tively transported and phosphorylation of the transported substances is not indispensable. Many L-isomers of amino acids are transported against a gradient whereas the D-isomers are usually not so transported. Competition may take place between amino acids for transport into the cell. Suppression of ATP brings about inhibition of energy-assisted transport into cells but there is a wide variety of substances that can accomplish such inhibition without affecting ATP level in the cell. Transport of an ion against a concentration gradient is an energy-dependent process requiring metabolic energy presumably in the form of ATP. Cardiac glycosides combine with a variety of transport carriers that are only able to act as transfer agents for sodium, potassium, or other substances when they are phosphorylated by ATP. (*Quastel, J. H.: Transport Reactions At the Cell Membrane. Introductory Survey, Canad. J. Biochem. 42: 907 (June) 1964.*)

ABSTRACTOR'S NOTE: This is the introduction to a symposium of six other articles on cell membrane transport.

HYPERGLYCEMIA Elevated arterial P_{CO_2} is believed to cause sympathetic stimulation resulting in secretion of epinephrine and mobilization of liver glycogen. A significant correlation was found between arterial P_{CO_2} and blood sugar level whereas mild hypoxia did not influence blood sugar levels. Positive-negative pressure breathing as well as ganglionic blockers or splanchnicotomy prevent hyperglycemia during anesthesia. (*Pflüger, H.: Role of Hypoxia or Hypercapnea as Causes of Hyperglycemia During Anesthesia, Der Anaesthesist 13: 129 (April) 1964.*)

PROLONGED INFUSION THERAPY Forty patients in prolonged coma due to organic or traumatic brain damage or under prolonged drug-induced sedation for treatment of tetanus were observed in respect to blood gases and acid-base balance. Most of the patients died from hypoxia and acidosis associated with pulmonary complications. All had been exclusively on parenteral fluids and exhibited anemia as expressed by a fall in oxygen capacity. As the anemia was considered partly responsible for the poor prognosis the cause of this anemia during prolonged intravenous alimentation was investigated in dogs. Hemoglobin, hematocrit and red cell count fell significantly in dogs undergoing prolonged barbiturate sedation while on intravenous fluid therapy. During oral fluid therapy these parameters remained normal under identical barbiturate sedation. Total body water increased significantly in animals under intravenous fluid therapy which was almost totally due to an increase in extracellular fluid volume. Intravenously perfused animals died in pulmonary edema and secondary pneumonia. Animals on oral alimentation failed to develop pulmonary edema or pneumonia. In man analogous findings were obtained. In comatose patients (cerebral contusion) intravenous fluid therapy caused a dilutional anemia through exclusive rise of plasma volume. The red cell mass remained normal. Since oral fluid therapy in comatose patients has been initiated, no fall of hemoglobin, hematocrit and red cell mass was observed. Likewise, pulmonary pathology developed less frequently. Severe degrees of hypoxia are consequently rarely seen in comatose patients on oral alimentation. Oral fluid therapy can increase the survival rate of comatose patients. (*Franke, D.: Pathophysiology of Infusion Therapy in the Unconscious State, Langenbeck Arch. Klin. Chir. 305: 428, 1964.*)