found in some cases of diabetes are such that there must be a marked diminution in the secretory activity of the gland. To this extent, in this one class of cases the results of autopsies are clearly in accord with the results of experiments. We have here examples of relative glandular inadequacy brought about by altered condition of the pancreas leading to diminution of internal secretion.

Examples of diabetes unassociated with disease of the pancreas are so well known that I need but refer to them. While such are difficult to explain from purely anatomical considerations the fact that they are found, and found relatively frequently is, in itself, an evidence that glycosuria is of at least a two-fold origin. That they are found is in conformity with the results of experiments, experiments which, on the whole, must be regarded as proving that there can be heaping up of sugar in the organism beyond the transforming power of the pancreatic internal secretion, or otherwise an incomplete burning up of the sugar. If this heaping up be in general due to increased glycogenesis, increased production of sugar, we should expect to find some evidence of increased activity of some glycogenetic organ, and here the recent researches of Glénard (5) and Triboulet (6) tend to show that this may be the case. Contrary to the older and generally accepted teaching, Glénard finds that clinically in over sixty per cent. of diabetics, there is evidence of some hepatic enlargement. Anatomically he finds that three conditions may be recognized, each possibly a stage in one morbid procees, namely, hyperæmia, general cellular hypertrophy (hyperplasia) and hypertrophy with cirrhosis (hypertrophic cirrhosis). Thus while I will not say that anatomical considerations prove the existence of my second subgroup in this connection, I must point out that the existence of this class of cases of diabetes without adequate recognisable pancreatic disturbance, is best explained on the supposition that there may be excessive production or assimilation of sugar with accompanying relative pancreatic inadequacy.

There is yet a third group of cases to be considered, that of extensive atrophic disease of the pancreas without diabetes. Here we have to proceed cautiously in our reasoning. As I have already indicated, Sandmeyer (7) has found that if only one-fifth to one-ninth of the organ be left in the dog, it may be months before sugar appears in appreciable quantities in the urine. Vaughan Harley (8) gives an even smaller amount, namely, one-fifteenth, but evidently he refers not to the eventual development of diabetes, but to its onset within a few hours. We can thus state that so long as from one-ninth to one-fifteenth of the glandular tissue of the organ is functional, for so long glycosuria