Closed-Chest Cardiac Massage, J.A.M.A. 193: 681 (Aug. 23) 1965.)

CARDIOVERSION Problems of electroconversion include several of direct concern to the anesthesiologist in addition to the obvious calculated risk of a short intravenous anesthetic in the presence of significant cardiac disease. Among these are unrecognized postshock hypoventilation from the anesthetic or premedication, synergy between muscle relaxants and concurrent quinidine therapy, and electrical burns due to arcing between defibrillator electrodes and monitoring electrodes which have been placed too close to each other. The advantages of transient narcosis, as compared to shocking heavily premedicated conscious subjects, outweigh the disadvantages, and the contribution of adequate ventilation supervised by an anesthesiologist is acknowledged. *(Paulk, E. A., and Hurst, J. W.: Clinical Problems of Cardioversion, Amer. Heart J. 70: 248 (Aug.) 1965.)*

COUNTERSHOCK Patients with mitral valvular disease and atrial fibrillation were restored to normal rhythm after external direct-current countershock. Systolic and mean pressures in the right and left ventricles and in the pulmonary and brachial arteries were not systematically altered, with a tendency, however, to a decrease in ejection pressures in the right heart and increase in the left heart. Mean right atrial and right ventricular end-diastolic pressures as well as left ventricular end-diastolic pressure decreased significantly. Cardiac output and index and stroke volume and index increased; pulmonary vascular and systemic resistances decreased. *(Reale, A.: Acute Effects of Countershock Conversion of Atrial Fibrillation Upon Right and Left Heart Hemodynamics, Circulation 32: 214 (Aug.) 1965.)*

ATRIAL SYSTOLE A properly timed atrial contraction results in a significant improvement in cardiac function in patients with heart block. There is not only augmented ejection time, mechanical systole, systolic pressure, isometric contraction time, tension time index and peak derivative of brachial pressure but also a significant decrease in the mean rate of systolic ejection. Although the contribution of atrial contraction on cardiac function occurs at all ranges of ventricular rates, its influence is greatest in the 50 to 80 beats per minute range. The optimal P-R interval is 1 to 300 milliseconds. *(Benchimol, A., and others: Contribution of Atrial Systole to the Cardiac Function at a Fixed and at a Variable Ventricular Rate, Amer. J. Cardiol. 16: 17 (July) 1965.)*

ATRIAL CONTRIBUTION The relationship between heart rate, synchronous and asynchronous atrial-ventricular activity and cardiac output was explored in patients with complete heart block. Studies were made of ventricular or His-bundle pacing at controlled rate, right ventricular outflow tract catheter electrode pacing, and both synchronous and atrial synchronous pacing. Thirty-four paired observations were made during equal atrial and ventricular pacing rates. The cardiac index is significantly decreased during ventricular as opposed to atrial pacing at the same heart rate. The average cardiac index during ventricular pacing was 2.63 liters/minute/m.², and during synchronous or atrial synchronous pacing the average was 2.90 liters/minute/m.². The contribution of atrial systole to the maintenance of cardiac output is demonstrated. *(Samet, P., and others: Atrial Contribution to Output in Complete Heart Block, Amer. J. Cardiol. 16: 1 (July) 1965.)*

ATRIAL TRANSPORT The quantity of ventricular filling and output is determined chiefly by the length of diastole, but the state of the myocardium and the temporal relationship between atrial and ventricular systoles are also important factors. Atrial contraction occurring after ventricular contraction and closure of the A-V valves is ineffective because at this time the ventricle is a closed cavity and there is merely a tendency to cause backflow and congestion of the systemic and the pulmonary veins. Normally, the piston-like downward movement of the A-V junction during ventricular systole enlarges the atrium and the vena cava. Whereas the grossly normal heart can compensate for loss of atrial contraction, the severely damaged heart may depend on atrial contraction to obtain good end-diastolic