due either to structural change in the liver itself or to some remote influence exercised on it through its nervous mechanism; (3) defective assimilation of the glucose in the system. Undoubtedly in some of this class of cases the pancreas is at fault.

Saunby summarizes the morbid anatomy of the disease as follows: "Diabetes has so profound an influence on the general nutrition of the body, that it tends to produce structural alterations in the various organs, which are for the most part of a secondary and degenerative character. The exceptions are (1) the tumors and growths in or near the medulla oblongata and the vagi nerves; (2) a few instances of primary liver disease; and (3) cirrhosis, and other destructive changes in the pancreas. The one important addition to our knowledge of the morbid anatomy which the last few years have yielded, is undoubtedly the lesions of the pancreas, and we are justified in regarding these changes when present as the cause of the symptoms of chronic Bright's disease." Experiments upon dogs prove that extirpation of the gland causes all the classical symptoms of the disease. On the other hand, mere obstruction of the pancreatic duct is not followed by any of these symptoms. Nor do they occur if a small part of the gland be left, even if the duct be removed. Grafting a small portion of the gland outside of the abdominal cavity in the muscles of the external walls will also prevent the onset of the symptoms. The relation of the pancreas to some cases of diabetes appears to be very analogous to the relation between the thyroid gland and myxedema. It has been suggested, on the one hand, that the pancreatic secretion in some way controls the sugar-forming function of the liver, and, on the other, that the pancreas secretes a sugar-destroying ferment, which passes directly into the blood. No one, however, has yet succeeded in obtaining such a ferment from the pancreas, and pancreatic extracts, however administered, have failed to control glycosuria. It is, however, to be hoped that further knowledge will place the treatment of pancreatic diabetes on the same satisfactory footing as that of myxedema.

With regard to the cause of the disease, in my case it would be idle, in the absence of a *post-mortem* examination, to speculate. However, I think it is safe to assume that rapid growth, close application to work and autointoxication from absorption of intestinal poisons, caused by prolonged constipation, had something to do with the onset of the trouble. In reference to the constipation, it is interesting to note that Charrin and Carnot have proved that it is possible to render a dog diabetic by injecting infective fluids derived from the bacillus coli, bacillus pyocyaneus, and streptococci, into the pancreatic duct,