usefulness of high or low carbohydrate intake in diabetes is evident. The first author considers carbohydrate limitation to be essential in diabetes. Though he recommends weight loss for obese diabetics, he does not for other obese persons. He then quotes studies showing that diabetics whose diet is not regulated do no worse than other diabetics. This is a confusing and poorly organized paper. A subsequent speaker deals with practical aspects of dietary treatment, but has nothing particularly new to say. The final paper is an interesting demonstration of the possible use and difficulties of teaching aids in the training of diabetics. Presented by someone actively engaged in teaching, it is well presented and will be of interest to those involved in teaching diabetics about the principles and practical aspects of diets.

The final section is entitled the "Provocation and Prevention of Diabetes by Nutrition." How the quantity and quality of the diet may affect laboratory rodents with spontaneously occurring hyperglycemic syndromes is discussed by the first speaker. Also, the effect of hyperphagia induced by destruction of hypothalamic structures on the appearance of diabetes in the animals is examined. Next is an extremely able discussion of the effect of diet restriction on the onset of diabetes in prediabetic Chinese hamsters. An interesting study of the dietary intake of diabetics and their nondiabetic siblings and other controls shows that diabetics eat significantly more than nondiabetic controls. The symposium ends with an article propounding the thesis that the vascular diseases of retinopathy and atherosclerosis are dissociated, the first being caused by hyperglycemia and the second being related to diet. Again, the hypothesis is interesting but little data is brought to bear on the proposition.

This book is a mixed bag. Though great emphasis is placed on the role of carbohydrates, proteins and fats on the induction and control of diabetes, and also on caloric restriction in the obese diabetic, other nutritional aspects of diabetes are not mentioned. In exchange for some of the poorer presentations in this symposium, one might have substituted discussions pertaining to the influence of potassium and calcium on insulin secretion and of chromium on glucose homeostasis. Interest in the micronutrients is certainly growing, and diabetologists could profit from such discussions.

In summary, the proceedings of this symposium will be of interest primarily to the investigator, though the practicing clinician who desires to read in depth may find many of the chapters valuable. The Ponte publishing house, which sponsored the conference, may be congratulated for preparing the text with clear attractive type and with illustrations and tables that are easy to read. The text is printed in English and Italian, side by side, and the English translations are, on the whole, excellently done.

ABSTRACTS

Biener, J.; Jansen, F. K.; and Brandenburg, D. (Diabetes Forschungs-Inst., Düsseldorf, and Deutsches Wollforschungsinstitut, Aachen, F.R.G.): INSULIN LABELLED BY COUPLING WITH PEROXIDASE. Diabetologia 9:53-55, February 1973.

Verbatim summary. By coupling purified horseradish peroxidase with glutaraldehyde and subsequent reaction with insulin, conjugates were obtained. These were partially purified by ion exchange chromatography on DEAE-cellulose. A fraction with an average molar ratio of peroxidase to insulin 1:0.37 was analyzed by electrophoresis and gel filtration. The peroxidase activity of this fraction was found to be 19 per cent of normal and the immunological reactivity 0.6 per cent as compared with that of insulin.

Birnesser, H.; Reinauer, H.; and Hollmann, S. (Inst. of Physiol. Chem. and Diabetes Res. Inst., Univ. of Düsseldorf, F.R.G.): COMPARATIVE STUDY OF ENZYME ACTIVITIES DEGRADING SORBITOL, RIBITOL, XYLITOL AND GLUCONATE IN GUINEA PIG TISSUES. Diabetologia 9:30-33, February 1973.

Verbatim summary. In guinea pig tissues the activities of the enzymes D-gluconokinase, sorbitol dehydrogenase, D-ribulo- and D-xylulokinase were measured. D-ribulose and D-xylulose were prepared by isomerization of D-ribose and D-xylose in pyridine and separated by preparative paper chromatography. The activity patterns of the pentulokinases were identical in all tested organs. The highest activities of these two enzymes were found in adipose tissue, when referred to soluble cell protein, and was higher than the activity in liver and kidney. The high enzyme activities of the pentulokinases in adipose tissue may explain the antilipolytic effect of these pentitols and pentoses in diabetes. The activities of sorbitol dehydrogenase and gluconokinase showed a similar activity pattern in all tested organs of the guinea pig. The highest activities were found in liver and kidney and the lowest in the adipose tissue. The direct metabolism of gluconate in adipose tissue seems impossible. The activity of the pentulokinases is diminished in the tissues of the diabetic rat.

Bloom, S. R.; Daniel, P. M.; Johnston, D. I.; Ogawa, Olivia; and Pratt, O. E. (Inst. of Clin. Res., Middlesex Hosp., Dept. of Neuropathol. Inst. of Psychiat., and Dept. of Child Health, King's Coll. Hosp., London, England): RELEASE OF GLUCAGON, INDUCED BY STRESS. Q. J. Exp. Physiol. *58*:99-108, January 1973.

Verbatim summary. When conscious, lightly restrained primates were startled by noise, the level of glucagon in the plasma rose rapidly and this rise was followed by an elevation of blood glucose but not of plasma insulin. In anesthetized animals similar effects were produced by unpleasant stimuli (rectal distension, drilling a burr hole in the skull or the passage of an electric current through the head). These experiments show that glucagon is rapidly released in response to various types of stress.

Bucher, W. H. (Medizinische Abteilung Interspital, Bern, Switzerland): MYOKARDINFARKT UND PLASMAFETTSAUREN. Schweiz. Med. Wochenschr. *103*:199-203, February 1973.

Verbatim summary. In a series of 100 consecutive patients with acute myocardial infarction the estimations of free fatty acids (FFA) did not reveal any relation between FFA levels and prognosis (mortality). The influence of nutrition on FFA levels is discussed. Especially high levels of FFA due to adrenergic activity in patients with an arteriovenous block suggest that the administration of catecholamines in this condition must be considered very carefully beforehand. The possible role of FFA in the etiology of acute and chronic occlusive vascular disease is mentioned.

Caspary, W. F.; and Creutzfeldt, W. (Div. of Gastroenterol. and Metab., Dept. of Med., Univ. of Goettingen, F.R.G.): INHIBITION OF INTESTINAL AMINO ACID TRANSPORT BY BLOOD SUGAR LOWERING BIGUANIDES. Diabetologia 9:6-12, February 1973.

Verbatim summary. The effect of blood-glucose lowering biguanides (phenethyl- and butylbiguanide) on active intestinal transport of different amino acids has been tested in hamster small intestine in vitro. Biguanides inhibited active transport of all amino acids tested. The inhibitory effect of biguanides increased with incubation time, was more pronounced after preincubation of intestinal tissue and was found to be noncompetitive. The minimal inhibitory concentration of phenethylbiguanide on amino acid transport was 5×10^{-4} M. 14-C-butylbiguanide was found to be transported into hamster small intestine by a concentration-independent, energy-independent uptake mechanism and was accumulated in intestinal tissue against a concentration gradient. In accord with earlier results on the inhibitory effect of biguanides on active intestinal hexose transport it is concluded that biguanides do not act as specific inhibitors for glucose transport, but rather affect active, energy-requiring intestinal transport mechanisms in general (hexose-, amino acid-, calcium- and myo-inositol transport), most likely due to their known inhibitory effect on mitochondrial respiration, thus depriving mucosal cells of ATP required to translocate substrates against a concentration gradient.

Connon, J. J. (Dept. of Med., Queen's Univ., Belfast, N. Ireland): A DIFFERENTIAL ACTION OF PHENFORMIN IN NORMAL AND DIABETIC RAT LIVERS. Diabetologia 9:47-49, February 1973.

Verbatim summary. Phenformin inhibited gluconeogenesis by livers from both normal and diabetic rats. However, the concentration of phenformin which inhibited gluconeogenesis by the diabetic livers was not effective in normal livers. It is suggested that an action which is differentially effective in the diabetic state is likely to be clinically relevant.

Felig, Philip (Dept. of Intern. Med., Yale Univ., New Haven, Conn.): THE GLUCOSE-ALANINE CYCLE. Metabolism *22*: 179-200, February 1973.

In this review, the central role of alanine as the key protein-derived gluconeogenic precursor is described; this amino acid, released from muscle, originates not only from catabolism of cellular protein but also from the transamination of glucose-derived pyruvate. Quantitatively, it is the primary amino acid released by muscle and extracted by the splanchnic bed in man in postabsorptive and fasting states. Hepatic gluconeogenesis from alanine exceeds that of all other amino acids Insulin inhibits gluconeogenesis by reducing alanine uptake, while in diabetes alanine extraction is augmented in the face of diminished circulating substrate. During prolonged fasting, alanine release is impaired and gluconeogenesis is reduced. Alanine deficiencies encountered in pregnancy, ethanol administration and ketotic hypoglycemia of infancy are associated with accentuation of fasting hypoglycemia. Hyperalaninemia and augmented glucose utilization are observed during exercise and accompany hyperpyruvemia associated with inborn errors of metabolism. Alanine may stimulate the release of glucagon unless gluconeogenic requirements are eliminated by infusion of glucose. Glucagon will reduce plasma alanine levels during stimulation of hepatic gluconeogenesis. Corticosteroids also increase gluconeogenesis from alanine, an effect demonstrable only at high substrate concentrations. Alanine, in addition to furnishing carbon skeletons for gluconeogenesis, provides a mechanism by which amino groups are transferred from muscle to liver for disposal as urea. In this connection alanine would represent a non-toxic alternative to ammonia in nitrogen transport. In the glucose-alanine cycle, the recycling of glucose carbon skeletons within the pathway appears to occur at approximately 50 per cent of that observed for the Cori (lactate) cycle. C.R.S.

Gabbay, Kenneth H. (Dept. of Med., Children's Hospital, and Dept. of Pediatr., Harvard Med. Sch., Boston, Mass.): THE SORBITOL PATHWAY AND THE COMPLICATIONS OF DIABETES. N. Engl. J. Med. *288*:831-36, April 19, 1973.

The author reviews the role of polyols in sugar-induced cataracts and motor nerve conduction velocity defects. An increase in aldose (sugar) in the lens secondary to an increase in the surrounding fluid leads to formation of the corresponding polyol, uptake of water and sodium and loss of potassium which eventually results in a loss of cellular integrity and cataract formation. Inhibition of polyol formation by an aldose reductase inhibitor prevents cataract formation. This inhibitor, 3,3-tetramethylene glutaric acid, also prevents the decrease in motor nerve conduction velocity seen in rats on a high galactose diet. The author indicates that in diabetes there is an increased sorbitol response to hyperglycemia which makes the diabetic more sensitive to blood sugar elevations. He does not discuss the recent studies of Clements, Morrison and Winegrad with regard to the role of polyols in altering the metabolic function of aortic tissue or their studies concerning other factors related to the polyols that may be important in diabetic and other neuropathies. This entire area is clearly an important one with respect to diabetic complications, and several investigators are now making notable contributions to our understanding of these complications. H.G.M.

Goodner, Charles J.; Koerker, Donna J.; Werrbach, Jon H.; Toivola, Pertti; and Gale, Charles C. (Robert H. Williams Lab. for Clin. Invest., Dept. of Med., Harborview Med. Center, Seattle; Univ. of Washington Sch. of Med., Regional Primate Res. Center; Dept. of Physiol. and Biophysics, U. of Washington, Seattle, Wash.): ADRENERGIC REGULATION OF LIPOLYSIS AND INSULIN SECRETION IN THE FASTED BABOON. Am. J. Physiol. *224*:534-39, March 1973.

Verbatim summary. Previous studies indicated that a glucoregulated pathway within the central nervous system participates in control of fasting lipolysis. To test the hypothesis that the efferent limb of this system is the sympathetic nervous system, we examined the effect of adrenergic blocking agents on the response to glucose in twenty-four-hour-fasted baboons. Ganglionic and beta-adrenergic blockade resulted in a 40 per cent decline in plasma free fatty acids and glycerol, whereas

insulin fell by 45 and 70 per cent, respectively. Superimposition of glucose infusion (intravenously or via the internal carotid artery) failed to further inhibit lipolysis in blocked animals, although injection of insulin did inhibit it. It is concluded that sympathetic tone is partially glucoregulated, contributes to fasting lipolysis and is one determinant of basal insulin secretion. Accordingly, we suggest that the rate of lipolysis is the resultant of the activity of two glucoregulated control systems, one the central nervous system and the other the endocrine pancreas via insulin secretion. These two systems may also be interrelated through sympathetic effects on basal insulin secretion.

Hofeldt, Fred D.; Dippe, Stephen; and Forsham, Peter H. (Dept. of Med., Univ. of Calif., San Francisco, Calif.): DIAGNOSIS AND CLASSIFICATION OF REACTIVE HYPOGLYCEMIA BASED ON HORMONAL CHANGES IN RESPONSE TO ORAL AND INTRAVENOUS GLUCOSE ADMINISTRATION. Am. J. Clin. Nutr. *25*:1193-1201, November 1972.

The authors offer a modified classification of reactive (postprandial) hypoglycemic states based upon plasma glucose, insulin, cortisol and growth hormone responses to oral and intravenous glucose tolerance tests. The five classes are: (1) Early alimentary hypoglycemia—includes patients who have had gastrointestinal surgery, and those who have peptic ulcer disease and vagotonic personality. (2) Late reactive hypoglycemia of early maturity-onset diabetes—may be insulinoplethoric or insulinopenic. (3) Hormonal hypoglycemia—includes deficiency of pituitary, thyroid and adrenal hormones as well as of epinephrine and glucagon. (4) Idiopathic. (5) Transitional—includes those anxious or hysterical patients with no cortisol or growth hormone response to low blood sugar. P.H.S.

Jivani, S. K. M.; and Rayner, P. H. W. (Univ. of Birmingham, Children's Hospital, Birmingham, England): DOES CONTROL INFLUENCE THE GROWTH OF DIABETIC CHILDREN? Arch. Dis. Child. *48*:109-15, February 1973.

Linear growth and growth velocity were studied in sixty girls and fifty-six boys with diabetes mellitus. The children were of normal height at the onset of diabetes, but were shorter than normal when examined three or more years after the onset of diabetes. The modest growth retardation in some children with diabetes is related to the duration of the disease before puberty and is due primarily to a delayed and reduced pubertal growth spurt. Since it does not appear to be related to the degree of diabetic control, linear growth is not a satisfactory criterion by which treatment may be assessed. J.M.F.

Johansen, K.; and Orskov, H. (Second Univ. Clin. of Int. Med., Kommunehospitalet, Aarhus, Denmark): THE STIMULATORY EFFECT ON INSULIN SECRETION IN LONG-TERM TOLBUTAMIDE TREATMENT. Acta Endocrinol. *71*:709-15, December 1972.

Verbatim summary. Previous studies of the long-term effect of sulfonylurea have consistently failed to reveal any chronic stimulation of insulin secretion. This failure is probably due to the inadequate design used.

In the present study an unquestionable long-term action of tolbutamide on the beta cell has been demonstrated. We have studied diabetic patients treated for several years during the days just before and just after experimental withdrawal of the drug and have ascertained by this means that the blood sugar is almost unchanged.

It has been found that the urinary insulin excretion decreased significantly after withdrawal of tolbutamide. It is concluded that the long-term clinical effect of tolbutamide is at least partly due to a stimulatory action of tolbutamide on the beta cell.

Johnson, J. D.; Hansen, R. C.; Albritton, W. L.; Werthemann, U.; and Christiansen, R. O. (Dept. of Pediatr. and Pathol., Stanford Univ. Sch. of Med., Stanford, Calif.): HYPOPLASIA OF THE ANTERIOR PITUITARY AND NEONATAL HYPOGLYCEMIA. J. Pediatr. *82*:634-41, April 1973.

Verbatim summary. Two infants presenting with hypoglycemia in the first day of life had no evidence of an anterior pituitary gland at autopsy. A third infant with neonatal hypoglycemia has survived with persistent hypoglycemia and hypopituitarism. Each of these infants represents a different clinical entity. One infant had congenital absence of the anterior pituitary with no other anomalies. Another had holencephaly at autopsy but did not demonstrate the facial characteristics of this syndrome, and there was no suggestion of this abnormality prior to death by family history or chromosomal analysis. The third infant had sept-optic dysplasia and pituitary dwarfism, which may represent a mild form of holencephaly. Studies of endocrine function revealed hypoadrenalism in all and absence of growth hormone response to hypoglycemia in two of the infants. An inappropriate antidiuretic hormone response was suspected in two infants providing evidence of posterior pituitary function.

Kanaghinis, T.; Iatromanolakis, N.; Ikkos, D.; Gatsou, P.; and Gardikas, C. (Professional Med. Unit and Radioisotope Lab. of the Evangelismos Hosp., Athens, Greece): INTRINSIC FACTOR SECRETION IN THE GASTRIC JUICE IN DIABETES MELLITUS. Am. J. Dig. Dis. *18*:85-91, February 1973.

Verbatim summary. The secretion of intrinsic factor (IF) in the gastric juice was studied in the basal and maximally stimulated gastric secretion of forty insulin-dependent, unselected diabetic patients and twenty-eight normal control subjects matched for age and sex. The pattern of IF secretion was studied in relation to (1) the presence of intrinsic factor antibodies and parietal cell antibodies in the serum and (2) the pattern of gastric secretion with respect to volume, pH and amount of hydrochloric acid. It has been shown that the incidence of IF deficiency is significantly higher in diabetic patients than in normal control subjects. Impaired secretion of IF is more pronounced in the basal than in the maximally stimulated secretion in diabetic patients. Deficiency of IF among diabetics is often, but not always, associated with circulating IF antibodies in the serum. It is suggested that impaired IF secretion resulting from atrophic gastritis in diabetes mellitus might not necessarily be associated with autoimmunity against IF.

Kerins, Craig; and Said, Sami I. (Dept. of Med., Med. Coll. of Virginia, Richmond, Va., and Dept. of Intern. Med., Univ. of Texas Med. Sch., and V.A. Hosp., Dallas, Tex.): HYPERGLYCEMIC AND GLYCOGENOLYTIC EFFECTS OF VASOACTIVE INTESTINAL POLYPEPTIDE. Proc. Soc. Exp. Biol. Med. *142*: 1014-17, March 1973.

A vasoactive intestinal polypeptide, isolated from the small intestine, showed 33 per cent hyperglycemic and 56 to 63 per cent glycogenolytic activities compared to those of pancreatic glucagon. The authors suggest that this polypeptide may be one of the components of "enteroglucagon." D.K.

Kompiang, I. P.; and Gibson, W. R. (Dept. of Physiol., Monash Univ., Clayton, Victoria, Australia): EFFECT OF HYPOPHYSECTOMY ON LIPOGENESIS AND GLYCOGENESIS IN COCKERELS. Am. J. Physiol. *224*:362-66, February 1973.

Verbatim summary. Hypophysectomy causes chickens to become obese even though their food intake is greatly reduced. To seek out possible differences in rates of lipogenesis by adipose tissue and liver and rates of glycogenesis by liver in vivo, we have measured the incorporation of glucose-14-C into lipids and glycogen in intact and hypophysectomized cockerels thirty minutes after the intravenous injection of the radioactive precursor. The hepatic lipid content for hypophysectomized birds was normal, but the lipids contained more than twice as much radioactivity as did lipids from intact birds. After hypophysectomy the plasma lipid concentration was elevated and there was an increase of more than ninefold in lipid radioactivity. Incorporation of glucose-14-C into adipose tissue lipids was low and was not affected by hypophysectomy. The glycogen content of liver was almost doubled, and the incorporation of glucose-14-C into liver glycogen was more than doubled. The obesity of hypophysectomized cockerels may be due, in part, to the deposition of lipids transported to adipose tissue from a liver in which the rate of lipogenesis is markedly increased.

Lieber, Charles S. (Dept. of Med., Mt. Sinai Sch. of Med., and Bronx V.A. Hosp., Bronx, New York): LIVER ADAPTATION AND INJURY IN ALCOHOLISM. N. Engl. J. Med. *288*:356-62, February 15, 1973.

This article is a review of the effects of alcohol on the liver and its metabolism. The oxidation of alcohol generates acetate and NADH. This leads to an increase in alpha-glycerophosphate and NADPH (via transhydrogenation) and spares the oxidation of fatty acids by mitochondria. All of these changes favor an increase in lipids. Alcohol also increases ketonemia and ketonuria in association with increased hepatic ketogenesis. Blood lactate levels are elevated, which leads to an increase in uric acid levels. The increase in lactate may also stimulate collagen formation. The author states that approximately 20 to 25 per cent of the ethanol is metabolized by the microsomal ethanol oxidizing system. The activity of this system increases with the chronic intake of either alcohol or drugs. This sharing of certain metabolic pathways accounts for some of the interaction between alcohol and certain drugs. The discussion and bibliography constitute a good review of a complex subject. H.G.M.

Malaisse, Willy J.; Brisson, Guy R.; and Baird, Larry E. (Lab. of Exp. Med., Univ. Libre de Bruxelles, Brussels, Belgium): STIMULUS-SECRETION COUPLING OF GLUCOSE-INDUCED INSULIN RELEASE. X. EFFECT OF GLUCOSE ON 45-CA EFFLUX FROM PERIFUSED ISLETS. Am. J. Physiol. *224*:389-94, February 1973.

Verbatim summary. Isolated islets of Langerhans preincubated in the presence of 45-Ca were perifused and the effluent radioactivity was continuously measured. Glucose-induced insulin increase was associated with a concomitant release of 45-Ca possibly enclosed within the secretory granules. When insulin release was abolished, namely at low extracellular calcium concentration or in the presence of heavy water, glucose provoked an immediate reduction in 45-Ca efflux. The effect of glucose was reversible. These data suggest that glucose might increase the intracellular concentration of calcium by inhibiting the outward transport of calcium across the membrane of the beta cell.

Marco, Jose; Calle, Consuelo; Roman, Dolores; Diaz-Fierros, Maruxa; Villanueva, Maria L.; and Valverde, Isabel (Clin. Puerta de Hierro, Univ. Autonoma de Madrid, Madrid, Spain): HYPERGLUCAGONISM INDUCED BY GLUCOCORTICOID TREATMENT IN MAN. N. Engl. J. Med. *288*:128-31, January 18, 1973.

The effect of short- and long-term administration of glucocorticoids on glucagon, insulin and glucose values was studied in healthy subjects. Prednisolone, 100 mg. intravenously, did not alter plasma glucagon or insulin levels. However, administration of prednisolone in doses of 40 mg. per day for four days increased the glucagon response to an arginine load. These values were approximately 50 per cent greater at the ten minute peak than they were before steroid administration. The insulin and glucose responses to arginine were also increased by prednisolone. The authors suggest that the increase in glucagon is due to hyperaminoacidemia, which is known to occur with steroids. H.G.M.

Melancon, S. B.; Khachadurian, A. K.; Nadler, H. L.; and Brown, B. I. (N.W. Univ. Med. Sch., The Children's Memorial Hosp., Washington Univ. Sch. of Med., St. Louis, Mo.): METABOLIC AND BIOCHEMICAL STUDIES IN FRUCTOSE 1, 6-DIPHOSPHATASE DEFICIENCY. J. Pediatr. *82*:650-57, April 1973.

Verbatim summary. A nine month old girl with recurrent episodes of hypoglycemia, metabolic acidosis and hepatomegaly was identified as having fructose 1, 6-diphosphatase deficiency on the basis of absence of activity of this enzyme in liver and in white blood cells. Fructose 1, 6-diphosphatase present in white blood cells of normal persons appears to be similar to the enzyme in liver and kidney with regard to pH optima, electrophoretic mobility, and inhibition by adenosine monophosphate. In contrast, fructose 1, 6-diphosphatase from muscle appears to be a distinct enzyme. The early diagnosis of this deficiency disorder is important as dietary restriction of fructose, sucrose and sorbitol appears to be of value in the management of this potentially fatal inborn error of gluconeogenesis.

Milner, R. D. G.; Chouksey, S. K.; Mickleson, K. N. P.; and Assan, R. (Dept. of Child Health, Univ. of Manchester and the Hôtel-Dieu, Paris, France): PLASMA PANCREATIC GLUCAGON AND INSULIN:GLUCAGON RATIO AT BIRTH. Arch. Dis. Child. *48*:241-42, March 1973.

Verbatim summary. At birth the mean plasma pancreatic glucagon concentration of maternal peripheral venous blood, umbilical arterial and venous blood was similar, and varied between 160 and 180 pg./ml. in thirty normal term deliveries. Plasma insulin levels and the insulin:glucagon ratio correlated with plasma glucose in each of the three samples. Plasma glucagon did not correlate with either plasma glucose or insulin. No evidence was found for biological significance of the insulin:glucagon ratio in mother or infant at the time of birth.

Murthy, V. K.; and Steiner, G. (Dept. of Med. and Physiol., Univ. of Toronto, Toronto, Ontario, Canada): HEPATIC ACETATE LEVELS IN RELATION TO ALTERED LIPID METABOLISM. Metabolism *22*:81-84, January 1973.

Using homogenized rat liver preparations, acetic-thiokinase activity has been shown to be depressed during starvation or in other conditions resulting in inhibition of lipogenesis. Free

acetate concentrations in liver preparations of fasted rats were increased. Acetyl CoA deacylase activities were unchanged during fasting both in whole mitochondrial extracts or in microsomal supernatants of liver. These results suggest that free acetate is a metabolic intermediate and that increased levels of acetate in the livers of fasted rats may be due to impaired conversion of acetate to acetyl CoA under conditions of inhibited lipogenesis. C.R.S.

Nordlander, Sune; Ostman, Jan; Cerasi, Erol; Luft, Rolf; and Ekelund, Lars-Goran (Dept. of Endocrinol. and Metab., and Dept. of Clin. Physiol., Karolinska Hospital, Stockholm, Sweden): OCCURRENCE OF DIABETIC TYPE OF PLASMA FFA AND GLYCEROL RESPONSES TO PHYSICAL EXERCISE IN PREDIABETIC SUBJECTS. Acta Med. Scand. *193*:9-21, January-February 1973.

The response of plasma FFA and glycerol to exercise was studied in healthy subjects, insulin-dependent diabetics and prediabetics. Although the prediabetics reacted normally to a glucose tolerance test, their insulin response to a glucose load was diminished. FFA values decreased slightly and glycerol values increased slightly in normal subjects during exercise. Four out of the ten prediabetics and all the diabetics had an increase in plasma FFA and glycerol levels that was significantly different from the normal response. The increase in glycerol and FFA was not correlated with either the lactate or growth hormone response to exercise. The authors presented no clear explanation for the diabetic type of metabolic response in the prediabetics. H.G.M.

Orskov, H.; and Johansen, K. (Second Univ. Clin. of Int. Med., Kommunehospitalet, Aarhus, Denmark): IMMUNOLOGICAL MEASUREMENTS OF URINARY INSULIN FOR THE EVALUATION OF INSULIN PRODUCTION. Acta Endocrinol. *71*: 697-708, December 1972.

A method for the measurement of immunoreactive insulin in human urine is described. The urine is collected at short intervals to minimize its exposure to body temperature. Albumin is added to the sample containers. Albumin-enriched urine is dialyzed across a visking membrane. Radioimmunoassay is performed on 200 μl. dialyzed urine by Wick chromatography or a double-antibody method. Using these modifications, the loss of insulin during dialysis is less than 10 per cent at all concentrations. The loss of immunoreactivity which occurs in undialyzed urine is eliminated completely by the dialysis procedure. The urinary clearance rate of endogenous insulin derived from measurement of arterial plasma and urinary concentrations of insulin during oral or intravenous administration of glucose or intravenous infusion of glucagon appears to be fairly constant in individual subjects during a test period as well as during oral glucose tolerance tests repeated at weekly intervals. The urinary clearance of insulin ranges from 0.15 to 0.47 ml. per minute in healthy subjects. The finding of a constant insulin clearance shows that urinary insulin excretion can be used as a valid expression of average plasma level of insulin during extended periods of time. S.P.

Podolsky, S.; Zimmerman, Hyman J.; Burrows, Belton A.; Cardarelli, John A.; Pattavina, Caterine G. (Med. and Nuclear Med. Services, Boston V.A. and Univ. Hosps., Boston Univ. Sch. of Med., Boston, Mass.): POTASSIUM DEPLETION IN HEPATIC CIRRHOSIS. N. Engl. J. Med. *288*:644-48, March 29, 1973.

Seven of seventeen patients with hepatic cirrhosis were found to be potassium depleted as judged by a 13 to 38 per cent increase in body potassium when given 180 mEq. of potassium chloride daily. Glucose tolerance and insulin response to a glucose load improved after potassium repletion in these seven patients; the growth hormone response to arginine also improved with potassium supplementation. In the ten patients who were not potassium depleted the parameters did not change after potassium chloride supplementation. H.G.M.

Rooth, G.; and Carlstrom, S. (Univ. Hosp., Lund, Sweden): DIURNAL VARIATIONS IN BLOOD GLUCOSE, 3-HYDROXYBUTYRATE, ACETOACETATE, PLASMA FREE FATTY ACIDS AND GLYCEROL IN DIABETES. Acta Med. Scand. *191*:559-63, June 1972.

Glucose, plasma free fatty acids (FFA), glycerol, 3-hydroxybutyrate (3-HB) and acetoacetate (AcAc) were measured throughout the day in seven diabetics receiving insulin. A fall in 3-HB, FFA and glycerol preceded the decrease in glucose. AcAc levels initially increased. As the insulin effect subsided the level of FFA and glycerol increased three to four hours before 3-HB and AcAc began to rise. One subject did not have any variation in the level of 3-HB, AcAc or FFA in spite of a swing in the glucose value from 50 to 300 mg./100 ml. during the twenty-four hour period. The measurement of these parameters did not substantially add anything further to the management of the diabetic with regard to altering his insulin dose above the blood glucose measurement (pointed out by one of the authors in the preceding paper in this journal). H.G.M.

Segal, Stanton; Genel, Myron; Holtzapple, Philip; and Ria, Claire (Div. of Biochem. Development and Molecular Dis., Children's Hosp. of Philadelphia, and Dept. of Pediatr., Med. Sch. of Univ. of Pennsylvania, Philadelphia, Pa.): TRANSPORT OF ALPHA-METHYL-D-GLUCOSIDE BY HUMAN KIDNEY CORTEX. Metabolism *22*:67-76, January 1973.

Alpha-methyl-D-glucoside is a nonmetabolizable sugar actively transported by human kidney cortex cells via a sodium- and energy-dependent system. D-glucose and D-galactose added to incubation media inhibited glucoside accumulation in human renal cortical slices. Inhibition by D-glucose was competitive and accelerated the efflux of the glucoside from preloaded cells. The glucoside appears to be transported by a mechanism shared to some extent with that of D-glucose. C.R.S.

Zarowitz, Harold (Diabetes Clin., Jewish Hosp. and Med. Center, New York, N.Y.): POSTPANCREATECTOMY INSULIN-RESISTANT DIABETES MELLITUS. New York J. Med. *72*: 3005-09, December 15, 1972.

Verbatim summary. Two female patients totally pancreatectomized because of carcinoma of the pancreas developed postsurgical diabetes mellitus that eventually became insulin resistant. No obvious cause for this resistance was apparent, except that exceedingly high insulin antibody titers were demonstrated in each instance. A rapid resolution of the insulin resistance quickly occurred following the use of prednisone in the first case, of 5-fluorouracil in the second. The former patient, still alive and well seven years following the removal of a cutaneous metastasis, remains very insulin sensitive. It is considered that in both instances immune resistance occurred and the therapy, serendipitously, in the response to the 5-fluorouracil, was responsible for its subsidence. The exact mechanisms involved are open to speculation in the absence of necessary laboratory data.