

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. Reduc-

tion 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*