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

seen in these conditions. (*Mitchell, J. R.: Radiology of Pulmonary Change During the Postoperative Period, Amer. J. Surg. 104: 54 (July) 1962.*)

**HYPOXIA** Oxygen uptake of four normal subjects was studied at several levels of exercise while breathing air and again while breathing a mixture of 11 per cent oxygen in nitrogen. Each subject took up less oxygen while breathing the low-oxygen mixture despite the accomplishment of similar work loads. Greater utilization of anaerobic metabolic sources while hypoxic was excluded as an explanation by measurement of oxygen debt after exercise. Muscular efficiency seems to be enhanced under conditions of induced arterial hypoxia. (*Cronin, R. F. P. and MacIntosh, D. J.: Effect of Induced Hypoxia on Oxygen Uptake During Muscular Exercise, Canad. J. Biochem. Physiol. 40: 717 (June) 1962.*)

**PEAK FLOW RATES** Good correlation exists between the one-second forced expiratory volume and the peak flow rate. The test is easily performed even by young children and provides objective evidence of disease and remission in those patients who cannot perform other tests. For example, the peak flow rate is greatly decreased in asthmatic children. (*Heaf, P. J. D., and Gillam, P. M. S.: Peak Flow Rates in Normal and Asthmatic Children, Brit. Med. J. 1: 1595 (June 9) 1962.*)

**RESPIRATION** Transesophageal electromyographic studies showed that active contraction of the diaphragm is the factor that limits a maximum exhalation. This mechanism prevents marked reductions in lung volume during maximum exhalation. Afferent impulses originating in lung and airway are the most probable stimuli to reflex diaphragmatic contraction. (*Agostoni, E., and Torri, G.: Diaphragm Contraction as a Limiting Factor to Maximum Expiration, J. Appl. Physiol. 17: 427 (May) 1962.*)

**DEAD SPACE** Changing from supine to sitting position increased anatomic dead space and physiologic dead space by corresponding amounts. Alveolar-arterial carbon dioxide gradients and alveolar dead space were unaffected by change in posture. This suggests that the change from supine to sitting caused no significant change in the number of nonperfused alveoli. Changing from air to oxygen breathing, regardless of posture, increased alveolar-arterial carbon dioxide gradients without significant alteration in the other variables. This is interpreted as diversion of blood to dependent portions of the lung, owing to vasodilating properties of oxygen on pulmonary vasculature, leaving non-dependent portions of the lung relatively underperfused. (*Larson, C. P., Jr., and Severinghaus, J. W.: Postural Variations in Dead Space and Carbon Dioxide Gradients Breathing Air and Oxygen, J. Appl. Physiol. 17: 417 (May) 1962.*)

**HIGH PRESSURE OXYGEN** Exposure of asphyxiated rats to 100 per cent oxygen at three atmospheres pressure without hypothermia increased survival times about 55 to 80 per cent over those rats breathing 100 per cent oxygen at atmospheric level. Oxygenation at three atmospheres pressure with hypothermia at 20° C. resulted in maximal prolongation of survival. However, this survival time was not significantly greater than that with hypothermia alone. Carbon dioxide added to the inspired oxygen under pressure gave additional protection even in the absence of hypothermia. The actions of oxygen at high pressure and hypothermia are additive rather than synergistic. (*Levy, J. V., and Richards, V.: Effect of Oxygen at High Pressure on Asphyxial Survival Time of Rats, Proc. Soc. Exp. Biol. Med. 109: 941 (Apr.) 1962.*)

**TRACHEOSTOMY COMPLICATIONS** Complications occurred in eight of 19 emergency tracheostomies, and in 11 of 61 elective tracheostomies, illustrating the value of the early operation. Hemorrhage at the operative site, with aspiration of blood, occurred seven times. Recurrent obstruction occurred four times. Ulcerative tracheobronchitis occurred three times, pneumothorax occurred twice, and tracheo-esophageal fistula occurred twice. Massive gastric distention due to aerophagy occurred twice. Attention is called to the sudden shift that occurs in blood gases when respiratory obstruction is relieved. Because of