

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 reserpine, 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 prominent. 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-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 pH have little effect on the ECG in themselves, although they may alter it by causing changes in extracellular K. (Suraicz, 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 atelectasis, 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 2/3 of the lumen causes right ventricular pressure elevation, right ventricular failure, reduced cardiac output, fall in systemic arterial pressure, and rise of pulse