effective antiarrhythmic dose suggests that reversal of the Ouabain-induced arrhythmia by propranolol is a result of a nonspecific effect of the drug and is not related to its ability to produce beta-receptor inhibition. (*Lucchesi, B. R., Whitsitt, L. S., and Brown, N. L.: Propranolol in Experimentally Induced Cardiac Arrhythmias, Canad. J. Physiol. 44: 543 (July) 1966.*)

ISOPROTERENOL During intravenous infusion of isoproterenol at rates of 1 μg./minute to 3 μg./minute, oscilloscopic monitoring for arrhythmias and frequent observation of blood pressure were maintained. Isoproterenol was effective in increasing cardiac output and in reducing left ventricular end-diastolic pressure in virtually all forms of heart disease, with the exception of some cases of severe coronary-artery disease or aortic stenosis. (*Elliott, W. C., and Gorlin, R.: Isoproterenol in Treatment of Heart Disease J.A.M.A. 197: 315 (Aug.) 1966.*)

CARDIAC OUTPUT With increasing age a continuous decrease of cardiac output can be demonstrated: 7.0 liters/minute for a group of men aged 16–28; 6.3 liters/minute in men aged 30–50 and 5.6 liters/minute for a group of men aged 51–61. The cause of the decrease of cardiac output seems to be the reduction of body cell mass with increasing age. In an unselected group of normal males, a definite dependence of cardiac output on body surface is not evident. It is consequently unfounded to term cardiac output as cardiac index in liters/minute/m.^{2} There seems to be, however, a fixed correlation between cardiac output and fat free body mass. (*Schroeder, R., and others: Cardiac Output—Its Dependence on Age and Various Body Measurements. Technique of Estimation of Cardiac Output by Dye Dilution Methods, Klin. Wschr. 44: 753 (July) 1966.*)

EXERCISE To determine whether the maximum increase in cardiac output during exercise was limited by cardiac or extracardiac factors, which might limit ventricular filling, the effect of acute expansion of blood volume was studied in 6 healthy men. Two weeks after 1,000 to 1,200 ml. of blood were withdrawn and stored, cardiac output was determined at rest, and cardiac output, oxygen consumption and oxygen debt were determined after exercise. The subjects were then reinfused with their own blood and the studies repeated. After infusion there was a small increase in central venous pressure and a substantial increase (average 1.47 liters/minute) in cardiac output at rest. During exercise, however, there was a substantial increase in central venous pressure but no significant increase in cardiac output or oxygen consumption, nor was there any reduction of the oxygen debt created by the exercise. This suggests that the heart itself rather than extracardiac factors limits the response to exercise. (*Robinson, B. J., and others: Circulatory Effects of Acute Expansion of Blood Volume, Cir. Res. 19: 26 (July) 1966.*)

HEART FAILURE Pulmonary blood volume (PBV) and central blood volume (CBV) were measured in normal patients, in patients with mitral valvular heart failure, and in patients with non-valvular heart failure. Results were: Normal patients—PBV 271 ml./m.^{2}, CBV 596 ml./m.^{2}, valvular heart failure —PBV 238 ml./m.^{2}, CBV 600 ml./m.^{2}; nonvalvular heart failure—PBV 292 ml./m.^{2}, CBV 679 ml./m.^{2} Cardiac index in the heart failure groups was 50 per cent of that in the normals, whereas pulmonary vascular resistance was three times normal in the nonvalvular group and 12 times normal in the valvular group. Pulmonary artery and left atrial pressures were significantly elevated in both heart failure groups. The findings indicate a marked alteration in pulmonary vasculature pressure volume relations in the presence of chronic pulmonary venous hypertension. In the pathogenesis of heart failure, elevations in pulmonary vascular pressures play the dominant role while changes in PBV are of secondary importance. (*Schreiner, B. F., and others: Pulmonary Blood Volume in Congestive Heart Failure, Circulation 34: 249 (Aug.) 1966.*)

PULMONARY CIRCULATION The effect of hypoxia on the diameter of the small muscular pulmonary artery accompanying a

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