

Literature Briefs

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Briefs were submitted by Drs. C. M. Ballinger, N. Bergman, D. R. Buechel, R. B. Boettner, R. B. Clark, J. Eckenhoff, J. Jacoby, R. L. Klein, W. Mannheimer, F. C. McPartland, D. Morrow, A. Patterson, R. E. Ponath, J. W. Pender, A. D. Randall, L. J. Saidman, P. Sechzer, A. D. Sessler, C. Wilkinson. Briefs appearing elsewhere in this issue are part of this column.

Circulation

ARRHYTHMIAS The effect of propranolol (Inderal) on atrial arrhythmias was determined in five patients in the immediate post-operative period. Three of the patients had atrial fibrillation and two, atrial flutter. In each patient the electrocardiographic pattern was converted to a sinus rhythm by propranolol. Digitalis, quinidine and DC counter-shock had been completely ineffective in three of the five patients. These observations suggest that the "quinidine-like" effect of propranolol may offer a safe and effective approach to the treatment of supraventricular arrhythmias. (Wolfson, S., and others: *Conversion of Atrial Fibrillation and Flutter by Propranolol*, *Brit. Heart J.* 29: 305 (May) 1967.)

DILANTIN AND DIGITALIS The electrophysiologic effects of diphenylhydantoin (dilantin) accounting for its antiarrhythmic properties were determined and compared with the effects of procainamide (Pronestyl). Studies were performed in normal and digitalis-intoxicated dogs. In both groups of dogs, dilantin (5 mg./kg.) decreased ventricular automaticity, had little effect upon intraventricular conduction, and increased A-V conduction. Pronestyl (30 mg./kg. in divided doses) also decreased ventricular automaticity but in addition decreased A-V and intraventricular conduction. The depression of conduction seen with Pronestyl might promote further, more serious, re-entry types of ar-

rhythmias. It is concluded that the electrophysiologic properties of dilantin upon the heart make it an excellent agent for the treatment of digitalis-induced arrhythmias. (Helfant, R. H., and others: *The Electrophysiological Properties of Diphenylhydantoin Sodium as compared to Procaine Amide in the Normal and Digitalis Intoxicated Heart*, *Circulation* 36: 108 (July) 1967.)

ABSTRACTOR'S NOTE: Two cases of fatal cardiac arrhythmias in digitalized patients following dilantin (*J.A.M.A.* 200: 335, 1967; *J.A.M.A.* 200: 337, 1967) might temper one's enthusiasm for its use in digitalis intoxication.

DILANTIN AND DIGITALIS Dilantin (5 mg./kg.) increased by 122 per cent the amount of acetylstrophanthidin needed to induce ventricular arrhythmias in dogs. The same dose of dilantin had no effect upon the rate of rise of intraventricular pressure. Thus, dilantin can dissociate the electrophysiologic and inotropic effects of the glycoside. Procainamide (30 mg./kg.) had no effect upon the arrhythmic dose of acetylstrophanthidin and decreased the inotropic effect of the glycoside. Since dilantin increased the toxic dose of digitalis without affecting the inotropic action, the therapeutic index of the glycoside was increased. This might be of benefit to patients in need of the inotropic effects of digitalis but sensitive to the arrhythmic action of digitalis. (Helfant, R. H., and others: *Protection from Digitalis Toxicity with the Prophylactic Use of Diphenylhydantoin Sodium*, *Circulation* 36: 119 (July) 1967.)

ALCOHOL AND THE HEART The effects of direct perfusion of the sinus node with ethanol and its degradation products, acetaldehyde and acetic acid, and methanol and its end products, formaldehyde and formic acid, were studied in 75 anesthetized dogs. None

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 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-to-extracellular K ratio of 30:1. Much greater changes in intracellular K_i 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. (Surauciz, 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