B., and Law, W.: Urinary Excretion of Adrenaline and Noradrenaline by Rats Under Various Experimental Conditions, Brit. J. Pharmacol. 13: 35 (Mar.) 1958.)

EPINEPHRINE The increase in cardiac output produced by infusion of epinephrine is due to an increase in force of contraction, rate, velocity of conduction and an increase in venous return. The latter is caused by an increased mean circulatory pressure. The rise in cardiac output is due more to the increased venous return than to the increased pumping ability of the heart. *(Guyton, A. C., and others: Mechanism of Increased Venous Return and Cardiac Output Caused by Epinephrine, Am. J. Physiol. 192: 126 (Jan.) 1958.)*

SEROTONIN-NOREPINEPHRINE The effectiveness of iproniazid (Marsilid) in treating depressed mental states may be due to the fact that this drug protects the neurohormones, serotonin and norepinephrine from monoamine oxidase, an enzyme that normally destroys them in the brain. Earlier studies had shown that the tranquilizer Resperine releases serotonin and norepinephrine from their stores in the brain. The theory is presented that serotonin and norepinephrine normally regulate brain centers that help govern bodily functions which are beyond voluntary control i.e. blood pressure, heart rate, emotional activity, etc. *(Spector, S., and others: Effect of Iproniazid on Brain Levels of Norepinephrine and Serotonin, Science 127: 701 (Mar. 28) 1958.)* (Recently a death has been reported to have resulted from a dose of Marsilid as originally recommended by the manufacturer—*Editor.*)

ADRENALINE RELEASE Plasma concentrations of adrenaline and noradrenaline and their changes after electroshock therapy (EST) were determined in psychiatric patients and in rats. EST caused a transient rise in both adrenaline and noradrenaline. Pretreatment with barbiturates and ganglionic blocking agents was found to suppress the adrenaline and noradrenaline responses; pretreatment with succinylcholine caused less inhibition. *(Griswold, R. L.: Plasma Adrenaline and Noradrenaline in Electroshock Therapy in Man, J. Appl. Physiol. 12: 177 (Jan.) 1958.)*

THORAZINE JAUNDICE Four jaundiced State Hospital patients on large doses of Thorazine were allowed to continue on the drug. Liver function studies and liver biopsies in three of the four subjects raised doubts as to the likelihood of chloropromazine jaundice being due to hepatotoxicity, chlorpromazine sensitivity, or to significant underlying hepatic disease. Other authors have suggested that biliary sphincter spasm occurs and leads to a marked slowing of bile flow. *(Schneider, E. M., Daugherty, C., and DeVore, J. K.: Chlorpromazine Jaundice, South. M. J. 51: 287 (March) 1958.)*

TRANQUILIZERS The use of tranquilizers in combination with reduced amounts of the older premedicant drugs has introduced a major change in preoperative regimen. Presently available phenothiazine derivatives, while not the ultimate in premedication, are worthy of appraisal because of their specific action on those areas of the central nervous system concerned with stress and emotional response and because of possible shock-sparing properties. Four potentially useful compounds, in order of decreasing effectiveness, are chlorpromazine (Thorazine), mepazine (Pacatal), promethazine (Phenergan) and diphenhydramine (Benadryl). The effective dosage range for chlorpromazine is 12.5–50 mg. intramuscularly, for mepazine 200–400 mg. orally, for promethazine 25–50 mg. intramuscularly and for diphenhydramine 50–100 mg. intramuscularly. These drugs are administered two hours preoperatively. One hour preoperatively meperidine 25–50 mg. intramuscularly combined with a belladonna derivative is injected. The resulting sedation has as its advantages (1) less over-all depression, (2) reduction in amounts of anesthetic drugs and undesirable reflex activity, (3) decreased incidence of emergence delerium and of postoperative nausea and vomiting, (4) reduction in dose and postponement of the postoperative reaction dose of narcotic. *(Lear, E., and others: Comparative Studies of Tranquilizers Used in Anesthesia,*

J. A. M. A. 166: 1438 (Mar. 22) 1958.) (An unusually fine and complete survey of the subject of tranquilizers which should be basic in an anesthesiologist's file.—*Reviewer.*)

CHLORPROMAZINE POISONING A case of acute chlorpromazine poisoning with resultant hypotension and respiratory depression responded to intravenous nalorphine. (*Sacks, N. Z: Acute Chlorpromazine Poisoning, Lancet, 2: 983 (Nov. 16) 1957.*)

ANTIHISTAMINES Sensitization to the cardio-accelerator action of adrenaline and noradrenaline by five antihistamines was examined on the acutely denervated heart of the cat. Antazoline (Antistin), chlorcyclizine and promethazine (Phenergan) increased cardio-accelerator response to both amines equally. Mepyramine (Anthisan) increased noradrenaline more than adrenaline action. Diphenhydramine (Benadryl) potentiated the responses to noradrenaline but not to adrenaline in a manner similar to that of cocaine and chronic denervation. Potentiating activity of the drugs was not related to their antihistamine potency or local anesthetic activity. (*Innes, I. R.: Sensitization of Heart and Nictitating Membrane of Cat to Sympathomimetic Amines by Antihistamine Drugs, Brit. J. Pharmacol. 13: 6 (Mar.) 1958.*)

SYNAPTIC TRANSMISSION Trimethadione (Tridione) administered to cats produced synaptic depression leading to a marked decrease in nervous transmission during repetitive stimulation. This was due to the action of the drug at a presynaptic site. All of the effects of a barbiturate were completely antagonized by appropriate doses of pentylenetetrazol (Metrazol). Conversely, the excitant effects of pentylenetetrazol could be completely antagonized by the barbiturates. (*Esplin, D. W., and Curto, E. M.: Effects of Trimethadione on Synaptic Transmission in Spinal Cord; Antagonism of Trimethadione and Pentylenetetrazol, J. Pharmacol. & Exper. Therap. 121: 457 (Dec.) 1957.*)

CORTICOSTEROID THERAPY Operative and postoperative complications were minimal during thirty-six operations done during long term treatment with either adrenocortical hormones or corticotropin. Operations during long-term treatment with adrenocortical hormones carry no increased risk of complications, provided there are no gross signs of overdosage with the hormones, and the administration of the hormones is not interrupted. (*Popert, A. J., and Davis, P. S.: Surgery During Long-Term Treatment with Adrenocortical Hormones, Lancet 1: 21 (Jan. 4) 1958.*)

ADRENAL ATROPHY Following prolonged parenteral administration to rats, prednisolone and hydrocortisone caused more adrenal atrophy than did prednisone. (*Hodges, J. R., and Vernikos, J.: Comparison of Pituitary Inhibitory Effects of Prednisone, Prednisolone, and Hydrocortisone, Brit, J. Pharmacol. 13: 98 (March) 1958.*)

NEOMYCIN PARALYSIS Neomycin given intravenously produced neuromuscular blockade in rabbits with one-tenth to one-twentieth the dose when the animal was anesthetized with ether. In dogs, neostigmine antagonized the ether-neomycin neuromuscular blockade. These studies suggest that reports of apnea following neomycin instillation in the peritoneum is due to the neuromuscular blocking activity of the neomycin. (*Pittinger, C. B., and others: Danger of Intra-peritoneal Neomycin During Ether Anesthesia, Surgery 43: 445 (Mar.) 1958.*)

STREPTOMYCIN Streptomycin injected intravenously in dose of 110 mg./Kg. abolished or diminished the amplitude of the contractions of some dogs' tibialis anticus when stimulated indirectly. This was interpreted as a neuromuscular blockade since it was abolished by neostigmine. (*Brazil, O. V., and Corrado, A. P.: Curariform Action of Streptomycin, J. Pharmacol. & Exper. Therap. 102: 452 (Aug.) 1957.*)

OPIOIDS Synthetic morphine-like compounds are called opioids. Those available for clinical use are meperidine, methadone, alphaprodine, levorphanol, anileridine, ethoheptazine, and darvon. All have their advantages, disadvantages and limitations. The narcotic antagonists of