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-