of slow and, it may be, intermittent extension of the disease elsewhere. The fact that both gross and miliary gummata may occur in the liver of the newly born is an absolute proof that the two forms are not characteristic of two different stages or periods of the disease. 'Absolute', that is to say, unless we are prepared to admit that while certain tissues such as the skin, present well marked secondary lesions, others may present either secondary or tertiary changes. Such an admission would make the terms 'secondary' and 'tertiary' valueless. For it must be kept clearly in mind that while the livers of these syphilitic infants show extensive fibrosis and indications which usually are recognised as of tertiary type, the cutaneous eruptions are secondary manifestations.

Over and above the granulomatous changes in the infant's liver it is most noticeable that a more generalised affection is peculiarly frequent, namely, fibrosis affecting either the whole organ or larger or smaller areas. Such fibrosis might be due to various causes; indeed, our knowledge of the etiology of cirrhotic changes in the liver, as in the kidney, and our knowledge of fibrosis in general is not sufficiently advanced to permit us to make positive statements. And, yet, since 1896 when, in this very room, although not before your Society, it was my privilege to deliver the Middleton-Goldsmith Lectures and I discussed the pathology of fibrosis in general, some little advance has been made in our conception of the process. For, on the one hand, Flexner* has shown that toxic substances (the blood serum of another animal), may lead to the development of cirrhosis, and, on the other, Weaver,† of Chicago, within the last few weeks, working (I think I may say) along lines suggested by certain publications of mine,‡ has demonstrated that bacteria exist which directly induce hepatic cirrhosis. Thus it would seem that whether in the process of excretion of toxic substances by the liver cells, or by the taking up of certain bacteria, and the influence of their toxins when so taken up, the liver cells may undergo a parenchymatous degeneration so intense that death ensues and, following thereupon, a replacement fibrosis occurs, more or less pure and unaccompanied by acute inflammatory change according as to whether the parenchymatous disturbance is unaccompanied by interstitial irritation or not. Where many miliary gummata are present, there eventually, much fibroid change is brought about by the tissue changes in their immediate neighbourhood.

We are not as yet in a position to state whether the fibroid change of this type in the liver of the syphilitic child is a consequence of the attempted removal of the syphilitic germs from the portal circulation by the agency of the endothelium of the hepatic blood vessels and by the liver cells, or whether it is the circulating toxins of the disease that

* Flexner, Trans. Path. Soc., Philadelphia, 1896.

† Weaver, Philadelphia Med. Jour., Feb. 4, 1899, p. 284.

‡ Montreal Medical Journal, July, 1898; British Med. Journal Oct. 22, 1898.