

# Literature Briefs

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## Circulation

**ARRHYTHMIAS** Edrophonium (Tensilon) was used to evaluate and treat supraventricular tachycardia. Of ten patients so treated, eight responded favorably. Edrophonium may be an important diagnostic aid without the inherent danger of vascular occlusion present with carotid massage. (Spitzer, S., and others: *Use of Edrophonium (Tensilon) in the Evaluation of Supraventricular Tachycardia*, *Amer. J. Med. Sci.* 254: 477 (Oct.) 1967.)

**ANTI-ARRHYTHMIC DRUGS** In addition to synchronized direct-current counter-shock, several drugs are available for the treatment of cardiac arrhythmias. Indications for the beta-adrenergic blocking agent propranolol include supraventricular arrhythmias which are either uncontrolled or caused by digitalis, where cardioversion is contraindicated; and supraventricular or ventricular arrhythmias associated with the administration of anesthetic agents. Diphenylhydantoin can be used in most situations where quinidine or procainamide is indicated and has the advantage of acting rapidly with few adverse hemodynamic effects. Lidocaine, effective in abolishing acute ventricular arrhythmias arising during surgery or cardiac diagnostic procedures, probably is the agent of choice in the treatment of ventricular arrhythmias in acute myocardial infarction. The average intravenous dose of lidocaine is 1 mg/kg. (Conn, R. D.: *Newer Drugs in the Treatment of Cardiac Arrhythmias*, *Med. Clin. N. Amer.* 51: 1223 (Sept.) 1967.)

**ARRHYTHMIAS** It has been reported that propranolol, given prophylactically, is not effective in preventing paroxysmal cardiac arrhythmias. Life-threatening paroxysmal arrhythmias were prevented in four patients by long-term therapy with oral propranolol. In two patients ventricular tachycardia due to beta-adrenergic stimulation caused syncope, and in the other two supraventricular tachycardia caused pulmonary edema. In each case the drug was given orally after other antiarrhythmic agents had been contraindicated, ineffective or poorly tolerated. In doses of 30-40 mg. daily, it produced no change in the ECG's of any of the four patients. This absence of ECG change suggests that propranolol does not produce the same electrophysiologic changes as do the quinidine-like drugs, and that its action in suppressing arrhythmias is by its beta-adrenergic blocking properties. This conclusion is supported by the observation that in isolated rabbit hearts the diastolic excitability threshold and the effective refractory period of the ventricular myocardium are not altered by perfusion with propranolol, but are altered by perfusion with quinidine. (Gettes, L. S., and Surawicz, B.: *Long-Term Prevention of Paroxysmal Arrhythmias with Propranolol Therapy*, *Amer. J. Med. Sci.* 254: 257 (Sept.) 1967.)

**CARDIAC OUTPUT** Cardiac output is regulated mainly by tissue oxygen requirements. Each individual tissue has a specific method for controlling its blood flow. The sum of all flows through various tissues determines the return of blood to the heart. The heart, so long as it is normal, serves as an automatic pumping station to propel the blood into the aorta. Under stressful conditions this basic mechanism of cardiac-output regulation is modified by a number of additional factors, including autonomic nervous system activity