dilator effect on the cerebral vessels, are probably chiefly secondary to their effects on blood pressure, cerebral metabolic rate, and respiratory gas tensions in the blood. Similarly, the effect of morphine in increasing cerebral blood flow (and hence elevating CSF pressure) is mediated by hypoxia and carbon dioxide retention secondary to respiratory depression. Homeostatic mechanisms for protection of cerebral blood flow and their modification by drugs are discussed. (*Sokoloff, L.: The Action of Drugs on the Cerebral Circulation, Pharm. Rev. 11: 1 (March) 1959.*)

CUTANEOUS CIRCULATION The controls acting on cutaneous circulation are adjusted primarily toward regulation of body temperature. Other reactions are due to direct sensitivity of cutaneous vessels to temperature and to vasoactive substances in a similar fashion as other vessels, or on the availability of their innervation to excitation by other than thermal stimuli. Thermal conductance and total cutaneous blood flow increase very little until the ambient temperature has risen to 28 C. Above 28 C. thermal conductance and cutaneous blood flow increase almost linearly with temperature. The dominant arteriomotor innervation appears to be arteriodilator. Vasodilatation of the hands (and feet), however, begins at an ambient temperature of 22 C. and is about one-third complete at 28 C. This is adjusted chiefly by variations in the activity of the arterio-constrictor innervation. The cutaneous venomotor innervation can greatly alter the blood capacity of the skin. Adjustments in cutaneous venomotor tone may be an important compensation for deficits in circulating blood volume. Since the cutaneous venous system is distended early in response to heat, mobilization of blood contained in the skin would be favored by cool surroundings. Sympathetic denervation probably exerts its principle vascular effect in the skin only on the veins and on palmar and plantar blood flow. (*Hertzmann, A. B.: Vasomotor Regulation of Cutaneous Circulation, Physiol. Rev. 39: 280 (April) 1959.*)

PULMONARY CIRCULATION The effect of hemorrhage to the extent of one-third of the estimated blood volume was studied in dogs anesthetized with pentobarbital. Reduction in blood flow resulted in continuous decline in systemic arterial blood pressure. Pulmonary arterial blood pressure, however, stabilized after an initial decline, dropping only slightly thereafter. Respiratory dead space increased following hemorrhage. This was accompanied by the appearance of a marked carbon dioxide tension gradient between the arterial blood and the end-tidal gas, indicating development of a significant "alveolar" dead space. Restoration of blood volume resulted in a rise in pulmonary blood pressure which was initially above the control level. The respiratory dead space decreased toward its control volume and the carbon dioxide tension gradient was reduced. With intermittent positive pressure ventilation, reduction of pulmonary blood flow may lead to complete closure of portions of the pulmonary bed. Following restoration of blood flow, there is some delay before these vessels again reopen. (*Gerst, P. H., Rattenborg, C., and Holaday, D. A.: The Effects of Hemorrhage on Pulmonary Circulation and Respiratory Gas Exchange, J. Clin. Invest. 38: 524 (March) 1959.*)

PULMONARY HYPERTENSION The pulmonary vascular bed is a complex system with fluctuating pressure and flow. Alterations in cardiac rate and stroke volume, changes in intrathoracic pressure associated with respiration and possible alterations in resistance to air flow all may affect the pressure-flow ratio across the pulmonary vascular bed without active changes in the caliber of the pulmonary vessels. Pulmonary vessels are constricted by serotonin, and the increase in pulmonary artery pressure seen with hypoxia may be due to a direct affect of lowered oxygen tension on the smooth muscle fibers. In patients with pulmonary hypertension associated with congenital heart disease, breathing mixtures low in oxygen increases the resistance while breathing oxygen decreases it. Also pulmonary hypertension is reduced when acetylcholine is injected into the pulmonary artery. An even greater reduction occurs when oxygen and acetylcholine are used simultaneously. Tone in pulmonary vessels has been demonstrated in every condition in which pulmonary hypertension occurs. The manner in which this tone is maintained is unknown. (*Shepherd, J., and*

Wood, E.: Editorial, The Role of Vessel Tone in Pulmonary Hypertension, Circulation 19: 641 (May) 1959.)

HEMODYNAMICS The hemodynamic effects of peripheral vasodilatation produced by Arfonad were studied in patients during congestive heart failure, in cardiac subjects after recovery of compensation and in noncardiac subjects. Vascular and intracardiac pressures were lowered by 20 to 30 per cent in both the systemic and pulmonic circulations. In most of the subjects with low output congestive heart failure the lowering of vascular pressures was associated with an increase in cardiac output of approximately 15 per cent. There was a concomitant increase in the oxygen content of mixed venous blood and decrease in arterio-venous oxygen difference. In noncardiac and compensated cardiac subjects the lowering of vascular pressure resulted in a decrease in cardiac output of approximately 15 per cent with a fall in oxygen content of mixed venous blood and a small rise in the arterio-venous oxygen difference. In all of the subjects there was a decrease in the excretion of water and sodium while renal hemodynamic functions changed variably and slightly. There was no difference between hypertensive and normotensive subjects. The increase in cardiac output during vasodilatation in the patients in congestive heart failure was achieved without an increase in cardiac minute or stroke work. In the noncardiac and compensated cardiac subjects minute and stroke work of the heart decreased during vasodilatation, this being due more to the fall in arterial pressure than to the decrease in cardiac output. Arfonad given intravenously in congestive heart failure produced effects similar to digitalis with respect to decreased intracardiac pressures, increased cardiac output and decreased arterio-venous oxygen difference. Following digitalis, cardiac work increased and diuresis of water and electrolytes was produced, whereas after Arfonad, cardiac work remained unaltered and water and electrolyte excretions decreased. It is not clear whether the increase in blood flow without increase in cardiac work, which follows peripheral vasodilatation by Arfonad, is beneficial to the circulation in those states where the heart is so impaired that myocardial activity cannot be intrinsically improved. (*Sobol, B. J., and others: Cardiac, Hemodynamic and Renal Functions in Congestive Heart Failure During Induced Peripheral Vasodilatation; Relationship to Starling's Law of the Heart in Man, J. Clin. Invest. 38: 557 (March) 1959.*)

BLOOD VOLUME The theoretical and technical limitations of blood volume measurement and of determination of normal values are discussed in detail. Results in one representative sample of male medical students were a mean plasma volume of 45 ml. per kilogram and a total blood volume of 77 ml. per kilogram, with a standard deviation of about 10 per cent. The major deviations were due to differences in adiposity and this accounts for the lower blood volume characteristically seen in females. In addition, blood volume normally varies with age, bed rest, exercise, athletic training, posture, temperature, altitude and pregnancy. Abnormal reductions occur with dehydration and salt depletion. The principle result of vasomotion in the high pressure arterial system is an altered peripheral resistance. However, in the low pressure venous system, where resistance to flow is a minor factor under most circumstances, the result is a change in capacity. Thus alteration in the caliber of arteries has far less potential effect on capacity than dilatation of veins. Plasma volume can be adjusted rather quickly to compensate for sudden changes in total blood volume and moment-to-moment adjustments in plasma volume serve to maintain constant the overall blood volume. The site of adjustment is the capillary, and the rate of transcapillary exchange of fluid is so great that relatively enormous shifts in plasma volume can occur rapidly with a shift in capillary osmotic and hydrostatic forces. The vasomotor system is presumably involved to a large degree; however, large reflex changes in arterial pressure can be brought about without change in capillary pressure or plasma volume. Also pressor doses of epinephrine have inconstant or only small effects on plasma volume (dog). Changes in venous pressure, however, have marked effects on plasma volume because of their influence on capillary pressure. (*Gregerson, M. I., and Rawson, R. A.: Blood Volume, Physiol. Rev. 39: 307 (April) 1959.*)