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

vascular resistance. When the work of the right side of the heart was taken over by a mechanical pump and a moderate negative pressure was applied to the intrathoracic veins, the cardiac output returned to normal. (*Caldini, R. C., and others: Effect of Thoracotomy on Cardiac Output and Pulmonary Hemodynamics in Dogs, J. Thor. Cardiov. Surg. 44: 104 (July) 1962.*)

VENOUS PRESSURE Effect of transfusion and hemorrhage on venous pressure was made in 22 dogs. Transfusions of 1 ml./kg./minute for 60 minutes were well tolerated in the group of animals not manifesting transfusion reactions. Elevations in inferior caval and portal pressures were 3.5 and 6.3 cm. of water after 30 minutes and 5.9 and 14 cm. of water after an hour. The hematocrit value rose 11 per cent during the transfusions. Of the 11 animals, eight died after removal of a volume of blood equal to that transfused. Systemic arterial blood pressure was diminished earlier and more significantly than the venous pressure in animals undergoing hemorrhage at 0.5 ml./kg./minute. There was no support for the contention that venous pressure appeared a poor indicator of volumetric overtransfusion; it may well be a valuable indicator of physiologic overtransfusion. (*DePena, B., and Dobell, A. R. C.: Venous Pressure Variations with Transfusion and Hemorrhage in Experimental Animals, Brit. J. Surg. 49: 449 (Jan.) 1962.*)

POSTURE The effects of posture and distribution of blood volume on cardiac output, stroke volume, and instantaneous pulmonary capillary blood flow were studied in normal subjects by modification of the nitrous oxide technique. The changes in cardiac output and stroke volume that ordinarily accompany changes in posture can be prevented if shifts in blood volume are prevented. The results support the contention that the thoracic blood volume is an important determinant of the stroke output of the heart in normal resting man. Blood flow through the pulmonary capillaries is pulsatile in both the upright and horizontal postures. The amplitude of the pulsations is increased in the horizontal posture. This increase is related to coincident changes in stroke volume and not to the posture itself. (*Naimark, A., and Wasserman, K.: Effects of Posture on Pulmonary Capillary Blood Flow in Man, J. Clin. Invest. 41: 949 (May) 1962.*)

MYOCARDIAL METABOLISM In dogs continuous oxygenation of the fibrillating heart protected it from significant metabolic disturbance and impaired function. Ischemic fibrillation depressed left ventricular function and resulted in serious metabolic aberrations. Thus normothermic, electrically-induced, ventricular fibrillation accompanied by continuous oxygenation fulfilled the requirements of an ideal method of cardioplegia. (*Stoney, R. J., and others: Myocardial Metabolism and Ventricular Function Before and After Induced Ventricular Fibrillation, Surgery 52: 37 (July) 1962.*)

HYPOTHERMIC HEART Review of available experimental data on work performance of the hypothermic heart indicates that cooling progressively reduces the work capacity of the heart through the bradycardia resulting from direct cold depression of the pacemaker. The stroke work capacity is unchanged by cooling to 25°–28° C. and myocardial contractility improves. The total work capacity of the isolated heart (heart-lung preparation) increases with moderate hypothermia. However, this is because isolated hearts tend to fail spontaneously and cold, by reducing myocardial metabolic rate, delays this failure and thereby increases the total work performance during the failure period. From a clinical standpoint, the most important conclusion is that the hypothermic heart has a reduced minute-work capacity and should not be overloaded as to either output or arterial pressure (*Badeer, H. S.: Work Capacity of the Hypothermic Heart, Amer. Heart J. 63: 839 (June) 1962.*)

COOLING IN INFARCTION To test the hypothesis that reduction of body oxygen requirements by induced hypothermia might be beneficial in managing recent myocardial infarction with intractable shock, 32 anesthetized dogs were subjected to acute myocardial infarction by means of plastic-sphere coronary embolization and then cooled to varying tem-