The enlarged cirrhotic liver / by Arthur Foxwell.

THE
ENLARGED CIRRHOTIC LIVER.

ARTHUR FOXWELL.

Alwyn A. Bradley.

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ENLARGED CIRRHOTIC LIVER.

BY THE SAME AUTHOR.

ESSAYS IN
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Unquestionably the best, and in our opinion that which gives the author a right to a niche in the temple of medical science, is the group of papers on the Condition of the Vascular System in Anæmic Debility, wherein he gives an excellent explanation of the production of the heart murmurs met with under these circumstances. It is without doubt the most satisfactory solution of the problem hitherto attempted, and appears to be very likely the true one. . . These admirable essays . . will be appreciated by all who care for writings which are eminently thoughtful, yet withal practical in their bearing.—*Brit. Med. Jour.*

THE

ENLARGED CIRRHOTIC LIVER.

BY

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PREFACE.

THE *raison d'être* of this little book is my conviction that an undue amount of uncertainty and difficulty surrounds the physical determination of the liver's condition in cirrhosis, especially of that form due to alcohol; and these essays are an endeavour on my part to make this diagnosis more easy and sure.

There is nothing *sui generis* in the cirrhosis resulting from alcohol: the anatomical nature of the deposit is identical with that occurring in other forms though its proportionate distribution throughout the lobule is naturally different from what occurs when the hepatic vein or bile duct acts as the conduit of the *materies morbi*. Clinically, the alcoholic, as well as the other forms, is characterised by an enlargement of the organ, those cases where diminution is noted being quite the exception. It is true that contraction when it does occur means an advanced stage of disease; but even in the severest forms enlargement remains the rule. In the early stage the increase in size is chiefly owing to active or passive congestion and, in a small degree only, to the pathological deposits of fibroid tissue. But when the disease is advanced this fibroid tissue forms the bulk of the enlargement and more than compensates for the loss in size resulting from wasting of the parenchyma.

During the last six years I have recorded the size of the liver in 202 private patients, in whom I suspected it was diseased. In 7 of the 202 I have noted that the liver was small; in 90 that its size was unchanged; and in 105 that it was larger than usual. That is, in *more than half* the cases where I suspected the liver to be involved in the diseased condition of the body I

found this organ to be larger than normal, and in only 7 was its size reduced. Moreover, of these 7, I find my diagnosis was based on the descent of the upper margin of superficial dulness below the sixth cartilage in the right nipple line, and that in no one of the seven did I satisfy myself that the *lower* limit was raised, and it may be that this descent of the upper margin of dulness was owing to the hyper-resonance of emphysema, or an unduly capacious lung which I failed to detect, though in each case I have recorded my endeavour to eliminate this kind of fallacy. Further, not one of these seven was a case of alcoholism.

It may be objected to my series of cases that the term cirrhosis is by no means applicable to all; but, with the exception of the phthisical ones, I think it best expresses the condition. They are at any rate instances of chronic congestion: and chronic congestion is probably always accompanied by permanent exudation with the formation of fibroid tissue and parenchymatous degeneration.

It must not be concluded that I have exhausted the causes of cirrhosis; I have only dealt with such as happened to come under my notice.

My thanks are due to my colleagues, Dr. Carter and Dr. Suckling, for permission to make use of their cases, and to my house physician, Dr. Simpson, for his help in abstracting these from the hospital reports.

ARTHUR FOXWELL.

Birmingham: July 1896.

THE ENLARGED CIRRHOTIC LIVER.*

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PAPER I.

NON-ALCOHOLIC FORM.

My remarks to-day are based upon the reports of 106 cases which I have extracted from my private note-books. With one exception I have had to rely entirely upon examination of the patient during life, for in no other of those cases with a fatal issue was I able to obtain *post mortem* evidence. I have considered the liver enlarged when it was felt below the ribs in the right nipple line. This paper is thus liable to two grave fallacies: 1st, that the facts stated depend upon the correctness of one observer; 2nd, that not all livers which reach below the ribs are necessarily enlarged. But this second fallacy tends to correct itself; for, its converse, not all large livers are felt below the ribs, is perhaps equally true. You will perceive I speak of palpation only. I think very little of the value of percussion in regard to the liver; often there is no dulness although the organ can be felt two inches or more below the ribs in the right nipple line.

In cases of any difficulty I always palpate with both hands; for bimanual examination aids the diagnosis between liver and

* Paper read before the Birmingham and Midland Branch of the British Medical Association, December, 1895.

kidney ; also without it many flaccid enlargements of the liver may be overlooked, as well as others where the organ has fallen away from the anterior abdominal wall. The left hand should be placed in the loin with the finger tips just outside the erector spinæ and pointing directly towards the lumbar vertebræ ; the right exercising a light but firm and steady pressure on the abdomen immediately to the right of the rectus muscle, with its finger tips pointing towards the right nipple. This hand I keep quite still, merely allowing it to exercise a steady passive resistance to the abdominal wall, trusting to the patient's inspiration to bring the liver edge down to my finger tips : this passivity I think is of considerable importance, as I find my fingers are more sensitive to delicate impressions when at rest than when actively seeking the liver. This does not prevent my taking up a fresh position with this hand should I fail to discover the organ in that assumed at first.

The patient I prefer to be lying on a couch, supine, with the trunk at an angle of 30° with the horizon and the head well supported. I do not find that flexing the thighs upon the pelvis is of much assistance.

It is obviously impossible for me to speak with any accuracy as to the frequency of the occurrence of enlarged liver in any given affection. The following statistics, therefore, simply indicate the number and nature of the cases amongst my private patients during the past five years in which I have observed, and noted down at the time of observation, undoubted hepatic enlargement.

TABLE A.

	Disease.	*Number of Cases.*	*Average Extent of Liver below Costal Arch in R.N.L.*
1.	Alcohol ...	21	... ... 3 inches
2.	Obstruction in Lung	16	... ... 1½ ,,
3.	Abdominal Catarrh	13	... ... 1⅓ ,,
4.	Gout	7	... ... 2 ,,
5.	Phthisis ...	11	... ... 1½ ,,
6.	Cardiac Debility	10	... ... 2 ,,

	Diseases.	*Number of Cases.*	*Average Extent of Liver below Costal Arch in R.N.L.*
7.	Valvular ...	5	... ... $3\frac{3}{8}$ inches
8.	Bright's ...	5	... ... $2\frac{1}{8}$,,
9.	Bile Obstruction	4	... ... $3\frac{1}{4}$,,
10.	Of Spleen ...	4	... ... $2\frac{1}{4}$,,
11.	Debility ...	3	... ... $1\frac{1}{2}$,,
12.	Syphilis ...	2	... ... $1\frac{1}{4}$,,
13.	Hepatitis ...	1	... ... $\frac{1}{2}$,,
14.	Rickets ...	1	... ... 1 ,,
15.	Scarlet Fever ...	1	... ... 3 ,,
16.	Typhoid ...	1	... ... 1 ,,
		105	2 inches.

The number is 105. Of these 21 gave trustworthy evidence of having taken, on an average daily for several years, a quantity of alcohol equal to or exceeding that contained in four ounces of whisky. Occasionally some laxity had to be allowed in the observance of this arbitrary limit; for instance, allowance must be made for age, occupation, and sex. Little difficulty was experienced however; the alcoholics were, as a rule, well beyond the four ounces. In these 21 the liver reached, on the average, three inches below the costal margin in the right nipple line. I would further point out, as regards alcohol, that these cases represent all those that I have notes of in which the diseased condition was one of alcoholism purely. In other words, so far as my experience of the past five years is concerned, in all cases of alcoholic cirrhosis of the liver, this organ is increased in size. These cases did not represent merely early stages of the disease, as no fewer than 7 of them died within two years of my first seeing them. But these cases I shall deal with more fully in Paper ii.

The remaining 84 were not alcoholic, *i.e.*, their daily intake was less than 3 ozs. of whisky. One or two have been omitted as on the borderland between excess and moderation, but it is only one or two. Few things are more striking than the sharp division which exists between drinker and non-drinker.

Of the 84, in 16 instances the enlargement was due to some obstruction in the lungs other than tubercle, and in these the average extent of the liver below the ribs was 1½ inches.

In 13 the cause is best described as abdominal catarrh, by which I mean some debility of the alimentary tract leading to repeated irritation. Chronic gastritis, gastro-hepatic catarrh, intestinal catarrhs with chalky stools, sick headaches with erythema papulatum, are some of the diagnoses included under this heading. 1¾ inches was the average extent of these livers below the costal arch.

Undoubted gout was the cause in seven, the liver edge reaching two inches below the ribs, oftener, it is true, visceral than arthritic in its manifestation, but in this respect agreeing with Trousseau when he says: "The liver which in regular gout is so often involved, is still oftener affected in abnormal (anomalous) gout." Between these cases of visceral gout and those previously spoken of under abdominal catarrh no hard-and-fast line can be drawn, yet still it seems advisable to attempt some division.

In 11 pulmonary tubercle was the disease suffered from. Here there does not seem any special reason why the liver should become irritated or passively congested; nor is cirrhosis or venous congestion the condition found after death. The amyloid liver used to be considered the special result of pulmonary tubercle, but now fatty infiltration and degeneration is recognised as by far the commoner. "Of 52 persons dying from tubercle, and whose autopsies I have recorded," says Murchison, "the liver was fatty in 20 and waxy in six, and in three of the six there was likewise caries of the bones." Caries of bone is a well-known cause of amyloid liver; hence in only three of Murchison's cases can we fairly say the waxy liver was due to pulmonary phthisis, and these three may well have been instances of prolonged suppuration of the lung.

Ten of the patients suffered from cardiac debility. By this I mean a weakness due to a primary defect of the heart itself,

other than peri- or endo-carditis; such weakness, for example, as accompanies fibroid and fatty degeneration, or is so frequent a sequela of influenza. Two inches below the ribs was the average position of the liver edge in these cases.

Here again there is a close connection between one or two of these and visceral gout, though none could be called cases of true gouty heart—that is, of heart feebleness occurring in a gouty subject.

Five suffered from valvular disease of the heart. These possessed the largest livers of all, $3\frac{2}{5}$ inches being the average extent of the organ below the ribs.

Five were the subject of chronic Bright's disease. The average extent of the liver below the costal arch in these cases being $2\frac{1}{5}$ inches.

Four had obstruction of the bile duct with an enlargement of $3\frac{1}{4}$ inches.

In four others the spleen was the organ chiefly diseased, and in these $2\frac{1}{4}$ inches was the extent of the liver below the ribs.

In three I failed to find any definite local disease, general debility being my diagnosis; yet in these the liver was enlarged to the extent of $1\frac{1}{2}$ inches.

Acquired syphilis was the cause in two, with an enlargement of $1\frac{1}{4}$ inches.

Single cases of acute hepatic congestion, rickets, scarlet fever, and typhoid were respectively the causes of enlargements of $\frac{1}{2}$, 1, 3, and 1 inches.

The condition of the spleen in these cases of large liver is a very interesting point. In estimating the size of this organ it must be remembered that normally it nowhere reaches the rib margin, but has already undergone considerable increase in size when it appears below the costal arch. On the other hand, it is extremely expansible, and too much stress must not be laid on what may be only a very temporary state.

For the purposes of this paper I have supposed that it had enlarged to an extent which might be represented by one inch

when it reached to the edge of the costal arch; also that it was normal in size when one failed to feel it at all. It is evident that there is no attempt here at anything approaching exactness, and any quantitative conclusions which may be drawn can only be vague. Especially must this be so since the spleen enlarges in such different directions, sometimes aiming straight for the umbilicus, and sometimes falling back into the loin parallel with the left kidney and simulating an enlargement of this organ.

The anatomy of its circulation would appear to insist that it should enlarge to a smaller extent than the liver when the condition is one of thoracic obstruction, as the liver then acts as a buffer between it and the thorax. But when the hepatic enlargement is due to an irritant conveyed by the systemic arteries, then the spleen should be the more notably affected, for (1) it is more freely supplied with arterial blood, (2) its structure is more expansile, and (3) it suffers from venous back pressure, as it has to send its blood through the liver, which is already congested by the same irritant as itself.

When the liver affection is due to an irritant conveyed by the portal vein the attitude of the spleen becomes somewhat complex. The liver evidently bears the brunt of the attack, and at first should alone be affected; but later, as it gets diseased and blocked by the cirrhotic growth, the effect of back pressure upon the spleen should become manifest by this latter's enlargement. Moreover, the more diseased the liver the less able, one would imagine, would it be to cope with the irritant and destroy it. This, therefore, would reach the systemic circulation, and in this way, also, would the spleen suffer injury. In cases of portal irritation it would thus seem that enlargement of the spleen points to an advanced stage of the disease.

An analysis of the cases under consideration fairly substantiate these suggestions.

TABLE B.

	Disease.	*Liver enlarged in*	*Liver & Spleen enlarged in*	*Ratio of Splenic Enlargement to that of Liver.*
1.	Valvular Heart	5 ...	1 ...	1 to 8
2	Obstructed Lung	16 ...	7 ...	1 to 4
3.	Syphilis ...	2 ...	1 ...	1 to 1
4.	Rachitis ...	1 ...	1 ...	1 to 1
5.	Bright's ...	5 ...	2 ...	5 to 4
6.	Typhoid ...	1 ...	1 ...	2 to 1
7.	Phthisis ...	11 ...	2 ...	3 to 10
8.	Portal Irritation—			
	A. Non alcoholic	20 ...	3 ...	1 to 3
	B. Alcoholic	5 ...	3 ...	3 to 7

Out of the five suffering from valvular heart disease in which the liver was enlarged, only one—a mitral regurgitation—showed increased size of spleen, and the increase was only one-eighth that of the liver.

Of the 16 cases due to pulmonary obstruction seven had enlarged spleens, the enlargement being one-fourth that of the liver. That is, out of 21 patients who suffered from hepatic enlargement due to thoracic obstruction, only seven had big spleens, and in these seven the average increase in size of the spleens was less than one-fourth that of the liver.

But when we turn to the other class, where the irritant is in the systemic circulation, we obtain very different figures. The child with Rickets, and one of the two cases of Syphilis, had a spleen as much enlarged as the liver. Two of the five patients with Bright's disease had spleens 1¼ times more increased in size than the corresponding livers; and the typhoid spleen was twice as much increased in size as the liver in the same patient. That is, out of nine cases with large livers due to systemic irritation five had large spleens, and the average increase in size of the spleen was 1¼ times that of the liver; that is, the spleen was enlarged in twice as great a proportion of cases, and the average increase of its size was 4½ times as great.

The 11 cases of Phthisis stand on a plane by themselves; as I have said, the change in the liver is chiefly a fatty one, and so far as I know the spleen is not susceptible to this degeneration. Hence it is, perhaps, that only 2 of these 11 had enlarged spleens; the increase in the spleen being then $\frac{3}{10}$ that of the liver. It may well be that in these two instances amyloid degeneration had taken place as, to this, we know the spleen is very liable. In this regard it would be very interesting to learn the experience of surgeons as to the condition of the liver and spleen in cases of long-continued suppuration.

Coming to the cases where portal irritation was the cause of the hepatic disease, we may with advantage make two classes—alcoholic and non-alcoholic. Of the 20 non-alcoholic, in only 3 were there big spleens, and in these 3 the splenic increase was but one-third the hepatic. Of the 5 alcoholic, in which the size of the spleen is definitely noted, in 3 it was increased, and the increase was three-sevenths that of the liver. That is, in 25 cases of portal irritation with enlarged livers, in 6 was the spleen also large; and in these 6 the ratio of splenic to hepatic enlargement was as 8 to 21.

These figures are very near those of thoracic obstruction; and if we take the non-alcoholic ones by themselves, the effect on the spleen is even less than in those due to venous back pressure. The irritants are apparently destroyed before they leave the liver, so that the spleen merely suffers from the obstruction in the liver which they produce, and, moreover, this obstruction does not seem to be great. But when the irritant is alcohol, the spleen suffers much more severely, not alone I think because this produces much greater obstruction in the liver, but because alcohol passes on beyond the liver through the pulmonary into the systemic circulation, and is thus more nearly allied to the class of systemic irritants. It must be remembered, too, that the non-alcoholic cases were not cases of severe disease; they were mostly instances of functional digestive troubles.

There are some noteworthy points in the pathology of hepatic

cirrhosis. The liver tissue can be irritated by the retention of waste products—passive cirrhosis—as in obstruction of the hepatic vein or bile ducts, or by the bringing to it of injurious material—active cirrhosis—either by the portal vein or hepatic artery. This *arterial* cirrhosis is not yet fully admitted, but the effect upon the liver of constitutional poisons, such as those of the acute specific fevers and of malaria, is strong evidence of its existence; moreover, Dr. Schupfer, of Rome (*Lancet*, 17, iii., 1894), has shown that the liver not only destroys poisons brought to it by the portal vein, but that it also does so when these are injected subcutaneously. There are thus four anatomical sources from which cirrhosis of the liver may arise, and to some extent the distribution of the resultant fibrous tissue depends upon the particular set of vessels involved. But this occurs, I think, to a much smaller extent than one would anticipate. In the early stages of disease no doubt the morbid anatomy is distinct; but soon overlapping must occur, and the conditions as we meet them in the *post mortem* room are not seldom indistinguishable under the microscope. Senator, *e.g.* (*Berl. klin. Woch.*, 18, xii., 1893), says that cardiac and portal cirrhosis cannot always thus be differentiated, the portal and hepatic radicles being jointly involved in both. And Sieveking (*Cent. f. allg. Path.*, 31, xii., 1894), as a result of a microscopical examination of 20 cases of atrophic cirrhosis (not all alcoholic), states that the proliferated connective tissue infiltrated the liver substance in quite an irregular manner, the variations being so numerous that no particular type of proliferation could be recognised.

Perhaps 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 and to have no etiological value,

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 limited experience goes there is one variety of fibroid change which is almost entirely 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 have little to add to this, but I would emphasize the fact that it is *translucent fibroid material* with little or no evidence of fibrillation which forms these masses of new growth—masses, 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 within 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; and Dr. Crooke has given me several sections where these nuclei are seen undergoing various changes through the fusiform connective tissue cell up to the fully-formed fibre.

Everyone who has had any experience in the treatment of the large livers due to portal irritation must have noticed how very rapidly they will increase in size from apparently slight causes. A liver which yesterday reached but two inches below the ribs

to-morrow may reach four, particularly if it had on some previous occasion reached as low or lower, this increase taking place far more rapidly than in a liver previously normal and exposed to the same evil influence. Moreover, the symptoms of ill-health are not so severe as those which would accompany an equal increase in the size of a healthy liver.

The explanation is obscure, but it may partly be that a liver once dilated dilates with greater ease and less pain the second time, and, also, partly to the fact pointed out by Prof. Hamilton, that the radicles of the *hepatic* vein, as well as those of the portal, are compressed by the fibroid tissue, while Ziemssen states that during gradual occlusion of the portal vein or of its chief branches the channels of the hepatic *artery* gradually widen to allow the entrance of blood sufficient both for the liver's functional needs as well as for its nutrition, hence hyperæmia may more suddenly arise and more slowly subside. On the other hand, we must remember that the smallest venules of the portal system convey blood from the hepatic artery as well as from the portal vein, and hence their occlusion must needs diminish the arterial supply.

In nearly every case of enlarged cirrhotic liver when the edge is smooth and rounded and the surface very firm and resistant, there is a considerable amount of hyperæmia or venous congestion : in such cases great benefit can be effected by efforts towards hepatic depletion. It is surprising what a decrease in size will occur in a few hours if meanwhile the bowels have undergone free watery purgation. This effect of a watery purge is, to a great degree, quite independent of any action the drug used may have as a hepatic stimulant or cholagogue, for sulphate of magnesia (*e.g.*) is well able to largely reduce the size of a congested liver. The exact nature of its action is not very clear. It appears to act by lessening the flow of the portal blood into the liver, but more importantly, I think, by encouraging the flow in the coronary artery of the stomach, and so lessening the pressure in the hepatic branch of the cœliac axis, and further, by drawing extra blood into the pyloric and

gastro-duodenal divisions of this hepatic branch it must greatly reduce the pressure in the remaining division, that is the hepatic artery itself. Thus the pressure in the hepatic artery undergoes a twofold reduction.

Hence, in all cases of hepatic congestion, the more or less constant use of some such saline as is contained in a natural aperient water is to be advocated. This of itself is often sufficient; but if a stronger remedy be required, it is well to supplement it by local leeching, blistering, or fomenting, or by the use of a hepatic stimulant. Of hepatic stimulants I have tried many, but the two which have given me the most constantly beneficial results are mercury and rhubarb. Nitro-hydrochloric acid has occasionally acted brilliantly, but far oftener has it failed. Chloride of ammonium has never shown anything but a feeble power in my hands. Podophyllin so often has upset the functions of the alimentary tract. Euonymin to my mind is much over-rated, and formerly I used it largely. On the other hand, I have faith in the stimulating action of the salicylates, especially when these are united to small doses of colchicin.

For all glandular organs calomel is a bad purge but splendid stimulant. Given in small doses of one-sixth to one-twelfth of a grain t.d.s., it so stimulates the abdominal glands that a mild saline aperient is able to freely open bowels over which this had no power at all previous to the administration of the calomel. But as a purge it often unduly depresses the patient, probably because so large a quantity too strongly stimulates the gastro-intestinal glands with which it comes into direct contact, whilst the single large dose so quickly passes out of the body that it fails to sufficiently stimulate the more remote glands (liver *e. g.*) as the less irritating fractional doses do. Who has not experienced the woeful debility and melancholy nausea with distaste for food following a too large dose of calomel? Perhaps this is why such a dose goes well with alcohol or opium, as these two drugs tend to inhibit glandular action.

If the condition be accompanied by gastro-intestinal catarrh,

with a dry and furred tongue, then I find a powder containing two to four grains each of rhubarb, soda, and ginger, given thrice daily before meals, most valuable. This is merely a modified Gregory: soda I prefer to light magnesia, as it is more easily mixed with liquid; the increased proportion of ginger I find advantageous to counteract the remarkably cold depressing sensation which rhubarb has on a sensitive stomach. If taken in milk instead of water, the repellant flavour of the rhubarb is almost entirely disguised. But if the tongue be flabby, with a white moist fur, and there be diarrhœa, bismuth often serves one's purpose better. In these cases there is often much want of systemic tone, and a vegetable bitter is wisely added. Some such prescription as this is useful :—

Bismuthi Carb. - -	gr. 5	
Tr. Chloroform Co. -	℥ 10	
Inf. Chiratæ	ad ʒ 1	t. d. s. p. c.

With so small a number of cases anything approaching dogmatism would be rash and foolish; and far from dogmatic is my intention. I have merely from the material I had at hand drawn such conclusions as this seemed most naturally to indicate.*

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* Several of the livers referred to in this paper would not be described as cirrhotic, but I have included them under the title, for though some of them represent too early a stage of disease for definite *fibrosis* to have appeared, yet fibrosis would arise later if the *causa causans* continued. I have, in fact, used cirrhosis as a pathological term, and not one of mere morbid anatomy. This statement does not hold good of the phthisical livers, for the essential pathology of these is not a cirrhosis; but I have included them as useful for comparison.

Paper II.*

THE ALCOHOLIC FORM.

I have for some years felt that not sufficient prominence was given in our text-books to the enlarged form of alcoholic cirrhosis of the liver. It has always seemed to me curious that, whereas in cirrhosis from other causes, *e.g.*, 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 Text-Books 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 following 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.

Let me read to you the description given by Dr. Frederick Taylor in his excellent manual.

"This fibrous tissue in course of time contracts, and thus compresses more and more the hepatic cells, the branches of the portal vein, and perhaps the bile-ducts. The varying size of the liver is thus in part, at least, dependent on the stage of the process. The organ is at first enlarged by the over-growth of connective tissue, and very large cirrhotic livers also contain a quantity of fat. If the patient lives long enough, the tissue

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* Read in the Section of Medicine at the Annual Meeting of the British Medical Association in London, August, 1895.

contracts, the liver-cells and fat disappear, and the organ is reduced much below its normal weight. But it is important to remember that many patients die of cirrhosis while the liver is still as large as normal, or even very much larger.

"Some writers have tried to distinguish a *hypertrophic* cirrhosis as a different form from the contracting or *atrophic* cirrhosis. They apply this term to the cases of great enlargement of the liver, in which jaundice is a marked symptom, ascites is absent, and death tends to take place with cerebral symptoms; and they state that this kind of liver is peculiar in that the new tracts of fibrous tissue contain double rows of small cubical cells, which are biliary ducts, newly formed to compensate for the obstruction of old ones; and this variety has also been called *biliary* cirrhosis. But, firstly, there is much doubt as to whether these rows of cubical cells are really bile-ducts; secondly, if they are, whether they are newly formed; and thirdly, it is certain that they have been found in contracted livers. It has also been argued in favour of there being two distinct forms, that the very large livers with jaundice as a symptom have not been observed to contract; but I have recorded a case in which a liver that reached below the umbilicus, in a patient with strongly marked jaundice and no ascites, was found fifteen months later to have contracted quite close under the edge of the ribs. On the whole, it seems more reasonable at present to consider the differences in size as only indications of different stages in the process. *Monolobular* and *intercellular* cirrhosis have also been spoken of; the latter occurs in congenital syphilis."—Taylor's Manual of the Practice of Medicine, p. 625.

This to us, with the light of our experience behind us, may seem a perfectly clear and reasonable account, but the student I fear it still leaves in uncertainty. At least so I find it in the Birmingham School where Dr. Taylor's book is a popular authority.

In the second edition of Fagge's medicine we find the following statement: "In advanced cases the liver is generally smaller

than natural, and sometimes it is very greatly reduced in weight. The *post mortem* records of Guy's Hospital record several instances in which the organ has weighed as little as 32 or 34 ounces, and cases have been recorded in which it has been still smaller. But not uncommonly a cirrhosed liver is found after death to be of the natural size, or even above it. *Whenever considerable enlargement is present, I believe that the organ is almost always also loaded with fat.*"

But this view is not confined to the statements of text books. Dr. Saundby in a valuable paper read before the Association in 1890 on the "Varieties of Hepatic Cirrhosis" gives the size of the liver in the alcoholic variety as "usually small;" in the cardiac, "usually large;" in the gummatous, "irregular;" in the scarlatinal, "normal;" and in the other six "large." He thus makes a broad clinical distinction between the alcoholic and other forms of cirrhosis. Such a distinction as this is not, I believe, in accord with the facts of the case.

It seems to me we have come to a stage when the student can quite justifiably be taught a simple, clear and unified doctrine on this condition. I will venture to put my meaning into words.

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 bear the brunt of the attack and 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 two or three 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. The evidence of this paper shows that the enlarged form is more commonly met with after death, whilst during life, in the great majority of cases, the liver is easily felt below the costal margin in the right nipple line.

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. Case 28.)

I have endeavoured to see how far facts agree 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, and I have also abstracted all similar cases from my private case-books 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 twenty-eight, and in twenty-six of them the liver reached below the costal arch in the right nipple line, it being felt on the average in these twenty-six cases 2¼ inches 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 inches. 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.

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: three have suffered from hæmatemesis; two from alcoholic neuritis; and number 28 is a type of some

of the others—this gentleman had taken alcohol, whiskey, to excess for over forty years, for thirty years his daily average being not less than 15 oz., and during the succeeding ten years not less than 6 oz. Yet the edge of his liver is felt thick, hard and rounded, 2½ inches 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 whiskey 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 dyspnœa, 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.

Class II. contains those cases where ascites had 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 eighteen in number, and in only nine 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 dulness 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 1⅔ inches; 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 twelve cases. In each of the five which died in Hospital ascites was present, and in the seven taken from my private case-books 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 two of the Hospital ones: in each of these latter it reached three inches beyond the costal margin, and in my cases the average extent of it below the ribs was also three inches. Taking the twelve cases together the liver extended on the average two and a quarter inches beyond the ribs.

The Fourth and last 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, only contains nine names and the size of the liver in one of these is not mentioned. We have, therefore, but eight 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 $43\frac{2}{3}$ oz.: taking Reid's statement that the average weight of the female liver is 45 oz., we find that in these three cases of cirrhosis the weight exceeded the average by $3\frac{1}{3}$ oz.

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 only weighed 35 oz., this brings to light another fallacy, viz.: 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 oz., giving an average weight of 64. Taking Reid's average again as 53, this gives an excess of weight of 11 oz.

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 $1\frac{6}{11}$ inches.

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, *viz.*, That in the large 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 twenty-six beer drinkers the average extent of the liver below the costal margin was only $1\frac{1}{13}$ inches; $1\frac{5}{7}$ inches was the corresponding measurement of those who indulged in beer and spirits; whilst of the sixteen who were pure spirit drinkers (nearly always whiskey), the liver reached on the average as much as $2\frac{1}{4}$ inches 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 whiskey 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.

Lastly, 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 eight cases whose cadavers were examined three had jaundice and in two of these the liver was large, but in no case was the jaundice marked. Only two cases are noted as dying with cerebral symptoms (out of twenty-one); one of these had a large and one a small 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 oz. 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 we have 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 have usually made the matter plain before extreme contraction takes place. But if it be dogmaticaly 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.

List of Cases.

Class I.—Non-fatal cases without ascites.

1. George L., 51.* "Fairly temperate." Gout, hæmatamesis. Liver two inches below costal margin in right nipple line; edge hard, regular, thick, and tender; size unchanged at date of discharge.

* These figures refer to the age of the patient.

2. Eliz M., 32. "Half-pint beer and occasional spirits."* Alcoholic neuritis. Liver almost to umbilicus; edge hard, firm, regular, and slightly tender.

3. Tom H., 32. "Six pints of beer and some spirits." Liver nearly to umbilicus; edge hard and rounded.

4. Harry B., 10. "A glass of beer every day." Liver one finger's breadth below † the ribs.

5. John L., 32. "Two glasses of spirits and one pint of beer." Syphilis. Liver two inches below, regular, tender, and rather hard. Later note: Less tender, softer, one inch below.

6. Ellen C., 40. "One and a half pints of beer daily, and two ounces of brandy twice a week for ten years." Spleen one inch and a half below the ribs. Liver at umbilicus: edge hard, rounded, rough, and rather tender.

7. Charlie H., 3. "An occasional sip of beer (never spirits), not more than a quarter of a pint in a week," is grudgingly admitted by parents. The child, however, prattles about whisky, brandy, and gin, and this morning asked for a taste of the last. Liver two inches below umbilicus, fairly firm, not hard, edge rounded. Five months later (still in hospital) the edge only reached one inch below the costal margin.

8. William J., 47. "Big beer drinker." Spleen not felt. Hæmatemesis. Liver not felt.

9. George S. W., 60. "Used to drink both beer and spirits in great quantity." Spleen not big. Hæmatemesis. Liver not felt in R.N.L. The edge is one inch below ziphoid in mid-line; not rough.

10. Samuel T., 32. "Two pints of beer; no spirits." Bright's disease at 30. Liver two inches below ribs; smooth and tender. In fourteen days the tenderness goes, but liver same size.

11. Joseph R., 42. "Three pints of beer." Some years in India. Bronchitis every winter. Œdema of legs now and twice before. Spleen felt. Liver an inch and a half below ribs.

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* The amount of alcohol stated represents the average *daily* consumption.

† The position of the liver edge is always estimated in the right nipple line.

12. Joseph H., 34. "Twenty to thirty glasses of beer and a little spirit." Heart rather big. Lungs have sibili. Œdema of legs. Spleen not made out. Liver to umbilicus: consistence normal.

13. Alfred W., 29. "Six pints of beer." Œdema of legs and scrotum. Liver to within one inch of umbilicus, softish and smooth; tender.

14. John C., 49. "Too much spirits." Liver hard, tense, prominent, almost to umbilicus, but in ten days only just felt.

15. Mr. T. W. V., 48. "One pint of beer." Creeping pneumonia; this nearly cured in a month. Liver not very hard nor prominent, two inches below ribs; the same size a month later.

16. Mr. A. C., 52. "Three and a half pints of beer." Four years of influenza and bad times. Diabetes intermittent for nearly four years. Insured as first-class life two years ago. Liver to umbilicus.

17. Mr. J. V., 43. "Many whiskies." Alcoholic skin. Large dilated heart. Chronic bronchitis. Liver softish, nearly to umbilicus; two years later half way to umbilicus and not hard.

18. Mr. J. J., 51. "Free liver." Arthritic gout. Large heart. Liver half way to umbilicus.

19. Mr. J. L., 40. "Free liver." Acute nephritis. Spleen felt. Liver soft, half way to umbilicus.

20. Mr. H. L., 35. "Four pints of beer, free liver." Heart big. Liver half way to umbilicus.

21. Mr. W. L. S., 43. "Half a pint of whisky." Liver to umbilicus; edge sharp, hard, and smooth; a month later one-third way to umbilicus, and softer.

22. Mr. E. L., 63. "Free liver in food and alcohol." Gout. Heart big, but returned to normal after five weeks' treatment. Liver half way to umbilicus; no smaller after five weeks' treatment.

23. Mrs. K. S., 37. "Spirit drinker." Alcoholic neuritis. Father died of gout, mother of diabetes. Liver just felt; edge thinnish, doubles up under finger.

24. Mrs. E., 62. "Spirit drinker." Liver nearly to umbilicus, hard and rugged; thick edge.

25. Mr. W., 43. "Heavy drinker, chiefly beer." Delirium tremens. Liver to umbilicus, hard and rounded.

26. Mr. E. S., 32. "Has drunk much for many years." Acute dilatation of heart. Spleen felt. Liver to umbilicus, hard and uniform.

27. Mr. J., 49. "Free drinker of spirits and beer." Heart very big. Hepatic glycosuria, which vanished under treatment. Liver to umbilicus; edge rounded, not very hard.

28. Mr. T. W., 67. "From 20 to 50 years of age, about three-quarters of a pint of whisky; from 50 to 60, about one-third pint; since 60, still less." Chronic bronchitis twelve winters; now permanent dyspnœa. Heart large and rapid. Thick cloud albumen. Slight œdema of legs. Spleen not felt. Liver two and a half inches below ribs; edge thick, hard, and rounded.

CLASS II.—NON-FATAL CASES WITH ASCITES.

1. John A., 33. "Seven pints beer for eighteen years." Liver dulness begins at fifth rib; edge not felt below.

2. Bruce N. 45. "Ten glasses of beer for ten years." After paracentesis the liver edge is visible to the eye as a sharp prominence below the ribs; surface nodular and tough.

3. Tom H., 56. "Moderate drinker." Liver not felt.

4. Ellen P. 40. "Friends supply strong history of drinking." After tapping the liver edge was felt distinctly one and a half inches below and to left of ensiform cartilage, but not in R.N.L. The left lobe was markedly nodular.

5. William H., 36. "Heavy rum drinker for six years." Mitral systolic murmur. Liver not felt.

6. Benjamin H., 41. "Three pints beer." Liver begins at fifth cartilage; not felt before paracentesis.

7. Alfred G., 40. "Two pints beer." Liver not felt; dulness for half an inch below ribs.

8. Jacob L., 62. "Moderate beer drinker." Liver not felt in R.N.L., but felt in epigastrium, where its surface is distinctly nodular.

9. William T., 34. "Heavy beer drinker for fifteen years." Liver vertical dulness one and three-quarter inches; not felt in R.N.L., but in epigastrium, where it is distinctly nodular.

10. Mary A. G., 47. "Whisky and gin drinker for seven years." Spleen not felt. Liver vertical dulness one and three-quarter inches.

11. Mary W., 49. "One pint of beer." History of scarlet fever, cirrhosis of liver and kidney. Mitral incompetence. Spleen just felt. Liver two inches below ribs, hard and rough.

12. Joseph P., 67. "No alcoholic history obtainable from the patient, but there is not the least doubt about undue indulgence in stimulants." Spleen not felt. Liver not felt.

13. Sarah C., 45. "History of alcohol doubtful." (?) Nephritis. Spleen not felt. Edge of liver felt two inches above umbilicus.

14. George J. W., 35. "Heavy spirit drinker." Alcoholic neuritis; delirium tremens. Liver large, smooth; edge firm and easily felt at level of umbilicus. Three months later (still in hospital) liver same size.

15. Henry E., 48. "Very heavy drinker." Delirium tremens once. Palpitation and dyspnœa. Œdema of legs. Liver one inch below ribs: surface and margin irregular, but not tender (on admission fourteen days before—only just felt).

16. Daniel H., 38. "One and a half pints of beer." Spleen just felt; hard. Liver one inch above umbilicus: edge hard, thin, and painless; appears to be contracted into a globular form. No ascites at this time. Two months later the liver was a little lower and some ascites had arisen.

17. M. Ann D., 50. "Has been occasionally the worse for liquor." Syphilis. Slight œdema of legs. Spleen felt; hard. Liver half way to umbilicus: edge extremely hard and rough.

18. Frank R., 48. "Four pints of beer." Hæmatemesis. Œdema of scrotum. Splenic dulness a little increased. Liver almost to umbilicus: tender.

CLASS III.—FATAL CASES IN WHICH NO POST MORTEM COULD BE OBTAINED.

1. Henry B., 38. "A rather heavy beer drinker." Ascites. Slight jaundice. Mitral systolic murmur, probably organic. Spleen felt. Liver two inches below ribs: not tender; edge hard and irregular; three months later, edge of liver one inch and a quarter above umbilicus; two months later still, dies.

2. Annie B., 37. "Has drunk spirits for at least ten years, spending as much as half a crown a day in gin." Ascites. Liver four inches below ribs.

3. Edward S., 49. "Has only drunk a little beer, and never to excess;" but his brother reports him as "a very heavy spirit drinker." Ascites. Liver not felt.

4. Eliz. W., 39. "A strong history of alcohol, but the kind not stated." Ascites. Hæmatemesis. Spleen easily felt. Liver not felt.

5. James F., 40. "Drank freely of beer till fifteen months ago; then had a fit, and since then has only taken a little spirit." Hæmatemesis. Ascites and anasarca. Pulmonary systolic murmur. Liver not felt (after tapping).

6. Mr. W. W., 76. "Free liver, chiefly as regards alcohol." Gouty. Weak heart. Frequent short attacks of jaundice for four years; at present a very slight one. Liver as low as umbilicus; left lobe especially hard. Two months later only half way to umbilicus. Died five months after first visit.

7. Mr. C. R., 46. "Four pints of beer and three whiskies." Heart big; double mitral murmur. Urine 1015, with thick cloud of albumen. Liver down to umbilicus. Died eighteen months later.

8. Dr. B., 43. "Heavy spirit drinker for many years."

August 13th, 1889. Seen for severe hæmatemesis. Liver felt a little below the umbilicus; edge very hard and tense.

October 2nd, 1889. Has recovered from his attack. Liver one inch below ribs.

February 3rd, 1891. As well as usual. Liver reaches to umbilicus; consistence not increased. Spleen felt one inch below ribs.

October 30th, 1891. Liver same size, large and hard.

December 3rd, 1891. Liver one and a half inches below ribs; consistence normal.

December 15th, 1893. Dies suddenly with fresh hæmatemesis.

9. Mrs. J., 52. "Wine and spirit drinker." Ascites and anasarca. Heart feeble; not big. Liver more than three inches below ribs; edge hard, rugged, and rounded. Dies suddenly in fourteen days.

10. Mr. A. H., 35. "Has drunk much beer and spirit for four years." Liver nearly to umbilicus. Dies in two months of acute phthisis.

11. Mr. K. K., 38. "Ten glasses of beer for fifteen years." Renal cirrhosis and purpura. Spleen not felt. Liver nearly to umbilicus; edge thinnish and tough. Dies in six months.

12. Mrs. A., 45. "Four years ago, after trouble, began chief excess, often amounting to a bottle of brandy a day." (Mother has had delirium tremens.) Alcoholic neuritis for two years. Heart fatty. Spleen one and a half inches below ribs. Liver down to umbilicus; edge tough and rather thin. Dies in a fortnight.

CLASS IV.—FATAL CASES WITH THE RESULTS OF POST MORTEM EXAMINATION.

1. Catherine H., 61. "One and a half pints of beer; also whisky and gin." Ascites and dyspnœa. Diarrhœa. Liver just below ribs: edge sharp, smooth, and tough.

Autopsy.—Fatty heart. Old perityphlitis. Thickening of intestine. Liver 35 ounces; surface pale, hobnailed, and outline irregularly contracted; many cicatricial but shallow depressions. Section not so rough; bile-stained with slightly prominent, well-marked bands of fibrous tissue. No adhesions.

2. Keziah M., 49. "Continuous beer-drinking for some time." Ascites for six weeks; œdema of legs for three weeks; vomiting, three weeks; and jaundice, two weeks. Spleen felt. Liver half way to umbilicus: the surface has small nodules and is very hard; not tender.

Autopsy.—Heart fatty and dilated; mitral constriction; calcareous aorta. Peritoneum thickened. Spleen 9 ounces, but soft. Veins dilated on both sides of cardiac orifice of stomach. Liver 43 ounces, *flattened out from above downwards*, typically hobnailed; parenchyma yellow. All the thick portion of the organ appeared to have suffered multilobular, but the anterior flattened portion monolobular cirrhosis.

3. Charlotte B., 42. "Has drunk spirits freely for the last four years, a pint or more a week." Mitral systolic murmur. Jaundice. Liver two and a half inches below umbilicus: edge firm and hard; surface smooth, regular, not tender. Dies in seven days, when liver only reaches half an inch below umbilicus.

Autopsy.—Much subcutaneous and subperitoneal fat. Spleen enlarged, too firm; trabeculæ well marked, but capsule not thick. Kidneys: both lax; marked yellowness of pyramids and some yellow streaks in cortex. Much broncho-pneumonia and œdema in lungs. Heart has much "tabby-cat" degeneration. Liver 68 ounces; right lobe pushed down and left lobe up; surface pale yellow, tough, and marked all over with small hobnailed excrescences. On section, there is evidence of marked cirrhosis with apparently a good deal of fatty change, and some bile-staining of the lobules. Microscope:—The fibrosis is moderate in amount but with a great tendency to be monolobular. There are in places distinct prolongations, even to the centres of the lobules. The fatty infiltration is in excess of that usually found in cirrhosis.

4. Edward C., 37. "Has been a heavy beer-drinker for ten years; also has drunk much of other forms of alcohol." Hæmatemesis. Ascites. Slight jaundice. Spleen four inches below ribs. Liver reaches to umbilicus; left lobe not so large; edge rounded, smooth; not tender.

Autopsy.—Spleen 20 ounces, firm and granular. Kidneys 21 ounces, congested, but otherwise normal. Liver 74 ounces. It has thick recent lymph adhesions, and the capsule is chronically thickened. Surface throughout moderately hobnailed. Section very bloodless; immense amount of fibrous tissue, the parenchyma standing out almost entirely in small yellowish islets. The radicles of the portal vein were dilated, and in some places scanty hæmorrhages were visible. The cirrhosis mostly took the multilobular, but in some parts the unilobular form.

5. William B., 25. "Three pints of beer." Hæmatemesis. Ascites. Slight jaundice. Spleen not felt, but dulness increased. Liver not felt.

Autopsy.—Spleen 12½ ounces. Liver extremely contracted and distorted. The capsule on the upper surface is very adherent and thick. The surface has large divisions with smaller secondary ones (thus producing raised nodules). On section the colour is seen to vary from dark olive to reddish brown, and the organ is intersected by fibrous bands which join the much thickened capsule. (An unusual case of capsular disease).

6. John M., 41. "In brewing trade for twenty-four years. Always moderate; never more than four glasses of beer and three whiskies." Ascites and œdema of the legs came on simultaneously. Liver "can be felt chiefly as to its left lobe, the rest being difficult to distinguish: friction heard over hepatic region."

Autopsy.—Chronic granular peritonitis. Veins near each side of cardiac orifice of stomach are distended and tortuous. Spleen 10 ounces, soft and flabby. Heart muscle soft and flabby; no evidence of organic disease. Kidneys 19¼ ounces, soft and flaccid; congested. Liver: typical hobnailed variety; bile-stained; 46 ounces.

7. James W., 51. "Two or three pints of beer; no spirits." Twelve and a half years in the army; five in India. Liver dulness from seventh rib to half an inch short of costal margin. Died comatose.

Autopsy.—Liver 40 ounces; a typical instance of the hob-nailed, contracted variety.

8. Joseph B., 34. "For three years six pints of beer; previously not so much, but 'pretty well.'" Œdema of legs and anasarca for four months. For fourteen days, ascites. Liver one inch and a half below ribs; begins at fifth rib.

Autopsy.—Kidneys: large, white, with some fibrosis. Spleen large and cirrhotic. Acute double pleurisy; pericarditis and peritonitis. Liver 68 ounces. Left lobe deformed by cicatricial contraction into a bossy puckered mass. Surface unusually smooth for cirrhosis. Capsule opaque, thickened and adherent by short adhesions to diaphragm and stomach. Cut surface smooth. Substance too yellow, and the lobules have a distinctly fatty outline.

Microscope.—There is hardly any fatty infiltration. The cirrhosis is very variable; in places moderate in amount and multilobular in distribution; in other places there is a mass of fibroid material with only scattered cells to remind one of the existence of the liver parenchyma. Much "bile-duct" formation.

9. James P., 50. "Long history of excess in all kinds of alcohol, chiefly wine and spirits." Shot himself; no examination prior to death. Liver 92 ounces; rounded, very hard and tough. The cut surface shows prominent islets with yellow surroundings.

Microscope.—There is an immense amount of fibrous tissue, chiefly interlobular, but also minutely and very extensively penetrating the lobules, sometimes isolating individual cells and often groups of two or three, the fibrillation being then often entirely absent, and areas equal in size to two or three lobules being completely transformed into grey translucent fibroid material. The liver cells found in these areas are mostly nuclei only, and on the outskirts of the masses is much "bile duct" formation. There is some fatty infiltration, but this is small in amount, and could account for only a very small portion of the increased weight.

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