age of .5 to 1 mg. Because it lacked sedative effect and suppressive action on cough, the intravenous administration of this drug prior to minor surgical procedures and oral endoscopic procedures proved unsatisfactory. With long-term usage the most frequent side reaction noted was dysphoria. The addicting properties of this drug are probably between morphine and dihydromorphinone. (*Samuels, M. L., and others: Critical Evaluation of Numorphan: New Synthetic Morphine-Like Alkaloid, South. M. J. 52: 207 (Feb.) 1959.*)

NEW ANALGESIC In a study of 15 patients with chronic pain d-propoxyphene (Darvon) and meperidine (Demerol) were compared. d-Propoxyphene appeared to be only slightly less effective in analgesia than meperidine. (*Sahagian-Edwards, A.: Comparison of Analgesic Efficacy of d-Propoxyphene (Darvon) and Meperidine (Demerol), J. Chron. Dis. 8: 645 (Nov.) 1958.*)

DEXTROMORAMIDUM Dextromoramidum was found in experiments on rats to be 32 times as potent as morphine. The therapeutic index is much larger than with morphine and other opiates (morphine 10:95, dextromoramidum 15:225). Onset of action is more rapid than with morphine. Normorphine (4 parts dextromoramidum–3 parts nalorphine) antagonizes respiratory depression. Experiments did not reveal side actions on heart, kidneys or gastrointestinal tract. (*Krueger, G. A., and Orth, P. H.: Animal Experiments Concerning New Analgesic, Dextromoramidum, Der Anaesthetist 8: 11 (Jan.) 1959.*)

PROLONGED PAIN RELIEF Thirty milligrams of amiphenazole (Deptazol) and 30 to 40 mg. of morphine were given every 12 hours for periods of 4 to 14 days to 40 patients suffering from severe pain following injuries or operations. This mixture was given even when the patient was in severe shock, with very superficial respiration and definite cyanosis. The results were excellent in all instances. The most striking feature was improvement of respiration and patient's general condition. (*Glatzl, A.: Clinical Experience with Pain Relief with Amiphenazole Morphine Combination in Surgery of Trauma, Der Anaesthesist 7: 341 (Nov.) 1958.*)

NERVE BLOCKADE Argon, methane, krypton, xenon, nitrous oxide, ethylene, cyclopropane and three gaseous fluorocarbons reversibly block impulse transmission in all myelinated fibers when a critical number of molecules exist in solution within a nonaqueous phase. (*Carpenter, F. G.: Kinetics of Blockade in Peripheral Nerve Fibers Produced by Anesthetic Gases, Fed. Proc. 18: 23 (March) 1959.*)

PAIN MECHANISMS The specificity of sensory end organs in the skin is now seriously questioned. Newer concepts postulate that impulses traveling in various nerve fibers will be interpreted according to the pattern of impulses entering the central nervous system. Both the spinal cord and the reticular substance of the midbrain may serve as centers for sorting and relaying sensory impulses to higher interpretive areas of the brain. (*Scott, J.: Physiological Basis for Pain, Canad. M. A. J. 80: 109 (Jan. 15) 1959.*)

PSYCHOGENIC PAIN In pain due to tension states and hysteria, there is no sensory loss and the pain threshold is lowered; pain, tenderness, and feeling is of normal quality but magnified; the reactions to stimuli are normal, but magnified, and there exists a true hyperalgesia. When true causalgia and hyperpathia exist, there is demonstrable sensory loss and the pain threshold is elevated. Pain and tenderness is altered in quality, being characteristically hyperpathic. (*Walters, A.: Differentiation of Causalgia and Hyperpathia, Canad. M. A. J. 80: 105 (Jan. 15) 1959.*)

ATROPINE There are three phases of atropine action on the heart–an initial vagotonic effect; a transient period of vagal imbalance at different levels of the conduction system; and a final prolonged parasympathetic blockade. In the usual doses, the cardiac actions of atropine are benign. However, serious arrhythmias and conduction defects may occur if action of atropine is combined with actions of drugs such as neostigmine. (*Averill, K. H., and Lamb, L. E.: Less Commonly Recognized Ac-*

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