and there is not a doubt that even in some of the most chronic and advanced cases great relief or permanent benefit is frequently obtained. Patients ought to be induced to visit one or other of these places year after year.

THE CAUSE OF SPLENIC ENLARGEMENT IN CASES OF HEPATIC CIRRHOSIS.

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It has astonished me that the enlargement of the spleen, so frequently found in cases of cirrhosis of the liver, is generally considered to be the result of passive congestion, consequent on obstruction to the blood flow through the cirrosed liver. Thus Osler\(^1\) mentions this view as an acknowledged fact: “Enlargement of the spleen occurs from the chronic congestion.” Strümpell\(^2\) says: “Neben dem Ascites ist das wichtigste von der Stauung im Pfortadergebiet abhängige Symptom der Stauungs-milztumor.” Here there can be no misunderstanding, for by the single word “Stauungsmilztumor,” which this author makes use of, he implies that congestion is the cause of the splenic enlargement in cases of cirrhosis of the liver.

If passive congestion be really the cause of splenic enlargement, why, it may be asked, is the spleen generally found small and hard in cases of chronic passive congestion of the abdominal viscera due to valvular heart disease? Surely, as far as the spleen is concerned, the chronic venous engorgement must be similar in the two cases, and yet in ordinary hepatic cirrhosis the spleen is generally found enlarged, and in chronic heart failure it is so often to be found small, that the term “cardiac spleen” has come to be often employed (in necropsy notes) for the small and hard condition of the organ found in cardiac cases. In fact, with a passive congestion so great as to cause atrophy of the liver cells from the inner portion of each lobule, the spleen is generally found to be small, or at all events not much enlarged, provided that no infectious disease was likewise present just before death. T. N. Kelynack\(^3\) fully recognises this fact, and in a very considerable post-mortem material found that the average weight of the spleen in hepatic cirrhosis was 12.93 oz., whilst in cardiac cases it only averaged 7.32 oz.

But there is another argument against the ordinary explanation

\(^1\)“Principles and Practice of Medicine,” 2nd edition, 1895, p. 447.
\(^2\)“Lehrbuch der Speziellen Pathologie und Therapie,” 8th edition, vol. ii. p. 251.
\(^3\)Birmingham Med. Rev., February 1897.
of splenic enlargement in cirrhosis of the liver: this is that the greatest enlargement of the spleen is not found in those cases of cirrhosis (as ordinary atrophic cirrhosis) where the portal obstruction is greatest, but precisely in those cases of cirrhosis (biliary cirrhosis, hypertrophic cirrhosis of Hanot, with chronic jaundice) where the portal obstruction is least, and where ascites is delayed to the last. Thus E. Auscher, speaking of the enlargement of the spleen in ordinary alcoholic cirrhosis, says: “Cette hypertrophie est moins marquée peut-être que dans la forme hypertrophique de la cirrhose alcoolique et surtout que dans certaines hépatites infectieuses (dans la cirrhose de Hanot en particulier).” In Hanot’s cirrhosis and biliary cirrhosis, I think it is now generally acknowledged that the enlargement of the liver is greater than in ordinary alcoholic cirrhosis. In children, in whom all vital reactions tend to be exaggerated, this enlargement of the spleen is best marked, and thus, in children with cirrhosis of the liver and chronic jaundice previously to the onset of ascites, the liver may be found to reach below the ileum, and in some cases in fact constitutes the chief feature of the case.2

What is then the real cause of the splenic enlargement in chronic jaundice? The most satisfactory explanation appears to me that the change is a “vital reaction,” called forth by the presence of some toxic substance circulating in the blood, a substance not necessarily of microbic origin, as in infectious diseases. The toxic material in question might be something normally excreted in the bile. In ordinary alcoholic cirrhosis there is often a slight degree of jaundice observable, especially in the final stages of the disease, showing that the bile pigments and salts manufactured in some parts at least of the diseased liver, find their way into the circulation and can exercise toxic effect. This auto-intoxication theory receives some support from what has already been mentioned, namely, that it is in the kinds of cirrhosis (with chronic jaundice) where the biliary flow is most hindered that the enlargement of the spleen is most marked; for instance, in cases of cirrhosis following prolonged obstruction by a calculus in the common duct,3 and in Hanot’s cirrhosis, where the minute biliary ducts are specially involved in the fibrotic process.

It has been suggested that the enlargement of the spleen, when it occurs, may be due to the same cause as the cirrhosis of the liver, but the microscopical investigations of H. Sieveking4 have lent no support to this view. He examined the spleen in several cases of hepatic cirrhosis, and could find no proliferation of

1 “Manuel de médecine” of Drs. Debove and Achard, tome vi. p. 91.
2 See A. Gilbert and L. Fournier, Rev. mens. d. mat. de l'enf., Paris, 1895, tome xiii. p. 310; F. P. Weber, Trans. Path. Soc. London, 1895, vol. xlvi. p. 71.
3 F. P. Weber, “Cirrhosis of the Liver and Icterus of Four Years’ Duration,” Brit. Med. Journ., London, 25th April 1896.
4 Centralbl. f. allg. Path. u. path. Anat., Jena, 1894, p. 1017.
connective tissue analogous to that in the liver. Moreover, E. Boix,' in describing a kind of cirrhosis (cirrhose dyspeptique), which he thinks is due to an auto-intoxication from the alimentary canal, specially draws attention to the fact that (at least in the earlier stages) the spleen is not found enlarged.

On the whole, the theory that the splenic enlargement is chiefly due to some auto-intoxication caused by the hepatic disease, seems more probable than that the affection of both organs occurs simultaneously and from the same cause. An exception, however, must be made for certain rare cases of splenic enlargement—splenic anæmia or "splénomégalie primitive"—in which, as the splenic disease advances, a cirrhotic process appears to commence in the liver. Here the hepatic and splenic affections may possibly both be due to the same (as yet unknown) cause; it has even been suggested, as an explanation of these cases, that a toxin is manufactured by microbes in the enlarged spleen, and that this toxin, carried by the blood stream to the liver, gives rise to a secondary cirrhotic process there.

SIMPLE RULES FOR THE ACCURATE DIAGNOSIS OF DIPLOPIA.

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In this article my motive is not so much to bring to light anything very new, or to pose as the discoverer of facts not hitherto made known, but rather I have set before myself the humbler task of explaining the method which seems to me the best and simplest of discovering which of the muscles of the eye is paralysed in any case of diplopia.

The diagnosis is not always very easy, on account of various circumstances, but it has long appeared to me that the current explanations of the mode of diagnosis are well calculated to make a subject which is necessarily a little complex, needlessly complicated. The reason is not far to seek, for I think it lies in this, that in the text-books the matter is regarded from the wrong side of the subject, or rather from only one side. In them it is—Given the muscle, find the situation, etc., of the double vision. For the purpose of recalling to the memory of one fairly well acquainted with the subject, the various features of such cases, this may be all very well, but this is not enough. The teacher or the writer of a text-book ought to regard the subject also from the point of view of a beginner, and of the practitioner who meets with a clinical problem—Given the diplopia, find the muscle. This is rarely done, and, when it is, there is a lack of definiteness.

1 "La Foie des dyspeptiques," Paris, 1895.