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*