of these substances except acetaldehyde had any effect like blood levels which might be found after alcohol ingestion. Acetaldehyde, however, markedly stimulated the node. This effect resembled that of tyramine in that it was not blocked by atropinization, could be reversed with propranolol, and was absent after reserpinization, suggesting a local release of norepinephrine from catecholamines stored in the myocardium as the mechanism of action. It is suggested that depletion of norepinephrine stores in the myocardium of the chronic alcoholic may be one of the factors leading to heart failure in these patients. (*James, T. N., and Bear, E. S.: Effects of Ethanol and Acetaldehyde on the Heart, Amer. Heart J. 74: 243 (Aug.) 1967.*)

**ECG AND ELECTROLYTES** Hyperkalemia decreases the normal resting membrane potential and the magnitude of the action potential. This is reflected in the ECG by an increase in the duration of the QRS complex, decreased or absent P waves, and peak T waves. Hypokalemia increases the resting membrane and action potentials. The S-T segment is shortened, T waves decrease in amplitude or are absent whereas U waves are prominant. Both hyper- and hypokalemia prolong A-V conduction and cause block through complex interplay of changes in resting membrane potential, threshold potential and slope of the depolarization curve. Relatively small changes in the extracellular K concentration can produce large transmembrane potential changes due to alteration of the normal intracellular-to-extracellular K ratio of 30:1. Much greater changes in intracellular K, required to alter the ratio significantly, probably do not occur clinically. Hypocalcemia prolongs the plateau of the action potential, causing an increase in the S-T interval, whereas hypercalcemia has the opposite effect. Intraventricular and atrioventricular conduction defects caused by K deficiency can be treated by lowering the Ca concentration with chelating agents. Those caused by excess extracellular K can be treated with Ca. Changes in Mg, Na, and *p*H have little effect on the ECG in themselves, although they may alter it by causing changes in extracellular K. (*Surawicz, B.: Relationship Between Electrocardiogram and Electrolytes, Amer. Heart J. 73: 814 (June) 1967.*)

**CARDIAC ARREST** The anesthesiologist assumes major responsibility in the prevention, recognition and treatment of cardiac arrest. The ABCD management denotes airway, breathing, circulation, diagnosis, drugs, defibrillation. In a patient receiving resuscitative therapy, death can be considered inevitable if there is no spontaneous respiration for 60 minutes and the electrocardiogram shows no systoles for 30 minutes. The electroencephalogram has not been a practical help. (*Pollard, J. W., and Cooley, J. C.: Modern Management of Cardiac Arrest, Selected Papers of Carle Hospital Clinic 20: 9 (Jan.) 1967.*)

**PULMONARY ARTERY OCCLUSION** Effects of pulmonary artery ligation on mechanical properties of the lung were studied in dogs. Three to seven days following unilateral pulmonary artery ligation, focal hemorrhagic atalectasis, increase in lung weight and decrease in ventilation volume occurred. Affected areas could not be inflated, but pressure-volume characteristics of inflatable portions of lungs whose pulmonary arteries had been ligated were normal during inflation and deflation with both gas and saline. Alveolar bubbles expressed from lung which appeared grossly and microscopically normal following occlusion were stable. Areas of normal lung persisted after pulmonary artery occlusion; pathological alterations were localized rather than generalized. Generalized change in alveolar surface forces was not observed. (*Edmunds, L. H., and Huber, G. L.: Pulmonary Artery Occlusion. I. Volume-pressure Relationships and Alveolar Bubble Stability, J. Appl. Physiol. 22: 990 (May) 1967.*)

**PULMONARY EMBOLISM** Fatal embolism occurs in 0.1 to 0.2 per cent of surgical operations, and accounts for 1 to 2 per cent of surgical deaths. There are 47,000 deaths from pulmonary embolism annually. In medical patients, it tends to occur most among those with cardiac or prostatic disease. A large proportion of the lumen of the pulmonary artery must be blocked before symptoms are produced. Obstruction of ⅔ of the lumen causes right ventricular pressure elevation, right ventricular failure, reduced cardiac output, fall in systemic arterial pressure, and rise of pulse

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,