créatitis may themselves be due to the action of one common and primary cause, and there may be one common factor causing blood destruction,—the liability to hæmorrhage and to morbid disturbances in the liver, pancreas, lymphatic glands, etc.; we cannot, I think, wholly pass over this possibility.

What has especially struck me, as it also impressed Hintze, is that common to all these cases we have evidence of more or less chronic intestinal disturbance. Indeed, as Hintze points out, the earliest stage of recognisable hæmochromatosis is that in which the walls of the small intestine alone are involved. We are forced also to recognise that in ordinary portal cirrhosis we have a similar history of chronic intestinal disturbance. Boix ([17]) has gone further, and has caused advanced cirrhosis in animals by feeding them with large quantities of the more deleterious products of intestinal digestion, and again by inoculating them with toxines developed from the bacteria inhabiting the intestinal tract. *There is then a certain amount of possibility, that the cause of hæmochromatosis is to be found in association with intestinal disturbance.*

In Cases Nos. 1, 2, 3, and 5 (Case 4, where the iron was only in the capillaries, may be omitted from consideration), and in the case of typhoid above mentioned, we found that the distribution of iron pigment differs from the case of general hæmochromatosis, which forms the subject of this paper, in the following points:—

1. In the liver, the pigment is found in the hepatic cell, where it lies in rows of fine granules along the margins of the bile capillaries; the connective tissue is free.

2. While there is pigmentation of both liver and spleen, the pancreas is quite iron free.

An identical distribution of iron pigment occurs in many cases in the literature, where there has been a history of local hæmorrhage.

The question arises, Is this a difference of degree only? or, is it essential? In other words, is hæmochromatosis a distinct morbid entity? Or is it simply a more advanced stage in the same pathological process at work in the cases of more localised iron pigmentation cited above?

The pigmentation of the connective tissue does seem to be a further step in the same process. In these slighter cases the pigment granules lie along the margin of the bile capillary, apparently being continuously excreted. In pigmentation cirrhosis, while the same appearance exists in the healthier cells, there are large areas of extensive pigmentation where the cell becomes overloaded, breaks down, and forms a mass of coarse pigment lying free in the newly formed connective tissue. The liver cell seems to have become unequal to its task of throwing off the insoluble granules which have been deposited in it, and succumbs at last to the accumulation of unrejected pigment which loads it down. Again, in the cases of cirrhosis, where there was hæmosiderosis of the liver cell without general hæmochromatosis, the