

is apparently closely connected with the carbohydrates. If the increased metabolism of fat is great enough to cause acid intoxication the condition becomes much more serious. The liver is unable to convert ammonia salts into urea. The diminished alkalinity of the blood renders it incapable of taking up as much CO_2 . CO_2 is therefore retained in the tissues and internal asphyxiation results.

A wasting diabetic is being starved as he cannot use his carbohydrates. Fats are broken down and fatty acids result, yielding diacetic acid and acetone, of which β -oxybutyric acid is the forerunner.

And acidosis may increase the wasting for autolysis proceeds faster in acid than in alkali media. Breaking down of fasting or oxygen-starved tissues produces acids which in their turn produce further breaking down of the tissues.

While the acids themselves are not directly toxic, but the associated diminished alkalinity of the blood may result in an internal asphyxiation of the tissues, expressing itself in vomiting, drowsiness, and coma. The acetonuria in delayed chloroform poisoning finds its explanation in a diseased liver which is unable to deal either with its own fat or with that reaching it from elsewhere.

Dr. J. Rose Bradford's contribution is a brief presentation of the present teaching regarding the pancreas and diabetes mellitus, with illustrative cases briefly sketched. He says diabetes is not an entity but a clinical label attached to a number of different conditions of different morbid anatomy and liable to follow different courses.

The interacinar or intralobar variety of chronic pancreatitis is that form with which diabetes is usually associated—diffuse intralobular fibrosis. It is in this form that the islets of Langerhans undergo degenerative changes. From Dr. Bradford's article one must infer that the conclusive proof is not yet adduced showing that diabetes can be attributed to the destruction of these islets, but experimental results certainly associate the glycosuria with the loss of an internal pancreatic secretion.

The paper by J. Walker Hall deals with the Basis of Therapy in this disease. He points out the necessity of arriving at a conclusion about the capacity of the tissue cells in diabetes, and regards undernutrition as the greatest danger of the diabetic subject. Abnormal, intermediate or by-products result in the unusual metabolic processes going on in the disease and these endanger the life of the cell. Already we have seen that these intermediate products express themselves in an excess of ammonia acetone and acetic acid, and quantities of these substances vary directly with the extent of the impairment of the assimilative powers of carbohydrates. Hence the necessity of increasing the activ-