Literature Briefs

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Briefs were submitted by: Drs. John Adriani, Norman Bergman, Peter P. Bosomworth, M. T. Clarke, H. S. Davis, John Hakes, Martin Helrich, J. J. Jacoby, F. C. McPartland, S. J. Martin, S. R. Oech, Alan Peterson, R. E. Ponath, Alan D. Randall, Wallace Ring, and H. S. Rottenstein. Briefs appearing elsewhere in this issue are a part of this column.

CARDIAC ARREST The most important source of energy for heart muscle is from phosphate bonds. The nucleotides provide this store of chemical energy, mainly as the adenosine phosphates. During arrest of the heart the level of adenosine triphosphate declines, while that of the monophosphate and diphosphate increases. During bypass, with or without cardiac arrest, some of the adenosine moiety is lost. Hypothermia minimizes the unfavorable changes. Biochemical recovery is slower than mechanical recovery; the chemistry of the heart is still abnormal a half hour after resumption of apparently normal contractions. (*Burdette, W. J., and Al-Shamma, A.: Changes in High-Energy Phosphates During Cardiac Arrest, A.M.A. Arch. Surg. 85: 4 (July) 1962.*)

CARDIAC ARREST Closed-chest massage and defibrillation were used successfully in the management of six acute cardiac emergencies occurring in a cardiac catheterization laboratory. Ventricular fibrillation was the most frequent emergency. The resilience of the rib cage in the infant permitted adequate arterial blood pressure to be obtained with relatively gentle manual compression. (*Dawson, B., and others: Closed-Chest Resuscitation in a Cardiac Catheterization Laboratory, Circulation 25: 976 (June) 1962.*)

CARDIAC EMERGENCIES A number of cardiac emergencies are discussed by a group of panelists. Therapy for sudden onset of auricular fibrillation with a fast ventricular rate is first digitilization followed by the use of quinidine, if necessary. Atropine 0.5 mg. should be given intravenously to block any anticipated vago-vagal response. Necessity for the repeated administration of atropine throughout extended surgery is emphasized. Certain patients seemed to be more obvious candidates for cardiac arrest, the anuric patient and the patient with obstructive jaundice. (*Russek, H. I., and others: Management of Cardiac Emergencies, Dis. Chest 41: 357 (Apr.) 1962.*)

CARDIAC OUTPUT During voluntary hyperventilation cardiac output increased by an average of 41 ml. of blood per liter-increase in ventilation. Increased cardiac output was maintained during sustained periods of hyperventilation up to 75 seconds in duration and occurred in both sitting and supine positions. Increased heart rate, rather than stroke volume, raised cardiac output during hyperventilation. Simulation of observed intrathoracic pressure changes during hyperventilation by imposition of inspiratory and expiratory resistances during normal ventilation failed to produce consistent changes in cardiac output. Increased cardiac output during hyperventilation may be, in part, caused by increased work of breathing but is not related to mechanical effects of intrathoracic pressure on venous return. (*Doneven, R. E., and others: Influence of Voluntary Hyperventilation on Cardiac Output, J. Appl. Physiol. 17: 487 (May) 1962.*)

CARDIAC OUTPUT In the closed chest, the venous return to the right heart is greatly enhanced by the emptying of blood from the extrathoracic veins into the thoracic veins during spontaneous respiratory movements in the presence of a negative intrathoracic pressure. When the chest is open, the respiratory movements and negative intrathoracic pressure are no longer present. The reduction in venous return is not due to an increased pulmonary