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