venous cannulae. With the availability of simple heart-lung machines, which may be primed with crystalloid solutions, it may be feasible to use extracorporeal circulation as an adjunct to cardiac resuscitation in a variety of clinical circumstances. (*Joseph, W. L., and Maloney, J. V.: Extracorporeal Circulation as an Adjunct to Resuscitation of the Heart, J.A.M.A. 193: 683 (Aug. 23) 1965.*)

RESUSCITATION Cessation of breathing movements and absence of pulse and heart sounds no longer can be considered signs of death. Death should be defined as evidence of irreversible cerebral destruction with unconsciousness. So, when a patient is found in acute respiratory or circulatory distress and he is not known to be in the terminal stages of an incurable disease, he should be considered salvable and treated promptly with a complete resuscitative effort. After the base line of cerebral oxygenation has been established, the situation should be assessed. Unnecessary prolongation of the act of dying should be avoided if the underlying disorder appears incompatible with survival or if there is clear-cut evidence that the hypoxic episode produced irreversible cerebral damage. (*Safar, P.: Cardiopulmonary Resuscitation Postgrad. Med. 38: 7 (July) 1965.*)

EXTERNAL CARDIAC RESUSCITATION External cardiac massage combined with artificial ventilation appears to be the method of choice for the management of cardiac arrest or ventricular fibrillation. Direct open cardiac massage would appear indicated where the chest is already open or where cardiac tamponade is suspected. It is well to bear in mind that the thoracic cage in a recently dead person is quite pliable, being completly relaxed and non-resistant. Fractures of the sternum produce instability of the chest, which may require tracheotomy and assisted ventilation. Too rigorous compression should likewise be avoided to prevent rupture of a dilated, diseased heart, or of the upper abdominal viscera. Intravenous or intracardiac administration of epinephrine hydrochloride (1 or 2 ml. of 1:1,000 or 1:10,000) is helpful for its cardiac stimulatory effect and also for its intense vasoconstrictive effects upon less vital circulatory areas. Once a spontaneous heart beat is restored, isoproterenol hydrochloride (1:50,000 intravenously in 0.5–2 ml. doses, depending on patient size) and sodium bicarbonate (2–4 mEq./kg., intravenously) or tromethamine are useful adjuncts. (*Lillehei, C. W., and others: Four Years' Experience With External Cardiac Resuscitation, J.A.M.A. 193: 651 (Aug. 23) 1965.*)

CARDIAC MASSAGE The overall results of cardiac resuscitation in 128 patients are summarized. This includes all patients for whom the cardiac resuscitation team was called because of sudden circulatory arrest manifest as loss of pulses and heart sounds and diagnosed by a physician. Instances of cardiac arrest occurring in the operating rooms have been excluded. Most of the patients were between 50 and 80 years of age, but survival was more common among those under 50. Of the 128 patients, 38, or 30 per cent, survived for more than 24 hours, and 23, or 18 per cent, were ultimately discharged from the hospital. Ventricular fibrillation was recorded in 44 of the patients and included 13 of those surviving. Asystole was diagnosed in 54 patients, and 7 of these survived. Metabolic acidosis and raised arterial-lactate levels were found in most patients. Correction of the acidosis with intravenous sodium bicarbonate was found beneficial. (*Smith, H. J., and Anthonisen, N. R.: Results of Cardiac Resuscitation in 254 Patients, Lancet 1: 1027 (May 15) 1965.*)

MASSAGE COMPLICATIONS Closed-chest cardiac massage is not without complications. Most of these are rib or sternal fractures, but bone marrow emboli, hemothorax, hemopericardium, pneumothorax, mediastinal emphysema, gastric rupture, and lacerations of spleen, liver, and blood vessels also occur. A case of aortic rupture secondary to sternal fracture emphasizes that extreme caution should be used to prevent overcompression of the sternum. There is a tendency to compress the sternum more than necessary during the emergency, and the risks are further compounded in the treatment of an elderly patient with a fixed chest. (*Nelson, D. A., and Ashley, P. F.: Rupture of the Aorta During*

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: 11 (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 control 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 relation 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