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Drugs and Their Actions

DIGITALIS AND PULMONARY VASCULATURE The effects of acute digitalization on pulmonary blood volume (PBV) and pulmonary vascular distending pressure (P_d) were studied in 23 adults during simultaneous right and left heart catheterization. Digitalization with acetylstrophanthidin (1.1-1.3 mg intravenously) was achieved over a 9-13-minute period. The subjects were divided into two groups according to the responses of left ventricular end-diastolic pressure (LVEDP) to digitalization. Group I (11 patients in whom digitalization reduced LVEDP 5 mm Hg or more) included five patients with ischemic heart disease, five with aortic valve disease (including two with aortic insufficiency), and one with mitral stenosis. Group II (12 patients in whom digitalis did not reduce LVEDP by more than 1 mm Hg) consisted of three patients with ischemic heart disease, six with aortic valve disease (including four with aortic insufficiency), two with mitral stenosis, and one normal patient.

In Group I, digitalis decreased the heart rate by 11 beats/min, increased the cardiac index from 2.39 ± 0.19 to 2.60 ± 0.19 l/min/m², and decreased LVEDP from 23.6 ± 3.2 mm Hg to 12.1 ± 2.7 mm Hg. Left atrial mean pressure dropped from 24.6 ± 1.7 to 13.1 ± 2.1 mm Hg, while mean pulmonary arterial pressure dropped from 28.1 ± 2.3 to 18.5 ± 2.3 mm Hg. The calculated pulmonary vascular resistance did not change (control, 123 dyne-sec cm⁻²; after digitalization, 120 dyne-sec cm⁻²). A decrease in the pulmonary distending pressure P_d from 27.8 ± 2.2 to 16.8 ± 2.3 mm Hg was observed, and was the result of the decline in both LA and PA mean pressures, since P_d is calculated by averaging mean LA and PA pressures.

Pulmonary blood volume, calculated by multiplying mean transit time from PA to LA by the cardiac index, decreased from 317 ± 27.6 to 262 ± 23.0 ml/m² after digitalization. A graph of pulmonary blood volume vs. pulmonary distending pres-

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sure indicated that the directions of decrease in PBV and in P_a coincided in all 11 cases, although there was considerable variation in the magnitude of these changes.

In Group II, although there was a significant decrease in heart rate and a significant increase in cardiac index, LVEDP did not change significantly. No significant changes were found in LA or PA mean pressures, in P_a , or in pulmonary blood volume. Hence, if digitalization has no effect on left ventricular dynamics, it apparently has no significant effect on the pulmonary vessels. In Group II—as in Group I—calculated pulmonary vascular resistance was unchanged.

Controls: In eight additional patients the injection of saline solution instead of acetylstrophanthidin resulted in no appreciable hemodynamic changes. (Murphy, G. W., and others: *Effects of Acute Digitalization on the Pulmonary Blood Volume in Patients with Heart Disease, Circulation* 43: 145 (Jan.) 1971.)

ERRON'S COMMENT: The authors note that pulmonary vascular resistance may not accurately reflect the vasoactive effects of drugs, since resistance measurements are affected by pulmonary arterial, left atrial, and airway pressures, and also by changes in cardiac index. In their Group II patients who showed no significant changes in cardiac output and LA and PA pressures after digitalization, there was no change in calculated pulmonary arterial resistance. In the Group I patients in whom changes in the hemodynamics of the left heart and pulmonary vessels did occur following digitalization, the pulmonary distending pressure (P_a) was decreased consistently (9/9), whereas the changes in pulmonary vascular resistance were inconsistent (four increased, seven decreased) and varied from as little as 16 to as much as 162 dyne-sec cm^{-2} . One wishes that the authors had discussed their pulmonary vascular resistance data in more detail, since a drop in P_a would result from any decrease in left atrial pressure.