tion of the pancreas. The experimental evidence of this is furnished by Ssobolen. It is remarkable that slight injury of the pancreas may produce diabetes and that extensive destruction may fail to produce such a result.

The test seems to rest as Opie has shown, not in the change in the pancreas, but the injury to the islands of Langerhans themselves. There are two types of interstitial inflammation affecting the developed pancreas. In the interlobular variety, the inflammatory process is localized at the periphery of the lobule, and implicates the island of Langerhans only when the sclerotic process has reached a very advanced stage.

In the inter-acinar pancreatitis the process is diffuse, invading the lobule and separating individual acini. The inflammatory change invades the island of Langerhans. This reminds us of another remarkable fact, that the security of the individual does not depend on the integrity of the whole pancreas. If one-quarter is left, this as surely and efficiently protects as does the whole pancreas.

It would seem as if the presence of even a very small quantity of secretion from the island of Langerhans were sufficient to stimulate some function of oxidation or combustion. Glycogen has been called the fuel of the body, stored not only in liver and muscle, but elsewhere, and the little islands of Langerhans, with minute quantities of secretion, are sufficient to start the combustion. W. H. Thompson refers to it in these terms: "The interesting researches of Otto Cohenheim show that an enzyme is produced in muscle which, if alone, does not act upon sugar, but when mixed with the secretion of the cells of Langerhans, becomes a very energetic solvent, in the same way that trypsin becomes effective only when joined with kinase ferment in the intestine. This muscle ferment is so energetic when mixed with juice of the pancreas that Cohenheim regards it as quite sufficient to account for the whole process of sugar combustion in the human body under ordinary conditions.

In the action of the adrenal glands, there has been a growing interest since 1855, when Addison, of Guy's Hospital, described a group of symptoms afterwards to be known as Addison's disease.

Interference with the function of this gland, whether by the fibro-caseous lesion of tuberculosis, simple atrophy or an affection of the abdominal sympathetic, produces, in Addison's words, "anemia, general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach and a peculiar change of color of skin." Whether this state is due to absence of a secretion from this gland or an affection of the