alter general hemodynamics but it greatly increased coronary blood flow, cardiac oxygen consumption and carbon dioxide production. In the intact anesthetized dog it appeared that the heart rate had a close relationship to coronary flow volume when the heart is electrically accelerated. In the circumstances described, a profound fall in heart efficiency occurs. It seemed that, within limits, it is more efficient to deliver the same amount of cardiac work at a slow heart rate than at a fast one. In the latter circumstance, energy is inadequately converted to useful work and must appear in some other form, e.g., heat. (*Maxwell, G. M., and others: Induced Tachycardia: Its Effect Upon the Coronary Hemodynamics, Myocardial Metabolism and Cardiac Efficiency of the Intact Dog, J. Clin. Invest. 37: 1413 (Oct.) 1958.*)

CORONARY CONSTRICTION Intra-coronary administration of epinephrine or norepinephrine into the beating, fibrillating or potassium-arrested dog heart, indicate that the primary action of these drugs is vasoconstriction. The secondary action of vasodilation is due in large part to the resultant hypoxia of the heart muscle. (*Berne, R. M.: Effect of Epinephrine and Norepinephrine on Coronary Circulation; Circulation Res. 6: 644 (Sept.) 1958.*)

CORONARY HEART DISEASE Twenty-four patients with coronary heart disease and without valvular disease or hypertension were studied by cardiac catheterization. Patients with previous infarction, enlarged heart and dyspnea all displayed evidence of left ventricular failure, increased pulmonary capillary and artery pressure, depressed mixed venous blood saturation, and elevated right atrial pressure. By contrast, patients with previous infarction, normal heart size, and angina showed normal hemodynamics at rest and when free of pain. During exercise in this latter group varying degrees of left ventricular failure occurred in 9 of 11 patients, and in all 6 patients in whom anginal pain was precipitated. Following administration of nitroglycerine in 4 patients exercise was tolerated without evidence of left ventricular failure. (*Muller, O. and Rorvik, K.: Haemodynamic Consequences of Coronary Artery Disease, Brit. Heart J. 20: 302 (July) 1958.*)

CARDIAC FAILURE Patients in various degrees of left ventricular failure as a rule showed respiratory alkalosis and low oxygen tension. Patients with severe pulmonary edema had variable findings, including severe acute respiratory acidosis. Patients with heart disease and pulmonary emphysema tended to have respiratory acidosis. (*Carroll, D.: Oxygen Tension and Acid-Base Adjustment in Cardiac Failure, Bull. Johns Hopkins Hospital 103: 242 (Nov.) 1958.*)

VENTRICULAR FIBRILLATION During closure of an atrial septal defect under hypothermia, a ten year old white female developed ventricular fibrillation lasting 60 minutes. Despite eight electrical shocks and adrenalin injected into the pulmonary artery fibrillation continued. The rhythm spontaneously reverted to normal sinus rhythm and then to atrial fibrillation when the body temperature rose to 32.5 C. The aortic arch was occluded distal to the left subclavian artery for 78 minutes, and the patient developed muscle weakness of the legs presumably due to spinal cord anoxia. With careful massage and rewarming, ventricular fibrillation of long duration can be overcome. (*Martinez, J. B., and others: Factors Involved in the Recovery of a Patient After Prolonged Ventricular Fibrillation During Hypothermia, J. Thoracic Surg. 36: 749 (Nov.) 1958.*)

RIGHT HEART PRESSURE In cats under chloralose anesthesia there was frequently a fall in systemic blood pressure and heart rate when the inflow of blood to the right atrium was increased so that venous pressure rose. Increase in the output resistance of the right heart resulted in no changes in the circulation until the right heart output began to decline and venous pressure rose. These changes were probably dependent on the integrity of the vagus nerves. (*Barer, G. R., and Kottegoda, S. R.: Changes in Heart Rate and Blood Pressure of the Cat in Response to Increased Pressure on the Right Side of the Heart, J. Physiol. 143: 1 (Aug. 29) 1958.*)