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*

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*in Human and Canine Plasma, J. Lab. & Clin. Med. 50: 769 (Nov.) 1957.)*

**CATECHOLAMINE METABOLISM** Epinephrine, norepinephrine and 3-Hydroxytryptamine were injected intraperitoneally in rats. Chromatographic examination of a 24-hour urine specimen showed that oxymethylation of the phenolic hydroxide (occupying the meta-position of the catechol nucleus with respect to the side chain) had occurred. In addition, a major portion was conjugated with glucuronic acid. Iproniazid increased the percentage of conjugated catecholamine at the expense of the free oxymethylated metabolite. These observations indicate that oxy-methylation of catechol amines occurs prior to oxidative deamination and that this pathway is a principle route of epinephrine and norepinephrine metabolism. Successive replacement of phenolic hydroxyl groups of sympathomimetic amines is known to shift pharmacologic activity from the peripheral to the central nervous system with concomitant loss of pressor action. Many of the physiological actions of epinephrine and norepinephrine may conceivably be mediated through oxy-methylated metabolites. *(Axelrod, J., Senoh, S., and Witkop, B.: O-methylation of Catecholamines in Vivo, J. Biol. Chem. 233: 697 (Sept.) 1958.)*

**CATECHOLAMINES** The properties are described of an enzyme that can carry out O-methylation of catecholamines by transferring the methyl group of S-adrenosylmethionine to the hydroxyl group in the metaposition of epinephrine and other catechols. The enzyme is in the soluble fraction of liver and other organs. It requires a divalent cation and is inhibited by sulfhydryl binding reagents. *(Axelrod, J., and Tomchick, R.: Enzymatic O-methylation of Epinephrine and Other Catechols, J. Biol. Chem. 233: 702 (Sept.) 1958.)*

**NOREPINEPHRINE** A patient with bulbar paralysis of undetermined etiology required continuous intravenous infusions of norepinephrine in concentrations of 8 to 32 ampules per liter to produce and maintain a systolic blood pressure of 100 mm. of mercury. In spite of the high dosage required for two weeks, the patient eventually made a full recovery without renal complications or gangrene of the extremities. *(Russell, K. P.: Selected Obstetrical Conferences from the Los Angeles County Hospital, West. J. Surg. 66: 240 (July–Aug.) 1958.)*

**ETHER** Administration of diethyl ether to patients for operation resulted in a marked increase in the plasma concentration of a substance resembling noradrenalin. The concentration of adrenalin was only slightly increased. Since bilateral adrenalectomy did not cause the elevated plasma concentration of noradrenaline to return to normal, the increased quantities of circulating noradrenalin likely do not originate in the adrenal medulla. *(Price, H. L.: Circulating Adrenaline and Noradrenaline During Diethyl Ether Anesthesia in Man, Clinical Science 16: 377 (May) 1957.)*

**ALDOSTERONE** Normal man is capable of oxygenating corticosterone to aldosterone. Further study is needed to establish whether or not this conversion occurs within the adrenal cortex. *(Seltzer, H. S., and Clark, D. A.: Evidence for Conversion of Corticosterone to Aldosterone in Man, Proc. Soc. Exper. Biol. & Med. 98: 674 (July) 1958.)*

**ADRENAL HORMONES** The cardiac effects, both functional and morphologic, of adrenal hormones were studied in thirty heart-lung preparations. Measurements were made of cardiac output, outflow pressure and the electrocardiogram. In the control series, cardiac work, output, and rate fell slightly and gradually in the course of the 70-minute experiments. No macroscopic or microscopic alterations were observed in the postmortem examination of these hearts. The infusion of epinephrine or norepinephrine resulted in a marked rise in cardiac work and rate which dropped below their control values after cessation of the infusion. Hydrocortisone alone did not cause any significant alteration in function. However, all the hearts receiving one of the adrenal hormones presented extensive lesions of the myocardium, valves, and coronary vessels. The lesions were more severe in the hearts in which both adrenal hormones were used. *(Nahas, G. G., and others: Functional and Morphologic Changes in Heart-Lung*

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