connective tissue is often pigmented, as in ten out of Kretz's fourteen cases, and in three out of our six.

On the other hand, the different distribution of iron in the organs seems at first sight to indicate that an essentially different process is at work. Not only in the one set of cases is the spleen pigmented, while the pancreas remains free; but in the other (pigmentation cirrhosis), the pancreas, with the other glandular organs, is loaded with pigment, while the spleen is relatively free. A possible explanation may be that the degenerating liver cells have lost much of their power of excretion, and, ceasing to act as a natural barrier, allow the blood pigment to pass on to the other organs. But this can hardly hold, for the lung is free from iron while pigmentation of the spleen is relatively slight. Neither those organs within the sphere of the portal circulation, nor those lying in a relation of contiguity to the liver, share evenly in the pigmentation, but certain elements which lie far removed from each other—for instance, the pancreas, thyroid, salivary glands, hypophysis cerebri, and heart muscle. These are all organs in which iron-free pigment is commonly formed. There would seem to be some degeneration of the parenchymatous cells of these organs, by which they become unable to throw off the altered blood pigment deposited in them.

The observations of Hunter would seem to us to possibly throw light upon the difference in these two sets of cases. Those observations in the first place show that there is a difference, in the extent to which the different organs become the seat of the removal of the hæmoglobin and modified blood pigment, according to the primary seat of the blood destruction. Where this is in the systemic circulation, it would seem that the kidney and the spleen are more active; where, on the other hand, we are dealing with a blood destruction in the portal circulation, there more especially is the liver affected. This, however, is not nearly all; thus Hunter points out there may be very extensive blood destruction, and the spleen itself, when its pulp is examined immediately after death, may show abundant evidence of the same, and yet microchemical tests may reveal no excess of pigment reacting to Perl's test.

This would seem also to be true in connection with the liver; even after extensive transfusion there may be little or no pigmentation of the organ; in other words, hæmoglobin, and it may be certain of its derivatives, do not react to the ordinary test for iron. Some further change is necessary in order to produce hæmochromatosis. While further, as Hunter again indicated, and as we have also pointed out in connection with pernicious anæmia, some definite and specific morbid condition of the cells of the liver (and of other organs) is very probably the factor in the development of the condition. It may, therefore, be that in these two different classes of cases, we are dealing, not so much with a different process as with a process originating in different areas, and