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

of diverse actions in the liver also affecting fatty acid metabolism. There is no demonstrable action of insulin in the brain. (*Stadie, W. D.: Aspects of Carbohydrate and Phosphate Metabolism in Diabetes, Bull. New York Acad. Med. 34: 5 (Jan.) 1958.*)

FAT METABOLISM The known disturbances of fat metabolism in diabetes fall into three categories: impairment of fatty acid synthesis, increased mobilization from tissue stores, and abnormal accumulation of lipid in various tissues. A reduction of fatty acid synthesis normally appears after a few hours of starvation and is reversed by feeding a relatively small amount of carbohydrate. The impairment of fatty acid synthesis in diabetes is an exaggeration of this normal response to carbohydrate lack. The synthesis of fatty acid *in vitro* requires a simultaneous oxidation of citrate or some equivalent component of the tricarboxylic cycle. The metabolic defect in diabetes appears to be related to the decreased activity of the tricarboxylic cycle. The mobilization of fatty acids from tissue is depressed *in vitro* if the animal be fasted in advance, and enhanced if it be fed. In the intact animal, feeding of glucose causes a reduction of the total serum fat; and if given after a fatty meal, it diminishes the amplitude of alimentary lipemia. The non-esterified fatty acid (NEFA) fraction, because of the exceptional velocity of its metabolic turnover, is probably intimately involved. In diabetic ketosis, the NEFA concentration is increased two or three fold. Its rise precedes the appearance of ketonemia, and its fall after insulin therapy parallels the fall of glucose. Although the evidence is indirect, the flow of fatty acids from blood to liver and other tissues may be increased when the concentration of NEFA rises above normal. In addition, insulin may act as an inhibitor of the mobilization of fatty acids from tissue stores. If this inference is confirmed, it might bear on the cause of ketosis; for the development of ketonemia probably requires an accelerated transfer of fatty acids from fat depots to liver tissue. (*Dole, V. P.: Fat Metabolism in Diabetes, Bull. New York Acad. Med. 34: 21 (Jan.) 1958.*)

GASTRIC EMPTYING Regulation of gastric emptying begins as soon as the evacuated material has accumulated in the intestine to the point where any one of numerous stimuli associated with the chyme reaches threshold value. The effect of these stimuli is inhibitory to further emptying. The inhibitory effect is exerted either through a vagal reflex (enterogastric) or through a hormone (enterogastrone) or both. The pyloric sphincter plays a part by preventing regurgitation. It also contracts rhythmically to limit the volume evacuated at each cycle but there is no evidence that it regulates the over-all rate of emptying. (*Thomas, J. E.: Mechanics and Regulation of Gastric Emptying, Physiol. Rev. 37: 453 (Oct.) 1957.*)

ASPHYXIA In the rabbit about 3 minutes after the arrest of circulation, there is a sudden increase in electrical resistance of the cerebral cortex. It is thought this is due to the passage of ions and water from extracellular spaces into cortical cells and fibers. Quick frozen brains were found to have an increase of 11 per cent diameter of the nerve cells. This represents a volume increase of about 40 per cent. (*Van Harreveld, A.: Changes in Volume of Cortical Neuronal Elements During Asphyxiation, Am. J. Physiol. 191: 233 (Nov.) 1957.*)

MEDIASTINAL EMPHYSEMA Although it may be the complicating tension pneumothorax which endangers life, severe uncomplicated mediastinal emphysema may produce death by compression of the great vessels of the mediastinum. Most cases of mediastinal emphysema are mild and require only expectant treatment. However, occasionally the condition is severe enough to threaten life even though complications such as tension pneumothorax and hemorrhage have been controlled. Most patients with severe mediastinal emphysema will respond to cervical mediastinotomy, however, a tracheotomy should be performed if the mediastinotomy fails. Tracheotomy not only decompresses the mediastinum but also prevents the development of excessively high intrabronchial pressures during cough, thus reducing the force which tends to propel the air