Hormonal Modifiers of Insulin Action
New York Diabetes Association Symposium, Part II
October 12, 1963

The Effects of Glucocorticoids on Insulin Action

James Ashmore, Ph.D., Indianapolis

SUMMARY

The effects of insulin and glucocorticoids on carbohydrate, fat and protein metabolism of various isolated tissues have been summarized. Insulin is required for maximum glucose utilization by liver, muscle and adipose tissue, but is not required by the brain and nervous tissue. Glucocorticoids do not oppose or antagonize the primary effect of insulin which is to increase the permeability of cells to glucose. However, glucose phosphorylation by muscle is decreased by the presence of pituitary and adrenal cortical hormones in the absence of insulin.

Insulin and the glucocorticoids do appear to play opposing roles in the mobilization of fatty acids (FFA). Many hormones promote FFA release from adipose tissue; however, the full lipolytic effect is not achieved in the absence of the adrenal cortex. Insulin, on the other hand, inhibits the lipolytic effect of catecholamines, glucagon, and ACTH.

Ketosis is not observed in experimental diabetes if FFA mobilization is prevented by removal of the adrenals or pituitary. On the other hand, mobilization of FFA does not lead to ketosis if adequate insulin is available. An important aspect of glucocorticoid action in diabetes is probably in the area of FFA mobilization and ketone body production. However, the role of the adrenal cortex in protein catabolism and gluconeogenesis is of equal concern. Overproduction as well as under-utilization of glucose remains the central problem in attempting to regulate the diabetic patient.

Insulin and the glucocorticoids have opposing actions in the regulation of carbohydrate, protein and lipid metabolism. Insulin is an anabolic hormone promoting glucose utilization and synthesis of protein and fatty acids. The glucocorticoids are catabolic, and stimulate protein breakdown, fatty acid mobilization, and glucose production. Many of the metabolic alterations observed in diabetes mellitus are not the result of insulin deficiency per se but are the consequence of unopposed action of the glucocorticoids. There is little evidence that in diabetes mellitus there is any increase in corticoid production;[1] however, the action of physiological quantities of steroids in the absence of insulin may produce many of the same metabolic responses of increased corticoid secretion in the presence of functional beta cells. To this extent, glucocorticoids may be considered to antagonize the action of insulin.

Presented at the Symposium on Insulin Antagonists and Hormonal Modifiers of Insulin Action sponsored by The Clinical Society of the New York Diabetes Association, Inc., on Oct. 12, 1963.

From the Department of Pharmacology, Indiana University School of Medicine, Indianapolis, Indiana.

DISCUSSION

That hormones of the adrenal are involved in diabetes mellitus was first demonstrated by Long and Lukens.[2] Removal of the adrenals from diabetic animals results in a lowering of blood glucose, decreased nitrogen excretion and return toward normal of a number of metabolic parameters. In order to focus more clearly upon the action of insulin and glucocorticoids, let us consider briefly the effects of these hormones upon a few specific biochemical processes.

I. *Glucose utilization*

Insulin is the hormone primarily involved in the regulation of glucose utilization. The rate of glucose metabolism by body tissues is governed by two processes: (1) the rate of glucose entry into the cell (permeability) and (2) the rate of glucose phosphorylation (glucokinase activity). Insulin immediately increases glucose utilization by increasing the rate of entry of glucose into cells.[3] This action is not opposed by the glucocorticoids.

A second defect in glucose utilization in diabetic muscle is due to a decrease in glucose phosphorylation.[4] This defect is not corrected by insulin in vitro, but is influenced by administration of insulin to the intact

animal. Adrenal and pituitary hormones also appear to be involved in the regulation of glucose phosphorylation by muscle. Phosphorylation of glucose is increased above the diabetic value in tissues from adrenalectomized or hypophysectomized diabetic animals, and treatment of adrenalectomized diabetic animals with cortisol results in a decrease in glucose phosphorylation by isolated muscle preparations. Cell permeability, however, remains the primary means of regulation of glucose utilization by muscle and the effect of glucocorticoids on glucose phosphorylation does not markedly influence the rate of glucose utilization in the absence of insulin. When insulin is present, phosphorylation rather than penetration, becomes rate limiting and a reduction in glucose utilization by steroids can be demonstrated.[4]

Hepatic cells are permeable to glucose even in the absence of insulin and phosphorylation is rate limiting in glucose utilization by liver.[5] Hepatic glucokinase activity is reduced in diabetes[6] and is increased to normal in insulin treated diabetic animals. The hormones of the adrenal cortex do not appear to be involved in the regulation of glucose phosphorylation by liver.

Glucose utilization by adipose tissue is decreased in diabetes.[7] This defect is not alleviated in adipose tissue from adrenalectomized diabetic animals.[8] Insulin markedly stimulates glucose utilization by adipose tissue but it is not known whether this effect is on cell permeability or glucose phosphorylation. Cortisol decreases glucose utilization by adipose tissue in vitro, but this effect can not be demonstrated in the presence of insulin.[9]

Glucocorticoid therapy results in impaired glucose tolerance in certain patients, and a test for prediabetes is based upon this phenomenon.[10] If an adequate insulin supply is available, pretreatment with steroids does not produce any alteration in glucose tolerance; however, in the presence of inadequate insulin reserve, reduced glucose tolerance may be observed.[11]

That utilization of a tracer dose of C-14 glucose is markedly impaired in both diabetic and adrenalectomized diabetic animals[12,13] would indicate that the defect in glucose utilization in the diabetic is due to the absence of insulin. However, C-14 glucose utilization by mice is impaired by pretreatment with glucocorticoids.[14] Although this could be interpreted to mean that steroids per se decrease glucose utilization, the more likely interpretation is that the effect of corticoids on glucose utilization is due to inadequate insulin reserves.

II. *Glucose production*

Overproduction of glucose in diabetes is due to the action of adrenal cortical hormones. Using incorporation of C-14 from CO_2 into blood glucose as an index of gluconeogenesis, it has been demonstrated that CO_2 fixation is markedly increased in the diabetic, and returned toward normal in the adrenalectomized diabetic animal.[15] Administration of cortisol to adrenalectomized diabetic rats results in a prompt increase in blood glucose[16] and this increase is accompanied by an increase in conversion of pyruvate to glucose as measured in the liver slice. Steroid treatment also results in an increase in the activities of a number of enzymes involved in carbohydrate formation.

Hepatic glucose-6-phosphatase and fructose diphosphate-phosphatase activities are increased in the liver in diabetes[16,17] and are also increased by cortisone administration to normal rats.[18,19] However, the increase in activity of the specific phosphatases observed in livers of diabetic or cortisone treated rats is thought to reflect an increase in gluconeogenesis, rather than a primary cause for increased glucose production,[20] since changes in the activities of the phosphatases are observed after glucose production has been increased.

Incorporation of C-14 from CO_2 or labeled amino acids into glucose is increased in liver slices from diabetic rats.[21] Similar increases in C-14 glucose production can be produced by addition of triamcinolone 8×10^{-5} M to liver slices.[22,23] An increase in CO_2 fixation and glucose production by livers from diabetic and steroid treated rats is due in part to an increase in hepatic phosphoenolpyruvate carboxykinase activity. Increases in the activity of this enzyme (table 1) are found within four hours after steroid treatment or twelve hours after the production of an insulin deficiency with anti-insulin serum.[24]

TABLE 1

Rat liver phosphoenolpyruvate carboxykinase activity

	No. of observations	cpm C-14-O_2 incorporated per gm. wet liver
Normal fed	6	11,500±1,100
Normal fasted	9	14,300± 810
Triamcinolone treated* fed	9	21,200±1,200
Triamcinolone treated fasted	9	18,400± 970
Alloxan diabetic	6	77,500±7,800
AIS diabetic*	6	62,000±7,100

*Phosphoenolpyruvate carboxykinase activity was determined five hours after the administration of 4 mg./kg. triamcinolone, and twelve hours after injection of anti-insulin serum (AIS).

While glucocorticoids increase hepatic glucose production, insulin acts to decrease gluconeogenesis. The mechanism of the insulin effect is not clearly understood; however, the activities of various hepatic enzymes associated with increased glucose formation in diabetes are decreased by insulin. The action of insulin in this respect requires the administration of the hormone over a period of several hours to several days.[16] There is also evidence that insulin restrains hepatic glucose production in vivo.[25-28] This effect may be due in part to insulin activation of UDP-glucose-α-glucan transglucosylase and increased glycogen synthesis.[29]

The protein catabolic action of glucocorticoids also contributes to increased glucose formation by: (1) increasing extra-hepatic protein catabolism and increasing plasma amino acid levels,[30,31] (2) increasing uptake of amino acids by hepatic cells[32] and (3) increasing deamination[33] and transamination[34] of amino acids in the liver. These actions are opposed by insulin which promotes protein synthesis[35-38] by extra-hepatic tissues.

TABLE 2

Fatty acid synthesis

Type of rat	Incorporation of C-14 from pyruvate-2-C-14 into fatty acids Per cent of normal* Liver slices	Epididymal fat pads
Diabetic	5-10	10-15
Adrenalectomized diabetic	80-100	10-15
Adrenalectomized diabetic + cortisol	10-20	—

*Incorporation of C-14 from labeled pyruvate into long chain fatty acids by liver slices[16] and epididymal fat pads[8] from diabetic, adrenalectomized diabetic and cortisol treated adrenalectomized diabetic rats has been expressed as a per cent of the incorporation obtained in preparations from normal fed rats.

III. *Lipid metabolism*

Fatty acid synthesis by liver and adipose tissue is stimulated by insulin and is markedly reduced in diabetes. In the liver, lipogenesis is restored by adrenalectomy of the diabetic animal[16,39] and is depressed by glucocorticoids (table 2). Fatty acid synthesis by adipose tissue of diabetic rats is not restored following adrenalectomy[8] and insulin in the absence of glucose does not stimulate fatty acid synthesis by adipose tissue in vitro.[8] Therefore, it would appear that insulin stimulation of lipogenesis in adipose tissue is mediated through the effect of the hormone on glucose utilization.

Mobilization of fatty acids from adipose tissue is increased in diabetes[40] and is restrained by insulin. The effects of insulin on FFA release are two-fold: (a) insulin promotes glucose utilization, α-glycerol phosphate synthesis and esterification of fatty acids and (b) insulin inhibits the lipolytic activity of the catecholamines, ACTH and glucagon on epididymal fat pads.[41,42]

The glucocorticoids are involved in some way in

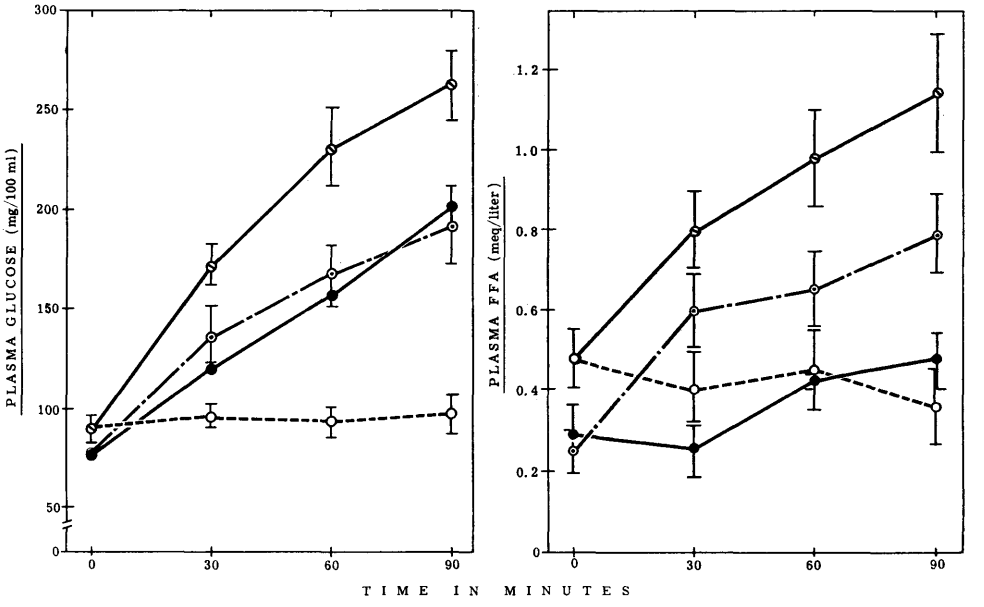

FIG. 1. Changes in plasma glucose and free fatty acids (FFA) in normal and hypophysectomized rats treated with anti-insulin serum (AIS) and epinephrine (2 mg./kg. subcutaneous). Normal + AIS (slash through circle), Normal + Epinephrine (dot in circle), Hypophysectomized + AIS (●), and Normal + Normal Guinea Pig Serum (O).

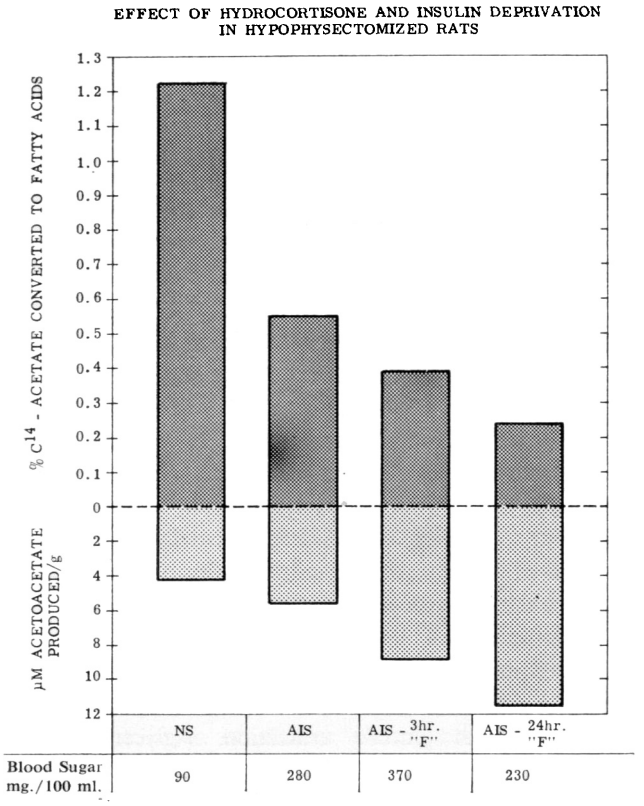

FIG. 2. Effects of hydrocortisone and anti-insulin serum (AIS) on fatty acid synthesis and ketone body production by rat liver slices. Top bars represent per cent of added C-14 from labeled acetate incorporated into fatty acids by liver slices. Lower bars indicate micromoles of acetoacetate produced per gm. wet liver per ninety minutes. NS, Normal guinea pig serum; AIS, anti-insulin serum, three hours prior to sacrifice; hydrocortisone (Compound F) was injected either three or twenty-four hours before sacrifice.

the release of FFA from adipose tissue and are said to have a permissive role. Cortisol stimulates the release of FFA from adipose tissue.[8] However, steroid induced release of FFA is not associated with increased oxidation of glucose or increased incorporation of glucose carbons into glyceride-glycerol as is epinephrine-induced lipolysis.[43]

Pancreatectomized rats usually die within forty-eight hours with severe metabolic acidosis. That ketosis does not occur in the pancreatectomized adrenalectomized rat is due in part to failure of lipid mobilization in the absence of the adrenal cortex.[44,45] Although acute insulin deficiency produced by anti-insulin serum results in a prompt rise in blood glucose and plasma FFA in normal rats, such increases are not observed when AIS is given to hypophysectomized animals (figure 1). However, pretreatment of the hypophysectomized rats with cortisol restores the hyperglycemic and

TABLE 3

Effects of insulin and glucocorticoids upon tissue meta

Effect	Liver	Muscle	Adipose
Glucose utilization			
Increased by insulin	Yes	Yes	Yes
Decreased by corticoids	No	No	Yes
Permeability to glucose			
Increased by insulin	No	Yes	Yes
Decreased by corticoids	No	Yes	?
Glucokinase activity			
Increased by insulin	Yes	No	?
Decreased by corticoids	No	Yes	?
Fatty acid synthesis			
Increased by insulin	Yes	?	Yes
Decreased by corticoids	Yes	?	No
FFA release			
Decreased by insulin		Yes	Yes
Increased by corticoids			Yes
FFA uptake			
Increased by insulin	Yes	No	Yes
Ketone body production			
Decreased by insulin	Yes		
Increased by corticoids	Yes		
Protein synthesis			
Increased by insulin	Yes	Yes	Yes
Decreased by corticoids	No	Yes	Yes

FFA response to anti-insulin serum. Acetoacetate duction by rat liver slices is increased following insulin deficiency (three hours) induced by AI creased acetoacetate production by liver from hyp sectomized AIS treated rats is observed only i animals have been pretreated with cortisol (figu The increase in acetoacetate production is asso with a simultaneous decrease in incorporation of active acetate into long chain fatty acids. Thes servations would suggest that lipid mobilizatior development of ketoacidosis in insulin deficien mals depends upon the presence of glucocorti The exact role of the steroids in this process i understood.

Table 3 has been prepared in an attempt to marize the effects of insulin and adrenal steroi various body tissues. The metabolic effects listed primarily to glucose and fat metabolism. The effe insulin and glucocorticoids are generally in oppo to each other, and the response to the hormone vary with the tissue.

SUMMARIO IN INTERLINGUA

Le Effectos de Glucocorticoides Super Insulina

Le effectos de insulina e de steroides adrenal su metabolismo de carbohydrato, grassia, e proteina in isolate tissus esseva summarisate in tabula 3. Insuli requirite pro le utilisation maximal de glucosa per

hepatic, muscular, e adipose, sed non es requirite per le cerebro e per tissus nervose. Glucocorticoides non antagonisa e non oppone le effecto primari de insulina que consiste in augmentar le permeabilitate de cellulas pro glucosa. Tamen, le phosphorylation per musculo es reducite per le presentia de hormones pituitari e adrenocortical in le absentia de insulina.

Insulina e le glucocorticoides non pare haber rolos opposite in le mobilisation de acidos grasse. Multe hormones promove le liberation de acidos grasse ab tissu adipose. Tamen, le plen effecto lipolytic non es effectuate in le absentia del cortice adrenal. Insulina, del altere latere, inhibi le effecto lipolytic de catecholaminas, glucagon, e ACTH.

Cetosis non es observate in diabete experimental si le mobilisation de acidos grasse es prevenite per le excision del glandulas adrenal o pituitari. Del altere latere, le mobilisation de acidos grasse non resulta in cetosis in caso que adequate quantitates de insulina es disponibile. Un importante aspecto del effecto de steroides adrenal in diabete es probabilemente a vider in le area del mobilisation de acidos grasse e del production de corpores cetonic. Tamen, le rolo del cortice adrenal in le catabolismo de proteina e in le gluconeogenese es de interesse non minus marcate. Hyperproduction si ben como hypoutilisation de glucosa remane le problema central in omne effortio de stabilisar le patiente diabetic.

REFERENCES

[1] Thorn, G. W., Renold, A. E., and Cahill, G. F., Jr.: The adrenal and diabetes. Diabetes *8*:337, 1959.

[2] Long, C. N. H., and Lukens, F. D. W.: The effects of adrenalectomy and hypophysectomy upon experimental diabetes in the cat. J. Exp. Med. *63*:465, 1936.

[3] Levine, R., Goldstein, M. S., Huddlestun, B., and Klein, S.: Action of insulin on the "Permeability" of cells to free hexoses, as studied by its effect on the distribution of galactose. Am. J. Physiol. *163*:70, 1950.

[4] Morgan, H. E., Henderson, M. J., Regen, D. M., and Park, C. R.: Regulation of glucose uptake in heart muscle from normal and alloxan diabetic rats: The effects of insulin, growth hormone, cortisone, and anoxia. Ann. N.Y. Acad. Sci. *82*:387, 1959.

[5] Cahill, G. F., Jr., Ashmore, J., Earle, A. S., and Zottu, S.: Glucose penetration into liver. Am. J. Physiol. *192*:491, 1958.

[6] Di Pietro, D. L., and Weinhouse, S.: Hepatic glucokinase in the fed, fasted and alloxan diabetic rat. J. Biol. Chem. *235*:2542, 1960.

[7] Winegrad, A. I., and Renold, A. E.: Studies on rat adipose tissue in vitro. I. Effects on insulin on the metabolism of glucose, pyruvate, and acetate. J. Biol. Chem. *233*:267, 1958.

[8] Jeanrenaud, B., and Renold, A. E.: Studies on rat adipose tissue in vitro. VII. Effects of adrenal cortical hormones. J. Biol. Chem. *235*:2219, 1960.

[9] Leboeuf, B., Renold, A. E., and Cahill, G. F., Jr.: Studies on adipose tissue in vitro. IX. Further effects of cortisol on glucose metabolism. J. Biol. Chem. *237*:988, 1962.

[10] Fajans, S. S., and Conn, J. W.: An approach to the prediction of diabetes mellitus by modification of the glucose tolerance test with cortisone. Diabetes *3*:296, 1954.

[11] Thorn, G. E., Renold, A. E., and Winegrad, A. I.: Some effects of adrenal cortical steroids on intermediary metabolism. Brit. Med. J. *2*:1009, 1957.

[12] Feller, D. D., Strisower, E. H., and Chaikoff, I. L.: Turnover acid oxidation of body glucose in normal and alloxan diabetic rats. J. Biol. Chem. *187*:571, 1950.

[13] Stern, M., Wagle, S. R., Sweeney, M. J., and Ashmore, J.: Studies on experimental diabetes. I. Effects of anti-insulin serum on C-14 glucose metabolism. J. Biol. Chem. *238*:12, 1963.

[14] Bontwell, R. K., and Chaing, R.: The acute effect of cortisone treatment on the utilization of glucose by the mouse. Arch. Biochem. and Biophys. *50*:461, 1954.

[15] Wagle, S. R., and Ashmore, J.: Studies on experimental diabetes. II. Carbon dioxide fixation. J. Biol. Chem. *238*:17, 1963.

[16] Ashmore, J., Hastings, A. B., Nesbett, F. B., and Renold, A. E.: Studies on carbohydrate metabolism in rat liver slices. VI. Hormonal factors influencing glucose-6-phosphatase. J. Biol. Chem. *218*:77, 1956.

[17] Weber, G., Banerjee, G., and Bronstein, S. B.: Role of enzymes in homeostasis. III. Selective induction of increases of liver enzymes in carbohydrate metabolism. J. Biol. Chem. *236*:3106, 1961.

[18] Weber, G., Allard, C., de Lamirande, G., and Cantaro, A.: Liver glucose-6-phosphatase activity and intracellular distribution after cortisone administration. Endocrinology *58*: 40, 1956.

[19] Mokrasch, L. C., Davidson, W. D., and McGilvery, R. W.: The response to glucogenic stress of fructose-1,6-diphosphatase in rabbit liver. J. Biol. Chem. *222*:179, 1956.

[20] Ashmore, J.: Physiology of glucose-6-phosphate utilization. Role of adrenal steroids. Rev. Canad. de Biol. *18*:229, 1959.

[21] Wagle, S. R., and Ashmore, J.: Interrelationships between amino acid metabolism and carbohydrate formation in insulin deficiency. J. Biol. Chem. *236*:2868, 1961.

[22] Haynes, R. C., Jr.: Studies of an in vitro effect of glucocorticoids on gluconeogenesis. Endocrinology *71*:399, 1962.

[23] Uete, T., and Ashmore, J.: Effects of triamcinolone on carbohydrate synthesis by rat liver slices. J. Biol. Chem. In Press.

[24] Ashmore, J., Wagle, S. R., and Uete, T.: Studies on gluconeogenesis. Advance in enzyme regulation. II. J. Biol. Chem. In press.

[25] de Bodo, R. C., Altszuler, N., Dunn, A., Steel, R., Armstrong, D. T., and Bishop, J. S.: Effects of exogenous and endogenous insulin on glucose utilization and production. Ann. N. Y. Acad. Sci. *82*:431, 1959.

[26] Weinhouse, S., Friedmann, M. A., and Reichard, G.: The effect of insulin on hepatic glucose production and utilization. Diabetes *12*:1, 1963.

[27] Madison, L. L., Combes, B., Adams, R., and Strickland, W.: The physiological significance of the secretion of endogenous

insulin into the portal circulation. III. Evidence for a direct immediate effect of insulin on the balance of glucose across the liver. J. Clin. Invest. 39:507, 1960.

[28] Leonards, J. R., Landau, B. R., Craig, J. W., Martin, F. I. R., Miller, M., and Barry, F. M.: Regulation of blood glucose concentration; hepatic action of insulin. Am. J. Physiol. 201:47, 1961.

[29] Villar-Palasi, C., and Larner, J.: Insulin treatment and increased UDPG glycogen transglucosylase activity in muscle. Arch. Biochem. and Biophys. 94:436, 1961.

[30] Long, C. N. H., Katzin, B., and Fry, E. G.: The adrenal cortex and carbohydrate metabolism. Endocrinology 26:309, 1940.

[31] Bondy, P. K.: Effect of adrenal and thyroid glands upon rise of plasma amino acids in eviscerated rats. Endocrinology 45:605, 1949.

[32] Christensen, H. N.: Action of cortisol on trapping of amino acids by the liver. Ciba Foundation Study Group No. 6. Metabolic effects of adrenal hormones. ed. G. E. W. Wolstenholme, F. M. O'Connor. Churchill, London, 1960, p. 56-64.

[33] Engel, L. L., and Scott, J. F.: Effects of steroid hormones on diphosphopyridine nucleotide-mediated enzymatic reactions. Recent Progress in Hormone Research 16:79, 1960.

[34] Rosen, F., Roberts, N. R., Budnick, L. D., and Nichol, C. A.: An enzymatic basis for the gluconeogenic action of hydrocortisone. Science 127:287, 1958.

[35] Wool, I. G., and Krahl, M. E.: Incorporation of C-14-amino acids into protein of isolated diaphragms. An effect of insulin independent of glucose entry. Am. J. Physiol. 196:961, 1959.

[36] Manchester, K. L., and Young, F. G.: The effect of insulin in vitro on the accumulation of amino acids by isolated rat diaphragm. Biochem. J. 75:487, 1960.

[37] Krahl, M. E.: Incorporation of (^{14}C) amino acid precursors into adipose tissue protein. An insulin stimulation not involving glucose or amino acid transport. Biochem. et Biophys. Acta. 35:556, 1959.

[38] Herrara, M. G., and Renold, A. E.: Hormonal Effects on glycine metabolism in rat epididymal adipose tissue. Biochem. et Biophys. Acta. 44:165, 1960.

[39] Brady, R. O., Lukens, F. D. W., and Gurin, S. J.: Synthesis of radioactive fatty acid in vitro and its hormonal control. J. Biol. Chem. 193:459, 1951.

[40] Frederickson, D. S., and Gordon, R. S.: Transport of fatty acids. Physiol. Rev. 38:585, 1958.

[41] Jungas, R. L., and Ball, E. G.: Studies on metabolism of adipose tissue. XII. The effects of insulin and epinephrine on free fatty acid and glycerol production in the presence and absence of glucose. Biochemistry 2:383, 1963.

[42] Mahler, R., Stafford, W. S., Tarrant, M. E., and Ashmore, J.: The effect of insulin on lipolysis. Diabetes. In Press.

[43] Cahill, G. F., Jr., Leboeuf, B., and Flinn, R. B.: Studies on rat adipose tissue in vitro. VI. Effect of epinephrine on glucose metabolism. J. Biol. Chem. 235:1246, 1960.

[44] Scow, R. O., Chernick, S. S., and Guarzo, B. N.: Ketogenic action of pituitary and adrenal hormones in pancreatectomized rats. Diabetes 8:132, 1959.

[45] Urgoiti, E. J., Houssay, B. A., and Rietti, C. T.: Hypophyseal and adrenal factors essential for ketoacidosis of pancreatectomized dogs. Diabetes 12:301, 1963.

Public Medical Forums

Six years ago, while searching for some method whereby the Niagara Falls Memorial and Mount St. Mary's hospitals and their medical staffs could render more service to the citizens of this area, one of us decided that a series of public medical forums might be of great value.

It was evident that some excellent medical information was available to the public in the newspapers, magazines, (especially in *Today's Health* published by the American Medical Association), on radio, and television. However, in spite of the efforts of the American Medical Association and the state and county medical societies much misinformation was reaching the general public. Public medical forums seemed to be an excellent way to present factual medical information, and the people in the community would hear the opinions of their own physicians on important medical subjects.

METHOD

A committee of twenty physicians was appointed to prepare a series of public forums. The original committee consisted of the chiefs and/or associate chiefs of the departments of medicine, surgery, pediatrics, otorhinolaryngology, roentgenology, neurosurgery, obstetrics and gynecology, and orthopedic surgery, plus several other specialists. Later, the chief of a department of general practice and two generalists were added. After a few years, it was evident that a smaller committee, consisting of those most interested in the public forums, was quite adequate, and the present size of this committee is fourteen physicians. The only lay member of this committee is the Director of the Niagara Falls Memorial Hospital.

It was decided that there would be three to six forums each winter, each forum to have a panel of five to eight physicians including the moderator. The members of the panels would be specialists in the subject to be discussed or outstanding generalists from the medical staffs of the two hospitals. Nationally known speakers could be obtained from outside the city (this has happened twice) if desired. Hospital personnel, other than the physicians, could appear on the panels (the chief dietitians of both hospitals and one physiotherapist have appeared). Each member of the panel would have five to eight minutes to present his part of the discussion. The audience would then be invited to submit the questions that they had written previously and to hear their questions answered. The time allowed for a single forum was two hours.

RESULTS

We are now in the sixth year of very successful public medical forums and would like to report some of our observations. Thirty-four forums have been held and the following topics discussed. They are listed according to their

(*Continued on page 361*)