

*Epinephrine and Norepinephrine by Sympathetic Nerves and Ganglia, Circulation* 17: 366 (Mar.) 1958.)

**PERIPHERAL VENOCONSTRICTION** Peripheral venoconstriction is found in a cool environment or after local cooling. Arterial constriction precedes venoconstriction while venodilatation precedes arteriolar dilatation. Congestion of the legs resulted in prompt arteriolar constriction and venous pressure reduction followed by peripheral venoconstriction in the forearm. Relief of congestion resulted in venous pressure "overshoot," venodilatation, and arteriolar dilatation. Reduction in venous volume of the forearm suggests that the remaining blood volume was moved centrally by venoconstriction in the periphery which was more intense than that in the central veins. (Wood, J. E., and Eckstein, J. W.: *Tandem Forearm Plethysmograph for Study of Acute Responses of Peripheral Veins of Man, J. Clin. Invest.* 37: 41 (Jan.) 1958.)

**CHRONIC ANEMIA** The relationship between severe uncomplicated chronic anemia and the size of the heart was studied in dogs. A significant hypertrophy of the heart was present in the chronically anemic animals. The presence of edema was ruled out as a factor in the increase in heart size. The hypertrophy was found to involve both right and left ventricles and to be accompanied by dilatation of both chambers. (Paplaunus, S. H., Zbar, M. J., and Hays, J. W.: *Cardiac Hypertrophy as Manifestation of Chronic Anemia, Am. J. Path.* 34: 149 (Jan.-Feb.) 1958.)

**METHYLENE BLUE** The respiration of non-nucleated erythrocytes may be greatly stimulated by the addition of methylene blue to the buffer system. Explanations offered for this phenomenon are: 1) increased oxidation of a degradation product of glucose, 2) oxidation of lactate, 3) increased reversible conversion of hemoglobin to methemoglobin, 4) and conversion of lactic acid to pyruvate. None of these explanations account for the magnitude of the increased respiration. *In vitro* experiments confirmed that oxygen consumption varied directly with increasing concentrations of dye, glucose

utilization was increased, and lactic acid formation was decreased. They also indicated that methylene blue activates a cyclic glucose oxidative pathway in mammalian red cells (hexose monophosphate shunt). This mechanism accounts for as much as 85 per cent of the oxygen consumed by human erythrocytes in the presence of methylene blue. (Brin, M., and Yonemoto, R. H.: *Stimulation of Glucose Oxidative Pathway in Human Erythrocytes by Methylene Blue, J. Biol. Chem.* 230: 307 (Jan.) 1958.)

**PRESSURE SUIT** Inflation around the lower half of the body of a tightly fitting pneumatic suit at a pressure of 75 mm. of mercury produces an acute increase in pulmonary arterial and wedge pressures of about 25 mm. of mercury in normal subjects. In none of these eleven series of experiments on four subjects was the diffusing capacity of the lungs for CO<sub>2</sub> significantly altered. (Lewis, B. M., Forster, A. E., and Beckman, E. L.: *Effect of Inflation of Pressure Suit on Pulmonary Diffusing Capacity in Man, J. Appl. Physiol.* 12: 57 (Jan.) 1958.)

**PULMONARY CIRCULATION** Human subjects under normal and hypoxic conditions were studied during the infusion of acetylcholine into the pulmonary artery. The infusion resulted in a fall in pulmonary arterial pressure which was more evident after hypoxia had produced pulmonary hypertension. The fall in pressure was not associated with a decrease in cardiac output and there was no change in pulmonary wedge pressure, heart rate, systemic blood pressure, or central blood volume. Apparently, acetylcholine causes pulmonary vasodilatation which is more marked in the presence of an increased vascular tone. (Fritts, H. W., Jr., and others: *Effect of Acetylcholine on Human Pulmonary Circulation Under Normal and Hypoxic Conditions, J. Clin. Invest.* 37: 99 (Jan.) 1958.)

**PULMONARY EMBOLISM** Review of the literature fails to provide convincing evidence of important reflex effects of pulmonary embolism. In experiments on anesthetized dogs with pulmonary emboli produced by starch and glass beads,

no evidence for important reflex phenomena could be found. The data are interpreted to indicate that such emboli cause pulmonary arterial bed obstruction with resultant pulmonary hypertension, followed by systemic hypotension as left ventricular output decreases. Late venous engorgement and right heart failure lead to death. (McEroy, R. K., Harder, A., and Dale, W. A.: *Respiratory and Cardiovascular Phenomena Associated with Pulmonary Embolism*, *Surg. Gynec. & Obst.* 106: 271 (Mar.) 1958.)

### SPINAL ARTERY OCCLUSION

Three main vessels supply the spinal cord: One anterior and two posterior spinal arteries which run the length of the cord and receive a variable number of radicular arteries, together forming a network of anastomosing vessels within the pia. Inasmuch as the anterior spinal artery supplies the anterior two-thirds of the cord, occlusion of this vessel produces a predictable clinical picture: Loss of pain and temperature perception below the level of occlusion (damage to lateral spinothalamic tracts), spastic muscular weakness (impairment of the descending motor pathways in the anterior and lateral funiculi), and loss of bladder and bowel control (interruption of supra-segmental pathways). The two posterior spinal arteries supply the posterior one-third of the cord: Their occlusion (or one involving a portion of the spinal venous system) does not appear to produce recognizable clinical syndromes. The onset of the syndrome of anterior spinal artery occlusion is usually abrupt and may be associated with severe pain. The typical clinical picture appears rapidly and progresses to its full evolution within minutes to a few hours. The cause of the syndrome may not be apparent, or it may be related to such conditions as sudden hypotension, coarctation of the aorta, prolonged shock, ischemia due to occlusion of the aorta during surgery, syphilis, arteriosclerosis, etc. With general supportive and intensive physical therapy, patients show early improvement in muscle strength and varying degrees of sensory recovery, but some neurologic deficit persists. (Peterman, A. F., Yoss, R. E., and Corbin, K. B.: *Syndrome of Occlusion of Anterior Spinal*

*Artery*, *Proc. Staff Meet. Mayo Clin.* 33: 31 (Jan. 22) 1958.)

**RENAL FUNCTION** The 15-minute or fractional phenolsulfonphthalein (PSP) test is a simple, accurate, rapid clinical method for assessing renal status which indicates not only tubular function but also the lowest glomerular function compatible with that level of tubular activity. Glomerular function of 45 per cent or more of normal is adequate for maintaining homeostasis during the most stressing of operative procedures. The equivalent of 45 per cent glomerular function is a PSP dye excretion of 15 per cent in 15 minutes, and thus in screening a patient for an operative procedure this PSP value is adequate. Under such circumstances no further assessment of kidney function need be made. However, if PSP excretion is less than 15 per cent in 15 minutes, then the urea or creatinine clearance test should be run since glomerular function may be sufficiently better than tubular function to permit the desired operative procedure to be performed. (Lapides, J., and Bobbitt, J. M.: *Preoperative Estimation of Renal Function*, *J. A. M. A.* 166: 866 (Feb. 22) 1958.)

### POSTOPERATIVE DIURESIS

Postoperative urine output of patients receiving the "lytic cocktail" as preoperative medication is greater than those receiving morphine and atropine as a premedicant. To determine the true effect of the lytic cocktail on urine output, a series of dogs were operated upon under ether anesthesia with normal and with the "lytic cocktail" as a preoperative medication. The "lytic cocktail" consisted of chlorpromazine, Synopen, Dolantin, and in 4 dogs, phenothalamine. Postoperative increased salt and water re-absorption was completely inhibited by the "lytic cocktail" administered 45 minutes before surgery. It is believed that this is due to the inhibition of increased aldosterone activity. It is probable that the peripheral sympatho-adrenalytic and/or the central depressant properties of the "lytic cocktail" is responsible for this inhibition. (Koracs, G. S., and others: *Effect of Autonomic Blocking Agents on Surgical*