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ABSTRACTS

Anderson, George E.; Kologlu, Yusuf; and Papadopoulos, Constantin (Dept. of Med., The Brooklyn-Cumberland Med. Center, affiliate of the State Univ. of New York, Downstate Med. Center, Brooklyn, N.Y.): FLUCTUATIONS IN POSTABSORPTIVE BLOOD GLUCOSE IN RELATION TO INSULIN RELEASE. Metabolism *16*:586-96, July 1967.

An hepatic vein and pancreatic vein were cannulated in dogs in order to measure glucose and immunoreactive insulin (IRI) levels in rapid succession. Hepatic-venous blood glucose values were determined at fifteen-second intervals and were correlated with IRI levels in the pancreatic and hepatic-venous blood at approximate periods. Pulsatile rises in the hepatic vein glucose levels were followed within fifteen seconds by corresponding rises in the insulin content of the pancreatic effluent blood. With an increase in insulin release the hepatic vein glucose concentration decreased. The reduction in hepatic vein glucose was accomplished by a simultaneous fall in hepatic-venous IRI. The latter increased with a rise in hepatic venous glucose levels. The results indicate that increased hepatic glucose output stimulates the release of pancreatic insulin which, in turn, causes a prompt reduction in hepatic glucose production and inhibits the circulation of insulin from liver into the peripheral circulation. The processes may represent a feedback regulatory mechanism whereby the liver controls the production of glucose as well as the availability of insulin to the tissues in accordance with needs. This control may be mediated by the liver through variations in its release of glucose which may, in turn, be influenced by glucagon or related substances. C.R.S.

Antonis, A.; Clark, M. L.; Hodge, R. L.; Molony, Margaret; and Pilkington, T. R. E. (Med. Unit, St. George's Hosp. Sch., and Dept. of Pharmacol., Royal Coll. of Surgeons, Lincoln's Inn Fields, London, England): RECEPTOR MECHANISMS IN THE HYPERGLYCAEMIC RESPONSE TO ADRENALINE IN MAN. Lancet *1*:1135-37, May 27, 1967.

The receptors responsible for the rise in concentration of blood glucose caused by administration of adrenaline have not been defined. In this study, the response to adrenaline was studied in subjects who had an alpha-receptor blockade with phentolamine, a beta-receptor blockade with propanolol, both blockades, or neither. Blockade of either receptor reduced responsiveness and blockade of both receptors abolished the hyperglycemic response to adrenaline. The usual rise in plasma lactate incident to adrenaline infusion was abolished only by beta-blockade. In obese subjects undergoing starvation the rise in glucose was prevented by beta-blockade alone. The results indicate that both alpha and beta receptors play a part in the hyperglycemic response to adrenaline. Since beta-blockade by propanolol abolished lactate response, the beta receptors to adrenaline lie only in muscle where glycolysis can cause lactate production but not hyperglycemia. Since beta blockade prevented hyperglycemic response in ketotic subjects with presumably depleted liver glycogen but not in subjects with adequate liver glycogen, the alpha receptors for adrenaline by inference must be situated in the liver. T.G.S.

Bagdade, John D.; Porte, Daniel, Jr.; and Bierman, Edwin L. (Univ. of Washington Sch. of Med., Seattle, Wash., and Veterans Administration Hosp.): DIABETIC LIPEMIA—A FORM OF ACQUIRED FAT-INDUCED LIPEMIA. New England J. Med. *276*:427-33, Feb. 23, 1967.

A report on the study of the pathogenesis of marked hyperlipemia (lactescent plasma) in five patients with chronic symptomatic diabetes and minimal ketoacidosis. The circulating triglyceride-rich fat particles obtained from the patients were found to have properties characteristic of particles of dietary rather than of endogenous origin. Levels of plasma lactescence and triglycerides were lowered by the isocaloric substitution of dietary carbohydrate for fat. Reinstitution of dietary fat resulted in massive hypertriglyceridemia. A possible explanation for the impaired assimilation of dietary fat was the finding of subnormal postheparin lipolytic activity. This suggested that tissue levels of the enzyme lipoprotein lipon were low. Decreased postheparin lipolytic activity also appears to be associated with diminished availability of insulin since immunoreactive insulin levels, obtained during lipemia development and after oral glucose administration were low. Prompt return of plasma triglycerides and lipolytic activity to normal levels occurred after insulin treatment. The triglyceride response to feeding and withdrawal of dietary fat, associated with de-