marily) may mediate the increase in pulmonary vascular resistance and the decrease in blood flow which occurs after collapse of a lung secondary to obstruction and hypoxia. (*Barer, G. R.: Reactivity of the Vessels of Collapsed and Ventilated Lungs to Drugs and Hypoxia, Circ. Res. 28: 366 (April) 1966.*)

CAROTID BODY The carotid bifurcation of cats was perfused at constant pressures with bicarbonate buffered Ringer's solution containing dextran. The response of the chemoreceptors to abrupt changes in *p*H and/or P_{CO_2} was measured as the change in impulse frequency recorded from Hering's nerve. An increase in P_{CO_2} of 30 mm. of mercury consistently resulted in a carotid body response only when accompanied by a decrease in *p*H. In some instances in which there was a response to an increase in P_{CO_2} at constant *p*H, the receptor discharge returned rapidly to control values despite maintained stimulus of constant intensity. When *p*H was decreased from 7.4 to 7.1 through either a reduction in bicarbonate ion or an increase in P_{CO_2}, the responses were of the same magnitude. However, the response to acidification by CO_2 occurred more rapidly than that to bicarbonate ion reduction. The data suggest that the receptor is responsive to and in equilibrium with extracellular *p*H but separated from the vascular space by a diffusion barrier more permeable to carbon dioxide than to hydrogen ion. (*Gray, B. A., Munroe, A. B., and Tenney, S. M.: Response of the Perfused Carotid Body to Changes in pH and P_{CO_2}, Fed. Proc. 25: 264 (March) 1966.*)

CEREBRAL BLOOD FLOW Man living at high altitude, when made normoxic, has normal cerebral blood flow (CBF) and cerebrospinal fluid (CSF) *p*H, despite low Pa_{CO_2} and high *p*H. Alkalemia does not influence CBF and CSF *p*H at constant Pa_{CO_2}. Thus, carbon dioxide may control CBF via *p*H in extracellular fluid (ECF) where bicarbonate ion (HCO_3^-) similar to that of CSF is subject to the *p*H regulating activity of the blood-CSF barrier. Measurement of CBF during step hyperventilation was made by computing CBF as per cent of control from the change in A-V oxygen saturation, utilizing internal jugular vein blood. Volunteers were taught to reduce their end-tidal P_{CO_2}, in 3 seconds to 23 mm. of mercury and to hold it constant for 2 hours. Pa_{CO_2} fell from 41 to 25 in about ½ minute and remained constant while Pv_{CO_2}, taken as an index of tissue P_{CO_2}, fell slowly from 50 to 36. It is concluded that Pa_{CO_2} controls CBF by means of its effect on *p*H of arteriolar smooth muscle ECF, and that this ECF *p*H, like CSF, can be reset to normal in chronic hypocapnia by transport across the blood cerebrospinal fluid barrier. (*Severinghaus, J. W.: Site and Mode of Action of CO_2 on Cerebral Blood Flow in Man, Fed. Proc. 25: 461 (March) 1966.*)

CEREBRAL BLOOD FLOW Experimental review led to the following conclusions concerning cerebral blood flow: (1) Cervical sympathectomy produces no significant increase of cerebral blood flow in normal dogs or in dogs with bilateral ligation of the internal carotid arteries. (2) In dogs, and in humans with obstructed carotid arteries, inhalation of 5 per cent carbon dioxide increases cerebral blood flow about 40 per cent during the period of inhalation and for about 30 minutes thereafter. (3) Administration of papaverine increases cerebral blood flow significantly in dogs, but to a far lesser degree than carbon dioxide inhalation. (4) Hypothermia decreases cerebral blood flow markedly by chemical changes resulting from reduced metabolism. (5) Circulation of blood in small cerebral vessels is under chemical rather than neurogenic control. These findings suggest the careful trial of carbon dioxide inhalation in the treatment of patients with strokes caused by partial obstruction of the cerebral arterial blood supply, providing that the patient is observed closely for evidence of carbon dioxide intoxication. Intermittent inhalations of carbon dioxide in air may beneficially supplement extracranial cerebral arterial surgery or, if operation is impossible, may have a beneficial effect when used alone. (*Shackelford, R., and Hegedus, S.: Factors Affecting Cerebral Blood Flow, Experimental Review: Sympathectomy, Hypothermia, CO_2 Inhala-*

tion and Papaverine, Ann. Surg. 163: 771 (May) 1966.)

VENOUS TONE The effects of various anesthetics, such as halothane, ether, methoxyfluorane, trichlorethylene, cyclopropane, thiopental and lidocaine, on the peripheral circulatory system were compared during passive tilting in human subjects. The blood content of the toe and the venous pressure at the ankle were measured simultaneously by means of a photoconductive plethysmography and an electromanometer. Generally, the blood content of the toe and the venous pressure at the ankle were increased more rapidly under general and epidural anesthesia with head-up tilting than under usual conditions. The change in the venous pressure preceded the change in the blood content of the toe. Furthermore, the administration of a muscle relaxant drug had no significant effect on the peripheral circulation of the lower extremities. These findings suggest that increase in venous distensibility occurred when the nervous system was blocked. *(Hayashi, K.: Clinical Studies on the Effect of Various Anesthetics on the Vascular Volume of the Human Toe, Evaluated by a Photoconductive Plethysmograph (Japanese), Med. J. Hiroshima Univ. 13: 235, 1965.)*

HEMODYNAMICS OF HEMORRHAGE Hemodynamic and metabolic effects of hemorrhage were studied in 11 unanesthetized human volunteers. Hemorrhage of 15–20 per cent of the blood volume in 35 minutes produced a significant reduction in splanchnic blood volume, indocyanine green dye clearance and hematocrit. About one-half of the blood loss was contributed by the splanchnic viscera as the splanchnic blood volume was reduced by 40 per cent while the central blood volume was reduced by only 10 per cent. This hemorrhage, however, produced no significant change in splanchnic blood flow, oxygen consumption, A-V excess lactate production or in splanchnic vascular resistance. Cardiac output, heart rate, mean arterial blood pressure and arterial excess lactate were unaltered. These results suggest that the splanchnic circulation functions as an important blood reservoir which can be preferentially depleted of blood in response to hemorrhage. *(Price, H. L., and others: Hemodynamic and Metabolic Effects of Hemorrhage in Man with Particular Reference to the Splanchnic Circulation, Circ. Res. 28: 469 (May) 1966.)*

CARDIAC SHOCK The responses to vasopressors in patients with acute myocardial infarction with shock were evaluated. Control measurements were cardiac output 2.2 liters/minute, mean arterial pressure (MAP) 53 mm. of mercury, systemic vascular resistance (SVR) 27 mm. of mercury/liter/minute. Methoxamine was given to seven patients. Cardiac output fell in four (19–28 per cent) and did not change in three. SVR was increased in all seven (9–212 per cent). Norepinephrine was given to 11 patients (7 of whom had received methoxamine). Cardiac output was increased in all except one (−8 per cent to +57 per cent), MAP increased in all (+3 per cent to 175 per cent). Low viscosity dextran given to two patients resulted in a 14 per cent and 54 per cent increase in cardiac output. Norepinephrine is felt to be preferable to a pure vasoconstrictor (methoxamine) in patients with cardiogenic shock. *(Gunnar, R. M.: Myocardial Infarction with Shock, Circulation 33: 753 (May) 1966.)*

HEMORRHAGIC SHOCK One hundred heparinized dogs, in groups of four, were bled into reservoirs until a mean systolic arterial blood pressure of 40 mm. of mercury was obtained. After 75 minutes at this blood pressure, treatment consisting of: (1) blood replacement (500–900 ml.), (2) infusion of dextran (500 ml., or (3) hydroxyethyl starch (500 ml.) was instituted in three of each four dogs. The fourth dog received no treatment. Survival rates were: blood infusion, 72 per cent; dextran infusion, 44 per cent; hydroxyethyl starch, 64 per cent; and no treatment, 20 per cent. The difference in survival between dextran and hydroxyethyl starch was not statistically significant. No adverse reactions to either dextran or hydroxyethyl starch were observed. The results indicate that hydroxyethyl starch is at least as effective a volume expanded as is clinical dextran. *(Bal-*