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Literature Briefs

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Literature Briefs were submitted by Drs. L. H. Cronan, A. Goldblatt, L. Brand, E. Lowenstein, H. Rackow, E. Salanitre, J. Bland, and L. C. Mark. Briefs appearing elsewhere in this issue are part of this column.

Circulation

PARASYMPATHETIC CONTROL IN HEART DISEASE This paper establishes the presence of deficient parasympathetic control of the heart in patients with heart disease. Parasympathetic control was studied by intravenous administration of atropine (0.04 mg/kg) after pretreatment with propranolol in patients with heart disease and control subjects. The decrease in R-R interval of the ECG after graded hypertension induced by increments of phenylephrine given intravenously was recorded. Both the increase in heart rate consequent to atropine and the decrease in R-R interval consequent to hypertension were less in subjects with heart disease and appeared to be independent of age, degree of ambulation, medication (including digitalis), and other systemic diseases. The documented abnormality of parasympathetic control may play a role in the limited cardiac response to exercise. (*Eckberg, D. W., Derabinsky, M., and Braunwald, E.: Defective Cardiac Parasympathetic Control in Patients with Heart Disease. N. Engl. J. Med. 285: 877–883, 1971.*)

BETA-ADRENERGIC BLOCKADE In 14 patients at rest and in nine during exercise the cardiovascular responses to the intravenous injection of alprenolol (0.2 mg/kg) in a 10-minute period were measured, and are given as mean ±SE. Significant decreases in heart rate (85 ± 7 to 75 ± 6 beats/min), aortic systolic pressure (131 ± 5 to 123 ± 5 mm Hg), cardiac index (2.3 ± 0.2 to 1.9 ± 0.1 l/min/m²), and left ventricular work (8.58 ± 1.10 to 7.17 ± 0.86 kg-m/min) were found. Mean right atrial (7 ± 1 to 10 ± 1 mm Hg) and pulmonary arterial (30 ± 4 to 33 ± 4 mm Hg) pressure rose. No significant change in aortic mean or diastolic pressure, left ventricular end-diastolic pressure, stroke index, or systemic or pulmonary vascular resistance occurred.

The negative chronotropic effect of alprenolol is again demonstrated. The decline in cardiac index was in large part related to the slowing of the heart rate, as indicated by the lack of a significant change in stroke index. In a previous study of five healthy volunteers, administration of 10 mg alprenolol produced no significant change in heart rate, arterial pressure, cardiac output, or right atrial pressure. These findings were attributed to the mild intrinsic beta-stimulating action of alprenolol, in contrast to the results of the present study, which involved patients who were generally in functional classes 3 and 4 (New York Heart Association). In the latter clinical setting, the mild sympathomimetic effects of alprenolol would appear inadequate to compensate for the hemodynamic consequences of the beta-adrenergic blockade.

Finally, the hemodynamic effects of alprenolol in the dose used in this study and propranolol in patients with heart disease in other studies are compared; it is demonstrated that the two agents are essentially similar. (*Kerber, R. E., and others: Circulatory Responses to Beta Adrenergic Blockade with Alprenolol, Am. J. Cardiol. 29: 26–32, 1972.*) ABSTRACTER'S NOTE: The increase in pulmonary arterial pressure is said to be statistically significant; however, the figures published do not make this significance apparent.

ETIOLOGY OF CONGENITAL HEART DISEASE From a prospective study of 56,109 births some incidence factors and etiologic correlates of congenital heart disease have been identified. The collaborative study involved many examiners, at 12 institutions.