

# Literature Briefs

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

**VENTRICULAR FIBRILLATION** The etiology of ventricular fibrillation during hypothermia was studied in dogs, cats, rabbits and rats anesthetized with pentobarbital, cooled by ice bath and artificially respired. Myocardial threshold, conduction time and refractory period were determined at 37° C., 30° C., 25° C. and 20° C. The findings of (1) a relatively greater increase in conduction time over increase in refractory period, below 30° C.; (2) the absence of ventricular fibrillation in the smaller animals with physiologically short conduction pathways as compared to the larger animals; (3) the prevention of fibrillation by anatomically shortening the conduction pathway through surgical incision or increasing the conduction velocity pharmacologically with certain sympathomimetic amines; and (4) the absence of demonstrable reduction in myocardial threshold during hypothermia favors the "circus movement" theory of etiology over the "ectopic focus" theory. (Covino, B. G., and D'Amato, H. E.: *Mechanism of Ventricular Fibrillation in Hypothermia, Circulat. Res.* 10: 148 (Feb.) 1962.)

**CARDIAC MASSAGE** Three-fourths of the children in this series showed traumatic injury following direct cardiac massage. The epicardium was diffusely ecchymotic and the ventricular myocardium had numerous gross hemorrhages. Microscopic examination revealed interstitial hemorrhages with fragmentation and disruption of myocardial fibers. The closed chest method of cardiac massage ap-

peared less traumatic when cardiac arrest occurred in infants and children. (Guevara, U., and others: *Traumatic Damage to the Heart from Cardiac Massage, Surgery* 51: 211 (Feb.) 1962.)

**CARDIAC ARREST** Incidence of anesthesia-related cardiac standstill declined from 1 in 1,062 in 1950 to 1 in 3,774 in 1959 as revealed by an analysis of 164 operating room-related cardiac standstills in a series of 118,552 patients. The incidence of total cardiac standstills did not vary significantly during this interval. In evaluating beneficial results, particular emphasis is placed on: (1) preoperative preparation of the patient, (2) value of teamwork, and (3) the constant clinical acumen of the anesthesiologist. (Stephen, C. R.: *Cardiac Arrest on the Decline, Ann. Surg.* 155: 345 (Mar.) 1962.)

**CARDIAC ARREST** Cardiac arrest occurring during the rapid administration of acidotic bank blood is not primarily related to alteration of the hydrogen ion concentration of the blood perfusing the heart. Rather it is due to the hyperkalemia associated with acidosis. (Le Veen, H. H., and others: *Role of pH in Myocardial Contractility, Surgery* 51: 360 (Mar.) 1962.)

**RESPIRATION DURING ASYSTOLE** In dogs whose hearts were driven by an artificial pacemaker, asystole was produced for 20 seconds and the effects on respiratory mechanics noted. There resulted a tachypnea, increased tidal volume, reduced functional residual capacity, reduced compliance, increased airway resistance, reduced end-tidal  $P_{CO_2}$ , and an increase in respiratory work. All of these changes were reversed in 30-60 seconds after restoration of the cardiac output. It was postulated that asystole resulted in immediate central pooling of blood, pulmonary congestion