ABSTRACTS

Abraham, J. M.; and Snodgrass, G. J. A. I. (Gen. Hosp., Ashton-under-Lyne, and Dept. of Pediat., Charing Cross Group of Hospitals, London, England): SOTOS' SYNDROME OF CEREBRAL GIGANTISM. Arch. Dis. Child. *44*:203-10, April 1969.

Metabolic studies were performed on a seventeen-year-old female with generalized lipodystrophy. Plasma immunoreactive insulin responses (IRI) to oral glucose or leucine and intravenous tolbutamide were increased above normal values. Fasting IRI concentrations were also approximately fourfold above control levels, but fasting growth hormone levels were normal. Plasma free fatty acid (FFA) concentrations tended to be elevated (700 to 800 μEq./L.) and were only slightly depressed during these tolerance tests as well as during a standard insulin tolerance test. Although a subcutaneous injection of epinephrine produced a significant rise in plasma glucose, plasma FFA remained relatively unchanged. Similar results were obtained with intravenous infusions of epinephrine. The authors concluded that adipose tissue of such patients has either a primary metabolic abnormality or else its defective responsiveness to insulin and epinephrine may be related to the action of an undefined circulating humoral agent. R.K.K.

Goldner, Andrew M.; Schultz, Stanley G.; and Curran, Peter F. (Biophysical Lab., Harvard Med. Sch., Boston, Mass.; Dept. of Physiol., Yale Univ. Sch. of Med., New Haven, Conn., and Dept. of Physiol., Univ. of Pittsburgh Sch. of Med., Pittsburgh, Pa.): SODIUM AND SUGAR FLUXES ACROSS THE MUCOSAL BORDER OF RABBIT ILEUM. J. Gen. Physiol. *53*:362-83, March 1969.

The mucosal surface of rabbit intestinal mucosa was exposed in vitro to various bathing fluids and the rates of penetration of sugars and sodium ions into the mucosa were measured. Entry of 3-o-methylglucose into the mucosa was markedly dependent on the presence of Na^+ in the mucosal solution; a decrease in the concentration of Na^+ caused a diminution of the maximal rate of sugar influx and little change in the apparent K_m. Conversely, the influx of Na^+ was enhanced by the presence of 3-o-methylglucose in the mucosal solution; at all concentrations of Na^+ tested there was a 1:1 molar ratio between the entry of sugar and the sugar-dependent influx of Na^+. It is postulated that sugar is transported across the intestinal mucosal cell membrane as a complex with a carrier, but that the rate of translocation is slow unless Na^+ is also bound to the carrier. H.T.W.

Kaess, H.; and Schlierf, G. (Med. Universitätsklinik (Ludolf-Krehl-Klinik) Heidelberg, Heidelberg, Germany): BLOOD SUGAR AND PLASMA INSULIN RESPONSE TO STIMULATION OF ENDOGENOUS SECRETIN RELEASE. Diabetologia *5*:228-32, 1969.

Verbatim summary. The effect of endogenous secretin released after intraduodenal instillation of 10 mval HCl/10 min. and of exogenous secretin (Boots) 1 U./kg. body weight, on exocrine and endocrine pancreatic function was examined in six subjects. Although stimulation of exocrine secretion was observed during instillation of HCl as well as after intravenous administration of secretin, a β-cytotropic effect, as estimated from blood sugar and plasma insulin concentrations in peripheral venous blood, was demonstrable only following exogenous secretin. In seven subjects, intraduodenal instillation of 2 × 3 mval HCl during intraduodenal glucose loading with 300 ml. 5 per cent glucose/20 min. did not result in any change of the blood sugar curve and of the insulin values compared with intraduodenal instillation of 2 × 30 ml. 0.9 per cent NaCl with the glucose. Lastly, no β-cytotropic effect could be detected following the intraduodenal instillation of 3 ml. ether, which stimulated secretion of a pancreatic juice rich in bicarbonate and enzymes. Thus endogenous secretin may be without appreciable physiological significance, with regard to the better assimilation of glucose and increased insulin secretion after oral glucose loading compared with intravenous glucose.

Katz, H. P.; Youlton, R.; Kaplan, S. L.; and Grumbach, M. M. (Dept. of Pediat., Univ. of California San Francisco Med. Center, San Francisco, Calif.): GROWTH AND GROWTH HORMONE. III. GROWTH HORMONE RELEASE IN CHILDREN WITH PRIMARY HYPOTHRYOIDISM AND THYROTOXICOSIS. J. Clin. Endocr. *29*:346-51, March 1969.

Verbatim summary. Growth hormone release was assessed utilizing insulin-induced hypoglycemia and arginine infusion in thirteen children with primary hypothyroidism and in ten patients with thyrotoxicosis. In the hypothyroid group there were six blunted and seven normal responses, with a mean peak concentration of serum growth hormone (SGH) of 7.9 ng./ml. which differed significantly from the controls ($p < .02$). The hypothyroid patients also exhibited a delayed peak in SGH compared to normal subjects. There was no correlation between the severity or duration of the hypothyroidism and growth hormone response. It was shown that thyroid deficiency is inconstantly associated with an abnormal growth hormone response to insulin-induced hypoglycemia and arginine infusion which was reversible after treatment with thyroid hormone. The impaired growth hormone release found in some patients with primary hypothyroidism may be indistinguishable from that observed in hypopituitary subjects. The growth hormone response in the group of children with thyrotoxicosis did not differ significantly from that of the normal controls.

Kipnis, David M. (Metabolism Div., Dept. of Med., Washington Univ. Sch. of Med., St. Louis, Mo.): INSULIN SECRETION IN DIABETES MELLITUS. Ann. Intern. Med. 69:891-901, November 1968.

Verbatim summary. Measurements of the plasma immunoreactive insulin responses to various insulinogenic stimuli in prediabetic, latent, juvenile, and maturity-onset diabetics indicate that impaired insulin secretion is a characteristic feature of the diabetic state. The development of overt diabetics depends, however, on both the degree of impairment of insulin secretion and the insulin sensitivity of the individual. The maintenance of normal carbohydrate tolerance in pre- and latent diabetics, despite decreased insulin secretion, indicates increased endorgan responsiveness to insulin.

Normal weight, maturity-onset diabetics also exhibit increased glucose utilization per unit insulin secreted, but insulin secretion is sufficiently impaired to result in overt diabetes. In these terms, conditions that increase insulin resistance—obesity, pregnancy, infection, excess glucocorticoids, and growth hormone—precipitate, and conditions that decrease insulin resistance—for example, glucocorticoid and growth hormone deficiency and unknown factors—delay the onset of overt diabetes to individuals possessing the inherited beta cell defect characteristic of diabetes mellitus.

Parker, D. C.; Sassin, J. F.; Mace, J. W.; Gotlin, R. W.; and Rossman, L. G. (Endocrine Div., Scripps Clin. and Res. Foundation, La Jolla, Calif.): HUMAN GROWTH HORMONE RELEASE DURING SLEEP: ELECTROENCEPHALOGRAPHIC CORRELATION. J. Clin. Endocr. 29:871-74, June 1969.

Plasma growth hormone (GH) levels were measured serially during sleep and correlated with electrophysiologic measurements of the brain in five young male volunteers. A maximum rise occurred within the first two hours (up to 36.5 ng.) reaching concentrations comparable to those seen after the infusion of arginine. The onset of enhanced GH secretion took place at the time of the first "slow wave sleep" (SWS). Smaller increments occurred fourteen times during later sleep, mostly associated with SWS. The authors feel that the results are consistent with the view that sleep has an anabolic function and is not just an overnight fast. O.V.S.

Paul, Oglesby; MacMillan, Anne; McKean, Harley; and Park, Heebok (Univ. of Illinois Coll. of Med., and Northwestern Univ. Sch. of Med., Chicago, Ill.): SUCROSE INTAKE AND CORONARY HEART DISEASE. Lancet 2:1049-50, November 16, 1968.

In July, 1964 Yudkin and Roddy reported that men aged forty-five to sixty-six with recent myocardial infarction habitually ingested twice as much sucrose as controls. In this study the authors analyzed daily sucrose intake in a prospective study of forty-two men who later suffered myocardial infarction and twenty-four men who died of coronary disease. The values were compared to eighty-five prospectively studied men who did not develop coronary disease. Subjects who ingested more than 2,800 gm. of sucrose in a twenty-eight-day period were arbitrarily classed as having high intakes; those ingesting less were classed as having low intakes. A logit analysis of the data showed that sucrose intake of coronary patients was higher but not statistically different from controls. Cigarette smoking appeared to be a far more important risk factor. T.G.S.

Swann, John C.; and Hammes, Gordon G. (Dept. of Chem., Cornell Univ., Ithaca, N. Y.): SELF-ASSOCIATION OF GLUCAGON. EQUILIBRIUM STUDIES. Biochemistry 8:1-7, January 1969.

Aggregation of glucagon molecules in concentrated aqueous solutions at pH 10 was studied by sedimentation equilibrium measurements and by gel filtration. The results suggest the existence of a monomer-dimer-hexamer mode of association. The finding of a large change in extinction coefficient near 250 mμ. associated with dimerization suggests that this transition is related to a conformational change in the glucagon molecule. It is postulated that this conformational change represents conversion from a random coil to a more helical structure. H.T.N.

Taylor, Alfred M. (Dept. of Intern. Med., The Cleveland Clin. Foundation, Cleveland, O.): THE CHALLENGE OF DIABETIC NEUROPATHY IN THE ELDERLY. Geriatrics 24:146-50, April 1969.

Verbatim summary. Diabetic neuropathy increases in frequency with age and manifests itself either as a symmetric sensory and motor polyneuropathy or as an asymmetric mononeuropathy multiplex. The most common symptoms are pain and sensory disturbances; the most common signs are loss of deep tendon reflexes and vibratory sensation. The pathological lesions are most frequently segmental demyelination with axonal degeneration of graded severity in the peripheral nerves, either on an ischemic basis from multiple infarcts or from genetically or metabolically induced lesions. The changes are not pathognomonic for diabetes, nor is there specific treatment. More study is necessary to adequately understand and treat diabetic neuropathy.

Updike, Stuart J.; and Harrington, Avery R. (Renal Sect., Dept. of Med., Univ. of Wisconsin Med. Center, Madison, Wisc.): ACUTE DIABETIC KETOACIDOSIS—A COMPLICATION OF INTRAVENOUS DIAZOXIDE TREATMENT FOR REFRACTORY HYPERTENSION. New Eng. J. Med. 280:768, April 3, 1969.

A twenty-nine-year-old woman with acute renal failure received intravenous diazoxide for treatment of refractory hypertension. Initial laboratory data revealed a fasting blood glucose level of 140 mg./100 ml. Over a ten-day period, nineteen injections of diazoxide were given. Blood pressure averages of 206/126 on conventional antihypertensive drugs were reduced to 180/76 during the period. On the tenth day diazoxide treatment was discontinued since the patient's blood glucose had risen to 840 mg./100 ml. Serum nitroprusside reaction was moderately positive for acetone bodies. Twenty-four hours later the blood glucose had reached 1,258 mg./100 ml. Radioimmunoassay failed to demonstrate insulin. Insulin treatment was initiated. Eight days after the last dose of diazoxide, insulin was discontinued. A two-hour postprandial blood glucose was 152 mg./100 ml., and an insulin assay twenty-six hours after the last insulin injection revealed 35 micro units of insulin per milliliter. Random blood glucose levels after discharge were within normal limits.

It was suggested the impaired renal function enhanced the diabetogenic effect of diazoxide. The authors recommend that patients receiving diazoxide for hypertension be followed carefully for development of diabetic ketoacidosis, especially in those with impaired renal function. B.R.B.