean pulmonary arterial pressure, mean left atrial pressure and functional classification, whereas there is little relation between PEV and cardiac output or pulmonary vascular resistance. (McCredic, Michael: *Measurement of Pulmonary Edema in Valvular Heart Disease, Circulation* 36: 381 (Sept.) 1967.)

AIR EMBOLISM Pulmonary air embolism occurred during insertion of a transvenous pacemaker in an 88-year-old woman. In reviewing the literature, it is felt that immediate therapy consists of placing the patient in the left lateral decubitus position, administering oxygen and vasopressors. More vigorous therapy includes closed-chest cardiac massage, aspiration of air from the heart and hyperbaric oxygenation. The pathophysiology of venous air embolism is such that a large bolus of air