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*

Should Know About Respirators, South. M. J. 50: 1497 (Dec.) 1957.) (Abstractor's note: These principles apply to post-anesthesia patients with depressed respiration.)

COMATOSE PATIENTS Regardless of the cause of coma, the results of treatment with adequate ventilation, blood pressure support, gastric lavage (following endotracheal intubation), fluid and electrolyte balance, administration of antibiotics, care to avoid pulmonary edema (by judicious use of positive pressure ventilation) etc. are superior to the use of stimulants. Use of drugs like megimide has not decreased the duration of coma. Hemodialysis has effectively lowered blood barbiturate levels. These patients are often best cared for in the recovery room. (*Crandell, D. L., and Page, W. G.: Comatose Patient, South. M. J. 51: 14 (Jan.) 1958.*)

UPRIGHT POSITION Patients who are anesthetized in the upright position, as for dental procedures, are subject to "fainting" or hypotensive episodes that may cause death or permanent brain damage. The semi-Fowler position immediately after anesthesia and operation is equally dangerous. Case histories from 14 patients are presented along with an excellent bibliography of the subject. The advisability of ever anesthetizing a patient in the upright position is questioned. (*Bourne, J. G.: Fainting and Cerebral Damage, Lancet 2: 499 (Sept. 14) 1957.*)

NEWBORN AS A RISK The newborn infant is a good risk on the operating table. He has a strength in his ability to survive cold, oxygen lack and infection. He cannot "come back" on his own, but must be "brought back." (*McCance, R. A., and Widdowson, E. M.: Physiology of Newborn Animal, Lancet 2: 585 (Sept. 21) 1957.*)

SEVERELY BURNED CHILD Clinical assessment of severely burned child should be based upon the functional efficiency of vital systems: the child's color, temperature, quality of pulse, thirst, capillary refill time, dryness or dampness of skin, rate and depth of respiration, muscle tone and restlessness. The volume of fluid lost in a burn is proportional to the extent of body surface burned. The "Rule of Nine" for estimating burn area gives these approximate figures: pubic area, 1 per cent; head and upper extremity (each), 9 per cent; back and front of trunk and lower extremity (each), 18 per cent. Fluid leakage from the burn continues for 48 hours, with the greatest loss occurring in the first eight of these hours. The formula employed in assessing the volume of fluid which must be replaced within this first 48-hour period is: 3 cc. colloid fluid/1 per cent surface area burned/ kg. body weight. The patient with a superficial burn should receive reconstituted plasma, but the patient with a deep burn must be given whole blood in addition to plasma in order to replace the red cells destroyed or damaged in the depths of the wound. In addition, the patient must receive sufficient water to satisfy metabolic fluid requirements as well as general supportive therapy. (*Shannon, D. W.: Resuscitation of Burned Child, Proc. Roy. Soc. Med. 50: 885 (Oct.) 1957.*)

CARBOHYDRATE METABOLISM The function of insulin varies among different tissues. In muscle, insulin may combine with the muscle, seemingly acting at the cell membrane to increase its permeability to glucose. The main metabolic effect would be an increase of glucose transport into the cell. Having entered the cell, impairment of phosphorylation by insulin lack does not appear to decrease the ability of the muscle to metabolize glucose. Other mechanisms seem to take place in the liver. Liver slices of diabetic rats show: diminished glucose uptake, diminished glucose oxidation, diminished glycogen formation, decreased ability to phosphorylate, and markedly increased gluconeogenesis. The permeability of liver cells to glucose is not influenced by insulin. The evidence suggests a metabolic block in the early stages of glucose metabolism possibly concerned with the oxidative phosphorylation (hexokinase) reaction. Other evidence suggests that by some unknown mechanism, insulin permits the liver to complete an enzyme system which in the absence of insulin has lost its function in whole or in part. This adaptation requires time and further suggests the possibility