rate. Microaggregation of platelets produces a gelatinous plug in the capillary circulation, as well as a rise in venous pressure, thrombocytopenia and hypocoagulability of blood. The most accurate method of diagnosis is by pulmonary angiogram. (*Khazei, A. H., Dembo, D. H., and Cowley, R. A.: Recognition and Management of Massive Pulmonary Embolism, Arch. Surg. 94: 884 (June) 1967.*)

AIR EMBOLISM Providing anesthesia for patients who are in the sitting position presents major problems of which circulatory insufficiency and air embolism are perhaps the most important. It seems prudent to approach cases in which air embolization appears likely with at least these three prophylactic measures: (1) place a catheter in or near the right side of the heart for measuring central venous pressure and aspiration of gas if it accumulates; (2) use some continuous monitor of cardiac sounds; (3) avoid nitrous oxide or use it in low concentrations (50 per cent or less). If embolization is suspected, nitrous oxide administration should probably be discontinued completely. (*Tisovec, L., and Hamilton, W. K.: Newer Considerations in the Air Embolism During Operation, J.A.M.A. 201: 376 (Aug.) 1967.*)

CARDIOVASCULAR NEURONS Peripheral nerve recordings taken from the inferior cardiac and phrenic nerves in mid-collicular decerebrate cats indicated that sympathetic activity was markedly influenced by the periodic discharges of the medullary inspiratory center. Cardiovascular neurons exhibited two spontaneous discharge patterns: steadily-firing and frequency-modulated. These neurons had low resting spike rates and were markedly influenced by blood pressure changes (30 per cent decrease to 100 per cent elimination of firing rate with 30 mm. Hg rise). They were found in the periventricular gray and adjacent dorsolateral reticular formation. Probably these neurons function to maintain and reflexly regulate sympathetic tone of the cardiovascular system. Much evidence suggests that baroceptor reflexes are primarily responsible for the marked changes in activity of the cardiovascular center in the case of either drug-induced or spontaneously-occurring systemic blood pressure variations. (*Przybyla, A. C., and Wang, S. C.: Neurophysiological Characteristics of Cardiovascular Neurons in the Medulla Oblongata of the Cat, J. Neurophysiol. 30: 645 (July) 1967.*)

CEREBRAL BLOOD FLOW Continuous measurements of cerebral blood flow (CBF), oxygen and glucose metabolism were made in patients with cerebrovascular disease. The effects of inhaled carbon dioxide, hyperventilation, nylidrin, low-molecular-weight dextran (LMWD), and endarterectomy upon the above parameters were noted. Control values were: CBF 35.5 ml./100 Gm./brain/min.; cerebral metabolic rate for oxygen (CMR_{O_2}) 2.37 ml./100 Gm./brain/min., cerebral metabolic rate for glucose (CMR_{GL}) 3.64 mg./100 Gm./brain/min. Significant correlation existed between CMR_{O_2} and CMR_{GL}, CBF and cerebral A-V glucose difference, mean arterial BP and CMR_{GL}. CBF was significantly increased by inhalation of 5 per cent carbon dioxide or carotid endarterectomy, and reduced by 100 per cent oxygen inhalation or hyperventilation, whereas no effect was seen after nylidrin or LMWD. (*Meyer, J. S., and others: Monitoring Cerebral Blood Flow, Oxygen and Glucose Metabolism, Circulation 36: 197 (Aug.) 1967.*)

JUGULAR OXYGEN SATURATION No reliable relationship could be found between jugular blood oxygen saturation and cerebral function during carotid occlusion under local anesthesia. It is suggested that the most reliable guide to cerebral oxygenation during carotid occlusion is the patient's state of consciousness. Jugular-blood oxygenation monitoring was useful, however, during general anesthesia and hypercarbia to indicate that total cerebral blood flow had been increased relative to oxygen consumption. (*Larson, C. P., and others: Jugular Venous Oxygen Saturation as an Index of Adequacy of Cerebral Oxygenation, Surgery 62: 31 (July) 1967.*)

CEREBRAL EDEMA The protective effect of intermittent hyperbaric oxygenation is the production of cerebral vasoconstriction and decreased blood flow in the presence of adequate or increased oxygenation of the brain. Both cerebral anoxia and increased cerebral blood flow are factors which increase cerebral edema. Expansion of an intracranial balloon in animals institutes a cycle of vasodilation,

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,