

Bradykinin, produced at birth, may be a mediator of neonatal circulatory changes, including constriction of the ductus arteriosus, dilatation of pulmonary vasculature and constriction of umbilical vessels. (McInnon, K. L., and others: *Kinins: Possible Mediators of Neonatal Circulatory Changes in Man*, *J. Clin. Invest.* 47: 1295 (June) 1968.)

**HEMOGLOBIN CATABOLISM** Varying quantities of normal canine erythrocytes damaged by incubation with N-ethylmaleimide were injected into the circulatory systems of anesthetized dogs. The quantities of hemoglobin injected ranged from 0.058 to 0.552 g/kg body weight. Although 0.364 g hemoglobin/kg body weight was associated with normal sequestration, above that amount the rate of catabolism of hemoglobin to carbon monoxide reached a maximum. As a result, large quantities of hemoglobin entered the plasma. These data indicate that: (a) the maximal rate of hemoglobin catabolism in these dogs averaged about 0.07 g/kg body weight/hr; (b) hemoglobinemia can result from overloading the reticuloendothelial system with damaged sequestered cells and, therefore, may not always reflect "intravascular" hemolysis; (c) the sequestering function of the reticuloendothelial system does not appear to limit the maximal rate of hemoglobin catabolism. (Coburn, R. F., and Kane, P. B.: *Maximal Erythrocyte and Hemoglobin Catabolism*, *J. Clin. Invest.* 47: 1435 (June) 1968.)

**PHEOCHROMOCYTOMA** Blood volumes in patients with pheochromocytoma, especially those with fixed hypertension, are low. Preoperatively, plasma volume and erythrocyte mass are determined. Deficits are corrected by blood transfusions and by the use of alpha- and beta-blocking agents. In patients thus prepared, no postoperative norepinephrine infusions have been necessary. Halothane is particularly suitable for anesthesia because of its depressant effect on catecholamine liberation and its ability to prevent hypertension during surgery. (Kalf, G.: *Anesthesiologic Problems in Patients with Pheochromocytoma*, *Der Anaesthetist* 17: 43 (Feb.) 1968.)

**VEINS** The veins have served medicine well. Their size and accessibility have made possible the development of our vast array of modern diagnostic and therapeutic methods which depend upon the simple technique of venipuncture. What are the functions of the veins? First, they act as conduits by which blood can return from the tissues to the heart. Second, they may contribute to the total peripheral vascular resistance, especially in the presence of precapillary vessel dilatation. Third, they probably play an important role in the control of fluid exchange between blood and extracellular spaces by altering the ratio between pre- and postcapillary resistance. Fourth, they almost certainly act as a blood depot, for a large quantity of blood lies in the postcapillary side of the circulation. (Browse, N. L.: *The Veins and Cardiovascular Reflexes*, *Ann. Roy. Coll. Surg. Eng.* 42: 307 (May) 1968.)

**TRANSFUSION** Hypotension and homologous blood transfusion can produce periarterial hemorrhage, alveolar hemorrhage, and edema in dog lungs. These do not occur with autologous transfusion. The underlying event is pulmonary arteriolar vasoconstriction associated with periarterial hemorrhage. This is followed by a compensatory vasodilatory phase characterized by capillary engorgement, diffuse pulmonary hemorrhage, and edema. (Verth, F. J., and others: *Pulmonary Microcirculatory Response to Shock, Transfusion, and Pump-Oxygenator Procedures*, *Surgery* 64: 95 (July) 1968.)

**HEMOPHILIA** Substances used for treating or preventing hemorrhage from hemophilia include fresh plasma or fresh blood, factor VIII concentrates prepared from animal plasma or concentrates of human origin such as Colin's fraction I and antihemophilic globulin. However, all of these substances have serious disadvantages. A new substance, antihemophilic cryoprecipitate, rich in factor VIII, was prepared from human plasma by cold precipitation and used to treat eight hemophiliacs on 12 occasions. Assays showed a steep rise in factor VIII activity after the infusion, and in some cases clotting function was restored to normal. The venous blood clot-

ting time proves to be a simple, rapid test for monitoring the therapeutic response. (Bruster, H., Glassner, K., and Riech, C. R.: *Antihemophilic Cryoprecipitate from Fresh Plasma and its Use in Hemophilia, German Med. Monthly* 13: 129 (March) 1968.)

**PARENTERAL FLUID THERAPY** In response to various stimuli associated with injury and operation, antidiuretic hormone and aldosterone are released into the circulation. Through their effect on the renal tubule, there is water and sodium retention. With continued injury there is translocation of extracellular fluid, chiefly an obligatory movement into the area of the injury. With loss of functional extracellular fluid volume, transcapillary filling is less effective and diminished blood volume leads to entry into the blood stream of more antidiuretic hormone<sup>1</sup> and aldosterone. Prompt repletion of the intravascular and extracellular fluid volumes can lessen this hormonal response, whereas continuing deficiency can increase the intensity of the response. (Crandell, W. B.: *Parenteral Fluid Therapy, Surg. Clin. N. Amer.* 48: 707 (Aug.) 1968.)

**POTASSIUM AND CALCIUM LEVELS** Serum calcium and potassium measured in 40 patients before and after induction of anesthesia with thiopental or halothane, decreased significantly after reaching plane I of the third stage. Administration of succinylcholine did not cause significant changes in calcium levels but did cause significant increases in serum potassium. A fluid shift in the extracellular space or respiratory or metabolic blood-gas changes are not believed to be responsible for the electrolyte changes. (List, W. F.: *Changes of the Serum Levels of Calcium and Potassium during Induction of Anesthesia, Der Anaesthesist* 17: 221 (July) 1968.)

### Respiration

**PULMONARY STRUCTURE AND FUNCTION** A cast of a normal human lung was made with a thermosetting resin. This preparation provided sufficient detail for

a study of patterns of branching of the respiratory system as well as for measurement of the dimensions of the various generations of bronchi and bronchioles. The pattern of branching in the human respiratory tract was asymmetrical, so that lengths of pathways and transit times to different pulmonary lobules varied. A system of designating subdivisions of the bronchial tree was proposed. Properties of the respiratory tract inferred from study of the case were used to explain respiratory phenomena such as the alveolar plateau. (Horsfield, K., and Cumming, G.: *Morphology of the Bronchial Tree in Man, J. Appl. Physiol.* 24: 373 (March) 1968 and *Functional Consequences of Airway Morphology, J. Appl. Physiol.* 24: 384 (March) 1968.)

**AIRWAY RESISTANCE** Disease of small airways may be common to various chronic obstructive lung diseases. Using a retrograde catheter technique for partitioning airway resistance, central ( $R_c$ ) and peripheral ( $R_p$ ) airway resistance were measured in five exercised normal lungs and the results compared with values in lungs from patients with emphysema, bronchiectasis or bronchiolitis. In the normal lungs,  $R_p$  accounted for only 25 per cent of the total airway resistance (averaging 0.18 cm water/liter/sec). In seven patients with emphysema,  $R_p$  was increased from four to 40 times.  $R_p$  was also increased in one patient with bronchiectasis and another with bronchiolitis. In all,  $R_c$  was scattered about the normal value. These observations support the conclusion that because  $R_p$  is normally so small, there may be considerable obstruction in peripheral airways, affecting ventilation, distribution and gas exchange with little effect on function tests designed to reveal obstruction. Elevation of airway resistance to a clinically detectable level by disease in the small airways may result in obstruction that is more severe than is generally recognized. (Hogg, J. C., Macklem, P. T., and Thurlbeck, W. M.: *Site and Nature of Airway Obstruction in Chronic Obstructive Lung Disease, New Eng. J. Med.* 278: 1355 (June) 1968.)