The Clinical Society of the New York Diabetes Association, an Affiliate of the American Diabetes Association, Fifth Annual Symposium Day on Diabetes Mellitus

Fat and Diabetes

The Fifth Annual All-Day Symposium of The Clinical Society of the New York Diabetes Association was held on Oct. 12, 1957, at Hunter College Playhouse Auditorium, New York City. Dr. Irving Graef, Chairman, presided.

CHAIRMAN GRAEF: May I introduce Dr. Alfred E. Fischer, President of the New York Diabetes Association, who would like to welcome you.

ALFRED E. FISCHER, M.D.: Today marks the Fifth Annual Symposium which has been sponsored by the New York Diabetes Association and its Clinical Society. This year the subject chosen is *Fat and Diabetes.*

A glance at the program will reveal that the speakers and their co-workers are, perhaps, the leading workers in the field of metabolism today.

It is particularly fortunate that a whole day can be spent having these investigators tell us the results of their latest endeavors.

I should like at this moment to express my personal thanks in advance to all of those who are participating in this Symposium for the time and the energy which they have spent in bringing their material before us.

I wish also to express our gratitude to Dr. C. J. O'Donovan and to The Upjohn Company, who have arranged to underwrite the cost of the Symposium. Without their help, this Symposium could not be held.

Ketogenesis

William C. Stadie, M.D., Philadelphia

The so-called ketone bodies are three in number: acetoacetic acid, β-hydroxybutyric acid, and acetone. The last, formed by the spontaneous nonenzymatic decarboxylation of acetoacetate, is, strictly speaking, the only true ketone. However, long usage has firmly fixed the term "ketone bodies" or "ketones" to designate these three substances. The precursors of the ketones are chiefly the long-chain fatty acids. Certain of the amino acids derived from protein are ketogenic; carbohydrates never yield ketone bodies. Ketogenesis is limited to the liver. The peripheral tissues do not produce ketone bodies from fatty acids. On the other hand, the kidney does form acetoacetate, but this is so rapidly oxidized by this organ that a net increase of ketone bodies does not occur. Ketogenesis occurs in the normal individual since traces of ketone bodies may be demonstrated in the blood and expired air. There are two conditions, however, in which ketogenesis is markedly increased, namely in starvation and in the diabetic animal.

The mode of formation of the ketone bodies from fatty acids in the liver is best understood by a brief review of the successive β-oxidation hypothesis originally developed

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