

# Literature Briefs

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Briefs were submitted by Drs. John Adriani, C. M. Ballinger, Peter P. Bosomworth, M. T. Clarke, H. S. Davis, Deryck Duncalf, J. E. Eckenhoff, Martin Helrich, G. Hohmann, J. J. Jacoby, F. C. McPartland, A. S. Paterson, R. E. Ponath, W. H. Ring, Norman Rosenbaum, H. S. Rottenstein, P. H. Sechzer, B. L. Vandermeer. Briefs appearing elsewhere in this issue are a part of this column.

**EXTERNAL CARDIAC MASSAGE** Patients treated for cardiac arrest over a two year period were classified according to the type resuscitative procedure employed: (1) closed-chest massage (2) open thoracotomy with massage, and (3) a combined closed and open massage. The survival rate in the operating room when the primary therapy was closed-chest massage was 90 per cent. However, when subsequent open-chest massage was required, this survival rate dropped to 35 per cent. The overall survival rate of patients treated in the operating room was 52 per cent. Three out of 19 patients treated outside of the operating room with closed-chest massage survived. (Coitlar, A. M., and others: *Increased Survival from Cardiac Arrest Since the Introduction of External Cardiac Massage, Dis. Chest* 44: 400 (Oct.) 1963.)

**EXTERNAL CARDIAC MASSAGE** Experiments were performed on 15 infant and child cadavers to determine the mechanism of liver rupture with closed-chest cardiac massage. Simultaneous compression of the chest and abdomen limiting the free movement of the liver accounted for its rupture. The ventricular surface of the heart underlies the middle third of the sternum in infants and young children and gradually descends during growth to a position behind the lower third of the sternum in adults. Compression experiments in 20 fresh, heparinized cadavers of infants and young children showed that effective circula-

tory pressures were produced by compression of the middle rather than the lower sternum. The technique of external cardiac compression suggested for infants and young children consisted of compression of the midsternum with superimposed thumbs while the fingers were linked behind the patient for additional support. (Thaler, M. M., and Stobie, G. H. C: *An Improved Technic of External Cardiac Compression in Infants and Young Children, New Engl. J. Med.* 269: 606 (Sept. 19) 1963.)

**CARDIAC MASSAGE** Incidence of pulmonary bone marrow emboli was studied in routine autopsies of patients on whom closed-chest cardiac massage had been attempted before death. Eleven such patients were investigated. Pulmonary bone marrow emboli were found in 10 of the 11 patients with 9 of 11 having a free-fat pulmonary embolization. (Yanoff, M.: *Incidence of Bone-Marrow Embolism Due to Closed-Chest Cardiac Massage, New Engl. J. Med.* 269: 837 (Oct. 17) 1963.)

**EXTERNAL CARDIAC MASSAGE** Observations were reviewed on 50 consecutive autopsies of patients who died after external massage during a 10 month period when there were 14,527 hospital admissions. Pre-existing cardiac disorders had been present in 72 per cent of the patients before cardiac arrest. Injuries resulting from the massage were ribs 14, sternum 1, heart 12, liver 3 and spleen 1. Pulmonary bone marrow emboli were present in peripheral regions of the lungs in 6 cases. (Bynum, W. R., Connell, R. M., and Hawk, W. A.: *Causes of Death After External Cardiac Massage, Cleveland Clin. Quart.* 30: 147 (July) 1963.)

**CARDIOVERSION** The patient in complete heart block who presents himself for cardioversion presents a special problem to the anesthetist. When normal rhythm exists,

a thiopental-nitrous oxide-oxygen-halothane sequence is simple and safe. However, during heart block, care must be taken during intravenous induction of anesthesia; and nitrous oxide and oxygen alone is safer, with a succinylcholine drip to provide relaxation and apnea. After pacing is satisfactory, halothane may be used safely in low concentration. (Howat, D. D. C.: *Anaesthesia for the Insertion of Indwelling Artificial Pacemakers*, *Lancet* 1: 855 (Apr. 20) 1963.)

**CARDIAC DENERVATION** Total extrinsic cardiac denervation was carried out on dogs. Their responses to hypoxia and hypercapnia were then tested and compared to those of normal dogs. Both groups had similar responses to hypercapnia: reduced blood pressure, heart rate and myocardial contractile force. During hypoxia the control dogs had the usual changes: elevated blood pressure, heart rate, and myocardial contractile force. The animals with cardiac denervation, however, had minimal responses to hypoxia. (Greenfield, L. J., and Ebert, P. A.: *Cardiac Denervation Effect in Hypoxia and Hypercapnia*, *Arch. Surg.* 87: 717 (Nov.) 1963.)

**CARDIAC SYMPATHETICS** The isolated heart can synthesize norepinephrine and the enzyme O-methyl transferase can inactivate norepinephrine. Norepinephrine at the sympathetic nerve ending is not in a homogeneous store but is partitioned into several pools or functional compartments. Guanethidine is able to block the adrenergic neuro-effector junction and is not dependent on the depletion of tissue norepinephrine stores, while these stores have to be lowered markedly in order for reserpine to block the effects of stimulating sympathetic nerves. Guanethidine releases free norepinephrine into the coronary venous blood, but its norepinephrine-depleting action is not dependent on this property. Reserpine, on the other hand, lowers tissue norepinephrine stores more rapidly than does guanethidine but does not release norepinephrine, as the free amine, into the circulation. (Braunwald, E., and others: *Studies on the Function of the Adrenergic Nerve Endings in the Heart*, *Circulation* 28: 958 (Nov.) 1963.)

**CARDIAC SYMPATHETICS** Norepinephrine is formed continuously regardless of sympathetic tone and is stored inside a lipid membrane. The amine is present in two pools—a reserve pool in granules in equilibrium with a mobile pool which is maintained against a concentration gradient by active transport. Monamine oxidase (MAO) controls the amount of norepinephrine in the nerve endings so that at the steady-state level the amine does not freely diffuse onto receptor sites. In the absence of sympathetic tone, norepinephrine can leave the storage compartments by simple diffusion through the lipid membrane onto the receptor sites and reaches the blood stream in the form of bases. (Brodie B.: *Recent Views on Mechanisms for Lowering Sympathetic Tone*, *Circulation* 28: 970 (Nov.) 1963.)

**HYPOTENSION** Circulatory reflexes of man have been investigated by standard procedures, such as intra-arterial measurement of pulse pressure curves, tilt table, and the Valsalva maneuver. These reflexes were found to be absent in neuritis due to alcohol, porphyria, and infective polyneuritis. Acute loss of circulatory reflexes was found in alcoholic intoxication and in poisoning due to barbiturates and drugs used in psychotherapy. Cerebrovascular accidents also caused acute interruption of the reflex pathways. Severe hypotension in the supine position was usual in the acutely ill group, was precipitated in chronic neuritis by minor decreases in blood volume, artificial respiration and therapeutic doses of hypnotics or drugs used for psychotherapy. (Barraclough, M. A., and Sharpey-Schafer, E. P.: *Hypotension from Absent Circulatory Reflexes*, *Lancet* 1: 1121 (May 25) 1963.)

**NERVOUS SYSTEM AND SHOCK** There are predictable effects at various sites of the central nervous system in "reversible" and "irreversible shock." There is a roughly linear depression on both monosynaptic and multisynaptic spinal reflex responses with graded hemorrhagic hypotension such that clear reduction of evoked responses is evident at 50 mm. mean arterial pressure and nearly complete loss of excitability at 30 mm. of mercury. This depression is reversed by reinfu-