Squatting reduces the blood flow in the legs with a consequent increase in flow to the upper part of the body, and in an increased venous oxygen content in this region. Benefits obtained by individuals with cyanotic congenital heart disease are thought to result from increased central nervous system and cardiac oxygen tension following squatting. (*Brotmacher, L.: Haemodynamic Effects of Squatting During Repose, Brit. Heart J. 19: 567 (Oct.) 1957.*)

Squatting causes kinking of the femoral arteries and veins in the groins and in the popliteal fossae. Blood flow in the legs is reduced, mainly as a result of obstruction to venous return. When patients with cyanotic heart disease exercise, the oxygen content of femoral venous blood falls precipitously. The desaturated blood reaches the right side of the heart and is shunted into the systemic arteries. The oxygen saturation of arterial blood falls in consequence. Squatting impedes venous return from legs and therefore minimizes the tendency of the arterial oxygen saturation to fall with exercise. (*Brotmacher, L.: Haemodynamic Effects of Squatting During Recovery from Exertion, Brit. Heart J. 19: 567 (Oct.) 1957.*)

HYPOTHYROIDISM Clinical and hemodynamic studies in twelve patients with myxedema were compared with those of seven euthyroid patients with chronic congestive failure from myocardial disease. In the hypothyroid patients the low cardiac outputs increased and elevated systemic resistances decreased with exercise in contrast to the patients with congestive failure. Mean arteriovenous oxygen differences in the patients with myxedema were not significantly different from those of healthy subjects. In view of the normal responses of cardiac output and arteriovenous oxygen differences in the patients with myxedema, the finding of elevated mean pressures in the right atria and pulmonary arteries, end diastolic right ventricular pressures, and diastolic dips in the right ventricular pressure curves in the patients with myxedema with enlarged cardiac silhouettes suggested pericardial effusion rather than cardiac dilatation as a cause of enlarged cardiac contour. (*Graettinger, J. S., and others: Correlation of Clinical and Hemodynamic Studies in Patients with Hypothyroidism, J. Clin. Invest. 37: 502 (April) 1958.*)

CORONARY CIRCULATION Chest pain in patients with aortic insufficiency has been ascribed classically to myocardial ischemia due to a decrease in the coronary blood flow. In the anesthetized dog acute aortic insufficiency, sufficient to lower markedly the mean arterial blood pressure and the aortic diastolic pressure, resulted regularly in an increase in coronary sinus blood flow and myocardial oxygen consumption. The increase in coronary flow must be due to a decrease in the resistance of the coronary bed. The decrease in the resistance was probably induced by the increase in work of the left ventricle. Whether the increase in coronary flow was sufficient to meet the demands made upon the left ventricle by the aortic insufficiency, and thereby prevent myocardial ischemia, cannot be determined from these experiments. (*Wegria, R., and others: Effect of Aortic Insufficiency on Arterial Blood Pressure, Coronary Blood Flow and Cardiac Oxygen Consumption, J. Clin. Invest. 37: 471 (March) 1958.*)

BLOOD VOLUME An average increase of 56 per cent in mean arterial pressure by levarterenol was associated with an average decrease of 15 per cent in plasma volume, no change in red cell mass, an average increase of 8 per cent in hematocrit and an average increase of 5 per cent in plasma protein. The fluid lost during hemoconcentration contains less protein than the plasma. These changes are quickly reversed when the levarterenol is discontinued. (*Finnerty, F. A., Jr., Buchholz, J. H., and Guillaudeu, R. L.: Blood Volumes and Plasma Protein During Levarterenol-Induced Hypertension, J. Clin. Invest. 37: 425 (March) 1958.*)

BLOOD VOLUME Values for blood volume (Evans blue technique) in a series of 100 patients suffering from lesions of considerable magnitude were from 2 to 30 per cent below normal in four-fifths of the patients when they were admitted to the hospital. No estimate of intravascular