

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 dorso-lateral reticular formation. Probably these neurons function to maintain and reflexly regulate sympathetic tone of the cardiovascular system. Much evidence suggests that baroreceptor 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, nyli-drin, 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 nyli-drin 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,