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-

peratures. Twenty dogs were cooled to the point of circulatory arrest or lower (5°–19° C.) and were then maintained on extracorporeal circulation for from two to three hours. Twelve dogs were cooled moderately (28°–30° C.) for from four to six hours. All animals were then rewarmed. A small group of controls were similarly infarcted but not cooled. The results of electrocardiographic, hemodynamic, blood gas, acid-base, and electrolyte studies on these animals lend some support to the protective concept. Upon rewarming, cooled animals showed more adequate hemodynamic recovery than normothermic animals. Furthermore, there was no more tendency for hypothermic animals to develop fatal arrhythmias than normothermic. The profoundly cooled group developed a metabolic acidosis and a less adequate return of cardiac output and aortic pressure than those maintained at 28°–30° C. (*Kuhn, L. A., and others: Hemodynamic and Metabolic Effects of Hypothermia and Extracorporeal Circulation in Experimental Myocardial Infarction and Shock, Circulat. Res. 10: 916 (June) 1962.*)

**EXTRACORPOREAL CIRCULATION** Hematologic changes were studied in dogs during and after a ten-hour period of cardiac bypass, employing several different pump oxygenators. Although no significant change in hematocrit occurred during perfusion, severe anemia of two to three weeks duration developed after bypass. Plasma hemoglobin increased linearly with time but had returned to preperfusion levels by the second day. Leukocyte counts fell initially but subsequently rose and exceeded control levels by termination of bypass. Thrombocytes decreased during perfusion, remained low in the immediate postperfusion period, and reached control values by the eighth postperfusion day. Leukocytosis and stabilization of thrombocyte count, correlated with bone marrow changes, suggested the presence of compensatory mechanisms which become operative during cardiac bypass. (*Brinsfield, D. E., and others: Hematologic Changes in Long Term Perfusion, J. Appl. Physiol. 17: 531 (May) 1962.*)

**PLASMA EXPANDER** Rheomacrodex, a low molecular weight dextran, was used to prime the pump in cardiopulmonary bypass. With cardiopulmonary bypass for ninety minutes using whole blood or macromolecular dextran solutions, a severe intravascular aggregation of cells can be detected. This intravascular aggregation can be prevented or reversed in early stages by administration of therapeutic doses of Rheomacrodex. The pathophysiologic revelance of intravascular aggregation has been disputed for years, however, diffuse renal, hepatic, and myocardial microinfarctions in dogs undergoing three hours of total cardiopulmonary bypass were demonstrated and attributed to intravascular aggregation. There is no reason why plasma expanders should not be used as partial blood substitutes in open-heart surgery. No additional bleeding tendency was noted by the administration of micromolecular weight dextran under these circumstances. (*Long, D. M. Jr.: Status of Plasma Expanders in Open Heart Surgery, Dis. Chest, 41: 578 (May) 1962.*)

**PROGNOSIS** Selection of patients with cardiopulmonary insufficiency for chest surgery is often difficult. Pulmonary artery pressure proved to be a more valuable prognostic tool than total and timed vital capacities. Electrocardiograms cannot be relied upon to indicate the presence of pulmonary hypertension, as changes tend to occur late. The mortality of patients with pulmonary arterial pressures of 36 or more is about ten times as great as that of patients with lower pressures. (*Pecora, D. V., and Brook, R.: Evaluation of Cardiopulmonary Reserve in Candidates for Chest Surgery, J. Thor. Cardiovas. Surg. 44: 60 (July) 1962.*)

**POSTOPERATIVE ECG** Electrocardiographic changes following surgery were studied in 220 patients, 190 with cardiac disease and 20 controls. There were three deaths, all in the cardiac group. About one half of the cardiac patients had coronary artery disease. The type of anesthetic was similar in both groups, being predominantly thiopental-nitrous oxide–curare. In the control group arrhythmias were seen in only two instances. In the cardiac series, 72 of the 190, or 38 per pent, had arrhythmias. Sixty per cent of these