Literature Briefs

John W. Pender, M.D., Editor

Briefs were submitted by Drs. John Adriani, C. M. Ballinger, Norman Bergman, M. T. Clarke, H. S. Davis, Martin Helrich, J. J. Jacoby, F. C. McPartland, S. J. Martin, Harold Nimer, S. R. Oech, R. E. Ponath, Alan Randall, Wallace Ring, H. S. Rottenstein, P. H. Sechzer, and Lynn Winchester. Briefs appearing elsewhere in this issue are a part of this column.

The Briefs of Russian Literature were taken from Excerpta Medica's "Abstracts of Soviet Medicine" which is supplied through the Public Health Service of the National Institutes of Health.

BAINBRIDGE REFLEX The change in heart rate produced by intravenous infusions of blood or saline is related to the initial heart rate before the infusion. In both the cat and dog there is a significant negative correlation between the initial heart rate and the proportion of experiments in which tachycardia occurred. Intravenous infusion usually increases the pulmonary arterial pressure, left atrial pressure and mean arterial blood pressure. However, it was not possible to relate the change in heart rate to any particular change in these pressures or to the character or volume of the fluid injected. It is suggested that intravenous infusions tend to change the heart rate to its optimal value for increasing the cardiac output and therefore restoring the venous pressure to its initial level. (Jones, J. J.: The Bainbridge Reflex, J. Physiol. 160: 298 (Feb.) 1962.)

CARDIAC MASSAGE A blood pressure cuff inflated to 90–100 mm. of mercury will record a brisk deflection of the needle during any effective cardiac compression whether open or closed massage is used. This method is more constant, does not require a second person and is not invalidated by heaving during closed chest massage. (O'Hara, V. S.: Assessing the Efficacy of Cardiac Massage, New Engl. J. Med. 266: 507 (Mar. 8) 1962.)

CARDIAC RESUSCITATION Attempts at conversion of chronic atrial fibrillation by means of quinidine in two patients with severe rheumatic heart disease led to toxicity characterized by repeated attacks of ventricular flutter and ventricular fibrillation. serious arrhythmias were controlled by external cardiac massage and external electrical defibrillation plus artificial respiration. One of the patients, who had 14 such episodes over a 14-hour period, ultimately recovered with persistent atrial fibrillation; the other patient, with 90 such episodes over a 28hour period, died in spite of control of the ventricular arrhythmias. (Rainer-Pope, C. R., and others: Treatment of Quinidine-Induced Ventricular Fibrillation by Closed Chest Resuscitation and External Defibrillation, Amer. Heart J. 63: 582 (May) 1962.)

EXTERNAL CARDIAC SHOCK External electric countershock has been of value in terminating supraventicular and ventricular tachycardia and atrial fibrillation. The technique is indicated when the patient's condition is desperate or intolerable and drug therapy is ineffective, too slow, or otherwise inadvisable. General anesthesia is necessary when the patient is conscious. There is a definite risk: ventricular fibrillation or standstill may follow. To prevent recurrent tachycardia, antiarrhythmic drugs, such as intravenous procaine amide, may be given concomitantly. (Zoll, P. M., and Linenthal, A. J.: Termination of Refractory Tachycardia by External Countershock, Circulation 25: 596 (Apr.) 1962.)

ATRIAL FIBRILLATION Atrial fibrillation with a rapid rate produces a reduction in cardiac output, coronary flow, cerebral, renal and mesenteric flows. This may, in spite of adequate digitilization and a slowed ventricular rate, prevent full cardiac compensation. Symptoms related to one or more of the systems with reduced flows may persist. Corrected.