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AN ADDRESS ON THE MORBID ANATOMY AND PATHOLOGY OF CHRONIC ALCOHOLISM.

DELIVERED AT THE PATHOLOGICAL SOCIETY OF LONDON, DECEMBER 4th, 1888.

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MORBID CHANGES PRODUCED BY ALCOHOL IN VARIOUS ORGANS.

Since it is obviously impossible to speak of all the organs which may be altered, I shall confine my remarks to a few, especially the liver and the nervous system, taking the morbid changes in these parts as types of the effects produced in the organs generally

Effects of Alcoholism as seen in the Liver.—It is generally recognized that one effect of alcohol is to produce accumulation of fat or steatosis in the liver. This change is produced especially by dilute forms of alcohol, and in those who are well fed. The explanation has already been suggested. Only one question occurs to me respecting this condition: Does it ever pass into cirrhosis? Are there not large livers, with a large amount of fat, which show commencing cirrhosis? Or does the fatty change in some way shield the liver tissues from the more serious and irritative action of the spirit? The accumulation of fat is, so far as it goes, evidence of the destruction of some alcohol, if the explanation given above be correct.

Cirrhosis of the Liver.—It would seem as if no pathological process were better known or explained in a more satisfactory way than this. It is generally accepted that concentrated forms of alcoholic drinks, brought into the stomach, are absorbed into the portal vein, and carried to the liver, where inflammation of the interstitial stroma is set up, by which new fibrous tissue is produced. In consequence of the pressure of this tissue, and its subsequent contraction, the liver cells are compressed and destroyed, and are found in various degrees of degeneration loaded with fat, yellow granules, and so on.

To this explanation I am inclined to demur. I would ask, Is a liver ever found with healthy hepatic cells and an inflamed stroma? In the very earliest stages of cirrhosis are not the cells decidedly degenerated? Is it not more reasonable to suppose that the injurious action of alcohol is exerted simultaneously on both parts of the organ; and that, if so, the parenchymatous elements, being more vulnerable than connective tissue, would suffer first? Dr. Lionel Beale, indeed, urged some years ago that the change is essentially atrophic, not inflammatory.

I am also led to raise the question by consideration of a certain very rare form of degeneration of the liver, which is really, I think, produced by alcohol, though the connection has not been recognized. I mean that called in England acute red atrophy. (In Germany the name "red atrophy" is often given to what we call the nutmeg liver.) It is generally admitted to be nearly allied to the acute yellow atrophy, but differs from it, in other respects besides color. The organ is much reduced in size; the liver cells, as in yellow atrophy, show advanced degeneration and necrosis. Other parts of the organ are of a deep red color, with little or no liver tissue, and consist chiefly of connective tissue and capillaries deeply engorged, inflamed with infiltration of leucocytes, and showing new formation of fibrous tissue. This short description, founded on a paper by Dr. Moxon in our *Transactions*, and on the only case which I have seen, proves, I think, that the same changes are displayed in an acute form, as cirrhosis shows in a chronic