pathologic Effects of Norepinephrine Infusions in Dogs, Naunyn Schmiedeberg Arch. Exp. Path. 248: 54, 1964.)

RESERPINE AND HEMORRHAGE Pretreatment of dogs with reserpine did not alter the bleeding volume, *e.g.*, the volume of blood that can be withdrawn before the arterial blood pressure falls to 40 mm. of mercury, or the maximal volume of blood expelled by the animal into a reservoir. The uptake of blood from the reservoir during hypotension was considerably slowed by pretreatment with reserpine. Control animals had taken up 40 per cent of the shed volume after three hours at 40 mm mercury, while reserpine treated animals had taken up an average of only 10 per cent within the same time. The large vessel hematocrit in both groups of animals fell during hemorrhage, but while the hematocrit of the control animals increased again during hypotension that of the reserpine treated animals remained low throughout hypotension. Inhibition or blockade of impulse transmission in the sympathetic nervous system by reserpine did not seem to impair the ability of the animals to compensate for loss of blood volume. *(Seifen, E., Flacke, W., and Alper, M.: Influence of Reserpine Pretreatment Upon the Hemodynamics of Dogs During Hemorrhagic Hypotension, Naunyn Schmiedeberg Arch. Exp. Path. 248: 27, 1964.)*

QUINIDINE Quinidine produces in certain individuals a specific sensitization of the myocardium by reducing the fibrillation threshold. Quinidine syncope probably represents attacks triggered by some as yet unrecognized precipitating factor. No clue as to the nature of the factor could be found in this series: the contributory effects of such possible additive factors as digitalis, procainamide, electric defibrillation or cardiac failure are not clear. Since the myocardial action of quinidine is related to potassium flux and membrane permeability, processes also influenced by other drugs, summation appears to be a good possibility. *(Selzer, A., and Wray, H. W.: Quinidine Syncope, Circulation 30: 17 (July) 1964.)*

MYASTHENIA GRAVIS An exacerbation of myasthenic symptoms occurred in 3 of 8 patients given metyrapone and 5 of 8 patients receiving ACTH. A more marked response followed ACTH than to metyrapone. Patients with excessive adrenocortical responses to stimulation experienced symptomatic exacerbation. Direct deleterious effect of cortisol or corticosteroids on the nueromuscular junction is suggested, although an abnormality of steroid metabolism peculiar to some patients with myasthenia gravis cannot be ruled out. *(Klein, J. J., and Kermit, E. O.: Studies in Myasthenia Gravis, New Engl. J. Med. 271: 177 (July) 1964.)*

MYASTHENIA Anesthesia was induced in a patient with myasthenia gravis with a small dose of thiobarbiturate and it was found that endotracheal intubation could be performed without the use of a muscle relaxant. Then a test dose of 10 mg. of succinylcholine increased the patient's respiration, a second dose of 10 mg. caused apnea without visible muscular fasciculation, while a third dose of 10 mg. failed to change the patient's respiration. *(Amaha, K., and Takahashi, T.: Anesthesia In a Patient With Myasthenia Gravis, Sapporo Med. J. (Japanese) 22: 263, 1962.)*

CSF PRESSURE During first plane of third stage of general anesthesia in dogs with thiamylal, ether, nitrous oxide or halothane, blood pressure, respiration rate and cerebrospinal fluid (CSF) pressures were stable. During light general anesthesia deep breathing, coughing or straining markedly affected CSF pressure. During deep general anesthesia with ether or halothane, CSF pressure increased. Hypoxia and anoxia likewise caused a marked rise while atropine and succinylcholine had little effect. *(Sadanga, Y.: Influence of Various Kinds of General Anaesthesia and Some Promoting Drugs on Cerebrospinal Fluid Pressure, J. Kumamoto Med. Soc. (Japanese) 37: 162, 1963.)*

CELL MEMBRANE TRANSPORT Glucose not only enters the intestinal mucosa by simple or passive diffusion but by an active process that is dependent on the normal aerobic metabolism of intestinal tissue. Only members of a certain class of structure among various carbohydrates and related substances are ac-

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.*)