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Studies on the pathological effects of organisms of the enteritidis paratyphoid B. group on the pancreas, liver and kidneys.

By BERNARD G. H. THOMAS (by invitation).

[*From the Pathological Laboratories, University of Pittsburgh, Pittsburgh, Pa.*]

During the fall of 1922, in an attempt to produce a streptococcic glomerulo-nephritis it was observed that pathological changes similar in character occurred in both the treated and the control animals. About the same time many of the stock guinea pigs began to die spontaneously and post mortem cultures of their organs and heart's blood yielded Streptococci, Staphylococci and organisms of the Enteritidis-Paratyphoid B. group, but the former were thought to be secondary invaders in a terminal septicæmia. The latter bacilli consisted of two distinct forms namely, *B. enteritidis* and *B. paratyphosus* of animal origin, separable by direct agglutination. Microscopic examination of the organs of the animals dying spontaneously showed that characteristic lesions were present in the kidneys, liver and pancreas. On injection of either killed or living bacilli of the Enteritidis-Paratyphoid B. group similar lesions, though more intense and widespread, were likewise obtained.

In the kidneys the most prominent changes occurred in the glomerulus, where considerable intra- and extra-glomerular hemorrhage, thrombosis and varying degrees of necrosis and disintegration were observed. Occasionally hyperplasia of the endothelial lining of the glomerular vessels was present. A marked degree of parenchymatous degeneration occurred primarily in the proximal convoluted tubules and to a less degree in the distal convoluted tubules and the ascending limb of the loop of Henle. In the very acute cases the congestion and hemorrhage had become extreme. Rarely small areas of round celled infiltration occurred.

The liver was characterized by marked congestion, focal necrosis and parenchymatous degeneration. The areas of focal necrosis varied considerably in size, the larger ones having been converted into typical abscesses with thin enveloping walls of fibrous connective tissue. These consisted of varying amounts of necrotic tissue, infiltrated with polymorphonuclear leucocytes

mainly, although occasionally endothelial cells were numerous. The liver cells were pale, swollen, and showed considerable fatty degeneration and pigmentation.

The pathological changes noted in the pancreas were confined chiefly to the islets of Langerhans. These were characterized by an extreme degeneration which caused them to appear as exceedingly pale translucent patches clearly demarcated under low magnification from the acinar tissue. Even small islets consisting of one or two cells could be readily distinguished at a glance. Two types of cellular degeneration were found within the islets, one hydropic in character, the other pycnotic. The cells showing the hydropic degeneration were enormously swollen, the cytoplasm was pale and consisted of a wide meshed finely granular net work enclosing vacuoles of different sizes. In many of the cells the vacuolation had become so extreme that the thread work looked almost invisible and the cell appeared to consist of large watery vesicles. Frequently these cells had become ruptured leaving large pale nuclei with shreds of adherent cytoplasm and other debris. In the second type of degeneration the cells were not much altered in size and the nucleus appeared shrunken and pycnotic, and stained intensely. Both types occurred in proportions varying with different animals. We were not able, by means of the technique of Lane and of Bensley for the identification of the alpha and beta granules, to determine definitely of what type of cell the degenerated islet tissue was composed. Since in some of the cells showing the earliest stages of degeneration granules of the beta type could be faintly recognized it was thought that the larger part of tissue affected belonged to this type of cell. In the markedly degenerated islet tissue no granule stain whatever was obtained. The hydropic degeneration observed by us, in so far as can be determined, is identical with the morphological alterations in the islets produced by Homans and by Allen through a depletion of the islet cells following partial pancreatectomy and subsequent overstimulation of the remaining pancreatic tissue by feeding a high carbohydrate diet.

Experiments now being carried on in our laboratories and shortly to be reported indicate that a correlation exists between the pathological changes in the islet cells and variations in the sugar content of the blood.