An Address

ON

SOME MISCONCEPTIONS WITH REGARD TO DISEASES OF THE LIVER.

Delivered before the Oldham Medical Society.

By W. HALE WHITE, M.D.,

Physician to, and Lecturer on Medicine at, Guy's Hospital.

GENTLEMEN,—I think it is due to you that I should apologize for the title of this address, but I can assure you I do not mean to imply that any of you have any misconceptions. I have, however, in teaching and examining found certain misconceptions exist, and I thought they might interest you.

THE CAUSE OF CIRRHOSIS.

The first hepatic disease I propose to take is cirrhosis of the liver, and by that I mean the disease which is characterized by the liver which we all of us know so well on the *post-mortem* table; the hard liver with a great deal of increase of uniformly-distributed fibrous tissue, which is easily recognizable to the naked eye.

Alcoholic Drinks.—In the first place, we will take its etiology. This is commonly set down to alcohol, and the disease is called alcoholic cirrhosis. Now, I find that in 100 cases of this disease it is quite easy to obtain a history of alcoholic excess in 60, so that probably there is such a history in 80 or more per cent.; for it is obviously not possible to obtain an accurate history in every case. But, striking as this high percentage of alcoholic histories is, I hope to be able to suggest to you reasons why we should not regard alcohol as the cause of this condition of liver. In the first place, no one has yet succeeded in producing cirrhosis of the liver by giving absolute alcohol to animals. At the most, a little fatty degeneration of the liver can be produced in that way. In the next place, there is this striking fact: that although an excess of alcoholic drink is very widely spread over the world, yet cirrhosis is not by any means widely spread. It is a common enough disease in London; it is uncommon in Scotland. Take, for instance, the experience of a friend of mine who was on the staff of a London hospital for some time, and is now on the staff of a large hospital in Scotland. After he had been many years on the staff of the Scottish hospital he had only seen 2 cases of cirrhosis of the liver, and one of those was in a woman who had come from Sussex. And another Scotch physician has told me that in Glasgow cirrhosis of the liver is not nearly so common as in London. Many other observers have noticed the same fact: that while an excessive consumption of alcohol appears to be distributed over a large area, cirrhosis of the liver is not coextensive with it.

Then, again, cirrhosis of the liver is sometimes found in children. Now very often, I quite admit, these children have taken alcohol. I have come across cases myself where the mother has boasted that she has given the child spirits. But there are other cases in which however carefully you go into the question, you cannot make out that alcohol has been given to the child. Only the other day I heard of a case in which the physician was driven to conclude that it was the alcohol contained in five minims of tincture of perchloride of iron three times a day that had caused the cirrhosis of the liver in the child. I think these facts do suggest that alcohol *per se* is not the cause of the cirrhosis. And lastly, the epidemic that occurred around Manchester of arsenical neuritis in people who were drinking beer opens our mind in the same direction. For the first few cases which were observed were set down to alcohol, and then, as the cases multiplied, the matter was gone into, and it was found not to be the alcohol in the beer that had caused the trouble, but arsenic. So I would suggest to you that it is not the alcohol alone in alcoholic drinks which causes cirrhosis of the liver, and that in order to be accurate we ought to say that it is the alcoholic drink and not the alcohol which causes cirrhosis. Or it may be that there are two factors present: one is alcohol, and the other is a factor which we have not yet got hold of. But it is clearly very important, if we are going to discover the cause of cirrhosis of the liver, to be accurate as to whether alcohol alone can cause cirrhosis, and I hope I have been able, if not to convince you, yet, at any rate, to suggest to you that something else must act in these cases.

The next so-called misconception that I want to draw your attention to, still connected with cirrhosis of the liver, is this. The books give many causes of cirrhosis of the liver, sometimes as many as twenty causes. It is true they put alcohol first, but I shall try and show that the disease which we recognize with the naked eye on the *post-mortem* table as cirrhosis of the liver, that we all know so well, has no other cause than alcoholic drinks. I believe the confusion has arisen in this way: that many other causes than alcohol will increase the fibrous tissue in the liver; and so the man writing a book in his study, and not writing from what he sees in the *post-mortem* room, lumps together all the conditions of increased fibrosis of the liver as cirrhosis, and great confusion ensues, because when it is said that a man has cirrhosis it is generally meant that he has alcoholic cirrhosis.

Syphilis.

I think all will agree with me—that if you see on the *post-mortem* table any condition of liver that can be put down to syphilis, you would expect a student who was only even a beginner to know that such an appearance of the liver was not due to alcoholic drinks. As far as I know syphilis can do the following things to the liver.

Acquired Syphilis.—There is that rare jaundice which occurs in the secondary stage, in which, in the very exceptional cases of death occurring while the patient is in this condition of jaundice from secondary syphilis, the liver is found to have a uniform increase of fibrous tissue throughout it. But there is none of the puckering and scarring and hobnailing that is so characteristic of the disease commonly known as alcoholic cirrhosis. This syphilitic cirrhosis is so rare as to be of little clinical importance. So leaving it aside, what do we usually see in the *post-mortem* room in the liver indicative of acquired syphilis? We sometimes see great fibrous bands cutting the liver up into areas; sometimes gummata, sometimes the liver is lardaceous, and sometimes we see a perihepatitis. All these things may be due to acquired syphilis. Not one of them ought, by the merest beginner, to be confused with what is commonly called alcoholic cirrhosis of the liver. They are all quite distinct from it. First, many of the fibrous bands are big and broad, often one-third of an inch wide, cutting the liver up into areas, and as big, sometimes, as the back of one's hand; and never little hob-nailed areas which are so characteristic of the ordinary cirrhosis of the liver. Further, none of these results of acquired syphilis ever cause the symptoms of alcoholic cirrhosis.

Congenital Syphilis.—We may see a liver hard, flint grey, full of fibrous tissue, but not puckered. But that is of very little clinical interest, for the children are usually stillborn, or only live a little while. Also, we may get in congenital syphilis gummata and fibrous bands, and very rarely lardaceous disease. I have never known the hard fibrous, steel-grey liver of congenital syphilis diagnosed during life. As to gummata and fibrous bands, I cannot help thinking that they must tend to get well in the natural course of events, for the latest age at which I have seen gummata and fibrous bands in the liver due to congenital syphilis was 12 years. I have spoken to many friends, and the oldest I have been able to hear of from them was a patient of 18. Rarely if ever do you see congenital syphilis of the liver producing fibrous bands and gummata which persist into adult life in the same way as you see the deep scarring about the mouth. Lardaceous liver due to congenital syphilis I have only seen once, in a child, aged 4 years, who died of diphtheria. We see, therefore, that syphilis does not cause symptoms or *post-mortem* appearances resembling alcoholic cirrhosis.

Malaria.

The next cause given in the books for cirrhosis of the liver is malaria. One French author enumerates nineteen varieties of cirrhosis of the liver due to malaria. I have never seen a cirrhosis of the liver at all like that due to alcoholic drinks, which could be certainly attributed to malaria; and many with a much larger experience than myself, for instance, Professor Osler, have no knowledge of such a malarial cirrhosis. Professor Osler would not allow cirrhosis of the liver to be malarial unless melanosis of liver and spleen was present also. Partly, I should think, malarial cirrhosis is largely imaginative. Probably it was so in the mind of the gentleman who described nineteen varieties, but chiefly the mistake has arisen from the fact that many people with malaria have taken more alcoholic drink than they should and the doctor perhaps has kindly wanted to whitewash them, and

consequently the cirrhosis of the liver has been attributed to malaria rather than to the alcoholic drink.

Other Alleged Causes.

Those are the principal causes of cirrhosis which are given; and you see that so far we have no genuine cause for that entity easily recognized during life and after death, and usually styled cirrhosis, except alcoholic drinks. But I take a recent textbook, in its second edition, and the following causes for cirrhosis are given: Rickets, diabetes, gout, cancer, tubercle, passive congestion from heart or lung. And another book adds lead, dyspepsia, and scarlet fever. Well, surely that is a misuse of histological knowledge as applied to clinical medicine. I quite admit that occasionally you may find in some of those conditions a slight increase of fibrous tissue in the liver, but it is not right in a book on clinical medicine to ascribe the condition clinically known as cirrhosis to all those causes. There is in these conditions only a little histological increase of fibrous tissue observable under the microscope; but there are none of the symptoms of cirrhosis, and the liver does not appear like it. Among these conditions increased fibrosis of the liver is most seen after passive congestion from heart or lung disease, commonly called nutmeg liver, for all passive congestion means sooner or later an increase of fibrous tissue. But the increased fibrous tissue in the liver under those circumstances, even after the longest standing heart case, never leads to anything which on the *post-mortem* table can be compared with cirrhosis of the liver due to alcoholic drinks. For my own part, if any student about to qualify could not separate by the naked eye a nutmeg liver from one diseased as a result of alcoholic drinks, I think he ought to be rejected.

Biliary Cirrhosis.

It is said that obstruction of the bile duct will cause cirrhosis of the liver. That is true experimentally. The bile duct has been ligatured in animals, and as a result there has been an increase of fibrous tissue in the liver. But common as obstruction of the duct is in man, all observers are agreed that cirrhosis of the liver due to it is very rare. Out of a considerable *post-mortem* experience I have only twice seen obstruction to the bile duct causing cirrhosis of the liver, and yet think how common cancer and other tumours pressing on the bile duct are. Therefore it is clinically of very little importance. To show you that I am not alone in this expression of opinion, let me give the figures by Ford, who has collected his cases very carefully. He searched through the Surgeon-General's Library at Washington from 1882 to 1900, and yet he could only find in those years 21 cases of cirrhosis of the liver associated with obstruction to the bile duct. He adds 3 which he observed himself, which makes the total 24. Of those, 13 were adults; in 8 the obstruction was due to gall stones, in 3 it was due to cancer; once it was caused by a band, and once by an undescribed tumour. All the rest were infants with congenital obstruction of the duct.

It is possible, he says, that in some of the thirteen adults alcoholic drink may have been the cause of the cirrhosis. So the sum total of the largest amount of evidence we can obtain on the subject is, inasmuch as clinically we can put aside infants with congenital obstruction to the duct—because they soon die—an exhaustive collection of cases from 1882 to 1900 only shows 13 adults in whom cirrhosis of the liver was associated with biliary obstruction; and even in some of them alcoholic drinks may have been the cause. The liver in these very rare cases is at first enlarged a little, and later it comes to look like ordinary alcoholic cirrhosis. The cirrhosis is usually at first interlobular, and later on it becomes perilobular and intralobular, and there are new ducts formed. I think that if it is as rare as I hope I have shown, all attempts to separate the clinical symptoms of biliary cirrhosis from the symptoms of the disease causing obstruction of the duct, must be futile, because no one can have had a sufficient experience of such a very rare condition. Take, for instance, cancer of the liver obstructing the bile duct, and causing cirrhosis of the liver—one man cannot have sufficient experience of such rare cases for him to be able to dissociate the symptoms due to cancer from those due to biliary cirrhosis. "But," you will say, "you said biliary cirrhosis can be easily produced in animals." That is true. It may be produced by ligaturing the common bile duct in animals; but undoubtedly it is very rare in man, although obstruction of the duct is so common. Clearly in man some other factor is wanting to produce it; there must

be obstruction to the duct together with something else, and what that something else is we do not at present know.

I hope to have shown you that the disease which we recognize clinically by its symptoms, and *post mortem* by the well-known condition of the liver as cirrhosis of the liver, and which we clinically call cirrhosis of the liver, really has only one cause, and that is alcoholic drinks. And I use the phrase alcoholic drinks rather than alcohol, because I hope to have suggested to you that alcohol by itself does not cause it, but is a very important contributory factor, and that there is some other contributory factor at present unknown.

Hypertrophic Cirrhosis.

The next misconception, if I may so call it, which I will take is one which is very frequently met with in textbooks. It is the attempt to draw a sharp distinction between atrophic cirrhosis of the liver and hypertrophic cirrhosis. In the first place, we must, in discussing this matter, put aside Hanot's hypertrophic cirrhosis of the liver. That has nothing to do with the subject under discussion now. It is a rare disease, in which the liver becomes markedly enlarged; the spleen is enlarged; it occurs almost exclusively in children, mostly in males, is very chronic, and the patient becomes deeply jaundiced. It is a rare and quite a separate disease, and it is better to leave it quite out of our discussion.

Leaving that aside, is there, as many people imply, any valid distinction in ordinary cirrhosis of the liver between hypertrophic and atrophic cirrhosis? Can we divide the cases into atrophic and hypertrophic? My argument will be that there is no distinction. I hope to be able to show that, as you would expect, the first stage is for the liver to become large; if an irritant acts upon it, the vessels dilate, the fibrous tissue hypertrophies, and more fat is laid down. Later on the fibrous tissue contracts and the organ becomes small. The following are the alleged points of difference between the hypertrophic and the atrophic forms. It is said that the hypertrophic form is smooth and not hobnailed, and that the atrophic form is hobnailed. But that is just what you would expect. First the growth of fibrous tissue enlarges the organ; then it contracts here and there and makes the organ hobnailed. That is only a restatement of the case that the disease is progressive. Then it is said that hypertrophic cirrhosis is perilobular and intralobular, whereas the atrophic is multilobular and extralobular. But this is only a question of degree: it is not a fundamental difference.

Again, it is urged that the hypertrophic liver is large, and the atrophic liver is small; that is obvious from the mere names. But let us look at some figures on this matter. A friend of mine, Dr. J. A. Price, found that the weight of the liver varied in 142 cases of cirrhosis of the liver, all so-called alcoholic cases, between 30 and 200 oz. In 29 it was under 50 oz., in 33 between 50 and 60 oz., and in 80 it was over 60 oz. If you are going to group the cases of the disease into atrophic and hypertrophic, where are you going to put the 33 between 50 and 60 oz. which is the normal weight of the liver? Surely the interpretation is that in 80 it was, at the necropsy, over 60 oz. and therefore was in the big stage. Some patients lived long enough for the fibrous tissue to contract, and so you get 33 between 50 and 60 oz. And if the patient survived the contracting stage in which the liver was the normal weight, it contracted below the normal weight, and so you get the 29 in which it was under 50 oz. These figures strongly suggest that there is no difference between the hypertrophic and the atrophic forms of cirrhosis of the liver. What they suggest is that the case is first hypertrophic and then atrophic, and so you have the largest number with heavy livers, for as some die fewer reach the stage of normal weight, and fewer still survive till the atrophic stage. In Guy's Hospital there was a case sufficiently long under my colleague, Dr. Frederick Taylor, for it to be observed that the liver contracted gradually, and the patient died of an ordinarily so-called atrophic cirrhosis of the liver. Then it is urged that ascites is slight or not present in hypertrophic cirrhosis, and there is much ascites in the atrophic form. This again is what would be expected, for ascites is usually a late symptom of the disease.

Again it is said that the jaundice is deep if the liver is big, and that there is little jaundice if it is small. I went through a number of cases in the hospital, and found that that distinction does not hold. I found that jaundice is common when the liver is small, and it is as common when the liver is big. And I was not able to find out that the statement that the spleen is large when the liver is hypertrophic, and small

when the liver is atrophic, held good. Again, I am sure your own experience will bear me out when I say that the assertion that an alcoholic history is given more often in the hypertrophic than in the atrophic form does not hold. Nor does the distinction that you get albumin in the urine less often in the hypertrophic form than in the atrophic. Then with regard to the distinction that the hypertrophic kind is found more in the young, and the atrophic more in old people: again that is what would be expected, for the hypertrophic is the early, and the atrophic is the late stage. Naturally people who die in the late stage on the average are older than those who die in the early stage. Moreover, the distinction has been made that the hypertrophic lasts longer than the atrophic. Of course it does, because a large number of people with the hypertrophic form pass into the atrophic form, and therefore they live longer after the diagnosis is made than those in whom the liver is small when they are first seen. So this part of my contention has been that there is no proper distinction between hypertrophic and atrophic cirrhosis of the liver in the cirrhosis of the liver due to alcoholic drinks: it is simply that they are different stages of one and the same disease.

SYMPTOMS.

Ascites.

We find that from half to one-third of all the cases that are entered in the *post-mortem* records as examples of cirrhosis of the liver presented no symptoms of it during life. An apparently healthy man is killed by an accident, and when he is brought into the hospital he is found to have a liver which is markedly cirrhotic. You inquire into the history and you find he has been intemperate. So we learn that a man may go on for many years with a cirrhotic liver without having any symptoms of it at all.

Now let us inquire what are the symptoms which may more or less rapidly develop in a man who has got this cirrhotic liver. He gets dilated venules and acne rosacea on the face, morning vomiting, loss of appetite, and indigestion and constipation. I shall not detain you over those symptoms, because it is difficult to say that they may not be attributed to the alcohol causing gastro-enteritis. But let us come to some symptoms which are not quite so easy to understand, taking first the ascites. Many books lead us to infer that the ascites is due to pressure on the portal vein by the fibrous tissue. I propose to bring forward reasons to show that that is an incorrect opinion. First, ascites may develop very rapidly. In one instance sixteen days after the patient first noticed distension of the abdomen twelve pints of fluid were drawn off. In a second case twenty-one days after distension was noted $23\frac{1}{2}$ pints of fluid were drawn off. I am sure, from watching many cases, that the patient complains of some distension as a rule after he gets three or four pints in his abdomen. So, allowing that he does not complain until a little fluid has collected, we must remember that the fluid must have collected at the rate of about a pint a day, possibly more than that, because probably we do not get it all off. If the disease were due to fibrous tissue pressing on the portal vein why should the onset be so sudden? There is no reason. You would expect it to occur gradually. The very last two cases of cirrhosis I have seen have not been tapped, but, judging by the great rapidity of the distension, the fluid must have poured out at the rate of quite a pint a day. Professor Starling and I have ligatured the portal vein in animals and yet no ascites has followed. This, which has been done (with the same result) by others, produces a great increase of pressure in the portal vein, but there is no ascites. In other parts of the body if you ligature veins you do not find oedema. Again, the portal vein is commonly pressed upon in malignant disease of the liver by enlarged glands in the portal fissure, but ascites is a comparatively rare symptom in malignant disease of the liver. Again, I have not been able to confirm the statement that ascites is most marked in those cases in which the branches of the portal vein in the stomach are most easily seen *post mortem*. We know that in cases of cirrhosis of the liver many branches of the portal vein are obviously dilated, but those are not particularly the cases in which you find most ascites. Lastly, if the ascites were due to pressure on the portal vein, it is difficult to understand why fluid is poured out from the peritoneum and not from the mucous membrane of the bowel. For these reasons we cannot regard ascites as merely a local result in the peritoneal cavity of pressure on the portal vein by an increase of fibrous tissue in the liver. What I would suggest is, that because of the disease of the liver there is some toxin circulating in the blood,

that it leads to an effusion into the peritoneal cavity, and that the effusion into the peritoneal cavity is more free than into the other serous cavities of the body, because the slight increase of pressure in the portal vein caused by the disease of the liver, determines the effusion there.

Jaundice.

Jaundice is often thought to be due to the pressure of the fibrous tissue on the bile ducts in the liver; but that will not hold for a minute, because we see jaundice commonly quite early. In the last two cases I have seen the patients had huge livers. The fibrous tissue had not begun to contract, but the patients were deeply jaundiced. The jaundice therefore is not due to simple obstruction of the bile passages by fibrous tissue, and I suggest again that it is probably toxic; the liver is diseased, its internal secretions are upset, and thus a toxin is poured into the blood, and consequently the patient becomes jaundiced. We may adopt the fashionable belief that there is no such thing as a purely haematogenous jaundice, and then you will probably accept the haemo-hepatogenous view, that when the toxin gets into the blood it destroys the blood cells, and that for a time the bile can deal with this excess of pigment in the blood and excrete it, but that the same toxin produces a catarrh of the bile duct, which leads to thickening of the biliary secretion and hence the bile gets held back and jaundice results. But whatever view we adopt of the cause of jaundice, I do not think we can maintain that the jaundice of cirrhosis of the liver is due exclusively to pressure of the fibrous tissue on the duct.

Swelling of the Feet.

The next symptom to which I want very particularly to direct your attention and try to explain, because it is so often overlooked, is swelling of the feet. If you take a large number of cases of cirrhosis of the liver, especially the bad cases, you will be surprised in what a number of them the patient complains of swelling of the feet. I have known this swelling extend up to the thighs and affect the penis and scrotum. Books which mention it dismiss it airily by saying it is due to the pressure of a big liver or ascitic fluid on the vena cava. Let us examine this theory. Over and over again I have cross-questioned patients, and I could bring you individual cases to show that the swelling of the feet has begun before the patient has noticed the abdomen enlarged. Indeed, we have had necropsies on patients with swollen feet in cirrhosis of the liver and there has been no ascitic fluid in the abdomen. Pressure of the ascitic fluid, therefore, will not explain it. Again, it certainly is not due to pressure of the big liver on the vena cava, for I have seen it when a liver has been small. Then it is said that when the liver is small it is contracting on the inferior vena cava, and so we get oedema of the feet. The objection fatal to that view is that in those cases the flow in the superficial veins of the abdomen is found to be in the normal direction, showing that the vena cava is not pressed upon. We cannot, therefore, explain the swelling of the feet which happens in cirrhosis of the liver by the notion that it is due to pressure on the inferior vena cava. I suggest again that it, like ascites and jaundice, is really toxic; that the internal secretion of the liver is upset and as a consequence the patient gets these general symptoms.

Anaemia and Haemorrhages.

Of the remaining symptoms we have, for instance, the sallow anaemia, with a tendency to epistaxis and purpura. No amount of ingenuity can maintain that they are due to a local disease in the liver. They are probably toxic in some way. And let me, in passing, remind you of the great importance of the tendency to bleed in diseases of the liver. It is no uncommon thing to be asked to see a man covered with purpuric spots and bleeding from his nose, and for this trouble to be solely due to cirrhosis of the liver. Only a few weeks ago one of my clinical assistants, because I had been urging this, when he saw a man who applied for admission to the hospital with a haematoma in his knee, examined the patient for cirrhosis of the liver and found it. People with cirrhosis of their liver, like people with Bright's disease, are liable to bleed from any part of the body. Again, we could hardly maintain that the coma is not evidence of a general toxæmia. If a man with cirrhosis of the liver die from it, certainly he usually dies comatose. Again, you will remember that very often people with cirrhosis of the liver get effusion into the pleural cavities quite apart from any tubercle. This again shows that you have to regard cirrhosis of the liver as more or less of a general, and not a local disease. And even

the piles and the haematemesis, although no doubt largely due to the dilated veins, may possibly be rendered more likely by the general liability to haemorrhages.

Cirrhosis in Cattle.

The last thing that I would bring forward as trying to prove my point that we must not look upon cirrhosis of the liver too much in the light of a purely local condition, is that in the remarkable disease of cattle which Adami worked out in Newfoundland the liver looks like that seen in human cirrhosis, and he draws attention to the fact that the cattle which may appear to have nothing the matter with them, suddenly show severe symptoms, and are dead within a few days. Their cirrhosis of the liver must have been present for weeks, and then quite suddenly these cattle got fatal symptoms.

HEPATIC CIRRHOSIS NOT A LOCAL DISEASE.

What I have tried, however imperfectly, to put before you with regard to the disease under consideration is this—that we must regard the disease we call alcoholic cirrhosis, because it is so frequently associated with the consumption of alcohol, although some other factor is present as a cause, not as a local disease of the liver any more than granular disease of the kidney is a local disease of the kidney, for mere increase of fibrous tissue in the liver—as, for example, that which we see in the big bands of syphilis—will not produce the symptoms which we are accustomed to see associated with the disease we call cirrhosis of the liver any more than heart disease causing an increase of fibrous tissue in the kidney leads to the symptoms of Bright's disease. Both in the disease of the liver and the disease of the kidney this fibrous change may exist for a long time without symptoms, and in both we must suppose that the chief trouble is not the mere development of the fibrous tissue but perversion of function, probably a perversion of internal secretion, so that the body gets filled with a toxin, causing in the case of the granular kidney uraemia, oedema, pleuritic effusion, and the many other well-known symptoms of chronic granular kidney; and in alcoholic cirrhosis of the liver the symptoms just mentioned—ascites, jaundice, coma, swelling of the feet, and a tendency to haemorrhages. If cirrhosis of the liver be regarded in this light, it is brought into line with the fibrous atrophic pancreas, in which, associated with fibrous atrophy, there is such a perversion of internal secretion as to lead to diabetes.

PROGNOSIS.

I want to urge that, in giving a prognosis, you must pay great attention to the ascites. If, in an uncomplicated case of cirrhosis of the liver, the patient has ascites he is near his end. I took from our hospital records a number of cases. Ten were cases of cirrhosis of the liver, with ascites, in which the patients died before paracentesis was necessary; and on analysing the history I found that the average interval between the time when the ascites probably began and death was only two months. Twelve were cases in which paracentesis was performed, and in which the data were sufficient to enable us to estimate the time of the collection of fluid. It worked out in these twelve cases that the time from the patient first noticing his distension to death was as follows: 12 days, 23 days, 30 days, 70 days, 84 days, 130 days, 56 days, 91 days, 140 days, 42 days, 63 days, 25 days; giving an average of 63 days, or two months from the time at which the patient first noticed enlargement of the abdomen to death, although paracentesis was performed.

As to the length of survival after tapping I have notes of 14 cases which survived the first and only tapping: 2 days, 9 days, 12 days, 11 days, 4 days, 8 days, 30 days, 2 days, 7 days, 4 days, 6 days, "a few days," 14 days, and 5 days. They do not survive tapping long, and this clearly proves that tapping is of little good, for if the patients only live about nine days or so after the tapping you can hardly contend that it does them good. I believe the only benefit which will follow tapping of the abdomen in cirrhosis of the liver is when the distension from the fluid is so great that it hampers respiration and the heart's action; in such case the withdrawal of the fluid will do good; otherwise it appears to cause no benefit at all. In passing let me remind you that these cases I have just read confirm my previous statement that the fluid may collect at the extraordinary rate of about a pint a day.

If you have a case of supposed cirrhosis of the liver, and you tap and the patient gets better, but the fluid re-collects and again you tap, such a case appears to contradict the statement that when the patient is ill enough to require paracentesis it means he is near his end, but at the necropsy it is

nearly always found that the diagnosis has been wrong, and that the patient has not got uncomplicated cirrhosis of the liver, but has chronic peritonitis. It is a very common mistake to confuse cirrhosis of the liver with non-tuberculous, non-cancerous, chronic peritonitis. My point is that patients with cirrhosis of the liver, if they have ascites due to it, are near their death, and tapping will not benefit them, except mechanically. I have been for many years trying to urge this at the bedside, and within the last few months Dr. Campbell Thomson has published a number of cases from the Middlesex Hospital which quite confirm what I have been telling you. Let me read you what he says: "Cases support these conclusions"—that is, the conclusions I have given—"entirely; of 18 cases, 12 died before tapping was necessary, or soon after it had been performed, 6 survived more than one tapping, and in all other cases than cirrhosis of the liver was found for the ascites." So that if the ascites is due to uncomplicated cirrhosis of the liver, that is to say, if there is no other cause for the ascites, the presence of the ascites means that the patient is near his end. But that is not the only confirmatory evidence I can give. Let me quote what Dr. Fagge said many years ago: "Moreover, when by means of diuretics and purgatives, one is able to clear the peritoneal cavity of this fluid, the successful action of such remedies seems often to avail the patient very little, for he presently becomes stupid and unconscious, and dies, although his abdomen may be perfectly flat and empty. The question has even been raised whether the removal of the fluid by medicines may not be sometimes concerned in the production of the fatal issue." Again, Murchison says, "When once it appears it persists and gradually increases." He holds out no hope of amelioration. Again, Wickham Legge says that the end soon arrives after tapping. As long ago as 1863 Austin Flint said "cirrhosis of the liver being an incurable lesion, when the dropsy is dependent thereon recovery is hardly to be expected." Taking all his cases of ascites together, Austin Flint says of 46 cases 24 ended fatally under his observation, or are known to have died subsequently. Of the remaining 22, there was in 8 no improvement, and after analysing the remaining cases he concludes by saying that "out of the 46 cases analysed I can positively state that recovery has taken place in but a single instance." I hope I have brought forward sufficient evidence to show the extreme gravity of ascites in cirrhosis of the liver. The reason I have brought it under the heading of misconceptions is that I am constantly having cases quoted to me which are supposed to show that the view is wrong. But whenever, or almost whenever they come to be examined *post mortem* it is found that some other cause than cirrhosis of the liver is present.

PERIHEPATITIS.

There was a man under my care in the hospital, 29 years of age, who had had syphilis. During the eighteen months preceding his death he was tapped thirty-five times, and on the average nearly 23 pints of fluid was taken away, giving a total of 790 pints of fluid. Sometimes the fluid re-collected at the rate of 2 pints a day. *Post mortem* the liver had a white jacket—a thick white jacket which could be peeled off. Sometimes by the contraction of the jacket the edge of the liver is turned up, so that it gets folded on itself. And that condition, as in this case, you will almost always find associated with a general chronic peritonitis. It is the *Zuckergussleber* of the Germans, and this is a very good name, for the white jacket looks very like the icing of a cake. I took from our hospital records 40 cases of perihepatitis for analysis. There were 18 cases of perihepatitis of partial character, and 22 complete. Of the 18, in 6 it was due to tubercle or to cancer, and of the remaining 12, in only one was peritonitis present; 9 of the 12 had capsulitis of both liver and spleen; in 10 of the 12 there was heart or lung disease, causing backward pressure. So we learnt that partial perihepatitis is comparatively unimportant, and that it is not associated with a general peritonitis, but usually with backward pressure from the heart or lungs. A much more important condition is shown in the 22 cases of complete perihepatitis, 22 cases of thick white jacket over the liver; 17 of those had general chronic peritonitis, never due to tubercle or growth. In regard to 3 there was no mention of the peritoneum; in respect of 2 it was stated there was no peritonitis. Those figures, I think, prove my point that perihepatitis, with a big thick white capsule over the liver, forming a great shell over the organ, is nearly always a part of a general chronic peritonitis not due to tubercle or to growth. The peritoneum gets thick and opaque, and

the intestines are all matted together, so that you can take them out as a whole, and when you cut across the mass, the intestines are held open by the thickening of the peritoneum around them, the mesentery shrinks and draws the intestines back, and the omentum gets puckered up into a great band which lies transversely across the colon. In these cases usually there is universal thickening of the capsule of the spleen as well, and in many of them granular kidneys are present, so that it must sometimes be regarded as a complication of granular kidney; sometimes there is an alcoholic history, and occasionally the patients have had syphilis.

You will say, "What is the misconception in regard to that?" It is this, there is a tendency to lump together the partial perihepatitis and the general. The partial is unimportant; in it there are little patches here and there; it is not part and parcel of a general peritonitis. But the universal perihepatitis, with thick white capsule over the liver is a much more important condition, for it is part of a general chronic peritonitis. For want of time I have had to confine myself to the peritoneum, but sometimes a zucker-gussleber is associated not only with chronic peritonitis but with chronic pericarditis and chronic pleurisy. To this condition the term multiple progressive hyaloserositis has been applied by Nicholls.¹ He, I may add, quite supports my contention that general perihepatitis is usually merely part of a chronic peritonitis.

PRIMARY MALIGNANT DISEASE OF THE LIVER.

There is a wide divergence of opinion as to the frequency of primary malignant disease of the liver. Friedreichs gives the proportion of primary to secondary malignant disease of the liver as one to two. On the other hand, you come across people who doubt whether there is such a thing as primary malignant disease of the liver. I went through our necropsy records. Among 10,000 necropsies we had 10 cases of undoubted primary malignant disease of the liver. That is to say every part of the body was carefully searched, but no other part of the body was found to contain malignant disease at all, except that sometimes the portal glands were a little affected by secondary deposits; and sometimes the few glands there are by the diaphragm on the upper surface of the liver. To show the care with which the cases were examined, I may say that in one case in which the patient had moles on the skin every mole on the body was subjected to a microscopical examination, to make sure that it was not the primary seat of the disease, because the growth was melanotic. The figures worked out in this way. Taking 10,000 necropsies, we had 1 case of undoubted primary malignant disease of the liver to every 24 in which the malignant disease was secondary to growth elsewhere. If we take the cases in which there could be no real doubt that the malignant disease in the liver was primary, but in which a few deposits secondary to the malignant disease of the liver had occurred in other parts of the body, it works out as 1 case of primary malignant disease in the liver to 17 in which the disease of the liver is secondary to malignant disease elsewhere. These figures, therefore, strike the balance between believing that it rarely, if ever, occurs, and the very high estimate given by Friedreichs of 1 to 2. I suspect that one cause at least of the mistake is that it has been overlooked that primary disease can occur in the gall bladder and biliary passages, and that malignant disease of the liver may be secondary to primary disease in those parts. While we are on the subject, I might point out a few clinical symptoms of primary malignant disease of the liver. It usually occurs at the same age as other malignant disease. In 17 cases, 10 were men and 7 women. After the first symptom was observed in those in which the date was sufficient to draw deductions, 2 lived four months, 2 lived three months, 1 lived two and a-half months, 2 lived two months. Therefore it is a disease very rapidly fatal, much more so than secondary malignant disease of the liver. Pain and tenderness were usual, and unless the primary malignant disease was of that very rare variety, scirrhus of the liver which contracts the organ, the liver was always enlarged, and weighed usually between 120 and 130 oz.; once the enormous weight of 267 oz. was attained. Now this is important; out of 10 cases there was no jaundice in four, and in two jaundice did not appear until just before death; in three of those affected it was slight. That is a very striking contrast to secondary malignant disease of the liver, in which jaundice is such a prominent feature. Never in primary malignant disease of the liver did jaundice become deep olive green, probably because the patient did not live long enough; and the absence of the

jaundice could not be entirely attributed to the fact that there was not time for the glands in the transverse fissure to be enlarged secondarily to the disease in the liver, because sometimes they were enlarged and no jaundice was present. Ascites was present in about half the cases. The temperature was often raised. We know it is raised occasionally in cirrhosis of the liver and in cancer of the liver, but it appeared to be particularly raised when the disease was primary in the liver. So I suggest that in attempting to diagnose whether malignant disease of the liver is primary or secondary we should pay attention to the following points: Is the disease very rapid? You see no patient lived more than four months after it was recognized. If it is rapid that fact is an argument in favour of the disease in the liver being primary. In the next place, if there is no jaundice it is an argument in favour of the disease being primary in the liver, and so perhaps also is an unusually high temperature.

Since I wrote the above many of the points urged have been confirmed by a paper by Acland and Dudgeon.²

LOCAL PERITONITIS DUE TO GALL STONES.

The only other point to which I have time to direct attention is a condition which I do not think we sufficiently often bear in mind in thinking of liver disease, and that is one of the after-effects of gall stones. It is not by any means an uncommon thing at a necropsy to find a good deal of local peritonitis around the gall bladder, and that may well cause the stomach and duodenum to become adherent to the gall bladder, and then the patient will have all sorts of vague pains in the abdomen, sometimes very severe, sometimes making life a misery and causing her to be called a hypochondriac and a valetudinarian. If we go carefully into the history, and find there is a distinct history of gall stones it may help us to come to a diagnosis. Perhaps I can best illustrate this point by two cases. In one case which I saw there was dilatation of the stomach, and inasmuch as a mass could be felt, it was thought that the dilatation was due to growth. *Post mortem* I found that the patient had much matting around the gall bladder, in which there were some stones, and that the gall bladder was adherent to the duodenum, and that all this matting around the first part of the duodenum and the pylorus had led to constriction at the pylorus and the consequent dilatation of the stomach, and all the symptoms of which the patient complained. In the next case, an old woman complained of great pain in the abdomen, which was much worse after food. She complained that she had indigestion, and sometimes she rolled about in pain. She gave a history of gall stones. The diagnosis made was that there were adhesions about the gall bladder implicating the end of the stomach and the duodenum. The abdomen was opened and the diagnosis was found to be correct. The adhesions were freed and the patient's pain was relieved.

REFERENCES.

- ¹ *Studies from the Royal Victoria Hospital, Montreal*, vol. 1, No. 3. ² *Lancet*, November 15th, 1902.

The Erasmus Wilson Lectures

ON

ADENOMA AND ADENO-CARCINOMA OF THE RECTUM.

Delivered before the Royal College of Surgeons of England.

By SIR CHARLES B. BALL, M.Ch., F.R.C.S.I.,

HON. F.R.C.S. ENG.,

Regius Professor of Surgery in the University of Dublin.

LECTURE III.

SURGICAL TREATMENT OF CANCER OF THE RECTUM.

HAVING determined that a given case of cancer of the rectum is suitable for removal, that is, that it is freely movable or at any rate only slightly invades the perirectal tissues; it is necessary to consider in the first place what route is the best adapted to the individual case, and this will largely depend upon the situation of the disease: (1) the growth may involve the anal canal only; (2) it may be situated low down in the rectal ampulla; and (3) it may originate high up above the peritoneal reflection; it must, however, be remembered that the disease may be so extensive as to occupy situations 1 and 2 or 3 and 4, while in some rare cases the entire rectum including the anal canal may be implicated. The routes we