ABSTRACTS

Andrews, Samuel S.; Lopez-S., Alfredo; and Blackard, William G. (Dept. of Med., Louisiana State Univ. Sch. of Med., New Orleans, La.): EFFECT OF LIPIDS ON GLUCAGON SECRETION IN MAN. Metabolism *24*: 35-44, January 1975.

The role of lipids in the control of IRG secretion in man was examined with use of oral lipid emulsion and intravenous lipid emulsion. Elevations of plasma FFA by either route demonstrated only a minor action of lipids in the control of IRG secretion. Plasma FFA and triglyceride increments failed to suppress either arginine- or hypoglycemia-induced plasma IRG elevations, but an inhibitory effect of intravenous lipid emulsion on basal IRG was demonstrated. Nicotinic acid administration was shown to elevate IRG despite a reduction in plasma FFA, an effect attributable to stress factors induced by the drug. The failure of lipids to inhibit IRG secretion at FFA concentrations inhibiting HGH secretion indicates a dissociation in secretory control mechanisms of the two hormones. C.R.S.

Dickerman, Richard M.; Twiest, Melvin W.; Crudup, James W.; and Turcotte, Jeremiah G. (Dept. of Surg., Sect. of Gen. Surg., Univ. of Michigan Med. Center, Ann Arbor, Michigan): TRANSPLANTATION OF THE PANCREAS INTO A RETROPERITONEAL JEJUNAL LOOP. Amer. J. Surg. *129*:48-54, January 1975.

Verbatim summary. A new technique of pancreatic transplantation was developed and assessed in sixty-nine dog experiments. The body and tail of the pancreas were transplanted into the side of a Roux-en-Y retroperitoneal limb of jejunum. A dual venous anastomosis of the splenic vein to the vena cava was utilized to avoid venous congestion. Mean survival with normoglycemia was 32.4 days in thirty dogs with autografts and 24.6 days in twenty-seven dogs with allografts. Two dogs with autotransplants remain alive at 106 and 128 days, and the longest normoglycemic survival achieved with an allograft was eighty-five days. With experience the incidence of pancreatitis and abscess formation decreased. The problem of venous thrombosis was eliminated and the patency of the pancreatic duct was maintained.

Dobbs, Richard; Faloona, Gerald R.; and Unger, Roger H. (V. A. Hosp., and Depts. of Intern. Med. and Biochem., Univ. of Texas Southwestern Med. Sch. at Dallas, Dallas, Tex.): EFFECT OF INTRAVENOUSLY ADMINISTERED GLUCOSE ON GLUCAGON AND INSULIN SECRETION DURING FAT ABSORPTION. Metabolism *24*:69-75, January 1975.

The intravenous administration of glucose was shown to alter markedly the glucagon and insulin responses to fat absorption in dogs. The rise in plasma glucagon induced by fat absorption was abolished during intravenous glucose administration while marked increases in plasma insulin levels were observed under these conditions. The peak insulin response to a fat meal plus glucose infusion was more than three times that observed when glucose was infused alone or with a nonabsorbable intraduodenal volume load in the form of mineral oil. This suggests that the absorption of fat elicits an enteroinsular signal that is greatly potentiated by exogenous glucose. It is speculated that glucose-induced changes in the hormonal response to a fat meal may influence significantly the metabolic effects of carbohydrate. C.R.S.

Eaton, R. Philip; Schade, D.S.; and Conway, M. (Dept. of Med., Univ. of New Mexico Sch. of Med., and Lovelace-Bataan Med. Center, Albuquerque, N. Mex.): DECREASED GLUCAGON ACTIVITY: A MECHANISM FOR GENETIC AND ACQUIRED ENDOGENOUS HYPERLIPEMIA. Lancet *2*:1545-47, December 28, 1974.

The authors propose that a resistance to the action of glucagon or a decreased secretion of glucagon plays an important role in the genesis of elevated very-low-density lipoproteins in man and animals. There are many lines of evidence that favor glucagon as a possible controlling factor of very-low-density lipoproteins. The administration of glucagon causes a reduction of VLDL in many human hyperlipoproteinemic subjects. Cobalt chloride injection in the rat is associated with a decreased sensitivity to glucagon and hypertriglyceridemia. Endogenous glucagon secretion is decreased in the genetically hyperlipemic Zucker obese rat. In human carbohydrate-induced hyperlipoproteinemia there is also a reduced secretion of glucagon. A similar state exists in women treated with oral hypoglycemic agents who develop hyperlipemia. Also, human obesity is associated with both a reduction in glucagon secretion and hyperlipoproteinemia. At the cellular level, glucagon inhibits both apoprotein and triglyceride synthesis by the liver, and a loss of this inhibitory action could account for elevated lipoprotein levels. Clofibrate therapy augments glucagon secretion in the rat and lower triglycerides. The aggregate of the data suggests that the normal hypolipoproteinemic action of glucagon may be compromised in hyperlipidemic states that are genetic, diet-induced, or drug-induced. T.G.S.

Fernandes, J.; and Blom, W. (Dept. of Pediat., Sophia Children's Hosp. and Neonatal Unit, Erasmus Univ., Rotterdam, The Netherlands): THE INTRAVENOUS L-ALANINE TOLERANCE TEST AS A MEANS FOR INVESTIGATING GLUCONEOGENESIS. Metabolism *23*:1149-56, December 1974.

Gluconeogenesis was tested by means of the intravenous injection of 0.5 gm. of L-alanine per kilogram body weight in children with glycogenoses, ketotic hypoglycemia, and fructose 1, 6-diphosphatase (FDP) deficiency. Each except the latter showed a marked increase in the initially low fasting blood glucose levels after alanine injection, while the patient in the last group showed only an increase in the initially elevated blood lactate without a rise in blood glucose. An intravenous glycerol-tolerance test produced the same responses. Blood alanine measurements showed delayed alanine elimination in the patient with FDP deficiency but a rapid decrease after alanine injection in the others. The results reveal unimpaired gluconeogenesis in all but the FDP-deficiency state. In the case of impaired gluconeogenesis an additional tolerance test, with a glucogenic substrate entering the pathway at a level different from alanine, facilitates the localization of the enzyme defect underlying impaired gluconeogenesis. C.R.S.

Fiser, Robert H., Jr.; Phelps, Dale L.; Williams, Paul R.; Sperling, Mark A.; Fisher, Delbert A.; and Oh, William (Dept. of Pediat., UCLA—Harbor Gen. Hosp., Torrance, Calif.): INSULIN-GLUCAGON SUBSTRATE INTERRELATIONSHIPS IN THE NEONATAL

SHEEP. Amer. J. Obstet. Gynec. *120*:944-50, December 1, 1974.

The pancreatic alpha and beta cell responses to glucose and deoxyglucose infusion were studied in nine healthy newborn lambs at twenty-four to thirty-six hours of age. Steady-state glucose infusion resulted in a significant, though obtunded, increase in plasma insulin concentrations within forty-five minutes; glucose/insulin ratios were higher than fetal values observed in similar studies (5.6 vs. 0.88, $p < 0.001$), indicating a more significant role of insulin in stimulating glucose disposal in the newborn period than in the fetal period. Glucose infusion resulted in a paradoxic rise in plasma glucagon concentrations (360 to 500 pg./ml., $p < 0.05$). Deoxyglucose infusions did not evoke significant changes in plasma insulin concentrations, but there was an increase in plasma glucagon levels (300 to 480 pg./ml., $p < 0.05$). The data indicate residual immaturity of insulin and glucagon secretion mechanisms in the neonatal period and suggest that such immaturity may be important in the abnormalities of glucose homeostasis in the newborn period. J.E.G.

Gabbiani, G.; Malaisse-Lagae, F.; Blondel, B.; and Orci, L. (Dept. of Path. and Inst. of Histology, Univ. of Geneva, Geneva, Switzerland): ACTIN IN PANCREATIC ISLET CELLS. Endocrinology *95*:1630-35, December 1974.

Verbatim summary. The cytoplasm of pancreatic beta cells contains a network of microfilaments 40-70 Å in diameter (cell web), which is particularly evident at the cell periphery. The use of immunofluorescent techniques to detect the presence of actin indicates that the cytoplasm of isolated and cultivated pancreatic endocrine cells contains significant amounts of actin, thus suggesting that this protein is a component of the cytoplasmic web. The correlation of immunofluorescence and ultrastructure may be useful in evaluating the role of contractile protein in beta cell function.

Hicks, T.; and Turnberg, L.A. (Dept. of Gastroenterology, Manchester Royal Infirmary, Manchester, England): INFLUENCE OF GLUCAGON ON THE HUMAN JEJUNUM. Gastroenterology 67:1114-18, December 1974.

Verbatim summary. Porcine pancreatic glucagon given by intravenous infusion reduced absorption of Na^+, Cl^-, and water in the human jejunum. A dose-response curve for glucagon indicated that the effect on ion transport was maximal to 0.6 μg./kg.-hr., and this effect decreased as the dose was increased, until, at a dose of 2.7 μg./kg.-hr., ion transport was unaffected. The mean transit time was increased by glucagon in a dose of 1.2 μg./kg.-hr., and the calculated volume and diameter of the jejunum was also increased. The levels of plasma concentration calculated to have been achieved by these infusions were above those reported under physiologic conditions, but they were within the range that could conceivably be found in pathologic conditions where these observations may have some relevance. F.G.B.

Jones, Calvin E.; Polk, Hiram C., Jr.; and Fulton, Robert L. (Dept. of Surg., Univ. of Louisville Sch. of Med., Health Sciences Center, Louisville, Kentucky): PANCREATIC ABSCESS. Am. J. Surg. *129*:44-47, January 1975.

Verbatim summary. Successful management of pancreatic abscess necessitates early diagnosis and prompt external surgical drainage. The infection is predominantly gram-negative and polymicrobic. Roentgenographic contrast studies are of particular diagnostic value. Prompt recognition and external drainage are associated most frequently with recovery. Multiple system organ failure is the typical pattern of death and should alert one to the possibility of occult sepsis, secondary to pancreatic abscess.

Kalhan, Satish, C.; Schwartz, Robert; and Adam, Peter A.J. (Dept. of Pediat., Case Western Reserve Univ., at Cleveland Metropolitan Gen. Hosp., Cleveland, Ohio): PLANCENTAL BARRIER TO HUMAN INSULIN-I^{125} IN INSULIN-DEPENDENT DIABETIC MOTHERS. J. Clin. Endocr. *40*:139-42, January 1975.

Although authors agree that insulin does not cross the placental barrier in normal women during early pregnancy, the data regarding the impermeability of the placenta to insulin at term gestation are conflicting. Three theories have been proposed: (1) that there is no transfer of insulin across the placenta at term, (2) that there is a significant transfer, and (3) that there is a bidirectional transfer. The first point of this article is to support the theory that there is no transfer of insulin across the placenta at term. Radioimmunoassay technics were used to study four normal and four insulin-dependent diabetic mothers. Secondly, this study supports the finding that anti-insulin antibodies in diabetic mothers on insulin therapy do cross the placenta at term. However, whether or not these antibodies serve as carriers for insulin into the fetus has never been studied in human beings in vivo. The last point of this report demonstrates that there is no transfer of insulin in these cases, implying that the fetal pancreas is the only source of the fetal insulin in both normal and diabetic women. S.L.A.

Lev-Ran, Arie (Diabetic Unit, Dept. of Intern. Med. "D", and Dept. of Obstet. and Gynec., Beilinson Med. Center, Petah-Tikva, and Central Endocrine Lab. of the Sick Fund, Tel-Aviv Univ. Med. Sch., Tel-Aviv, Israel): SHARP TEMPORARY DROP IN INSULIN REQUIREMENT AFTER CESAREAN SECTION IN DIABETIC PATIENTS. Am. J. Obstet. Gynec. *120*:905-08, December 1974.

Twelve consecutive subjects with severe diabetes undergoing elective cesarean section were given infusion of isotonic saline without insulin for forty-eight hours starting on the morning of the operation. In all but one case, blood glucose remained relatively stable and insulin was not resumed until fifty-four to eighty-four hours after its last injection. Three patients, in spite of the temporary withdrawal of insulin, developed hypoglycemia necessitating glucose infusion. Serum free insulin showed no definite changes after the operation, but antibody-bound insulin and insulin-binding capacity showed clear temporary drop by about half their preoperation values. J.E.G.

Liechty, R. Dale; Alsever, Robert N.; and Burrington, John (Depts. of Surg. and Med., Univ. of Colorado Med. Center, and Children's Hosp., Denver, Colo.): ISLET CELL HYPERINSULINISM IN ADULTS AND CHILDREN. J.A.M.A. *230*:1538-43, December 16, 1974.

This paper reviews thirteen patients with benign and malignant tumors or hyperplasia of the pancreas; the clinical presentation and laboratory data of these patients are presented. It is pointed out in the review that the single most reliable diagnostic test was the presence of an increased insulin concentration in the presence of a decreased glucose concentration. False positive or negative values were seen in the tolbutamide tolerance test, leucine tolerance test, and glucose tolerance test. They suggest calculating a fasting insulin/glucose ratio as follows:

$$\frac{\text{serum insulin } (\mu\text{U./ml.}) \times 100}{\text{plasma glucose (mg./100 ml.)-30.}}$$

They reported that a value <3 was found in normal subjects and a value >2 was found in six of seven cases. C.M.C.

MacCuish, A.C.; Barnes, E.W.; Irvine, W.J.; and Duncan, L.J.P. (Diabetic and Dietetic Dept., Royal Infirmary, Edinburgh, and Clin. Immunology, Univ. Dept. of Therapeutics, Edinburgh, Scotland): ANTIBODIES TO PANCREATIC ISLET CELLS IN INSULIN-DEPENDENT DIABETICS WITH COEXISTENT AUTOIMMUNE DISEASE. Lancet *2*:1529-31, December 28, 1974.

There are several lines of evidence to suggest that autoimmunity may play a role in the pathogenesis of diabetes mellitus. First, diabetes is significantly more prevalent in patients who have diseases believed to be autoimmune in their etiology (pernicious anemia, thyrotoxicosis, lymphocytic thyroiditis, idiopathic adrenal insufficiency). Second, circulating antibodies to specific organs or tissue are commonly found in subjects with diabetes. Third, in-vitro studies such as demonstration of inhibition of leukocyte migration and blastogenic transformation of lymphocytes can be shown with materials from diabetics. In the past it has not been possible to show that the sera of diabetics contain humeral antibodies directed against the endocrine pancreas. This finding has dampened enthusiasm for favoring an autoimmune origin of diabetes. The authors used an indirect immunofluorescent technic to detect circulating antibodies to pancreatic islet cells and now describe the detection of antibodies to the islets in the sera of five subjects with insulin-dependent diabetes. Their method employs thin sections of fresh human pancreas to which antibodies from the sera of patients were detected by fixation of antihuman IgG fluoresceinisothiocyanate. One hundred and five sera from diabetics were tested, and five sera from twenty subjects with both juvenile diabetes and idiopathic primary adrenal insufficiency contained islet cell antibodies. The role of anti-islet antibodies in the pathogenesis of diabetes has not been established by this work, but the data suggest that the presence of such antibodies may account for instances of insulinitis previously reported. T.G.S.

Martin, F.I.R.; and Warne, G.L. (Univ. of Melbourne, Dept. of Med., Royal Melbourne Hosp., Melbourne, Australia): FACTORS INFLUENCING THE PROGNOSIS OF VASCULAR DISEASE IN INSULIN-DEFICIENT DIABETICS OF LONG DURATION: A SEVEN-YEAR FOLLOW-UP. Metabolism *24*:1-9, January 1975.

Insulin-dependent diabetic patients with known duration of diabetes of fifteen years or more were restudied after seven years of treatment. Those with severe vascular disease, hypertension, or hypertriglyceridemia at initial examination were found to have a bad prognosis. Serum cholesterol, age, fasting blood sugar, and duration of diabetes had poor predictive value. The presence of insulin insensitivity to intravenous insulin was correlated with the presence of vascular disease and was found to be associated with a higher incidence of death and clinical deterioration in either large or small vessels over the period of study. Insulin sensitivity was reproducible and may be helpful in predicting the potential for progression of long-term vascular changes in the insulin-dependent diabetic patient. C.R.S.

Navalesi, Renzo; Pilo, Alessandro; Lenzi, Silvia; and Donato, Luigi (2nd Med. Clin., Univ. of Pisa, and C.N.R. Clin. Physiol. Lab., Pisa, Italy): INSULIN METABOLISM IN CHRONIC UREMIA AND IN THE ANEPHRIC STATE: EFFECT OF THE DIALYTIC TREATMENT. J. Clin. Endocr. *40*:70-85, January 1975.

This paper concerns the controversy over what causes the reduction in the rate of insulin degradation in uremic patients. Insulin catabolism in uremia is determined by obtaining the metabolic clearance rate and the exchangeable pool of the hormone in patients with chronic renal failure before and after dialysis compared with dialyzed anephric patients and controls. Both lack of renal tissue and toxic factors inhibiting both secretion and catabolism combine to determine the insulin metabolic pattern in uremia. The technical aspects of the methodology in obtaining measurements and the logic used in supporting the conclusions are thoroughly explained. S.L.A.

Olefsky, Jerrold M.; and Reaven, Gerald M. (Dept. of Med., Stanford Univ. Sch. of Med. and V.A. Hosp., Palo Alto, Calif.): DECREASED INSULIN BINDING TO LYMPHOCYTES FROM DIABETIC SUBJECTS. J. Clin. Invest. *54*:1323-28, December 1974.

Circulating lymphocytes isolated from adult, nonketotic, nonobese diabetics with fasting hyperglycemia were shown to have a decreased ability to bind insulin. This decreased binding was shown to be the result of a decrease in the number of available receptor sites rather than a decreased affinity of the receptor for insulin. Lymphocytes from patients with hyperglycemia secondary to chronic pancreatitis had normal insulin binding. Furthermore, this abnormality could not be transferred to normal lymphocytes by in vitro incubation with diabetic serum. The authors suggest that this defect in binding may be a primary phenomenon. R.R.

Rivlin, Richard S. (Dept. of Med., Francis Delafield and Presbyterian Hosps., and Inst. of Human Nutrition, Columbia Univ. College of Physicians and Surgeons, New York, N.Y.): DRUG THERAPY: THERAPY OF OBESITY WITH HORMONES. N. Engl. J. Med. *292*:26-29, January 2, 1975.

The author thoughtfully reviews the present methods of treating obesity by the pharmacologic use of various hormones. He begins with human chorionic gonadotropin and comments that the data showing weight loss with this agent are controversial. He also points out the recent finding that human chorionic gonadotropin has some intrinsic thyroid-stimulating hormone activity. Therefore, it may act by increasing the secretion of thyroid hormones. Administration of this hormone in children may lead to precocious puberty. Thyroid hormones certainly stimulate weight loss, but this is not sustained, and weight gain rapidly follows stopping the drug. Thyroid hormones have some unwanted effects, such as increasing oxygen consumption by the heart and increasing nitrogen and calcium loss by the body. In children they increase skeletal development and dental eruption, which may have some long-term unwanted results. Bray showed that when human growth hormone is given along with thyroid hormone oxygen consumption increases but nitrogen excretion is normal. Progesterone has been reported to be of value in the treatment of alveolar hypoventilation associated with obesity and may well have a place in the treatment of this complication of obesity.

In summary, none of the available hormonal agents available at the present time is of real value in the treatment of obesity. H.M.

Sapir, D.G.; and Owen, O.E. (Dept. of Med., General Clin. Res. Center, and O'Neill Labs. of Johns Hopkins Univ. Sch. of Med., Baltimore, Md., and Dept. of Med., General Clin. Res. Center and Fels Res. Inst., Temple Univ. Health Sciences Center, Philadelphia, Pa.): RENAL CONSERVATION OF KETONE BODIES DURING STARVATION. Metabolism *24*:23-33, January 1975.

During starvation in obese human subjects, the concentration of ketone bodies increases approximately seventy-fold, thus furnishing a significant portion of the body fuels. Renal handling of

acetoacetate and beta-hydroxybutyrate during starvation was studied to determine clearance and reabsorption rates. While renal clearance of ketone bodies remained constant, reabsorption rates increased markedly, with progressive starvation measured serially from day 3 through day 24. Both acetoacetate and beta-hydroxybutyrate reabsorption rates increased linearly when plotted against their filtered loads, and no tubular maximum transport rates could be demonstrated during ketonemia. Ketone-body conservation provides an important mechanism for prevention of large urinary losses of cations during starvation. Since ammonium becomes the major cation excreted during prolonged fasting, the increased ketone renal reabsorption of ketone bodies reduces body protein loss and aids in maintaining the elevated blood concentrations of acetoacetate and beta-hydroxybutyrate. C.R.S.

Saudek, Christopher D.; Finkowski, Michael; and Knopp, Robert H. (Thorndike Memorial Lab. and Harvard Med. Unit, Boston City Hosp., Boston, Mass.): PLASMA GLUCAGON AND INSULIN IN RAT PREGNANCY: ROLES IN GLUCOSE HOMEOSTASIS. J. Clin. Invest. 55:180-87, January 1975.

The role of glucagon and insulin in the fall in blood glucose during pregnancy was studied in mid- (twelve-day) and late (twenty-one-day) gestation in fed and fasted pregnant rats. In fed rats in midgestation, insulin levels were significantly elevated while glucose and glucagon levels were unchanged; this elevated insulin-glucagon ratio suggests insulin resistance. In fed rats in late gestation, glucose levels were significantly depressed, with a further elevation of insulin and a proportional glucagon elevation such that the insulin-glucagon ratio was unchanged. In rats fasted for forty-eight hours in late gestation the glucose levels fell even further; while insulin appropriately decreased, there was no rise in glucagon as was seen in control, nonpregnant rats. The pancreatic glucagon content or secretory reserve was not felt to be responsible for this relative reduction in glucagon levels. The authors suggest that reduced glucagon secretion in pregnancy may limit hepatic gluconeogenesis, resulting in relative hypoglycemia but protecting amino acid stores. R.R.

Unger, Roger H.; and Orci, Lelio (V.A. Hosp. and Univ. of Texas Southwestern Med. Sch., Dept. of Med., Dallas, Tex., and Inst. of Histology and Embryology, Geneva, Switzerland): THE ESSENTIAL ROLE OF GLUCAGON IN THE PATHOGENESIS OF DIABETES MELLITUS. Lancet *1*: 14-16, January 4, 1975.

The traditional concept of the pathogenesis of the hyperglycemia of diabetes is that a relative or absolute deficiency of insulin causes both a decreased uptake of glucose by the peripheral cells and an increased release of glucose by the liver. The authors propose that hyperglycemia is bihormonal in origin. An insufficient amount of insulin permits decreased uptake by the peripheral cells, and an inappropriate or elevated amount of glucagon causes hyperglycemia by stimulation of gluconeogenesis and hepatic glycogenolysis. They cite three lines of evidence to support the glucagon hypothesis. The first is that endogenous hyperglycemia has never been reported in the absence of glucagon. The second is that hyperglycemia does not occur in animals given sufficient somatostatin to suppress both insulin and glucagon secretion. The third is that suppression of glucagon secretion by somatostatin in diabetic man treated with a fixed dose of insulin permits normoglycemia. T.G.S.

ORGANIZATION SECTION

SEVENTH ALLIED HEALTH COURSE ATTRACTS 321 REGISTRANTS

Attendance at the Seventh Allied Health Postgraduate Course in Diabetes, held in Kansas City April 14-16, totaled 321 registrants including dietitians, nutritionists, nurses, social workers, program directors, and administrators from thirty-nine states and Canada. Richard A. Guthrie, M.D., and Diana W. Guthrie, R.N., M.S.P.H., were Codirectors. The Course was presented under the auspices of the Committee on Professional Education of the American Diabetes Association, Karl E. Sussman, M.D., Denver, Chairman, and was held in cooperation with the American Diabetes Association, Greater Kansas City Chapter, in conjunction with the University of Kansas School of Medicine, Wichita State University Branch; the University of Missouri at Columbia; the University of Missouri at Kansas City; and Wichita State University, Department of Nursing.

THIRTY-FIFTH ANNUAL MEETING

A complete account of the Thirty-fifth Annual Meeting of the American Diabetes Association, which was held in New York June 15-17, will be published in a coming issue of this Journal. Comparative attendance at this and previous meetings will be reported, as will the roster of Officers and Directors for the 1975-76 organizational year.

THIRTEENTH RESEARCH SYMPOSIUM

The American Diabetes Association will present its Thirteenth Research Symposium, entitled "Perspectives in Current Diabetes Research," on October 16-17 in Indianapolis. The Symposium is a project of the Committee on Research, of which Albert I. Winegrad, M.D., is Chairman. Philip Felig, M.D., New Haven, John A. Galloway, M.D., Indianapolis, and Gerold M. Grodsky, Ph.D., San Francisco, are Codirectors of the Symposium.

The program, which includes a registration form, will be sent to all members of the Professional Section. Fee for the Symposium is $50.00. Fellows, residents, and interns in a training status who have not completed five years of formal training may register for a fee of $25.00.

NOVEMBER IS DIABETES MONTH

November 1975 will be Diabetes Month, an expansion of the Association's annual Diabetes Week, to permit an all-out public-information program on diabetes. As in the past, television, radio, and newspaper materials will be available to Affiliates for use in their own communities.