need not manifest itself in the dog. There is no valid reason why we should not apply these facts to the human being. Hence so long as a very small proportion of fairly healthy gland tissue is left, we have a satisfactory explanation why diabetes should not show itself, even though the major portion of the pancreas exhibits fibroid and atrophic or neoplastic changes. There are, however, aspects of that subject not capable of so simple an explanation. I remember my friend, Dr. H. D. Rolleston, of St. George's Hospital, showing to me a series of sections of the fibroid pancreas taken from both diabetic and non-diabetic cases, from which the only conclusion to be reached was that a given extent of fibrosis might or might not be associated with diabetes. Again, Hansemann has called attention to cases of complete replacement of normal pancreas by a diffuse cancerous infiltration. He seeks to explain these by the hypothesis already indicated, that the cells of a primary new growth of a ductless gland may continue to furnish an internal secretion. This may or may not be the case. Where there is primary cancer of the hepatic parenchyma, the new growth in the liver may be devoid of bile, the secondary growths are without exception free from bile. A more simple explanation of these and other examples of complete or almost complete destruction of the pancreatic glandular tissue is that of compensation, whether by vicarious function of Brunner's and other glands (the duodenal glands have frequently been found enlarged) or by diminished assimilation or production of sugar.

THE SUPRARENAL BODIES AND ADDISON'S DISEASE.

We meet with an identical series of cases in connection with another organ in which experimentally the existence of an internal secretion has been fully demonstrated. We may have (1) Addison's disease associated with disease of the supra-renal bodies, (2) Addison's disease with intact supra-renals, and (3) extensive, if not complete, destruction of the supra-renal bodies without the symptoms of Addison's disease.

Here, as with the pancreas in diabetes, the affection of the gland in Addison's is some form of atrophy or destruction of the specific gland tissue. Most frequently, I need scarce say, the change is tuberculous and necrobiotic, resulting in the disappearance of the gland tissue and its replacement by caseous material. But cases are on record of simple atrophy, hæmorrhagic necrosis and malignant growth of the bodies associated with or leading to all the symptoms of Addison's disease. In the vast majority of cases both glands are affected, but cases are on record (I have come across one such) where only one of