tration of sodium bicarbonate. Increments in renal PEPC activity following ammonium chloride in normals or in acidotic diabetic animals were similar for any given reduction in plasma carbon dioxide. It was concluded that the increase in hepatic PEPC is secondary to the defect in carbohydrate metabolism, while in the renal cortex the increase in PEPC activity during diabetes is secondary to acidosis and is independent of the defect in carbohydrate metabolism. C.R.S.

Niki, Atsushi; Niki, Hatsumi; Miwa, Ichitomo; and Okuda, Jun (Dept. of Intern. Med., Sch. of Dentistry, Aichigakuin Univ., and Dept. of Clin. Biochem., Faculty of Pharmaceutical Science, Meijo Univ., Nagoya, Japan): INSULIN SECRETION BY ANOMERS OF D-GLUCOSE. Science *186*:150-51, October 11, 1974.

Verbatim summary. Isolated rat islets were incubated for five minutes in the media containing either the α or β anomer of D-glucose (2 mg. per milliliter). The amounts of secreted insulin and changes of anomer ratio were concomitantly determined. In spite of rapid mutarotation, significantly greater stimulation of insulin secretion was observed by α-D-glucose than by β-D-glucose.

Pagliara, Anthony S.; Stillings, Susan N.; Hover, Barbara; Martin, Duane M.; and Matschinsky, Franz M. (Edward Mallinckrodt Depts. of Pharmacol. and Pediat., Washington Univ. Sch. of Med.; St. Louis, Mo.): GLUCOSE MODULATION OF AMINO-ACID-INDUCED GLUCAGON AND INSULIN RELEASE IN THE ISOLATED PERFUSED RAT PANCREAS. J. Clin. Invest. *54*:819-32, October 1974.

Glucose- and amino-acid-induced glucagon and insulin release were studied in the isolated perfused rat pancreas. The study showed that (1) glucagon and insulin were both secreted at a low basal rate, (2) a biphasic pattern of glucagon release to amino acids as well as for insulin was identified, (3) there was a graded stimulation of insulin and glucagon release to arginine or an amino acid mixture, (4) physiologic glucose concentrations (5 mM) markedly augmented insulin release and inhibited glucagon release induced by arginine or an amino acid mixture, and (5) the threshold glucose concentration for glucagon inhibition was lower than for insulin release, the first phase for both being more sensitive to glucose than the second phase. The authors discuss these data with reference to the postulate that there are receptors for both glucose and amino acids in the pancreatic alpha and beta cells. RR.

Peracchi, M.; Reschini, E.; Cantalamessa, L.; Giustina, G.; Cavagnini, F.; Pinto, M.; and Bulgheroni, P. (First Inst. of Clin. Med. and Second Inst. of Med. Path., Univ. of Milan, Milan, Italy): PRELIMINARY REPORT: EFFECT OF SOMATOSTATIN ON BLOOD GLUCOSE, PLASMA GROWTH HORMONE, INSULIN, AND FREE FATTY ACIDS IN NORMAL SUBJECTS AND ACROMEGALIC PATIENTS. Metabolism *23*:1009-15, November 1974.

Somatostatin, administered by infusion, lowered the plasma growth hormone (GH) and IRI levels in seven acromegalic patients. There was no effect observed on blood glucose, while plasma free fatty acid levels were increased. In normal subjects, somatostatin inhibited plasma GH and IRI responses during an arginine-stimulation test. Similarly, hypoglycemia-induced GH release was blocked by somatostatin in two normal subjects. C.R.S.

Rado, Janos P.; Szende, Laszlo; and Marosi, Judit (Isotopic Dept. and Metabolic Unit, Janos Hosp., Budapest, Hungary): INFLUENCE OF GLYBURIDE ON THE ANTIDIURETIC RESPONSE INDUCED BY 1-DEAMINO-8-D-ARGININE VASOPRESSIN (DDAVP) IN PATIENTS WITH PITUITARY DIABETES INSIPIDUS. Metabolism *23*:1057-63, November 1974.

Glyburide was demonstrated to have a diuretic action in six patients with diabetes insipidus ingesting an ad-libitum fluid intake. Graded doses of arginine vasopressin were administered and produced a progressive decrease in the diuretic action of glyburide. The antidiuretic action of arginine vasopressin was significantly decreased by simultaneous administration of glyburide. Apparently, glyburide competitively inhibits the action of vasopressin, and its diuretic action in patients with diabetes insipidus may be mediated through inhibition of the peripheral action of residual antidiuretic hormone. C.R.S.

Walter, Robert M.; Dudl, R. James; Palmer, Jerry P.; and Ensinck, John W. (Dept. of Med., Univ. of Washington Sch. of Med., Seattle, Wash.): THE EFFECT OF ADRENERGIC BLOCKADE ON THE GLUCAGON RESPONSES TO STARVATION AND HYPERGLYCEMIA IN MAN. J. Clin. Invest. *54*:1214-20, November 1974.

There are conflicting data concerning the effect of the adrenergic system on glucagon secretion; in this study the effects of alpha and beta receptor blockade with phentolamine and propranolol, respectively, did not modify the augmented glucagon levels seen during an eighty-four-hour fast and during insulin-induced hypoglycemia in healthy nonobese young men. Thus, the adrenergic system does not appear to modulate glucagon secretion during fasting or insulin-induced hypoglycemia. R.R.

Weindling, Howard; and Henry, John B. (Dept. of Pathology, Upstate Medical Center, State Univ. of New York, Syracuse, N.Y.): LABORATORY TEST RESULTS ALTERED BY "THE PILL." J.A.M.A. *229*:1762-68, September 1974.

This is a review of the metabolic effects of oral contraceptives. It contains concise and up-to-date information regarding the various metabolic alterations that occur following the oral administration of the pill. These include the physiologic effects on the renin-angiotensin system, which sometimes leads to hypertension, the occasional side effects of hepatic dysfunction, changes in binding proteins leading to changes in serum adrenal steroid concentration, and alterations in thyroid function tests. The influence of the oral agents on carbohydrate metabolism and lipid metabolism and the occasional effects on the hematologic system are also mentioned. This is an excellent review for the practicing physician on the metabolic alterations that occur in patients on oral contraceptives. C.M.C.

Erratum

The paper "Persisting Enhanced Proinsulin-insulin and Protein Biosynthesis (^{3}H-Leucine Incorporation) by Pancreatic Islets of the Rat after Glucose Exposure," by P. Zucker and J. Logothetopoulos, in DIABETES 24: 194-200, February, 1975, omitted a phrase on page 198. The full heading of table 2, column 3, should read "Leucine conc. (n moles per 150 μl.) in incubation medium."