Literature Briefs

C. Philip Larson, Jr., M.D., Editor

Briefs were submitted by Drs. R. B. Boettner, R. C. Clark, M. I. Gold, W. Mannheimer, F. McPartland, D. H. Morrow, A. D. Randall, L. J. Saidman, P. Sechzer, and A. D. Sessler. Briefs appearing elsewhere in this issue are part of this column.

Circulation

DIGITALIS ARRHYTHMIAS Both hypothermia and pronethalol increased the threshold toxic dose level for cardiac glycosides in dogs. At the same time, both hypothermia and pronethalol prevented ouabain-induced changes in myocardial sodium and potassium. Without the protection of hypothermia or pronethalol, an arrhythmic dose of ouabain was always accompanied by a significant fall in myocardial potassium and increased myocardial sodium, both of which are theorized to be the basis of glycoside-induced arrhythmias. (*El-Fiky, S. B. I., and Katzung, B. G.: Effects of Hypothermia and Pronethalol on Ionic Correlates of Ouabain Arrhythmias in Dogs, Circ. Res. 24: 43 (Jan.) 1969.*)

ATROPINE AND BETA BLOCKADE Beta antagonists, with or without atropine, significantly depressed myocardial function in dogs, as measured by downward displacement and shift to the right of left ventricular function curves. *dl*-propranolol had a much greater depressant effect than Ciba 39,089-Ba when given in equipotent beta-adrenergic blocking doses. Atropine did not antagonize this depression, in that the heart could not respond to increasing work loads without elevation of filling pressure. However, atropine did prevent the reduction in coronary blood flow associated with beta blockade. Even though the beta blockers depressed left ventricular function, efficiency of myocardial contraction was improved in that less oxygen was consumed by the heart for a given amount of work performed. Atropine did not alter this. (*Nayler, W. G., and others: The Combined Effect of Atropine and Beta-adrenergic Receptor Antagonists on Left Ventricular Function and Coronary Blood Flow, Amer. Heart J. 77: 246 (Feb.) 1969.*) ABSTRACTER'S COMMENT: Like the ability to protect against digitalis-induced arrhythmias, the myocardial depressant action of the currently available beta blockers is not necessarily related to their ability to produce beta blockade.

COUNTERPULSATION Counterpulsation, or postsystolic augmentation, is a form of mechanical assistance to the failing circulation which works by withdrawal of blood from the central arterial system during cardiac systole and its reinjection during diastole. It lowers systolic ventricular ejection pressure and at the same time maintains peripheral flow and diastolic pressure. Counterpulsation decreases all the parameters of left ventricular work and maintains peripheral perfusion. (*Dormandy, J. A., and others: Hemodynamics and Coronary Blood Flow with Counterpulsation, Surgery 65: 311 (Feb.) 1969.*)

MYOCARDIAL INFARCTION AND OXYGEN Fifty subjects with acute myocardial infarction were given humidified oxygen by mask, 90 per cent oxygen being delivered by this method. Hypoxemia, as determined by arterial blood tensions, and tissue hypoxia, as determined by elevated blood lactate levels, were reversed by oxygen treatment. A rise in cardiac output occurred following oxygen therapy in patients with low cardiac output, though in most patients cardiac output and stroke volume ultimately decreased. Blood pressure increased, as did systemic vascular resistance, without any increase in left ventricular work. This may