rence of large periodic irregular discharges is a poor prognostic sign. Deterioration of cerebral function is indicated by an increase in irregular, slow activity. (*Pampiglione, G.: Electroencephalographic Studies After Cardio-Respiratory Resuscitation, Proc. Roy. Soc. Med.* 55: 25 (*Aug.*) *1962.*)

BLOOD COAGULATION A significant thrombocytopenia often develops during extracorporeal circulation. This may be transitory or may last for about a week, apparently related to the degree of megakaryocyte exhaustion. Clinical manifestations are not seen in most cases. Slight prolongations of the one-stage prothrombin time occur. These are short-lived and unlikely to cause hemorrhage. Of the thromboplastic plasma factors, most attention has been given to the reduction in factor VIII. The more likely mechanisms for its loss are consumption during intravascular clotting and the use of deficient bank blood. Fibrinogen depletion is seldom marked. When severe reductions occur they are usually associated with impaired hemostasis and significant alterations in other coagulation factors. Intravascular clotting or fibrinolysis activation or both are responsible in most cases. Activation of the fibrinolytic system usually can be demonstrated by sensitive tests, but its importance in relation to postoperative hemorrhage is uncertain. The level of plasma hemoglobin provides a rough measure of the degree of traumatization of the blood, generally increasing with duration of bypass. Undesirable consequences of hemolysis are numerous and complex, not the least being disturbances of the coagulation and hemostatic mechanisms. The depletion of clotting factors that may occur during bypass is thought to be due primarily to intravascular clotting, but to some extent may be due to plasma-protein denaturation. The latter process can lead also to widespread damage to the microcirculation of vital organs. (*Kendall, A., and Lowenstein, L.: Alterations in Blood Coagulation and Hemostasis during Extracorporeal Circulation. Canad. Med. Ass. J.* 87: 786 *and* 839 (*Oct. 13 and Oct. 20*) *1962.*)

OXYGEN NEED In aortic-valve surgery under hypothermia, analysis of perfusion records demonstrates the fallacy of basing tissue oxygen requirements on oxygen consumption. The amount of oxygen consumed is known to depend upon the flow rates, the temperature level, the oxygen tension, and the *pH* of the blood. In addition, other factors difficult to specify determine whether complete oxygen protection has been provided. If flow rates lowered to 25 ml. kg. minute to minimize blood trauma lasted for over an hour hypoxic complications ensued. In the presence of an elevated mean arterial pressure, there was a tendency to perfuse at the lower rates. An elevated pressure at 25° C. is not necessarily an index of a satisfactory perfusion. It may indicate a precarious perfusion in the face of undue vasoconstriction and indicate either rewarming to lessen the effect of vasoconstriction or increasing further the flow rate to push through this added vascular resistance. (*Kay, E. B., and others: Operative Results in Aortic Valve Surgery, Circulation 26: 484 (Oct.) 1962.*)

OXYGEN TENSION Oxygen tension of liver, brain, kidney, and muscle decreases at perfusion rates below 60 ml. kg./minute. At normal temperature myocardial oxygen tension falls significantly with decreasing perfusion rates but, at 16° C. it remains at control levels with perfusion rates of 20 ml. kg. minute. Coronary perfusion with cold blood allows myocardial oxygen tension to remain at near normal levels during 30 minutes of arrest at 8° C. Intermittent perfusion should be used if the arrest is prolonged beyond 30 minutes. (*Mahoney, E. B., and others: Tissue Oxygen Tension During Total Body Perfusion and Hypothermic Cardiac Arrest, J. Thor. Surg. 44: 658 (Nov.) 1962.*)

PERIPHERAL RESISTANCE In ten dog-perfusions lasting 30 to 65 minutes with the Gaertner-Kay heart-lung machine, there was an initial increase in peripheral vascular resistance. During the course of perfusion the resistance fell progressively, and this was accompanied by a gradual reduction in arterial oxygen saturation due to an inefficient oxygenator. In seven other perfusions lasting 35 to 50 minutes in which a Melrose machine was used, the arterial oxygen saturation was well maintained throughout the experiments. Again, however, there was an initial rise in

resistance followed by a small fall during the course of the perfusion. In all 17 experiments the final value for peripheral vascular resistance was higher than the estimated value before perfusion began. In eight human perfusions with the Melrose machine, lasting 35 to 97 minutes, a considerable decrease in resistance occurred at the start of perfusion, which was followed by a progressive increase as perfusion continued. In all the patients the final value for peripheral vascular resistance was lower than the estimated value before perfusion began. (*Hunt, R. L.: The Change in Peripheral Vascular Resistance during Total Body Perfusion in Dog and in Man, J. Physiol. 161: 22P (April) 1962.*)

CARDIAC OUTPUT Cardiac difficulties which follow sustained tachycardia are not due to work and power output per stroke, but rather to the results of "fatigue" due to a short diastolic period, that is, a short period for physicochemical recovery. This reduced recovery period becomes even more important in the presence of a diseased myocardium, hypertension, valvular disease, or coronary artery disease. The maximal rate at which an undiseased heart can function well as a pump is about 180 beats per minute. In tachycardia, the necessary chemical exchanges must occur quickly if the needs of the myocardium are to be met. Studies with radioactive elements show that some substances can exchange quite rapidly, the limiting factor being the rate of blood flow. In order to maintain a constant cardiac output at a high cardiac rate, the peak tension necessary to pump blood is much less that at slow rates, but the tension is maintained relatively longer at high rates since diastole is so short. (*Burch, G. E.: Relationship of Heart Rate to Cardiac Output, Work, Power, and Tension in Man, J. A. M. A. 182: 339, (Oct. 27) 1962.*)

VENOMOTOR ACTIVITY Forearm circulation is occluded by rapid inflation of a cuff to a tension well above the subject's systolic pressure. Upon occlusion, pressures in the veins rise for a few seconds before levelling at a height of 10 to 15 mm. of mercury. Subsequent changes are regarded as reflecting changes in venomotor tone. Venomotor activity is increased by many different stimuli including increased respiratory effort (either voluntary or due to carbon dioxide inhalation), performing a Valsalva or Mueller maneuver, positive-pressure breathing, and emotional disturbance. When intrathoracic pressure is increased by positive-pressure breathing or by Valsalva maneuver, there is an appropriate linear relationship between intra-esophageal pressure and the peak pressure reached in the isolated vein. (*Bergel, D. H., and Gould, D. W.: A Method for Studying Reflex Venomotor Changes in Man, J. Physiol. 161: 3P (April) 1962.*)

ARRHYTHMIAS In 146 consecutive surgical repairs of uncomplicated atrial septal defects of the secundum type, the total incidence of postoperative arrhythmias was 43 per cent. Benign arrhythmias (nodal rhythm, temporary A-V dissociation) occurred in about one third of the younger patients with few serious arrhythmias. In the group over 36 years of age, serious arrhythmias (nonparoxysmal supraventricular tachycardias, atrial flutter and atrial fibrillation) were found in the majority of cases (12 out of 17). These latter lasted from a few hours to permanently, most of them for at least several days. Childhood is the best time to close atrial septal defects, since atrial arrhythmias in adults may increase morbidity and convalescence time significantly. (*Papper, R. W., and others: Arrhythmias After Cardiac Surgery, Amer. Heart J. 64: 455 (Oct.) 1962.*)

HYPOXIA The area of the conduction system of the heart is the most heavily vascularized portion of the heart, and hypoxia produces capillary wall changes and increased capillary permeability. Hemorrhage and cardiac arrhythmia often result. Autopsy and experimental evidence often revealed the presence of hemosiderin, suggesting that permanent injury occurs making the conduction system even more susceptible to hypoxia. (*Thung, N., and others: Hypoxia as the Cause of Hemorrhage into the Cardiac Conduction System, Arrhythmia and Sudden Death, J. Thor. Surg. 44: 687 (Nov.) 1962.*)

CORONARY OCCLUSION Male surgical patients over 50 years of age were studied for preoperative coronary occlusions and cere-