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ABSTRACTS

Grande, Francisco; and Prigge, William F. (Jay Phillips Res. Lab., Mount Sinai Hosp., and Lab. of Physiol. Hygiene, Univ. of Minnesota, Minneapolis, Minn.): GLUCAGON INFUSION, PLASMA FFA AND TRIGLYCERIDES, BLOOD SUGAR, AND LIVER LIPIDS IN BIRDS. Amer. J. Physiol. *218*:1406-11, May 1970.

Verbatim summary. Glucagon infusion produced elevations of plasma free fatty acids (FFA), triglycerides (TGL), and blood sugar in geese and ducks. Free fatty acids rose promptly after the beginning of the infusion, reaching a level which was maintained constant until the end of infusion. Blood-sugar rose continuously throughout the infusion. The effect of doses between 0.05 and 0.5 ug./kg. per min. on the plasma FFA of geese showed significant correlation ($r = +0.74$, $P < 0.01$) with the logarithm of the glucagon-infusion rate. In ducks, glucagon infusion (0.5 ug./kg. per min. for 2 hr.) caused significant elevation of the TGL content of the liver, but epinephrine and norepinephrine, infused at the same rate as glucagon, failed to elevate plasma FFA and caused no significant change of liver TGL. The results demonstrate that glucagon has a marked adipokinetic effect in birds, which is associated with elevations of plasma TGL and deposition of TGL in the liver. By contrast, catecholamines, which elevate plasma FFA and produce elevations of liver TGL in mammals, have no such effects in ducks.

Greene, Harry L.; Schubert, William K.; and Hug, George (Dept. of Pediat., Univ. of Cincinnati Coll. of Med., Cincinnati, Ohio): CHRONIC LACTIC ACIDOSIS OF INFANCY. J. Pediat. 76:853-60, June 1970.

Chronic metabolic acidosis in a six-month-old girl resulted in three hospital admissions and eventual death eight months later despite vigorous therapy. Lactic acidosis was established and lactate and pyruvate serum levels exceeded normal levels as much as ten-fold. Chronic lactic acidosis of infancy was reviewed, since many features of this rare disorder existed in the patient: convulsions, tetany, tachypnea, muscular hypotonia, and recurrent acidosis. Central nervous system lesions are also common and at postmortem examination, dilated ventricles, necrotizing encephalopathy and brain degeneration were noted. Of interest were the new findings of cataracts and anemia in this subject. The authors speculate that this condition may result from defective decarboxylation of pyruvate as a consequence of abnormal thiamine metabolism. R.K.K.

Huttenlocher, Peter R.; Hillman, Richard E.; and Hsia, Yujen E. (Sect. of Neurol. and Div. of Med. Genetics of the Depts. of Med. and Pediat., Yale Univ. Sch. of Med., New Haven, Conn.): PSEUDOTUMOR CEREBRI IN GALACTOSEMIA. J. Pediat. 76:902-05, June 1970.

Three infants presented with histories of poor feeding and development and signs of increased intracranial pressure with bulging fontanelles. These findings obscured underlying galactosemia and initially central nervous disease was primarily suspected. Following documentation of galactosemia, galactose-free diets led to prompt improvement in each case. Fontanelles were less full and head circumference diminished with amelioration of cataracts and reduction of hepatomegaly. It was suggested that cerebral edema may result from abnormal accumulations in brain of a metabolite of galactose, dulcitol. Since this sugar-alcohol derivative is not metabolized further and does not freely diffuse out of cells, its osmotic action may draw water into brain tissue and lead to edema. R.K.K.

James, W. P. T.; and Coore, H. G. (Tropical Metabolism Res. Unit and Dept. of Physiol., Univ. of West Indies, Mona, Kingston, Jamaica): PERSISTENT IMPAIRMENT OF INSULIN SECRETION AND GLUCOSE TOLERANCE AFTER MALNUTRITION. Amer. J. Clin. Nutr. *23*:386-89, April 1970.

Insulin secretion and glucose tolerance were studied in three groups of Jamaican children. Twenty-six children with malnutrition aged six to eighteen months were tested an average of twelve days after admission. Patients received a milk diet (100 kcal./kg. body wt.) with additional glucose for at least forty-eight hours prior to testing. A group of twenty-eight malnourished children underwent glucose tolerance testing after a prolonged period (average twelve weeks) of dietary treatment. A third group of five well nourished children served as control group.

All tests were performed after a six-hour fast. Blood glucose was measured by the glucose-oxidase method and plasma insulin by the double antibody method of Hales and Randle. All insulin assays were performed in triplicate using pork insulin standards for calibration. Results indicated impaired glucose tolerance after oral glucose (2 gm./kg. body wt.) and intravenous glucose (0.5 gm./kg. body wt.) in the children with malnutrition, even in those treated for three months with an adequate protein and calorie intake.

The malnourished children exhibited practically no increase in plasma immunoreactive insulin levels and the rise in plasma insulin was less in the treated than the control group, even after three months treatment.

Persisting reduction in the rate of glucose uptake by the periphery in the treated group was thought to be partly due at least to defective insulin production or release by the pancreas, or both. It was further suggested that malnutrition may pro-