tity of precursor. The active fibrinolysin formed destroys the fibrinogen by intravascular hydrolysis and it is then itself rapidly denatured. The lability of the active enzyme makes it difficult to detect "in vitro." Many disease states such as cirrhosis of the liver, advanced carcinoma, benign prostate hypertrophy and infection appear to present their own patterns in the fibrinolytic enzyme system. These changes are probably secondary in nature. (*Phillips, L. L., and Skrodelis, V.: The Fibrinolytic Enzyme System in Normal, Hemorrhagic and Disease States, J. Clin. Invest. 37: 965 (July) 1958.*)

ARTERIOSCLEROSIS In a review of 3,360 autopsies in zoo mammals and 7,660 autopsies in zoo birds over a period of 40 years, arteriosclerosis has increased 10 to 20 fold. The social pressure of an expanding zoo population and also inactivity are considered major factors in this increase. This change has involved 45 families of birds. (*Ratcliffe, H. L., and others: Changing Frequency of Arteriosclerosis in Mammals and Birds at the Philadelphia Zoological Garden, Circulation 18: 41 (July) 1958.*)

MYOCARDIAL INFARCTION Preoperative diagnosis, type and extent of surgery and anesthesia were studied in 35 patients with postoperative myocardial infarction. Predisposing factors include arteriosclerotic and hypertensive cardiovascular disease, polycythemia, and emotional tension and anxiety. Precipitating factors include hypotension, shock (hemorrhagic), arrhythmias and tachycardia with poor coronary filling, respiratory depression and hypoxia, decreased venous return, postoperative absorption of toxic tissue degradation products with changes in blood viscosity, volume and coagulability, trend toward increased blood coagulability on seventh to tenth postoperative days characterized by increased thromboplastin activity and platelet count and increased tolerance to heparin. Differential diagnosis must include electrolyte imbalance, acute cor pulmonale, acute pericarditis, digitalis and quinidine effects, and knowledge that effect of vasopressor drugs can invert T waves. In addition to active treatment of myocardial infarction, prophylactic use of anticoagulants should be used between the third to tenth postoperative day. (*Feruglio, G. M., Bellet, S., and Stone, H.: Postoperative Myocardial Infarction, A. M. A. Arch. Int. Med. 102: 345 (Sept.) 1958.*)

HEART BLOCK Chronotropic responses to *l*-epinephrine and levarterenol in dogs with chronic complete heart block produced surgically, produced a change in ventricular rate of the heart which was inversely related to the resting rate. Levarterenol was superior to *l*-epinephrine as a ventricular cardioaccelerator agent in the unanesthetized and anesthetized animals. It is believed that reflex activity of the A-V nodal pacemakers in some humans with complete heart block may account for differences in dog and man to responses of levarterenol. Pentobarbital transiently increased ventricular rate and persistently enhanced responsiveness to *l*-epinephrine and levarterenol. Hydrocortisone has no acute effect on the ventricular rate. Acidosis of either metabolic or respiratory origin slowed the resting ventricular rate and decreased the sensitivity to *l*-epinephrine and levarterenol. CO_2 decreased ventricular rate more than acidosis induced by hydrochloric acid. Alkalosis of metabolic or respiratory origin increased ventricular rate and enhanced responses to *l*-epinephrine and levarterenol. Prefibrillatory activity of the heart was commonly seen. (*Boyer, S. H., and Chisholm, A. W.: Chronotropic Responses to Sympathomimetic Amines in Experimental Complete Heart Block: The Influence of Pentobarbital, Hydrocortisone and Acid-Base Changes, Bull. Johns Hopkins Hosp. 103: 47 (Aug.) 1958.*)

EPINEPHRINE ESTIMATION As determined by a new sensitive fluorometric method concentrations of epinephrine and norepinephrine in normal human arterial plasma are 0.10 and 0.20 micrograms per liter respectively. The concentration of epinephrine in antecubital venous plasma is lower, and that of norepinephrine higher, than in arterial plasma due to the secretion of the latter from sympathetic nerves in the forearm. (*Price, H. L., and Price, M. L.: The Chemical Estimation of Epinephrine and Norepinephrine*