

tions of Atropine on Cardiac Rhythm, Am. J. M. Sc. 237: 304 (March) 1959.)

DIGITALIZATION Eight human volunteers were studied during rest and exercise before and after intravenous administrations of 1 to 1.2 mg. of digoxin or 1.6 to 1.8 mg. of acetyl strophanthidin. At rest there was a decrease of cardiac output, pulse rate, and stroke volume, and an average rise of blood pressure from 129/73 mm. Hg to 165/86 mm. Hg following digitalization. Similar changes occurred during exercise except that there was no change in blood pressure after digitalization. It was concluded that any augmentation of myocardial contractility resulting from digitalization in resting or exercising normal subjects is overshadowed by the peripheral action of the drug. (*Williams, N. H., Jr., Zohman, L. R., and Ratner, A. C.: Hemodynamic Effects of Cardiac Glycosides on Normal Human Subjects During Rest and Exercise, J. Appl. Physiol. 13: 417 (Nov.) 1958.*)

ISOPROTERENOL Despite mild hyperventilation, intrapleural pressure changes were not significant during intravenous infusion of isoproterenol. Atrial pressure fell regularly as did transmural pressure. Fall in transmural pressure was caused by the fall in atrial pressure because intrapleural pressure was not changed appreciably. Fall in atrial pressure is probably not a ventilatory effect. Forearm venous pressure fell but venous constriction occurred regularly. The large shift of blood from the forearm which occurred was caused primarily by the venous constriction and not by the fall in intraluminal pressure. (*Eckstein, J. W., and Hamilton, W. K.: Effects of Isoproterenol on Peripheral Venous Tone and Transmural Right Atrial Pressure in Man, J. Clin. Invest. 38: 342 (Feb.) 1959.*)

STEROIDS Testosterone, cortisone, desoxycorticosterone, estrone, and progesterone act as haptens when they are conjugated with bovine serum albumin. Antibodies with steroid specificity are formed in rabbits with each of the five steroid-hormone-protein conjugates. (*Beiser, S. B., and others: Antigenicity of Steroid-Protein Conjugates, Science 129: 564 (Feb.) 1959.*)

ASTHMA Chronic bronchial asthma produces a marked increase in the mean airway resistance during periods of acute and chronic respiratory distress. The inspiratory airway resistance may be almost as high as the expiratory resistance. Following therapy, the majority of patients show a greater improvement in the inspiratory resistance than in the expiratory resistance. The so-called "check-value" mechanism of expiration, described in patients with emphysema, may be operative in many patients with bronchial asthma during acute attacks. The compliance or the elastic resistance of the lungs appears to decrease as nonelastic (airway) resistance increases. There is a marked increase in the work of breathing in patients during attacks of bronchial asthma. Such increase is due almost entirely to overcoming resistance. Therapy directed toward decreasing elevated airway resistance is rational and justified. (*Wells, R. E., Jr.: Mechanics of Respiration in Bronchial Asthma, Am. J. Med. 26: 384 (March) 1959.*)

MYASTHENIA Plasma and serum samples from 22 patients with myasthenia gravis were bioassayed by the frog sciatic nerve-sartorius muscle preparation *in vitro*. As compared to the results on controls, 5 of these samples caused a reduction of maximum tetanus tension and 13 produced an appreciable augmentation of the twitch or end tetanus tension. Discussion is presented concerning the possibility of a circulating neuromuscular blocking agent in myasthenia gravis, a method of bioassay and possible properties of such a blocking agent. (*Nastuk, W. A., and others: Search for Neuromuscular Blocking Agent in Blood of Patients with Myasthenia Gravis, Am. J. Med. 26: 394 (March) 1959.*)

MYASTHENIA GRAVIS The defect in myasthenia gravis is probably a defect in muscular transmission, probably due to some alteration in acetylcholine. It is similar in many ways to the block produced by *d*-tubocurarine in normal subjects. While decamethonium and succinylcholine are better tolerated than other muscle relaxants even these drugs may aggravate weakness and should be avoided during anesthesia of myasthenia gravis patients. Procaine and its derivatives have neuromuscular