ABSTRACTS

Auzuki, Tadao; Ferris, Robert K.; and Gordon, Edwin E. (Dept. of Med., Diabetes & Metabolism Division, New York, Univ. Med. School, New York): INHIBITION OF RENAL GLUCONEOGENESIS BY QUINOLINATE AND HYDRAZINE IN DIABETIC RATS. Endocrinology 97:1058-60, October 1975.

Renal as well as hepatic gluconeogenesis is inappropriately accelerated in the diabetic state when plasma glucose levels are elevated. Known regulatory mechanisms influence gluconeogenesis in both organs. However, under certain circumstances gluconeogenesis may be affected in one organ and not the other. Recent studies with the tryptophan metabolite quinolinate suggest that hepatic gluconeogenesis in the diabetic is unaffected by this agent, whereas gluconeogenesis is blocked in the normal liver. These experiments have been interpreted as providing evidence for the lack of a specific physiologic repressor for gluconeogenesis in diabetic liver. In the present study, quinolinate and hydrazine are shown to be effective inhibitors of the accelerated gluconeogenesis in the renal cortex of diabetic rats. Thus, the rental gluconeogenic mechanism in diabetics retains the capacity to recognize quinolinate as an inhibitor but may be influenced by the depressed conversion of tryptophan to quinolinate in the intact diabetic organism. J.E.G.

Ensinck, John W.; Walter, Robert M.; Palmer, Jerry P.; Brodows, Robert G.; and Campbell, Robert G. (Dept. of Med., Univ. of Washington, School of Med., Seattle, Wash., & the Endo.-Metab. Unit, Monroe Community Hosp., Dept of Med., Univ. of Rochester, Sch. of Med., Rochester, N.Y.) GLUCAGON RESPONSES TO HYPOGLYCEMIA IN ADRENALECTOMIZED MAN. Metabolism 25:227-32, February, 1976.

Glucagon responses to insulin-induced hypoglycemia were measured in adrenalectomized patients and normal subjects to determine the role of epinephrine in the mediation of these responses. The mean nadir of plasma glucose and the rate of ascent to baseline levels were similar in both groups. Glucagon responses in the adrenalectomized patients were not different from those of normal volunteers. These data show that epinephrine does not contribute to the augmented glucagon release during glucopenia and is not required for recovery from hypoglycemia. C.R.S.

Frankel, Barbara J.; Gylfe, Erik; Hellman, Bo; and Idahl, Lars-Åke (Dept. of Histology, Univ. of Umeå, Sweden): MAINTENANCE OF INSULIN RELEASE FROM PANCREATIC ISLETS STORED IN THE COLD FOR UP TO FIVE WEEKS. J. Clin. Invest. 57:47-52, January, 1976.

In-vitro preservation of functionally intact islet tissue may become an important factor in the transplantation of islets for the treatment of diabetes. The authors, therefore, studied the conditions under which hand-dissected pancreatic islets from obese-hyperglycemic mice (ob/ob) would maintain their insulin content and secretory capacity during storage. Cells were stored at 37°, 22°, and 8° C. in tissue culture medium with a glucose concentration ranging from 1-18 mM for one to five weeks. The results showed that the insulin secretory capacity was best maintained when islets were stored in a high glucose concentration at 8° C. with short weekly periods at 37°; glucose-stimulated insulin release in islets stored in this manner after five weeks was one-third that of fresh islets and similar to that of islets stored for only one week at 37° C. R.R.

Gavin, James R. III; Kahn, C. Ronald; Gorden, Phillip; Roth, Jesse; and Neville, David M. Jr. (Diabetes Branch, National Institute of Arthritis, Metab., and Digestive Diseases, N.I.H., Bethesda, Md.): RADIORECEPTOR ASSAY OF INSULIN: COMPARISON OF PLASMA AND PANCREATIC INSULINS AND PROINSULINS. J. Clin. Endocrinol. Metab. 41:438-45, 1975.

Porcine proinsulin, related intermediates, and plasma immunoreactive insulin components have been studied by radioreceptor assay. In the purified rat-liver membrane or cultured human-lymphocyte radioreceptor assay, porcine proinsulin is 5 per cent, split proinsulin 6 per cent (54-55 split in connecting peptide), desdipeptide proinsulin 20 per cent (deletion of amino acids 62 and 63 of connecting peptide), and desnonapeptide proinsulin 27 per cent (deletion of amino acids 55-63 of connecting peptide) as active as porcine insulin in both assay systems; these values closely parallel the in vitro bioactivity of these preparations. These findings support the conclusions of a recent study by Freychet showing that hormone receptor binding occurs by way of the insulin moiety. There is no evidence for an additional unique binding site for compounds containing the connecting peptide. The lymphocyte radioreceptor assay has the same ease of application as radioimmunoassay and is much simpler to carry out than the standard bioassay, which measures glucose oxidation in fat cells.

In the lymphocyte radioreceptor assay the human plasma immunoreactive insulin-like component has the same potency as porcine insulin per immunoreactive unit, whereas the plasma immunoreactive proinsulin-like component is only 15 per cent as active. Since both plasma immunoreactive components are somewhat less reactive than would be expected from purified human insulin and proinsulin, the data suggest that both plasma components contain immunoreactive molecules that do not react in the radioreceptor assay. If the assay is to be used as an estimate of biologic function, it must be shown that the relative affinity of hormone preparations for binding sites correlates with a known biologic property of the preparation. The cultured lymphocyte and liver membrane receptors appear to satisfy this requirement and under appropriate conditions may be used to estimate the in-vitro biologic potency of pancreatic and plasma insulins and proinsulins. S.L.A.

Kahn, C. Ronald; Megyesi, Klara; and Roth, Jesse (Diabetes Branch, NIAM&DD, N.I.H., Bethesda, Md. 20014): NONSUPPRESSIBLE INSULIN-LIKE ACTIVITY OF HUMAN SERUM—A POTENT INHIBITOR OF INSULIN DEGRADATION. J. Clin. Invest. 57:526-29, February, 1976.

Nonsuppressible insulin-like activity soluble in acid ethanol (NSILA-S) is a peptide chemically distinct from insulin but similar to insulin in biologic activity. Both insulin and NSILA-S can occupy the insulin receptor in liver, fat, and cultured lymphocytes and the distinct NSILA-S receptor in liver membranes. In the present study the authors have demonstrated that the insulin-degrading enzyme in the liver also recognizes both insulin and NSILA-S. Furthermore, NSILA-S can act as a competitive inhibitor of insulin degradation and appears to be about 100 times as potent as insulin in this action. It is likely that NSILA-S in concentrations known to exist in the plasma significantly reduces insulin degradation in vivo. R.R.

Koncz, Lajos; Davidoff, Frank; DeLellis, Ronald A.; Selby, Mark; and Zimmerman, Clarence E. (Depts. of Med. & Surg., Beth Israel Hosp. & Harvard Med. Sch., & Dept. of Path., Tufts Univ. Med. Sch., Boston, Mass): QUANTITATIVE ASPECTS OF THE METABOLIC RESPONSE TO PANCREATIC ISLET TRANSPLANTATION IN RATS WITH SEVERE KETOTIC DIABETES. Metabolism *25*:147-56, February, 1976.

Severe, ketotic diabetes was produced by the injection of streptozotocin in adult rats. Transplantation of intact islets of Langerhans from syngeneic adult donors into a muscle pocket or a pouch created from pancreatic tissue eliminated ketonemia in the immediate postoperative period while ketonemia persisted in the sham-operated controls. Transplanted animals manifested weight gain and a decrease in blood sugar levels compared with controls; the differences were most marked in animals receiving the largest numbers of islets. In one animal whose metabolic indices returned to normal for eight weeks, the reappearance of diabetes was again reversed by a second transplantation. These studies demonstrate that islet tissue from adult donors can function in severely diabetic ketotic hosts and metabolic response to transplantation is a function of the ratio of islet tissue to body mass. C.R.S.

Lefebvre, Pierre J.; and Luyckx, Alfred S. (Div. of Diabetes, Inst. of Med., Univ. of Liège, Belgium): EFFECT OF ACUTE KIDNEY EXCLUSION BY LIGATION OF RENAL ARTERIES ON PERIPHERAL PLASMA GLUCAGON LEVELS AND PANCREATIC GLUCAGON PRODUCTION IN THE ANESTHETIZED DOG. Metabolism *24*:1169-76, October, 1975.

Bilateral renal exclusion in the dog resulted in a marked increase in arterial plasma glucagon. Ligation of the renal arteries resulted within 30 minutes in a 200 per cent elevation and within 90 minutes a 357 per cent elevation in plasma glucagon above the mean basal level. Comparable increments were observed when basal plasma glucagon was suppressed by intravenous infusion of glucose. The rate of production of glucagon was not significantly increased by kidney exclusion. These findings demonstrate that the abrupt cessation of renal glucagon uptake is the major factor in the rise in plasma glucagon levels observed after ligation of renal arteries and provide an explanation for the high plasma glucagon values reported in cases of severe renal failure. C.R.S.

Lickley, H. L. A.; Chisholm, D. J.; Rabinovitch, A.; Wexler, M.; and Dupre, J. (Fraser Lab. for Res. in Diabetes Mellitus and the McGill Univ. Clinic, Royal Victoria Hosp., Montreal, Quebec, Canada): EFFECTS OF PORTACAVAL ANASTOMOSIS ON GLUCOSE TOLERANCE IN THE DOG: EVIDENCE OF AN INTERACTION BETWEEN THE GUT AND THE LIVER IN ORAL GLUCOSE DISPOSAL. Metabolism *24*:1157-68, October, 1975.

Glucose infusions were given into the duodenum (ID), the portal vein (IP), or a peripheral vein (IV) to intact dogs or dogs with portacaval anastomoses. In the intact animals, ID glucose tolerance was better than IV and IP glucose tolerances. Portacaval anastomosis with ligation of the portal vein resulted in impairment of ID glucose tolerance and did not affect IV glucose tolerance. The impairment in tolerance to enterically administered glucose in dogs with portacaval shunts and the similarity in IP and IV glucose tolerance in intact dogs suggest that both the liver and the gut are implicated in determination of oral glucose tolerance through mechanisms that have negligible effects on responses to parenterally administered glucose. The results demonstrate that the hepatic contribution is not dependent on portal venous perfusion of the liver and that a humoral interaction between the gut and the liver occurs which is not dependent on pancreatic endocrine responses. C.R.S.

Long, C. L.; Kinney, J. M.; and Geiger, J. W. (Dept. of Surg., College of Physicians and Surgeons of Columbia Univ., New York, N.Y.): NONSUPPRESSABILITY OF GLUCONEOGENESIS BY GLUCOSE IN SEPTIC PATIENTS. Metabolism *25*:193-201, February, 1976.

Labeled alanine administered to septic patients provided data on the incorporation of alanine into glucose and the extent of alanine oxidation in the presence of severe infection. A twofold increase of alanine into glucose was noted in sepsis. This increase was observed while patients received a constant glucose infusion prior to and during the injection of alanine. Failure of glucose to decrease the gluconeogenic response during sepsis indicates that control mechanisms for glucose synthesis are modified following injury and influence the abnormal carbohydrate metabolism observed in stressful states. The contribution of alanine carbon to oxidation was the same in the control and septic groups as determined by per cent of the labeled alanine dose expired. Since control subjects received glucose during the study with and without amino acids, it was apparent that nutritional intake and injury had minimal effect on oxidation of alanine. These data suggest that transamination is not affected by sepsis and that there is no inhibition of pyruvate oxidation in this condition. An increased flux of amino acids from muscle to liver in injury provides substrate for increased gluconeogenesis. The nonsuppressibility of this effect during injury and infection requires further study to define the stimulus for this response. C.R.S.

Mitsuhashi, O.; Schucher, R.; and Kalant, N. (Lady Davis Inst. for Med. Res. of the Jewish Gen. Hosp., Montreal, Quebec, Canada): RELATION BETWEEN INSULIN AND GLUCOSE FLUX RATES IN THE DOG. J. Lab. Clin. Med. *86*:683-89, October, 1975.

This study was designed to examine the relationship of the plasma insulin concentration to the insulin secretion rate and the relationship between glucose utilization and the rate of disappearance of insulin from the plasma. Adult dogs were anesthesized with pentobarbital, and infusions were made into a femoral vein, while blood for insulin radioimmunoassay and glucose was sampled from the contralateral femoral artery; glucose and insulin fluxes were determined under steady-state conditions. For insulin flux rates, the dogs were prepared by removal of the intestinal tract, pancreas, and spleen and then infused with unlabeled and ^{131}I-labeled insulin at predetermined rates. Insulin specific activity was determined. It was shown that the rate of removal of labeled insulin was identical to that of unlabeled insulin and that iodinated insulin was a reliable metabolic tracer of native insulin in plasma. Also, the concentration of plasma insulin was found to vary directly with its rate of secretion and degradation (with concentrations up to 80 μU./ml.). In each animal there was also a linear relationship between glucose flux rate and insulin concentration, but there was considerable interanimal variation. T.G.S.

Norwich, K.H.; Fluker, G.; Anthony, J.; Popescu, I.; Pagurek, B.; and Hetenyi, Jr., G. (Dept. of Physiol. & Inst. of Biomed. Eng., Univ. of Toronto, Dept. of Physiol. & Computing Center, Univ. of Ottawa, & Dept. of Systems Eng., Carleton Univ., Ottawa): THE DEVELOPMENT OF A GLUCOSE CLAMP. Metabolism *24*:1221-30, November 1975.

A control system has been devised for clamping (i.e., holding at a steady level) the concentration of blood glucose in the hyperglycemic region of a normal unanesthetized dog. The operation of the clamp does not require the use of radioisotopes but is based on a model of the canine glucoregulatory mechanism assembled from the results of experiments in which radioisotopes were used. In the clamping procedure, blood glucose is measured continuously and calculations are derived for computerized estimation of the required rate of glucose infusion. Possible clinical applications of the glucose clamp include measurement of glucose turnover rates and the study of the effects of glucose turnover on insulin secretion with constant glucose levels. C.R.S.

Olefsky, Jerrold; Bacon, Virginia C.; and Baur, Sonia (Div. of Metab. & Gastroenterology, Stanford Univ. Sch. of Med., & Palo Alto V. A. Hosp., Palo Alto, Calif.): INSULIN RECEPTORS OF SKELETAL MUSCLE: SPECIFIC INSULIN BINDING SITES AND DEMONSTRATION OF DECREASED NUMBERS OF SITES IN OBESE RATS. Metabolism *25*:179-91, February, 1976.

A membrane preparation obtained from rat striated muscle was found to bind ^{125}I-insulin rapidly and specifically. The optimal steady-state conditions are described and are associated with insignificant degradation of insulin. Binding of regular insulin was readily inhibited at physiologic concentrations of unlabeled insulin. The specificity of the receptor sites for insulin was demonstrated by the finding that high concentrations of other peptide hormones were without effect on insulin binding and that insulin analogues inhibited binding in proportion to their biologic activity. When membranes from older, fatter rats were compared with those from young, lean animals, it was found that insulin binding to membrane receptors was significantly decreased in the former group. These studies demonstrate that specific insulin receptors exist in skeletal muscle plasma membranes and that the membranes of older, fatter rats have fewer receptors than those from young, lean animals. C.R.S.

Palmer, Jerry P.; Henry, David P.; Benson, James W.; Johnson, David G.; and Ensinck, John W. (Dept. of Med., Univ. of Wash., Sch. of Med., Seattle, Wash.): GLUCAGON RESPONSE TO HYPOGLYCEMIA IN SYMPATHECTOMIZED MAN. J. Clin. Invest. 57:522-25, February, 1976.

Insulin-induced hypoglycemia in subjects with neurologically complete traumatic cervical cord transections failed to induce a rise in norepinephrine levels, providing strong evidence for total interruption of the hypothalamic-sympathetic outflow. Nevertheless, peak and total immunoreactive glucagon responses were similar to those of control subjects. It was therefore concluded that the glucagon response to hypoglycemia is not mediated by the sympathetic nervous system and is likely to be a direct response of the alpha cell to hypoglycemia. R.R.

Phelps, Richard L.; Bergenstal, Richard; Freinkel, Norbert; Rubenstein, Arthur H.; Metzger, Boyd E.; and Mako, Mary (Center for Endocr., Metab., and Nutrition, Northwestern Univ. Med. Sch., Dept. of Med., Univ. of Chicago and Pritzker School of Med., Chicago, Ill.): CARBOHYDRATE METABOLISM IN PREGNANCY: XIII. RELATIONSHIPS BETWEEN PLASMA INSULIN AND PROINSULIN DURING LATE PREGNANCY IN NORMAL AND DIABETIC SUBJECTS. J. Clin. Endocrinol. Metab. *41*:1085-91, 1975.

Twenty-seven women with normal carbohydrate metabolism or diabetes mellitus were studied for the relationship between circulating insulin and proinsulin during late pregnancy. They had varying degrees of carbohydrate intolerance. It was found that most of the normal gestational increase in basal and glucose stimulated total insulin immunoreactivity (TIR) can be ascribed to insulin rather than disproportionate increments in proinsulin-like components. Mild carbohydrate intolerance during pregnancy is not attended by abnormalities in plasma proinsulin. This is contrasted with subjects who required subsequent insulin therapy. In this group, the basal proinsulin levels were elevated in four of the nine subjects. Pregnancy per se does not modify appreciably the relationship between plasma insulin and proinsulin in normal and mildly diabetic subjects, although there may be some tendency for proinsulin to account for a smaller proportion of TIR. S.L.A.

Pozefsky, Thomas; Tancredi, Robert G.; Moxley, Richard T.; Dupre, John; and Tobin, Jordan D. (Dept. of Med., Johns Hopkins Univ. School of Med., Baltimore, Maryland; The Fraser Labs., Royal Victoria Hosp., McGill University, Montreal; Gerontology Res. Centr. N.I.H. on Aging, Baltimore City Hospitals, Baltimore, Maryland): EFFECTS OF BRIEF STARVATION ON MUSCLE AMINO ACID METABOLISM IN NONOBESE MAN. J. Clin. Invest. 57:444-49, February, 1976.

Prolonged starvation has been clearly demonstrated to result in a decrease in amino acid release from skeletal muscle and, hence, a decrease in gluconeogenesis; this adaptive response is coincident with a change in the major brain substrate from glucose to ketone bodies. Since brief fasting is associated with an increase in gluconeogenesis, the authors examined the amino acid release across forearm muscles in postabsorptive and three-day fasted normal male subjects. The release of the principal glycogenic amino acid, alanine, was increased by 59 per cent, while the release for all amino acids was increased 69 per cent. Basal insulin levels were reduced and glucagon levels increased as expected. The authors suggest that since the observed changes in glucagon levels would not account for the increased amino acid release the decrease in insulin levels appears to be a more important controlling mechanism. R.R.

Rossini, Aldo A.; Aoki, Thomas T.; Ganda, Om P.; Soeldner, J. Stuart; and Cahill, George F. Jr. (E.P. Joslin Res. Lab. Dept. of Med., Harvard Med. School, and Peter Bent Brigham Hosp., Boston, Mass.): ALANINE-INDUCED AMINO ACID INTERRELATIONSHIPS. Metabolism *24*:1185-92, October, 1975.

Normal subjects receiving alanine by oral and intravenous routes manifested significant increases in whole-blood levels of threonine, serine, glutamine, proline, glycine, and alpha-amino-n-butyric acid. These increments were due to increased concentrations in the plasma compartment while whole blood levels of leucine, valine, and isoleucine declined because of decreases in the cell compartment. Plasma glucagon levels rose in both studies, while insulin levels rose significantly only during the oral study; glucose concentrations were not affected in either study. Plasma FFA and blood glycerol levels declined, while lactate and pyruvate increased. The data demonstrate that administration of large amounts of alanine is capable of inducing significant alterations in levels of other amino acids and substrates as well as changing hormone levels. C.R.S.

Sacca, L.; and Perez, G. (Inst. of Med. Path. & Clin. Methodology, II Faculty of Med., Univ. of Naples, Naples, Italy): INFLUENCE OF PROSTAGLANDINS ON PLASMA GLUCAGON LEVELS IN THE RAT. Metabolism *25*:127-30, February, 1976.

The intravenous infusion of prostaglandins (PGE_1 and PGA_1)

in sympathectomized and propranolol-treated rats was studied with respect to their effect on plasma glucose and glucagon. PGE_1 increased glucose and glucagon levels while PGA_1 had no effect. Because PGE_1 reduced the arterial blood pressure by 20 per cent, this compound may have acted through reflex sympathetic nervous system hyperactivity. The increases in plasma glucagon induced by PGE_1 were found to occur in sympathectomized or in beta-blocked animals. Thus, mediation through sympathetic nervous system activity or during stimulation of pancreatic beta receptors appear to be excluded as the mechanism of PGE_1 action. The data suggest that PGE_1-induced increases in plasma glucagon may be due to increased secretion of plasma glucagon stimulated directly by this compound. C.R.S.

Singh, Sant P.; and Patel, Dhanooprasad G. (V. A. Hosp., Downey, Ill., and the Dept. of Med., Chicago Med. Sch., Downey, Ill.): EFFECTS OF ETHANOL ON CARBOHYDRATE METABOLISM: I. INFLUENCE ON ORAL GLUCOSE TOLERANCE TEST. Metabolism *25*:239-43, February, 1976.

Glucose tolerance in rats was determined following the intragastric administration of saline, glucose, ethanol, and ethanol with glucose. Saline or ethanol alone produced no significant changes in blood glucose or plasma insulin concentrations. Addition of ethanol to glucose load resulted in glucose intolerance and to lower insulin responses than those obtained with glucose alone. These data suggest that ethanol per se has no effect on blood glucose or plasma insulin. Ethanol given together with glucose, however, produces glucose intolerance as well as inhibition of glucose-mediated insulin response. C.R.S.

Starr, Jerome I.; Juhn, Doojung D.; Rubenstein, Arthur H.; and Kitabchi, Abbas E. (Dept. of Med. and Diabetes Endocrinology Center, Univ. of Chicago, Chicago, Illinois; Pritzker Sch. of Med., Chicago; and Depts. of Med. & Clinical Res. Cntr. Univ. of Tennessee, Memphis): DEGRADATION OF INSULIN IN SERUM BY INSULIN SPECIFIC PROTEASE. J. Lab. Clin. Med. *86*:631-37, October, 1975.

Since proinsulin cross-reacts with antibodies to both insulin and C-peptide, the direct measurement of proinsulin in serum is at present impossible. It is possible to estimate serum proinsulin by radioimmunoassay if proinsulin is separated from insulin by gel filtration. An alternative proinsulin assay has been described that destroys insulin by incubation of serum with an insulin-specific protease and designates the residual insulin reactivity as proinsulin. However, proinsulin concentrations estimated by the protease method have been shown to overestimate proinsulin, especially in the range of 0-50 μU./ml. In this study, it was found that insulin specific protease failed to degrade insulin completely and that residual insulin reactivity measured after protease treatment represented both proinsulin and undegraded insulin. The failure of the enzyme to completely degrade insulin may be due either to inhibition of the enzyme by a substance in serum or to products formed by insulin degradation. Since the enzymatic method is not specific, the authors recommend caution in using this technic for measurement of proinsulin-like components. T.G.S.

Tattersall, Robert B.; Pyke, David A.; Ranney, Helen M.; and Bruckheimer, Sally M. (King's Coll. Hosp., London, and State Univ. of New York at Buffalo): HEMOGLOBIN COMPONENTS IN DIABETES MELLITUS: STUDIES IN IDENTICAL TWINS. N. Engl. J. Med. *293*:1171-73, Dec. 4, 1975.

Because of the reported increase in fast-moving hemoglobin in diabetics, the authors looked at this in concordant twins with juvenile diabetes and in discordant twins. They confirmed the finding that diabetics had a higher proportion of hemoglobin A_{1a-c}. However, the normal levels in identical twins without diabetes led the authors to conclude that the increase in fast hemoglobin was not genetically linked to diabetes but resulting from the metabolic derangement of diabetes. H.M.

Wands, J. R.; LaMont, J. T.; Mann, E.; and Isselbacher, K. J. (Dept. of Med., Harvard Med. Sch., and Mass. General Hosp. Boston, Mass.): ARTHRITIS ASSOCIATED WITH INTESTINAL-BYPASS PROCEDURE FOR MORBID OBESITY. New Engl. J. Med. *294*:121-24, January 15, 1976.

Five patients with intestinal-bypass procedures were studied with regard to circulating cryoproteins. In three of the patients, cryoprotein complexes were found. (These three had arthritis, while the other two did not.) They were composed of IgG, IgM, and IgA and complement components C_3, C_4, and C_5. IgG antibodies against E. coli and B. fragilis were found in the cryoprotein complexes. No antibacterial antibodies were found in the IgM and IgA components of the cryoproteins. C_3 activator fragment of C_3 proactivator was shown in acute-phase serum of two of the patients with cryoprotein complexes. The finding of antibodies against bacterial flora that are known to overgrow in the blind loop after bypass surgery associated with arthritis and cryoprotein complexes indicates that bypass surgery for obesity is not without hazard. Other hazards of this procedure are pointed out in an editorial in this same issue by W. Faloon. These are oxalate renal stones and amino acid deficiencies. H.G.M.

Young, James B.; Landsberg, Lewis; and Knopp, Robert H. (Thorndike Mem. Lab., Boston City Hosp. & Beth Israel Hosp., Dept. of Med., Harvard Med. Sch., Boston, Mass., & Harborview Med. Center, Dept. of Med., Univ. of Wash. Med. Sch., Seattle): EFFECT OF INTRAVENOUS GLUCAGON ON URINARY CATECHOLAMINE EXCRETION IN NORMAL MAN. Metabolism *25*:233-37, February, 1976.

Urinary epinephrine and norepinephrine were measured at two-hourly intervals after intravenous injection of saline, glucagon, or insulin. Urinary epinephrine was increased only slightly by both saline and glucagon but rose 24-fold following insulin hypoglycemia. No rise was detected in norepinephrine following any test. These results suggest that epinephrine secreted after glucagon injection is due to the stress of the test and that insulin hypoglycemia remains the only proved method of assessing adrenal catecholamine reserve. By contrast, in pheochromocytoma, glucagon may be specific for catecholamine secretion. C.R.S.

Erratum

The Editors regret that a word in the title of a paper that appeared on page 223 of the March issue was misspelled. The word "maternity" should have been "maturity." The correct title is: "Peripheral T-Lymphocytes in Juvenile-onset Diabetics (JOD) and in Maturity-onset Diabetics (MOD)."