assured themselves of a more happy life among the Otaheitans than they could possibly have in England, which, joined to some female connections, has most likely been the leading cause of the whole business."

Further investigation of ackee has indicated that hypoglycin B is a peptide of hypoglycin A and glutamic acid; hypoglycin A, however, is not a peptide but an amino acid ($C_7H_{11}N_1O_2$) of a molecular weight of 141, which apparently represents the hypoglycemic principle.[9] If the emetic and hypoglycemic activity can indeed be separated, perhaps derivatives of *Blighia* will yet find usage in therapy.

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ABSTRACTS

Aloia, J. F. (Dept. of Med., Sect. of Endocrinology, Nassau County Med. Center, East Meadow, N.Y.): MONOSACCHARIDES AND POLYOLS IN DIABETES MELLITUS AND UREMIA. J. Lab. Clin. Med. *82*:809-17, 1973.

Recent evidence has suggested that the hyperglycemia of diabetes may cause increases in tissue and plasma concentrations of polyols, glucosamines and monosaccarides other than glucose. This publication describes the efficacy of gas-liquid chromatographic quantitation of serum and urine fructose, mannose, sorbitol and myo-inositol. The method involves deproteinization of serum or urine samples by ultrafiltration, lyophilization, passage through an anion exchange resin and injection into a gas chromatograph. Comparisons were made between thirty-two normals, thirty diabetics and twelve uremics. Normal patients had a mean glucose concentration of 80.8 mg. per cent; fructose was 0.56 mg. per cent and sorbitol was 0.27 mg. per cent. Diabetics had a mean glucose of 193 mg. per cent but their fructose was similar to normal patients and averaged 0.53 mg. per cent. Their sorbitol level was about twice the normal and averaged 0.59 mg. per cent. The uremic subjects had near normal glucose levels, but also had elevated sorbitol concentrations. There was correlation between glucose and sorbitol in the diabetics suggesting that the elevated serum sorbitol may have been derived from the polyol pathway. T.G.S.

Baber, J. C., Jr.; Hayden, W. F.; and Thompson, B. W. (Dept. of Surgery, Univ. of Arkansas Med. Center, and Surg. Svc., V.A. Hosp. and St. Vincent Infirmary, Little Rock, Ark.): INTESTINAL BYPASS OPERATIONS FOR OBESITY. Am. J. Surg. *126*:769-72, 1973.

Verbatim summary. Ninety jejunoileal bypass procedures were performed in eighty-six patients to control morbid obesity. The fifteen and ten-inch anastomosis does not assure adequate weight loss, but it is relatively free of complications. The fourteen and four-inch bypass produces better weight loss but has many complications. The fifteen and five-inch jejunoileal bypass seems to give adequate weight reduction and is relatively free of complications. Although diarrhea, electrolyte imbalance, progressive liver disease, and an occasional death are seen, end to side jejunoileal bypass produces adequate weight reduction and prevents death from the complications of morbid obesity.

Bagdade, J. D.; Porte, D., Jr.; Brunzell, J. D.; and Bierman, E. L. (Dept. of Med. and V.A. Hosp., Univ. Washington Sch. of Med., Seattle, Wash.): BASAL AND STIMULATED HYPERINSULINISM: REVERSIBLE METABOLIC SEQUELAE OF OBESITY. J. Lab. Clin. Med. *93*:563-69, 1974.

Ten ambulatory obese male subjects were studied by measuring basal and glucose-stimulated immunoreactive insulin (IRI) before and after weight reduction. Prior to weight reduction (obtained by feeding a 600 kcal/day diet) their mean weight was 174 per cent of ideal. After losing an average of 28.6 kg., their weight decreased to 137 per cent of ideal. Their preweight-loss basal IRI averaged 48 and fell to 29uU/ml. The IRI decrement was observed in each subject and suggests that basal hyperinsulinism is a reversible consequence and not a cause of obesity. Also, weight reduction was associated with nearly uniform improvement of glucose tolerance. Subjects with normal or mildly impaired glucose tolerance displayed improvement despite a reduced IRI response to glucose challenge. This suggests that improved peripheral resistance to insulin may be a consequence of weight reduction. In contrast, the more severely diabetic subjects improved their glucose tolerance in association with increased insulin secretion. This may have resulted from increasing the pool of insulin participating in basal secretion and making it available for response to glucose challenge. T.G.S.

Blanks, M. C.; and Gerritsen, G. C. (Diabetes and Atherosclerosis Research, Upjohn Co., Kalamazoo, Mich.):AN ULTRA-MICRO IMMUNOASSAY FOR INSULIN. Proc. Soc. Exp. Biol. Med. *146*:448-52, 1974.

To measure micro quantities of insulin the authors have described a modification of the conventional double antibody technic of Morgan and Lazarow. This ultra-micro method is capable of determining insulin contents as low as .02 microunits (μU.). D.K.

Block, M. B.; Pildes, R. S.; Mossabhoy, N. A.; Steiner, D. F.; and Rubenstein, A. H. (Dept. of Med., Univ. of Chicago Sch. of Med. Dept. of Pediat., Cook County Hosp.; and Abraham Lincoln Sch. of Med., Univ. of Illinois, Chicago, Ill.): C-PEPTIDE IMMUNOREACTIVITY (CPR): A NEW METHOD FOR STUDYING INFANTS OF INSULIN-TREATED DIABETIC MOTHERS. Pediatrics *53*:923-28, 1974.

Verbatim summary. Proinsulin is converted to insulin and C-peptide in the pancreatic islet cells; the latter two polypeptides are then secreted in equimolar concentration. Thus, measurement of C-peptide may provide an alternative means of studying β-cell function in pregnant, insulin-treated, diabetic patients and in their newborn infants (IDM). Using this approach, β-cell function was studied in eight normal, ten juvenile onset and five gestational diabetic patients at the time of delivery. Normal maternal and cord CPR (C-peptide immunoreactivity levels, mean ± S.E.M.) were 1.5 ± 0.3 and 0.9 ± 0.1 ng./ml., respectively. In six of ten juvenile-onset diabetics, CPR levels were undetectable, while in four CPR was elevated. Five gestational diabetic women had elevated CPR levels. Cord CPR levels were significantly higher in all IDM than in the normal controls. A prompt rise in CPR was seen following intravenous glucose load in four IDM. Gel filtration of selected sera demonstrated both C-peptide and proinsulin, the latter protein being bound to circulating insulin antibodies which have crossed the placenta. Thus, active β-cell secretion composed of proinsulin, C-peptide and presumably insulin is present during insulin therapy in gestational, in some juvenile onset-diabetic, pregnant women and in their offspring.

Boxer, J.; Kirby, L. T.; and Hahn, P. (Depts. of Pediat. and Obstet. and Gynec., Univ. of British Columbia, Vancouver, British Columbia, Canada): THE RESPONSE OF GLUCOSE-6-PHOSPHATASE IN HUMAN AND RAT FETAL LIVER CULTURES TO DIBUTYRYL CYCLIC AMP. Proc. Soc. Exp. Biol. Med. *145*:901-03, 1974.

Verbatim summary. Glucose-6-phosphatase was determined in fetal human and rat livers. It increased in activity with fetal age in both species. Culturing fetal livers for twenty-four hours in Eagle's essential medium had no effect on the enzyme. Addition of glucagon or dibutyryl cyclic AMP for the last eighteen hours of culturing increased enzyme activity significantly in both man and rat. However, in fetal livers from rat fetuses weighing more than 4.6 gm., this effect was not observed. Fructose-1,6-diphosphatase in human fetal liver also increased in activity between the twelfth and fifteenth week of gestation, but neither glucagon nor dibutyryl cyclic AMP had any effect in cultures.

Brown, R. G.; O'Leary, J. P.; and Woodward, E. R. (Dept. of Surg., Univ. of Florida College of Med., Gainesville, Fla.): HEPATIC EFFECTS OF JEJUNOILEAL BYPASS FOR MORBID OBESITY. Am. J. Surg. *127*:53-58, 1974.

Verbatim summary. Experience with thirty-seven intestinal bypass operations in thirty-six patients is reported. Documented severe liver failure occurred in six patients with one death. The failure is manifested by the onset of anorexia, nausea, crampy abdominal pain, and vomiting. The earliest detectable functional abnormality appears to be a decrease in uptake of technetium sulfa colloid by the liver. Bromsulphthalein retention is followed by hypoalbuminemia and hypokalemia. Elevation of the SGOT, SGPT, and alkaline phosphatase levels may occur at this time. Fluid retention with weight gain, pheripheral edema, and ascites is rapidly followed by hyperbilirubinemia of the conjugated type. Lesser degrees of abnormal liver function were discovered in eight other patients. Halothane anesthesia, hepatitis-associated antigen, alcoholism, and diabetes do not appear to be factors.

Buchwald, H.; Lober, P. H.; and Varco, R. L. (Depts. of Surg. and Path., Univ. of Minnesota Med. School, Minneapolis, Minn.): LIVER BIOPSY FINDINGS IN SEVENTY-SEVEN CONSECUTIVE PATIENTS UNDERGOING JEJUNOILEAL BYPASS FOR MORBID OBESITY. Am. J. Surg. *127*:48-52, 1974.

Verbatim summary. To clarify the status of hepatic morphology existent at the time of jejunoileal bypass in morbidly obese patients (those more than 100 pounds overweight) and the subsequent changes in liver architecture after jejunoileal bypass, we are performing intraoperative and yearly postoperative liver biopsies in all of our patients managed in this manner. This is the first report of our liver studies and this paper is an analysis of the histologic aspects of the liver at the time of bypass in seventy-seven consecutive patients.

This study clearly demonstrates that the majority of markedly obese patients undergoing jejunoileal bypass have fatty metamorphosis of the liver at the time of their operative procedure. Previous similar reports from the literature are reviewed, and theories for the orgin of hepatic fatty infiltration in obesity per se and after jejunoileal bypass are discussed.

Christensen, N. J. (Second Clin. of Intern. Med. and Dept. of Pediat., Kommunehospitalet, Arhus, Denmark): HYPOADRENALINEMIA DURING INSULIN HYPOGLYCEMIA IN CHILDREN WITH KETOTIC HYPOGLYCEMIA. J. Clin. Endocrinol. Metab. *38*:107-12, 1974.

Verbatim summary. Employing a precise and sensitive double-isotope derivative technique, plasma adrenaline was measured in adults and in children with and without spontaneous hypoglycemia before and after intravenous injection of insulin. In adults the rise in plasma adrenaline during hypoglycemia was dependent on the blood glucose concentration attained. Children without spontaneous hypoglycemia showed about the same rise in plasma adrenaline as the adults. Four children with spontaneous hypoglycemia had adrenaline concentrations during hypoglycemia of less than 5 per cent of the expected values. It is suggested that ketotic hypoglycemia and spontaneous hypoglycemia associated with hypoadrenalinemia are the same disease.

Craighead, J. E.; Kanich, R. E.; and Kessler, J. B. (Dept. of Pathol., Univ. of Vermont Coll. of Med., Burlington, Vt.): LESIONS OF THE ISLETS OF LANGERHANS IN ENCEPHALOMYOCARDITIS VIRUS-INFECTED MICE WITH DIABETES MELLITUS-LIKE DISEASE. Am. J. Pathol. *74*:287-300, 1974.

Verbatim summary. Mice infected with the M variant of the encephalomyocarditis (EMC) virus develop lesions of the islets of Langerhans associated with a diabetes mellitus-like disease. Ultrastructural alterations become evident in capillaries and beta cells at a time when large amounts of virus are present in the pancreatic tissue. Although some beta cells become necrotic, degranulation and contraction of intact cells is the prominent lesion. Changes in the capillaries appear early in the course of the infection and later are associated with interstitial fibrosis in and around the islets. During early convalescence, beta cells are degranulated and exhibit striking alterations of cytoplasmic organelles. These changes appear to be consequent to increased metabolic activity by the residual insular tissue. Interestingly enough, specific lesions of the alpha cells are not observed.

Dweck, H. S.; and Cassady, G. (Perinatal Research Lab., Dept. of Pediat., Univ. of Alabama in Birmingham, Birmingham, Ala.): GLUCOSE INTOLERANCE IN INFANTS OF VERY LOW BIRTH WEIGHT. Pediatrics, *53*:189-95, 1974.

Verbatim summary. Forty-three of fifty babies with birth weights 1,100 gm. or less were hyperglycemic with serum glucose levels exceeding 125 mg. per 100 ml. while receiving parenteral glucose. Serum glucose levels exceeded 300 mg. per 100 ml. in thirty-six of these fifty babies. Hyperglycemia was most frequent in the twenty-four hours after birth and was also related to high rates of glucose infusion (> 0.4 mg./kg./hr. from birth) as well as parenteral glucose administration in the absence of oral supplement. These data attest to the fragile nature of glucose metabolism in infants of very low birth weight.

Feller, J. H.; Brown, R. A.; Toussaint, G. P. M.; and Thompson, A. G. (McGill Univ. Surg. Clinic, and Dept. of Surg., Montreal General Hosp., Montreal, Quebec, Canada): CHANGING METHODS IN THE TREATMENT OF SEVERE PANCREATITIS. Am. J. Surg. *127*:196-201, 1974.

Verbatim summary. A review of recent experiences with 200 cases of acute pancreatitis is presented. The prevention and management of the major complications consisting of nutritional depletion, abscess and acute pseudocyst, respiratory failure, and gastrointestinal hemorrhage are discussed. A broader approach to the disease has led to improved results in terms of decreased mortality.

Frankel, B. J.; Gerich, J. E.; Hagura, R.; Fanska, R. E.; Gerritsen, G. C.; and Grodsky, G. M. (Metabol. Res. Unit and Depts. of Med. and Biochem. and Biophys., Univ. of Calif., San Francisco, Calif., and The Upjohn Co., Kalamazoo, Mich.): ABNORMAL SECRETION OF INSULIN AND GLUCAGON BY THE IN VITRO PERFUSED PANCREAS OF THE GENETICALLY DIABETIC CHINESE HAMSTER. J. Clin. Invest. *53*:1637-46, 1974.

Hereditary insulin-deficient diabetes mellitus occurs in certain sublines of nonobese Chinese hamsters which is remarkably similar in many respects to insulin-deficient human diabetes. The authors studied the insulin and glucagon secretory response of the isolated perfused pancreas of these animals using nondiabetic Chinese hamsters as controls. They found in the diabetic animals that there was: (a) a decrease in both first and second phase insulin secretion following perfusion with glucose, (b) an impaired suppression of glucagon to glucose, (c) a normal insulin and glucagon stimulation during theophylline perfusion and (d) a reduced insulin/glucagon molar ratio. The authors suggest that these results support the concept of a decreased sensitivity to glucose of both alpha and beta cells in the diabetic pancreas. R.R.

Friesen, S. R.; Hermreck, A. S.; and Mantz, F. A., Jr. (Depts. of Surg., Physiol., and Pathol., Univ. of Kansas Medical Center, Kansas City, Kans.): GLUCAGON, GASTRIN, AND CARCINOID TUMORS OF THE DUODENUM, PANCREAS, AND STOMACH: POLYPEPTIDE "APUDOMAS" OF THE FOREGUT. Am. J. Surg. *127*:90-101, 1974.

Verbatim summary. The clinical and investigative findings in eleven selected patients are presented, which illustrate various degrees of functional activity of polypeptide-secreting APUD cell endocrinopathies of the foregut. Six patients with carcinoid tumors presented with as many different clinical pictures, including the Zollinger-Ellison syndrome, hyperparathyroidism, the carcinoid syndrome, the familial multiple endocrine adenomatosis (MEA) syndrome, Cushing's syndrome, and hyperglucagonism with diabetes. Four patients with diabetes and nonbeta islet cell carcinoma are presented in whom presumpative and assay evidence implicates glucagon activity of the tumors. Two patients with APUD cell abnormalities (one with diarrheogenic islet cell hyperplasia and one with duodenal paraganglioma) and in whom no known polypeptides have been identified are also presented. The enbryologic cytochemical and functional capabilities of the neuroendocrine cells of the foregut are interrelated, and when these cells are hyperplastic or neoplastic, they may produce unusual clinical pictures which are more easily diagnosed by assay evaluations than by histologic methods.

Gerich, J. E ; Schneider, V.; Dippe, S. E.; Langlois, M.; Noacco, C.; Karam, J. H.; and Forsham, P. H. (Metabolic Res. Unit and Dept. of Med., Univ. of California, San Francisco, Calif.): CHARACTERIZATION OF THE GLUCAGON RESPONSE TO HYPOGLYCEMIA IN MAN. J. Clin. Endocrinol. Metab. *38*:77-82, 1974.

Verbatim summary. The present investigation was undertaken to characterize glucagon responses to hypoglycemia in man. Using a highly specific antiserum, plasma immunoreactive glucagon levels were determined during episodes of insulin-induced hypoglycemia in fifteen normal subjects and during oral glucose tolerance tests in four subjects with reactive hypoglycemia. During insulin-induced hypoglycemia, plasma glucagon rose from a mean (± S.E.M.) basal level of 143 ± 6.5 pg./ml. to a maximum of 471 ± 15 pg./ml. at forty-five minutes, $p < .001$. Initial glucagon responses were evident fifteen minutes after insulin administration and preceded those of cortisol and growth hormone. Total glucagon responses (area under curve) correlated positively with the total decrease in plasma glucose (area below basal), $r = 0.852$, $p < 0.0005$, rather than the nadir or absolute level of hypoglycemia achieved. Hyperglycemia induced by sixty-minute glucose infusion, which suppressed basal glucagon secretion, did not prevent glucagon responses to subsequent insulin-induced hypoglycemia. Moreover the fall in plasma glucose from a mean of 187 mg. per 100 ml. to 112 mg. per 100 ml. was sufficient to stimulate glucagon secretion. In all subjects with reactive hypoglycemia plasma glucagon rose at least two times basal.

Thus, in man, hypoglycemia is a potent stimulus for glucagon secretion. The rate of fall of plasma glucose as well as the degree and duration of hypoglycemia are important determinants of the

glucose response. These findings suggest that pancreatic glucagon may function physiologically as a hypoglycemic counter-regulatory hormone, and that glucagon responses to hypoglycemia may provide an additional parameter with which to assess pancreatic alpha-cell function in man.

Gossain, V. V.; Matute, M. L.; and Kalkhoff, R. K. (Metabol. Div., Dept. of Med., Med. Coll. of Wisconsin, the Clin. Res. Ctr. of Milwaukee County Gen. Hosp., and Deaconess Hosp., Milwaukee, Wisc.): RELATIVE INFLUENCE OF OBESITY AND DIABETES ON PLASMA ALPHA-CELL GLUCAGON. J. Clin. Endocrinol. Metab. *38*:238-43, 1974.

Verbatim summary. Conflicting reports exist regarding the effects of obesity and diabetes on pancreatic alpha-cell function. For this reason plasma glucagon was measured in obese and lean nondiabetic and diabetic women during thirty-minute intravenous infusions of arginine (1.0 gm. per minute) and glucose (1.5 gm. per minute) and after a 400 gm. lean beef meal.

Basal levels of glucagon were comparable in lean and obese nondiabetic groups, suppressed significantly during glucose infusions and rose to similar peak values after oral protein meals. Fasting glucagon concentrations in diabetic women were 20 per cent higher than control levels despite fasting hyperglycemia and did not suppress after glucose administration. Glucagon responses to oral protein resemble those of nondiabetic subjects, although concentrations were higher throughout the procedure in association with abnormally elevated plasma glucose curves. Paradoxically, arginine infusions elicited greatly augmented increments of glucagon in nondiabetic obese women as well as in both groups of diabetic subjects relative to lean control values.

It is concluded that pancreatic alpha-cell function is generally normal in nondiabetic obese states. The physiologic meaning of arginine-induced hyperglucagonemia in simple obesity in this context is uncertain. Plasma glucagon disturbances observed in human diabetes can exist in the presence or absence of obesity.

Hagenfeldt, L.; and Wester, P. O. (Depts. of Clin. Chem. and Med., Karolinska Institutet, Stockholm, Sweden): PLASMA LEVELS OF INDIVIDUAL FREE FATTY ACIDS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION. Acta Med. Scand. *194*:357-62, 1973.

Free fatty acid levels were measured by gas chromatography in patients with acute myocardial infarctions. The admission value was 830 micromoles/liter and tended to decrease slightly over the next forty-eight hours although there was a great deal of variation in the free fatty acid level in a given patient throughout the day. These levels tended to be higher in patients with pain, arrhythmias and larger infarctions leading the authors to speculate that their levels were related to their epinephrine levels. Some of the patients with arrhythmias had very low free fatty acid levels so that there was no correlation of any significance between the free fatty acid levels and arrhythmias. H.M.

Hayashi, K.; Boucher, W.; and Notkins, A. L. (Lab. of Oral Med., Nat'l Inst. of Dental Res., N. I. H., Bethesda, Md.): VIRUS-INDUCED DIABETES MELLITUS. Am. J. Pathol. *75*:91-102, 1974.

Verbatim summary. Infection of DBA/2 male mice with the M Variant of encephalomyocarditis virus resulted in a diabetes-like syndrome. Histologic examination of the pancreas revealed damage to the beta cells with little involvement of the acinar cells. The severity of the hyperglycemia correlated closely with the degree of beta-cell damage. By immunofluorescence, viral antigens could be detected in the beta cells during the first ten days of the infection. In contrast to the response found in male DBA/2 mice, infection of DBA/2 female mice and male mice of several other strains resulted in little if any elevation of blood glucose concentration. Histologic examination of the pancreas of these animals revealed only minimal damage to the beta cells. It is concluded that differences in the severity of the hyperglycemia between DBA/2 males and females and among the different strains of male mice tested are directly related to the degree of beta-cell damage produced by the viral infection.

Hedner, P.; Schersten, B.; and Thulin, T. (Dept. of Int. Med., Univ. Hospital, Lund, and Community Health Research Centre, Dalby, Sweden): DEXTROSTIX REFLECTANCE METER AS AN AID IN DIAGNOSTIC HYPOGLYCEMIA. Acta Med. Scand. *195*:29-31, 1974.

The accuracy of the dextrostix reflectance meter in measuring blood sugar values was studied in patients undergoing insulin hypoglycemia to test their pituitary and adrenal function. The reflectance meter was compared with the standard glucose oxidase method. The random error in the blood sugar range of 15 to 85 mg. per 100 ml. was 1.8 mg. with the greatest difference between duplicate determinations being 8 mg. per 100 ml. In the range from 15 to 40 mg. per 100 ml. the reflectance meter systemically underestimated the blood glucose values by 3 mg. per 100 ml. The authors found that if the dextrostix strips were more than one year old they gave values which were much too low. They also found that using blood from a indwelling catheter rather than capillary blood facilitated making an accurate measurement because the strip was much more easily covered with blood in the required time (three seconds). H.M.

Joram, A.; Fessard, Cl.; Tron, Ph.; de Menibus, C. H.; and Dailly, R. (Hopital Charles-Nicolle, Rouen, France): TYPE II GLYCOGENOSIS. Sem. Hop. Paris *50*:489-95, 1974.

Verbatim summary: The authors review cases of type II glycogenosis in infants and recall that in all cases of Pompe's and Gunther's disease, there is cardiac and muscle involvement. Diagnostic certainty is obtained by the demonstration of the glycogen deposits and a deficiency in acid maltase in the various samples. There is at present no effective treatment, but genetic advice and ante-natal detection should be carried out during any future pregnancy. More recently, have been recognised late myopathic forms of the disease. There are several theories to explain this within a single disease defined by acid maltase deficiency, the coexistence of forms in the infant, usually of the same type, and late forms which are very different and more variable in presentation.

Loubatieres, A.; Mariani, M. M.; Ribes, G.; and Alric, R. (Lab. de Pharmacol., Inst. de Biologie, Montpellier, France): PHARMACOLOGICAL COMPARISON BETWEEN TOLBUTAMIDE AND TWO SECOND GENERATION HYPOGLYCEMIC SULFONYLUREAS (GLIBENCLAMIDE AND GLISOXEPIDE). Acta Diabetol. Lat. *10*:261-82, 1973.

Verbatim summary. The authors conclude that administered intravenous in the normal conscious dog, glibenclamide and glisox-

epide are considerably more active on glycemia than tolbutamide. Administered per os, glibenclamide acts less rapidly on the glycemia than tolbutamide and glisoxepide. The dynamic study of the insulin secretion shows that the secretion of insulin begins more slowly and lasts longer with glibenclamide than with the two other products. On the isolated and perfused rat pancreas, glisoxepide and tolbutamide more rapidly stimulate the secretion of insulin than glibenclamide; with the latter substance the duration of the secretion of insulin is longer and the total quantity of insulin secreted is greater. As far as the beta-cytotrophic action is concerned, all three sulfonamides stimulate the neogenesis of the islets of Langerhans in Swiss mice.

Lundbaek, K. (Kommunehospitalet, Aarhus Univ., Aarhus, Denmark): DIABETIC ANGIOPATHY. Acta Diabetol. Lat. *10*:183-207, 1973.

Verbatim summary. In this lecture it was attempted to sketch the present situation in diabetes angiopathy research. Three major areas are discussed: 1) the growth and consolidation of the morphometric principle as applied to the elucidation of vascular structure changes in diabetes mellitus. Morphometric studies have shown that the vascular basement membrane is normal at the onset of diabetes; 2) the first studies in basement membrane biochemistry which have opened up an extremely important new field in diabetology. It has been shown that enzymatic abnormalities of diabetic basement membrane synthesis are reversible phenomena; 3) the realization that in the course of life the large blood vessels of the diabetics suffer changes that cannot be called simple arteriosclerosis. Diabetic macroangiopathy will be taken up as an important new research field. The situation today is also characterized by experimentation and speculation about the possible role of various metabolic pathways and various hormones (inter alia growth hormone) for the development of diabetic vascular disease. The lively activity in the field of diabetic angiopathy research gives some hope for practical ameliorations of diabetes therapy.

Munk, P.; Freedman, M. H.; Levison, H.; and Ehrlich, R. M. (Research Institute, Hospital for Sick Children; and Dept. of Pediat., Univ. of Toronto, Toronto, Ont., Canada): EFFECT OF BICARBONATE ON OXYGEN TRANSPORT IN JUVENILE DIABETIC KETOACIDOSIS. J. Pediatr. *84*:510-14, 1974.

Verbatim summary. In ten children with diabetic ketoacidosis, the red cell 2,3-diphosphoglycerate (2,3-DPG) was decreased to 2.93 ± 0.90 μmole per mililiter of RBC compared with a normal value of 4.41 ± 1.24; in contrast, in twenty-three children with controlled diabetes mellitus, the 2,3-DPG was increased to 6.97 ± 1.49. Five patients had their acidosis corrected with insulin, intravenous fluids, and sodium bicarbonate, and five with insulin and intravenous fluids alone. The 2,3-DPG returned to normal values in all ten patients within twenty-four hours of the beginning of therapy in contrast to the four- to five-day recovery period seen in adult patients with diabetic ketoacidosis. With respect to the oxyhemoglobin dissociation curve, the calculated in vivo P_{50} was high (31.6 ± mm. Hg) in all ten ketoacidotic patients on admission. Patients who were treated with insulin and fluids but without intravenous bicarbonate had a decrease of the in vivo P_{50} similar to that of those who received bicarbonate. This decrease was never significant enough to alter tissue oxygenation. We conclude from these results that bicarbonate therapy does not affect oxygen transport adversely and, consequently, the possibility of such an effect is not a reason for omission of bicarbonate from the therapeutic regimen.

Norton, L.; and Eiseman, B. (Dept. of Surg., Denver Gen. Hosp., Univ. of Colorado Sch. of Med., Denver, Colo.): NEAR TOTAL PANCREATECTOMY FOR HEMORRHAGIC PANCREATITIS. Am. J. Surg. *127*:191-95, 1974.

Verbatim summary. Four patients judged to be dying from acute hemorrhagic pancreatitis were treated by near total pancreatectomy of 65 to 80 per cent. Excision was suprisingly easy and rapid because of thrombosis of the small veins.

Three patients with disease of two days' duration or less survived despite postoperative complications of respiratory distress syndrome (two patients) and delirium tremens (one patient). They were discharged from the hospital eleven to twenty days after surgery. The only late complication was diabetes in one patient. The fourth patient, who was in terminal condition when operated on after one week of the disease, died three weeks after pancreatectomy as a result of sepsis due to intestinal leakage. Recovery was dramatic before the occurrence of sepsis.

The primary indication for near total pancreatectomy is failure of nonoperative treatment. The optimal time for surgery appears to be twenty-four to forty-eight hours after onset of disease.

In view of the high mortality of acute hemorrhagic pancreatitis and the relatively low mortality and morbidity of near total pancreatectomy, we regard excisional surgery in patients dying from hemorrhagic pancreatitis as reasonable and effective treatment.

Passa, P.; Gauville, C.; and Canivet, J. (Dept. of Nutrition-Endocrinology, Hopital Saint Louis, Paris, France): INFLUENCE OF MUSCULAR EXERCISE ON PLASMA LEVEL OF GROWTH HORMONE IN DIABETICS WITH AND WITHOUT RETINOPATHY. Lancet *2*:72-74, 1974.

The concentration of growth hormone in the plasma of human diabetics has been shown to rise higher after exercise in poorly controlled insulin requiring patients than in well controlled diabetics. For a number of reasons it appears plausible that elevations of growth hormone could serve as a causative factor in the genesis of diabetic microvascular disease. In this study the plasma growth hormone levels were determined during moderate controlled muscular exercise in two comparable groups of ten male nonobese insulin dependent diabetics. A group of ten normal subjects was used for control comparison. The average basal growth hormone levels were similar for both diabetic and control groups. In controls after moderate exercise the plasma growth hormone rose moderately but less than 5 ng/ml. In diabetics it was significantly higher during exercise. In diabetics without retinopathy the growth hormone increment peak of 6.6 ng./ml. was not different than controls. In diabetics with retinopathy the growth hormone increment was significantly higher than either controls or diabetics without retinopathy. The increase in growth hormone was independent of blood glucose and was seen in diabetics whose mean glucose was twice that of controls. During the periods of observation there was no transient decrease in blood glucose which exceeded 20 mg. per cent and could have been considered a stimulus to growth hormone release. The study suggests that there is an abnormality of growth hormone in diabetics with vascular disease but it does not distinguish whether the defect might be due to decreased removal or increased secretion of

growth hormone. However, it broadly fits the hypothesis that growth hormone hypersecretion may be a causal factor in the genesis of diabetic retinopathy. T.G.S.

Pi-Sunyer, F. X. (Med. Serv., St. Luke's Hosp. Center, and Dept. of Med., Coll. of Physicians and Surgeons, Columbia Univ., New York, N. Y.): STARVATION-INDUCED KETOSIS: REDUCTION IN DOGS ENRICHED WITH ODD-CARBON FATTY ACIDS. Proc. Soc. Exp. Biol. Med. *145*:786-89, 1974.

Dogs were fed a nutritionally complete diet containing 40 per cent of its calories as fat. Four experimental animals were given a mixture of equal portions of cottonseed oil and triundecanoin, while the three control dogs received cottonseed oil only. After thirteen weeks, 23 per cent of the total adipose tissue fatty acids were odd numbered in experimental animals. During an eight-day fast it was observed that the experimental animals had significantly lower acetoacetate and betahydroxybutyrate concentrations. Their serum glucose levels were significantly higher and they had greater mobilizable liver glycogen reserves. It appeared that the feeding with odd-carbon fatty acids inhibited the ketogenesis. D.K.

Reaven, G. M.; and Olefsky, J. M. (Dept. of Med., Stanford Univ. Sch. of Med. and Veterans Administration Hosp., Palo Alto, Calif.): INCREASED PLASMA GLUCOSE AND INSULIN RESPONSES TO HIGH-CARBOHYDRATE FEEDINGS IN NORMAL SUBJECTS. J. Clin. Endocrinol. Metab. *38*:151-54, 1974.

Verbatim summary. Plasma glucose and insulin responses to two different diets were measured in twenty adult nondiabetic men. The diets were isocaloric, and differed in amount of calories as fat and carbohydrate. One diet contained 42 per cent fat and 43 per cent carbohydrate, while the others contained 30 per cent fat and 55 per cent carbohydrate. On the fourth day of each dietary period plasma glucose and insulin levels were measured before the noon feeding, and hourly for the next three hours. The low-fat high-carbohydrate diet led to postprandial increases in plasma glucose and insulin concentrations. The increase in plasma glucose response was relatively modest, but the high-carbohydrate diet resulted in an approximate 40 per cent increase in plasma insulin response. Since ingestion of a low-fat high-carbohydrate diet led to increases in glucose and insulin levels, the advisability of recommending such diets as general prophylaxis against the development of atherosclerotic heart disease must be reconsidered in this light.

Sladek, C. D.; and Sharr, J. F. (Dept. of Physiol., Univ. of Ill. Med. Center, and Dept. of Physiol., Northwestern Univ., Chicago, Ill.): CONCENTRATION DEPENDENT INHIBITION OF HEPATIC GLUCONEOGENESIS BY INSULIN. Proc. Soc. Exp. Biol. Med. *146*:194-99, May 1974.

Verbatim summary. The rate of gluconeogenesis from U [^{14}C-alanine was determined at physiological insulin concentrations in the isolated rat liver perfused with a nonrecirculating medium. There was a significant negative relationship between gluconeogenesis and the insulin concentration over the range of 10 to 500 μU/ml. Increasing the insulin concentration to 1000 μU/ml. did not result in any further reduction of gluconeogenesis. Insulin did not alter the gluconeogenic response of the liver to increasing substrate concentrations in these perfusions.

Snyder, N.; Scurry, M.; and Hughes, W. (Gastroenterology Sect. and Dept. of Med., Univ. of Texas Medical Branch, Galveston, Texas): HYPERGASTRINEMIA IN FAMILIAL MULTIPLE ENDOCRINE ADENOMATOSIS. Ann. Intern. Med. *80*:321-25, 1974.

Verbatim summary. Fasting serum gastrin levels of forty-six members of five families with familial multiple endocrine adenomatosis were measured; fourteen patients had hypergastrinemia (a serum gastrin level greater than 200 pg./ml.), including six patients with intractable peptic ulcer disease. Eight patients had asymptomatic hypergastrinemia, and twelve of the patients with hypergastrinemia had a history of findings of hyperparathyroidism. Fasting gastrin levels were not significantly correlated with calcium levels. Normal gastrin levels were found in six patients with hyperparathyroidism. Hypergastrinemia was found in six patients with normal or low serum calcium values who were studied after parathyroid surgery. Basal gastric acid hypersecretion and a marked rise in serum gastrin levels on intravenous calcium infusion were associated with fasting hypergastrinemia, but were not found with hyperparathyroidism alone in the few patients tested.

Warshaw, A. L.; Imbembo, A. L.; Civetta, J. M.; Daggett, W. M. (General Surgical Services, Massachusetts General Hosp., and Dept. of Surg. Harvard Medical School, Boston, Mass.): SURGICAL INTERVENTION IN ACUTE NECROTIZING PANCREATITIS. Am. J. Surg. *127*:484-91, 1974.

Verbatim summary. Thirty-eight patients with acute pancreatitis were treated with an operation that included cholecystostomy, gastrostomy, feeding jejunostomy, and sump drainage of the peripancreatic lesser sac. Of eleven patients judged to be dying of fulminant pancreatitis in the first forty-eight hours of treatment, nine improved immediately and seven survived. Of eighteen patients who underwent operation because of persistent or increased signs of inflammation two weeks after admission, seven had an immediate favorable response, mainly due to drainage of abscesses, necrotic pancreas, or pseudocysts, and twelve survived. Nine patients with pancreatitis of lesser severity received no apparent benefit from the operation.

Gastrostomy and jejunostomy were used for prolonged treatment of intestinal dysfunction in nine patients. In six patients the cholecystostomy helped to control bacterial cholangitis. In seven patients the peripancreatic sumps provided tracts for the late drainage of abscesses, pancreatic fistulas, and sloughed pancreas.

The major problem after surgery was the development of intra-abdominal abscesses in sixteen patients. Of the thirteen deaths, ten were directly related to these abscesses. An aggressive approach to re-exploration and drainage of late pancreatic abscesses in patients with necrotizing pancreatitis should improve still further the survival rate of patients.

The survival of patients with severe pancreatitis resistant to standard medical measures appears to be improved by this operative approach.
