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

arrhythmias appeared only postoperatively. A number of tracings indicated changes in conduction. About two thirds of the patients in both the cardiac and control series showed some postoperative electrocardiographic changes. Many of these were minimal ST depression and lowered T wave, which were probably not of great clinical significance. Complete electrographic restitution occurred in 70 per cent of the controls but in only 41 per cent of the cardiac series. Extra systolic arrhythmias were the most common of the observed manifestations occurring after surgery. Persistent tachycardia, congestive heart failure, and angina followed in that order as postoperative cardiac manifestations. There is rather good correlation between the incidence of electrocardiographic changes and the duration of surgery up to three hours. The overall prognosis in cardiac patients undergoing surgery is good; however, cardiac changes do occur, some being readily detectable clinically and some being suggested only by electrocardiographic changes. (*Hurwitz, M. M.: Electrocardiographic Changes Following Surgery, Geriatrics 17: 275 (May) 1962.*)

ENDOTOXIN SHOCK In order to determine the site of the cardiovascular defect in so-called "endotoxin shock," 46 dogs were monitored for cardiovascular responses following the intravenous injection of 5 mg./kg. of fresh *Escherichia coli* endotoxin. One or two minutes following injection of the endotoxin the blood pressure fell to 30–40 mm. of mercury mean pressure, remained so for approximately five minutes, then gradually rose to a pressure of approximately two thirds of the control values. The blood pressure tended to be maintained at this level for some time before it gradually declined and death occurred. During this same period, ventricular contractile force actually increased or remained unchanged as long as arterial blood pressure was maintained; however, it was extremely sensitive to blood pressure falls. Right atrial pressure varied directly as blood pressure until just before death, when there was a transient abrupt rise associated with ECG evidence of a shift to right axis deviation. Cardiac output averaged 30 per cent of control value during the period of partial recovery of blood pressure. Hematocrit tended to rise. Changes in total blood volume were insignificant. The electrocardiogram characteristically showed ST-segment depression. It was concluded that endotoxin shock is the direct result of failure of peripheral vascular resistance and that cardiac failure *per se* is the indirect result of hypotension and insufficient coronary perfusion. (*Alican, F., Dalton, M. L., Jr., and Hardy, J. D.: Experimental Endotoxin Shock, Amer. J. Surg. 104: 702 (June) 1962.*)

SHOCK Administration of catechol amines with endotoxin in normal rabbits produces a greater degree of inflammation and necrosis of cardiac muscle than does that resulting from the use of either alone. Pretreatment with dibenamine prevents or markedly alleviates such damage. The data do not demonstrate that a specific cardiac lesion attributable to endotoxin is a key factor in the development of irreversibility to transfusion in hemorrhagic shock. However, such data are not inconsistent with such a hypothesis. (*Palmero, C., and others: Cardiac Tissue Response to Endotoxin, Proc. Soc. Exp. Biol. Med. 109: 773 (Apr.) 1962.*)

SHOCK No significant improvement in the survival of dogs subjected to irreversible hemorrhagic shock followed a postganglionic mesenteric sympathectomy as compared to a control series. Some hemodynamic changes occurred which simulated those produced in a series of dibenzyline-pretreated animals. (*Berger, R. L., Healey, P. J. M. and Byrne, J. J.: Effect of Postganglionic Mesenteric Sympathectomy in Irreversible Hemorrhagic Shock, Proc. Soc. Exp. Biol. Med. 110: 225 (June) 1962.*)

POSTOPERATIVE CHEST ROENTGENOGRAM The presence or absence of shadows indicative of segmental changes is of major importance. Segmental changes are found in atelectasis and pneumonia but are not present in pulmonary edema or pleural effusion. Pulmonary infarcts may be segmental or of unusual shapes and invariably extend to contact one or more pleural surfaces. The underlying pathophysiology is correlated with configuration of the roentgenographic shadows