and a high-lying diaphragm makes accurate placement of pressures critical. (*Thaler, M. N., and Crause, V. W.: Serious Trauma in Children after External Cardiac Massage, New Engl. J. Med. 276: 500 (Sept. 6) 1962.*)

**PLASMA VOLUME** Plasma volume is restored to control values four hours after a hemorrhage regardless of the magnitude of the hemorrhage and regardless of the availability of exogenous water following hemorrhage in animals. Total blood volume four hours after a hemorrhage is the resultant of the amount of blood withdrawn plus totally restored (initial) plasma volume. Since the plasma protein concentration is decreased following hemorrhage, and since the resultant decrease in oncotic pressure would tend to allow water loss from the vascular compartment, this mechanism cannot be invoked to explain what has been observed. The decrease in capillary pressure initiated by hemorrhage promotes movement of fluid into the vascular compartment, and this phenomenon seems the most likely possible explanation. (*Pareira, M. and others: Plasma Volume Response to Graded Hemorrhage, Surgery 52: 378 (Aug.) 1962.*)

**BODY PERFUSION** During perfusion of patients with tetralogy of Fallot, when the aorta is cross-clamped, the flow perfusing the systemic tissues is equal to total perfusion flow minus the considerable flow through bronchial collateral channels. In four patients the "runoff" through the bronchial circulation averaged 22 per cent of the total flow. When perfusion flow rate is reduced, the percentage of runoff through collateral vessels does not fall but may even increase. Reduction of flow rate may improve visibility, but it will not effect a more favorable ratio between systemic and total perfusion. (*Moffitt, E. A., and others: Physiologic Studies During Whole-Body Perfusion in Tetralogy of Fallot, J. Thor. Cardiov. Surg. 44: 180 (Aug.) 1962.*)

**BLOOD–BRAIN BARRIER** Rates of accumulation of blood-borne solutes within the extravascular compartments of the central nervous system are slower than the interstitial compartments of most other tissues. This phenomenon is referred to as the blood-brain barrier. Water, highly lipoid soluble substances, and a few non-dissociated water soluble compounds penetrate rapidly into the central nervous system from the plasma. Most solutes which display the blood-brain barrier effect appear to enter central nervous system tissue more readily from the cerebrospinal fluid than from the plasma. Various explanations for this decreased permeability between plasma and extravascular compartments of the central nervous system have been suggested. Some of these are: (1) A structural wall separating the plasma from the interstitial fluid has variously been described as composed of capillary endothelium, the endothelial basement membrane or the perivascular glial sheath. (2) Lack of an interstitial space might restrict the movement of plasma solutes by the nature of the cell membranes adjacent to the capillaries. In this case the blood-brain barrier becomes a special case of inter-intracellular solute transfer. (3) The metaabolic activities of the central nervous tissue determine the rate of accumulation of blood-borne solutes. The central nervous system is thus assumed to contain compartments of low metabolic turnover which exchange slowly with the plasma. (4) Evidence has accumulated to suggest that certain important solutes may be transferred between plasma and extravascular fluids of the central nervous system by an active transport process. This mechanism appears to have similarities with analogous systems in the kidney tubules. (*Tschirgi, R. D.: The Blood-Brain Barrier: Fact or Fancy? Fed. Proc. 21: 665 (May–June) 1962.*)

**MYOCARDIAL FATTY CHANGE** Many infants dying shortly after delivery show clinical and postmortem evidence of heart failure. The findings of stainable fat in the myocardium of these infants is in contrast to its absence in those neonates who die without evidencing heart failure. This is related to the deranged metabolism in these failing hearts by which fatty acids are oxidized for energy. (*Scott, J. M.: Fatty Change in the Myocardium of Newborn Infants, Amer. Heart J. 64: 283 (Aug.) 1962.*)