maintenance should not be in the control of Boards of Guardians who frequently do not realise their responsibilities, but rather yield to their prejudices.

A COMMISSION OF INQUIRY NEEDED.

Well, how is this state of things to be corrected? The agitation against it has gone on for years. Government after Government has been approached on some special point, and with a few exceptions, with little effect. Chief Secretaries have occasionally been sympathetic; more commonly they have raised up a barrier of difficulties behind which to shield themselves; and so the struggle has proceeded, until the sufferers from this wretched system have grown weary and almost hopeless. I think the future plan of action needs very deliberate consideration. There is little good to be gained by periodic visits to the Government, asking for the removal of this or that wrong. We must take the higher ground of charging against the whole system that it is bad to the core, and that there is no use in a policy of tinkering. It is waste of money, of time, of temper, for, after trifling improvements have been made, the service remains unsatisfied and thoroughly discontented. If changes are to be effected which shall be really lasting, and of advantage to the departments and to the public at large, there must be a thorough inquiry by competent men. We must ask the Government to appoint a Commission to investigate the working of the Poor-law medical relief system in this country in its relation to the poor, the ratepayers, and the medical officers. By this means we shall be able to prove our case, and to fix public attention upon the glaring evils which have become interwoven with the system.

We shall be able to gather evidence as to the best method of reorganising the whole Poor-law Medical Service, so as to make it most effective—at once satisfactory to the officers, a benefit to the sick poor, and a safeguard to the public health. I should like also to see arising out of it a scheme which would, as in the Colonial services, secure a pension, not only for the medical men themselves, but for their wives and children, who, too often, are left derelict to seek the uncertain charity of the profession.

HOW THE ASSOCIATION CAN HELP.

To help in gaining all this, I suggest that every public medical body in this country should be invited to join in a common demand, the form of which can be determined by this Branch; that it should also have the support of the Council of the Association, and, if necessary, of the Association itself, by resolution submitted at the general annual meeting. Such a request so supported could not be lightly treated, and we should then have the opportunity of showing what are the wrongs and what are the remedies.

I will not further detain you. I knew from the sympathy which you have always shown on this subject that I could count upon your forbearance. The greater number of us do not belong to the service in whose interests I have spoken. But you have never forgotten the brotherhood of medicine, and I know you are ready to-day, as always, to send to our kin, even at the uttermost parts of the land, a message of hope and of sympathy. If you and they approve of what I have suggested, I am sure you will give it hearty support. What we want is continuous and concerted action, and the suppression of ridiculous jealousies, which can only irritate and harm. I shall indeed be gratified if, at the close of my term of office, we can say that the cause for which I speak has been advanced, and that the dispensary doctors are within sight of those reforms for which they have so long and patiently pleaded.

ALCOHOLIC CIRRHOSIS OF THE LIVER.¹

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I HAVE for some years felt that not sufficient prominence was given in our textbooks to the enlarged form of alcoholic cirrhosis of the liver. It has always seemed to me curious that whereas in cirrhosis from other causes—for example, cardiac weakness and syphilis—increased size has been recognised

as a common condition, to the cirrhosis of alcohol has been attributed a peculiar contractile force. I admit that opinion has changed much of late, and that the existence of the enlarged variety is freely admitted; but the authors of textbooks are, quite rightly, very conservative, and they still largely qualify this admission by the suggestion that the enlargement may be merely an antecedent state to atrophy, or, granting its individuality, they couple with it a clinical state quite different from that of ordinary alcoholic cirrhosis, including such symptoms as marked jaundice, absence of ascites and delirium. It seems to me that we have come to a stage when the student can quite justifiably be taught a simple, clear, and unified doctrine on this condition.

Prolonged excess in any variety of alcohol produces a chronic form of inflammation, which, as is usual in such inflammations, selects for special attack the supporting structure rather than the parenchyma of the organ. As the alcohol is brought to the liver by the portal vein it is only natural that the portal spaces should suffer most, but the intralobular connective tissue is also affected, and the parenchyma itself not seldom undergoes injury. After any special indulgence the inflammation is of a more acute nature, and is accompanied by acute hyperæmia, which latter greatly and suddenly increases the size of the liver, its edge descending as much as 2 or 3 inches in the course of twenty-four hours, giving to the finger the sense of great rigidity and hardness. But with abstinence from alcohol this sudden swelling soon subsides and the edge retires to nearly the position it held before the attack. As the case progresses the liver either permanently enlarges or grows smaller.

Clinically, then, alcoholic cirrhosis of the liver is accompanied by enlargement, the edge being felt hard, firm, tough and blunt, somewhere between the ribs and the level of the umbilicus. This enlargement, though increased by, is not solely due to hyperæmia, for it continues permanently long after alcohol has ceased to be taken (Class I, No. 28, *vide infra*). Though the irritation of the parenchyma—as shown by the division of the nuclei and wasting of the cell substance, thus producing the so-called new bile ducts—and the intralobular fibrous growth are more frequently observed in those livers which show increase of size, yet these morbid changes are by no means confined to this class, but are to be found in extremely contracted forms. The difference in size appears to be due to purely mechanical conditions arising partly from the amount of fibrous growth and partly from the amount of compression the parenchyma undergoes owing to the disposition of this growth. And yet perhaps this is not entirely the case; so far as my experience goes there is one variety of fibroid change which is almost confined to the enlarged liver. I cannot do better than quote Dr. Bristowe's clear description of it:

The liver is extremely dense in texture and on section is found to be largely infiltrated with dense, greyish, slightly translucent, connective tissue. This does not form a distinct network as in the hobnailed liver, circumscribing definite groups of hepatic lobules, but is rather a diffused growth in which the remnants of the hepatic parenchyma are scattered irregularly in yellowish masses from the size of a poppy seed to that of a pea. In some parts the hepatic tissue predominates, and there is an approach to the hobnailed condition; in some almost every remnant of the natural structure has disappeared, and dense fibroid tissue alone remains.

I would emphasise the fact that it is translucent fibroid material with little or no evidence of fibrillation which forms these masses of new growth, each of which not seldom reaches the size of six or more lobules. Within them may usually be seen, scantily scattered, groups of not more than two, three, or four cells of the parenchyma; these are wasted, very often the nuclei alone remaining. At the circumference of the masses the fibrous network again assumes its sway, but with it the "bile-duct" formation is especially frequent. It is generally allowed now that these "bile-ducts" are but double rows of degenerated and irritated liver cells, or rather double rows of the nuclei of these. My friend Dr. G. F. Crooke has shown me several instances where these nuclei are undergoing various changes through the fusiform connective tissue cell up to the fully formed fibre. Hence there appear to be two ways in which these fibroid masses may arise: either owing to a peculiar instability of the parenchyma cells, a special liability to the action of alcohol, inherited or acquired; or to the interlobular growth developing in such a manner that no great compression of the contained parenchyma results not

¹ Read in the Section of Medicine at the Annual Meeting of the British Medical Association held in London, July-August, 1895.

enough to cause its annihilation, but sufficient to produce its irritation and degeneration into fibroid material.

I have endeavoured to see how far facts agreed with this view of the question, and for this purpose have collected all the cases bearing on the point from the reports of the Queen's Hospital during the last six years; I have also abstracted all similar cases from my private casebooks during the same period. The numbers are small, partly because several cases had to be omitted owing to the existence of other chronic lesions or causes which may have had serious effect on the state of the liver, and partly, I think, because hepatic cirrhosis is not common in Birmingham, the kidney being our more vulnerable organ. Moreover, I have included no cases which had not definite evidence of chronic alcoholic excess. I have divided up the cases into four classes:

The First Class contains those in which there was no ascites, and which were relieved by treatment. The number of these is 28, and in 26 of them the liver reached below the costal arch in the right nipple line, it being felt on the average in these 26 cases $2\frac{1}{4}$ in. below the ribs. Thirteen were private patients who would correspond to hospital out-patients; in these the liver was enlarged in all, the average increase being 2 in. Of hospital out-patients I have no records, but I can safely say that it is very rarely that I fail to feel the liver edge in alcoholic subjects.

It may reasonably be said of this class that it contains only early cases of disease. This may indeed be true of the majority, but by no means of all; 3 have suffered from hæmatemesis; 2 from alcoholic neuritis; and No. 28 is a type of some of the others; this gentleman had taken alcohol (whisky) to excess for over forty years; for thirty years his daily average was not less than 15 oz., and during the succeeding ten years not less than 6 ozs. Yet the edge of his liver is felt thick, hard, and rounded, $2\frac{1}{2}$ in. below the costal margin in the right nipple line. Moreover, during the past six years, he has been moderate as regards alcohol, probably taking not more than one glass of whisky a day, hence the increase of size is not likely to be due to hyperæmia. It is true he is stout and troubled with cardiac dyspnoea, but I think the condition of the heart points rather to fibroid than to fatty degeneration, and no one feeling the hard tough edge of the liver would suggest that this had suffered much fatty infiltration.

The Second Class contains those cases in which ascites has ensued. It thus represents a more advanced stage of the disease. These cases also were relieved by treatment, though if I had been able to follow them up I should no doubt have found that by this time many had reached a fatal termination. They are 18 in number, and in only 9 is the liver noted as having been felt below the ribs, but in several instances no note is made of the liver after paracentesis, and we know how hard it is to detect enlargement of this organ when the abdomen is tensely distended with fluid; moreover, the hepatic dullness is sometimes stated as being raised an inch or more. Still, taking the reports as they stand, we find the liver to be increased in size in one-half the cases, the average increase in these being 1½ in.; further, we must not conclude that because the liver is not felt that, therefore, it is contracted, as normally the liver is not palpable in the right nipple line.

The Third Class—in which the result was fatal, but where no *post-mortem* examination was obtainable—consists of 12 cases. In each of the 5 which died in hospital ascites was present, and in the 7 taken from my private casebooks there appears to me to be excellent evidence of advanced hepatic disease. The liver was felt below the ribs in all my private cases, and in 2 of the hospital ones; in each of these latter it reached 3 in. below the costal margin, and in my cases the average extent of it below the ribs was also 3 in. Taking the 12 cases together, the liver extended on the average $2\frac{1}{4}$ in. below the ribs.

The Fourth Class consists of those fatal cases where a *post-mortem* examination was obtained. Here we are at once on surer ground, and to this class I wish to draw your special attention. It unfortunately contains only 9 names, and the size of the liver in one of these is not mentioned. We have, therefore, but 8 cases to deal with. I have chosen to estimate size in these cases by weight. Three of them were female, and the average weight of the liver was 48½ ozs.;

taking Reid's statement that the average weight of the female liver is 45 ozs., we find that in these 3 cases of cirrhosis the weight exceeded the average by 3½ ozs.

Still livers may exceed in weight and yet be contracted, the excessive weight being due to increase in density, but I find that in each of these cases the organ was felt during life. As in one instance the liver weighed only 35 ozs., this brings to light another fallacy—namely, that because the liver is felt below the ribs it does not necessarily follow that the organ is enlarged; but this fallacy only seems to exist for women, as in each case amongst the males where the liver was felt it was also found to be of excessive weight. The five male livers weighed 320 ozs., giving an average weight of 64 ozs. Taking Reid's average again as 53, this gives an excess of weight of 11 ozs.

From these figures it is evident that in the large majority of these 67 cases the liver was detected by palpation below the ribs in the right nipple line. The actual number is 46—that is, over two-thirds. If we lump the whole 67 together, and allow fully for those instances where diminution was noted, we find that the average extent of the cirrhotic liver below the costal margin in the right nipple line was 1½ in.

Some authorities have expressed their belief that when the liver is enlarged in advanced cirrhosis the increase in size is due to fatty infiltration. If this were so it would not invalidate the clinical portion of my position—namely, that in the vast majority of cases of hepatic cirrhosis at all stages of the disease the liver is felt below the ribs.

But perusal of the *post-mortem* reports recorded in my list and an examination of the sections I submit to you will show that such is not the case. There is no more fatty infiltration in the large than in the small livers. It is true that macroscopically it was recorded that there was an appearance of fatty change, but the microscope refutes this statement. The explanation of the discrepancy is probably due to the fact that the parenchyma is more bile-stained than the fibrous new growths, and hence gives the appearance of irregular fatty deposit. Fatty infiltration, of course, does occur, but in no larger proportion than in the small cirrhotic liver.

This leads me to another point which the list of cases demonstrates. I at one time surmised that the large livers belonged to beer drinkers, whilst the small ones were the especial property of the consumers of spirits. But analysis of the cases shows otherwise. Of 26 beer drinkers the average extent of the liver below the costal margin was only 1½ in.; 1½ in. was the corresponding measurement of those who indulged in beer and spirits; whilst of the 16 who were pure spirit drinkers—nearly always whisky—the liver reached on the average as much as $2\frac{1}{4}$ in. below the ribs. Further, the largest liver of all those which were subjected to *post-mortem* examination—one weighing 92 oz.—belonged to a gentleman who had for at least twenty years drunk very largely to excess of whisky and scarcely at all of beer, though he had no doubt indulged in wine.

Wine I have taken no special note of; no one of my cases could be said to have been a wine drinker, though several spirit drinkers partook freely of wine, yet the excess was nearly always due to excess of spirit, and with hospital patients wine played no part at all.

Further, in the reports of the cases I have collected there is nothing to show that jaundice, cerebral symptoms, etc., were more marked in large than in small livers, whilst ascites is certainly as common in the large. Indeed, the first case that drew my attention to the large liver was a woman whom I tapped in 1884 for extreme ascites. Of the 8 cases whose cadavers were examined 3 had jaundice, and in 2 of these the liver was large, but in no case was the jaundice marked. Only 2 cases are noted as dying with cerebral symptoms (out of 21); one of these had a large and one a small liver.

Lastly, the spleen is mentioned as being felt below the ribs in 17 cases, and it is noteworthy that 14 of these belonged to patients whose livers were enlarged. Again, the spleen was specifically reported as not enlarged in 8 cases, only one-half of whom had large livers. In other words, those patients who have enlarged livers show a greater tendency to enlargement of the spleen than do those whose livers are contracted. If it be admitted that enlargement of the spleen is due to portal obstruction, then this obstruction must occur more frequently with the enlarged liver. It is generally supposed

that obstruction and cirrhosis vary directly. Hence this condition of the spleen is a fresh proof of the extreme degree of cirrhosis attained by the large cirrhotic liver.

I think, therefore, examination of these cases goes far to show the truth of my contention that in alcoholic cirrhosis the liver is generally enlarged at all stages of the disease, and that whether enlarged or contracted the clinical symptoms and course of the disease are much the same, and the pathology of both forms identical; and, further, that there is no particular line of demarcation between the two, but that cirrhosis from alcoholic excess produces all shades of sizes from a liver of 100 ozs. to one weighing but 30.

Insistence on this fact of cirrhotic enlargement will prove of great value to the diagnostic power of the ordinary medical man. Hitherto he has sadly neglected close examination of the liver as an aid to diagnosis, knowing how impossible it is to perceive a small diminution in its size, while hæmatemesis or ascites has usually made the matter plain before extreme contraction takes place. But if it be dogmatically asserted, without qualification, that the cirrhotic liver is generally an enlarged one, then the whole attitude of the examiner will be altered. Attempted palpation of the liver will be the ordinary routine, and perception of the offending edge will bring a comfortable assurance of certainty of diagnosis instead of leading to confusion, as it must do now.²

A POINT IN THE DIAGNOSIS BETWEEN CHRONIC RHEUMATIC AFFECTIONS AND CHRONIC GOUT.¹

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THE chronic changes found in the various joints and due to rheumatic and gouty conditions, are at first sight so similar that much difficulty often arises in making a differential diagnosis. So-called chronic rheumatic affections may be divided into those due to true rheumatoid arthritis and those which have been left by acute rheumatic fever. Although these are probably entirely different affections, they will be treated for the purpose of this communication under the one head of chronic rheumatism, and an attempt will be made to show a fairly easy method of differentiating between the changes found in chronic rheumatism and those produced by true gout. The importance of a careful distinction between rheumatism and gout for purposes of treatment is sufficiently obvious.

The point which I wish to bring forward does not hold good in acute rheumatic or acute gouty conditions, nor can it necessarily be applied to other joints than those of the hands. It must be remembered then that I shall deal only with chronic changes in the hands.

In chronic rheumatism it will be found as a rule, to which there are very few exceptions, that the joints of the hands are symmetrically affected, that they are affected to an equal degree on the two sides, and that the deformities produced are similar and symmetrical. These statements hold good, however *bizarre* the deformities may be. Very rarely, although the joints affected are symmetrical, the deformities are asymmetrical, as in two cases I have seen. In one of these the deformities of the fingers of one hand showed flexion, extension, and flexion of the three joints of the fingers; whereas in the other hand they showed extension, flexion, extension. In the second case the middle finger of the left hand showed extension, flexion, extension; all the other fingers of the two hands being in a position of flexion, extension, and flexion. It should be mentioned that in examining deformities of the hands, care must be taken to exclude those produced by Dupuytren's contractions or by old injuries, which, as a rule, produce asymmetrical deformities.

When we examine the chronic changes in the hands produced by gout we shall find an entirely different state of affairs. If only a few joints are affected we shall see that they

are quite asymmetrical, the extent of the affection is unequal, and the deformities produced are dissimilar. If all

Fig. 1.—Symmetrical lesions and deformities. Chronic rheumatoid arthritis. Marked ulnar flexion of both hands

Fig. 2.—Chronic rheumatoid arthritis. Symmetrical lesions and deformities.

Fig. 3.—Chronic rheumatism. Symmetrical lesions and deformities.

the joints of both hands are affected, as occasionally happens, then it will still be found that the amount of affection is very unequal, and the deformities are also quite different. This is a rule in gout, to which I have seen no exception. The photographs shown have been taken only from those cases of gout in which the diagnosis, as obtained from the history, the presence of uratic deposits, or of gouty kidney, was perfectly clear.

Incidentally it may be remarked that three of the slides show that ulnar flexion, or the "seal-fin" hand may occur in gout as well as in rheumatism. Many elaborate theories have been brought forward from time to time to explain the ulnar flexion of rheumatism, but some of them will certainly not hold when applied to gout. I think a simple theory will explain to a large extent this peculiar deformity, in both affections. The healthy but inactive hand held in front of the chest will be found to take up a position of slight flexion of the fingers, together with slight inclination to the ulnar side. Now if the hands are affected with a painful condition they will naturally be held in a position of rest, which is one

² Want of space prevents the publication of abstracts of the sixty-seven cases. These will appear in the *Birmingham Medical Review*, April, 1896.

¹ Read in the Section of Medicine at the Annual Meeting of the British Medical Association, held in London, July-August 1895.