probably caused by anoxia and increased cerebral blood flow. The increased blood flow causes further rise in intracranial pressure, additional anoxia, and eventual vasomotor paralysis. Hyperbaric oxygenation serves to break this cycle, presumably by combating the anoxia while at the same time decreasing cerebral blood flow by vasoconstriction. (*Sukoff, M. H., and others: The Protective Effect of Hyperbaric Oxygenation in Experimentally Produced Cerebral Edema and Compression, Surgery 62: 40 (July) 1967.*)

NOREPINEPHRINE The effects of neuronally-transmitted and blood-borne norepinephrine (NE) on the isolated perfused hind limb and on an isolated segment of the splanchnic circulation were studied in dogs. A comparison was made between intra-arterial injection of NE in the isolated segments and reflex stimulation of these sites produced by carotid body hypotension before and after alpha and beta adrenergic blockade. Results in the two circulations were similar. Intra-arterial NE produced vasoconstriction in the untreated animals, as did reflex release of NE. However, after alpha blockade intra-arterial NE produced vasodilation, whereas reflex stimulation still produced some vasoconstriction although of a much smaller degree than in the controls. In other words, the beta adrenergic effects were unmasked by alpha blockade with injection of NE but not by reflex stimulation. The administration of beta blockers re-reversed the response to NE in the intra-arterial injection, causing vasoconstriction. There was, however, no augmentation of the vasoconstrictor effect on neuronal stimulation after beta blockade. It is postulated that NE released from nerve terminals gains access to alpha receptors but not to beta receptors, whereas blood-borne NE is effective at the alpha receptors reached by nerve stimulation and possibly at other alpha receptor sites not reached by nerve stimulation, and is also effective at beta receptor sites. (*Glick, G., and others: Physiological Differences Between the Effects of Neuronally Released and Bloodborne Norepinephrine on Beta Adrenergic Receptors in the Arterial Bed of the Dog, Circ. Res. 21: 219 (Aug.) 1967.*)

CAVAL OBSTRUCTION Twelve dogs were anesthetized and pressure recordings were made from catheters placed in the peritoneal cavity, in the vena cava just below and just above the diaphragm. Glycerine, saline or air was introduced intraperitoneally in stepwise fashion. When intraperitoneal volume reached 200 ml./kg., intra-abdominal pressure was greater than 30 mm. Hg. Thoracic vena caval pressure rose to more than 30 mm. Hg. There was narrowing and angulation of the vena cava at the level of the diaphragm. Blood flow through the vena cava diminished to less than half the control value. All values returned to normal when intra-abdominal pressure was reduced. (*Rubinson, R. M., and others: Inferior Vena Caval Obstruction From Increased Intra-abdominal Pressure, Arch. Surg. 94: 766 (June) 1967.*)

IPPB AND CIRCULATION Ventilation and cardiac output were measured simultaneously in human volunteers during breathing against graded increments of positive pressure. Cardiac output decreased as positive pressure increased, and when ventilation was maintained constant at 10 l./min., magnitude of the decrease in cardiac output was 0.2 l./min. per additional centimeter of positive pressure imposed. At any level of positive pressure, increases in ventilation counteracted the circulatory effects of positive pressure in the airway; the data suggest that magnitude of tidal volume is an important factor. (*Cruz, J. C., Cerretelli, P., and Farhi, L. E.: Role of Ventilation in Maintaining Cardiac Output Under Positive Pressure Breathing, J. Appl. Physiol. 22: 990 (May) 1967.*)

ARTERIOVENOUS SHUNTS In 44 healthy dogs, continuous infusion of epinephrine in amounts capable of being produced endogenously in shock caused highly significant increases in portal flow and oxygen pressure and femoral venous oxygen pressure. These findings, unexplained by changes in cardiac output or metabolic rate, were thought to be caused by the opening of multiple arteriovenous shunts in these vascular systems. It is suggested that A-V shunts, instead of excessive vasoconstriction due to epinephrine,