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)."