Literature Briefs

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Circulation

ARRHYTHMIA A study of 150 patients treated with lidocaine for ventricular arrhythmias was made. One hundred and ten of the patients were premedicated with phenobarbital or Librium as prophylaxis against convulsions. The use of a single intravenous injection of 1 to 2 mg/kg of lidocaine resulted in complete suppression of ventricular arrhythmias in 82 per cent of cases. Many patients required repeated doses, and one patient received a total of 1,400 mg in 14 hours. Hypotension was uncommon. Central nervous system signs occurred in 7 per cent of the patients. The ventricular arrhythmias accompanying heart failure at times responded to lidocaine alone, but treatment of the heart failure with digitalis improved results. In patients in shock, prior treatment of the hypotension also improved the response to lidocaine. (Malach, M., Kostis, J. B., and Fischetti, J. C.: Lidocaine for Ventricular Arrhythmias in Acute Myocardial Infarction, Amer. J. Med. Sci. 257: 52 (Jan.) 1969.)

GLUCAGON Glucagon (3 or 5 mg) given intravenously resulted in significant improvement in ventricular function in 13 patients with heart disease. The maximum increased inotropic effect seen five minutes after administration, declined to control values by 15 to 30 minutes. In addition, blood glucose increased and plasma potassium decreased following glucagon, which may make digitalized patients more susceptible to arrhythmias, although no arrhythmias were seen in this study. Despite the brief duration of the improvement, glucagon may, because of its notable absence of significant side-effects, be useful in certain clinical conditions. (Williams, J. F., and others: Hemodynamic Effects of Glucagon in Patients with Heart Disease, Circulation 39: 38 (Jan.) 1969.)

DIGITALIS AND HYPOXIA Hypoxia (average PaO2 40 mm Hg) reduced by 19 per cent the amount of acetylstrophanthinidin needed to induce ventricular tachycardia in dogs. Possible explanations for the reduced glycoside tolerance include: 1) reflex catecholamine stimulation; 2) hypoxia-induced alterations in blood flow; 3) effects of hypoxia on the cellular sodium-potassium exchange pump, with resultant changes in the extracellular potassium gradient and changes in the binding of intracellular glycosides. (Harrison, D. C., and others: Role of Hypoxia in Digitalis Toxicity, Amer. J. Med. Sci. 256: 352 (Dec.) 1968.)