the rise in blood pressure due to the administration of epinephrine, reduces the sympathetic and parasympathetic stimulating effects of nicotine and inhibits vagal reflexes. Given subcutaneously, it has no antihistaminic properties. Sedation was obtained following oral administration. Subcutaneous administration of methopromazine controlled the vomiting induced by apomorphine. It potentiates barbiturate and ether anesthesia and the analgesic effects of morphine. (*Courvoisier, S., and others: Pharmacodynamic Properties of Methopromazine, New Phenothiazine Derivative Related to Chloropromazine, C. R. Soc. Biol. 151: 689 (Oct.) 1957.*)

THIAMINE BLOCK When a nerve is stimulated, thiamine chlorhydrate (vitamin B-1) appears at the neuromuscular junction at the same time as acetylcholine. Occasionally accidents have occurred when large doses of thiamine have been given intravenously, and certain observers have related those accidents to its curarizing properties. The neuromuscular block produced is a curare type of block (nondepolarizing). In the cat, small doses of thiamine (20 mg./kg.) which do not produce a neuromuscular block antagonize the action of tubocurarine, but repetition of these doses sensitizes the neuromuscular junction to the action of tubocurarine. The antagonistic action of thiamine to depolarizing agents is also demonstrated. Cocarboxylase (active enzymatic form of thiamine) and acetyl-thiamine have a marked neuromuscular blocking action of the same type as tubocurarine and thiamine; they antagonize the neuromuscular block of the depolarizing agents. The pyrimidine fraction and the thiazol fraction of the thiamine molecule do not show any neuromuscular blocking action. Pyramine (anti-vitamin B-1) has, as thiamine, a neuromuscular blocking action. (*Cheymol, J., and others: Upon Curarizing and Anticurarizing Actions of Thiamine and Its Derivations, Arch. internat. pharmacodyn. 3: 36 (June) 1957.*)

CURARE AND BLOOD PRESSURE D-tubocurarine chloride (0.1 to 0.75 mg./kg). was given intravenously to 136 anesthetized patients. Blood pressure decreased in 117 patients, the rate and degree of fall being directly proportional to the dose of relaxant. The fall was greater in acutely sick patients. The individual variation to the same dose was considerable. (*Thomas, E. T.: Effect of d-Tubocurarine Chloride on Blood Pressure of Anaesthetized Patients, Lancet 2: 772 (Oct. 19) 1957.*)

CURARE PAIN RELIEF Tubadil (1.2 to 1.6 cc.) was given to 33 post-hemorrhoidectomy patients to help control pain. The effectiveness was compared with a group of 33 additional patients not given the relaxant. The narcotic requirements of the group given the relaxant were less than the control group. Muscle weakness and diplopia developed in 2 patients of the treated group. (*Rovner, H., and Haskell, B.: Study of Long Acting Muscle Relaxant (Tubadil) in Anorectal Surgery, Surg., Gynec. & Obst. 105: 488 (Oct.) 1957.*)

PRESTONAL A curare-like shorter acting muscle relaxant drug, Prestonal (Geigy), was used in 88 cases both in divided doses and continuous intravenous drip. A single injection of 30–70 mg. was sufficient for intubation, the larger dose giving more satisfactory cord paralysis. Smooth anesthesia was difficult to maintain with divided doses. Using Prestonal to supplement d-tubocurarine when more profound relaxation was desired was quite successful. Intravenous drip administration often provided relaxation short of the dose necessary to cause apnea. Complications encountered were tachycardia, hypertension, four instances of prolonged apnea, nine instances of disturbing restlessness in the recovery room, and a variable response to neostigmine and tensilon. (*Woodside, J. R., and others: New Muscle Relaxant Drug, Prestonal, South. M. J. 51: 80 (Jan.) 1958.*)

CITRATE INTOXICATION Plasma citric acid levels were measured by the pentabromacetate method in preoperative patients, in patients with liver disease, during gestation, during infusion of ACD solution, and during and after blood transfusion. Citric acid produces toxic effects by lowering the concentration of plasma

ionized calcium. Citric acid is readily metabolized and most adults can mobilize calcium stores rapidly so that citrate intoxication under ordinary conditions does not exist. (*Howland, W. S., and others: Massive Blood Replacement. V. Failure to Observe Citrate Intoxication, Surg., Gynec. & Obst. 105: 529 (Nov.) 1957.*)

EPINEPHRINE AND NOREPINEPHRINE Epinephrine is almost completely bound to plasma albumin, norepinephrine seems to be partly unbound. The increase of epinephrine and norepinephrine activity following acid hydrolysis suggests the presence of a conjugated form, bound to albumin, which is released by acid hydrolysis. (*Antoniades, H. N., and others: Transport of Epinephrine and Norepinephrine in Human Plasma, Proc. Soc. Exper. Biol. & Med. 97: 11 (Jan.) 1958.*)

EPINEPHRINE AND NOREPINEPHRINE The actions of these two drugs upon various parameters of the heart of the conscious and anesthetized dog were studied. Both drugs diminished cardiac rate in the conscious state; norepinephrine also diminished the rate in the anesthetized dog, whereas epinephrine led to an increased rate. The left ventricular systolic pressure was increased more by norepinephrine. There were no major differences between the drugs on ventricular contractility. (*West, T. C., and Rushmer, R. F.: Comparative Effects of Epinephrine and Levarterenol on Left Ventricular Performance in Conscious and Anesthetized Dogs, J. Pharmacol. & Exper. Therap. 120: 361 (July) 1957.*)

NOREPINEPHRINE SLOUGH Soft tissue necrosis associated with intravenous administration of norepinephrine solution is apparently the result of extravascular infiltration. Such tissue necrosis can be minimized or prevented by injecting a solution of Regitine (R), 10 mg. in 20 cc. of saline, about the margins of the extravasation. (*Berben, J. Y., Bryant, M. F., and Howard, J. W.: Etiology and Prevention of Sloughs Produced by L-Norepinephrine (Levophed), Ann. Surg. 146: 1016 (Dec.) 1957.*)

NOREPINEPHRINE NECROSIS Mechanisms responsible for the cutaneous necrosis following intravenous infusion of norepinephrine are (1) extravasation or marked spasm and ischemia of the infusion vein with diffusion of the drugs through its wall, or (2) a more intense ischemia in the presence of hypotension associated with hemorrhage or trauma due to an increased sensitivity of vessels to norepinephrine-induced constriction. Both prevention and treatment are remarkably facilitated with the local use of Regitine (R) and hyaluronidase solution. Priscoline in 15.0 mg. dosage apparently provides similar protection but with slower and less striking beneficial action. (*Close, S. A., Frackelton, W. H., and Kory, R. C.: Cutaneous Necrosis Due to Norepinephrine. II. Mechanism and Prevention, Ann. Surg. 147: 44 (Jan.) 1958.*)

POLIOMYELITIS Incipient respiratory failure in patients with severe acute poliomyelitis may be heralded in part by (1) shallow, rapid, regular respirations; (2) dilatation of nares and use of other accessory muscles; (3) preoccupation with breathing effort; (4) decreased duration of phonation in counting; (5) decreased or absent cough reflex; (6) diminished movement of diaphragm and intercostal muscles, etc. It is better to err on the side of early use of respiratory aids rather than waiting until asphyxia ensues. Of the respiratory aids, the tank is used early in the disease while the rocking bed, cuirass respirator, positive pressure equipment and glossopharyngeal breathing are used during the recovery period. Tracheotomy is not always needed. The techniques of respiratory failure care developed in regional centers can well be adapted to diseases other than poliomyelitis. (*Riley, H. P., Jr., and Batson, R.: Poliomyelitis Patient with Respiratory Failure, South. M. J. 50: 1357 (Nov.) 1957.*)

RESPIRATORS With pictures, charts, and detailed descriptions all of the commonly used tank and cuirass respirators are explained and criticized. Such information will be helpful for anyone caring for polio patients or chronic respiratory cripples. (*Kent, H.: What Physicians*