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