THE LUMLEIAN LECTURES ON SOME CIRRHOSSES OF THE LIVER.

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[ABSTRACT.]

LECTURE I.

CIRRHOSIS of the liver occurs in various forms, and in different degrees. It does not, as some would appear to conceive, arise suddenly fully developed—in the extreme form of the final stage of atrophic degeneration. It is my purpose to deal mainly with the three chief forms of cirrhosis, and those with which ascites in its most pronounced and characteristic form is especially associated. It is on these—distinguished as atrophic cirrhosis, hypertrophic cirrhosis, syphilitic cirrhosis—that the main interest of the subject centres.

Before proceeding further, however, it will be well to clear the ground by putting aside certain minor forms which lie outside the strict lines of the present inquiry. Amongst these are those conditions of fibrosis which are due to the obstruction of venous escape from the hepatic vein—as in mediastinal tumours, mediastinitis—or associated with general dropsy in mitral and tricuspid disease of the heart. The hepatic fibrosis which is set up by chronic venous congestion of this kind is as a rule only a slight cirrhosis, not usually sufficient in itself to interfere materially with nutrition, or to cause such serious portal obstruction as to give rise to abdominal dropsy. The peritoneal effusion is not great, and forms merely a part as it were of the general dropsy which follows the damming back of blood in the systemic venous system. Yet in some instances of this congestive cirrhosis we meet with an ascites as a distinct feature, which is out of all proportion to the effusion elsewhere—a true ascites due to added difficulty in the portal area, the systemic obstruction, insufficient alone to produce a general dropsy—reinforced by the hepatic obstruction causes peritoneal dropsy. I wish to draw attention to this, because it has, I think, an important bearing upon certain cases of hypertrophic cirrhosis of which I have to speak later.

1. In *atrophic cirrhosis* the fibrous invasion is described as spreading along the main branches of the interstitial tree, embracing groups of lobules and compressing them, and thus causing atrophy and destruction of cells. The new fibrous growth between the enclosed groups of lobules contracts, and, drawing in the surface at these points, causes hobnailing of the surface and shrinkage of the whole organ, so that it is much smaller than normal, decreased in weight, tough and hard. Amongst the clinical features which are laid down as characteristic of atrophic cirrhosis, as contrasted with other forms, are signs of portal obstruction, evidenced by the frequency of ascites and the occurrence of hæmorrhage from the stomach and bowels, the rarity of jaundice, the short course of the disease after it can be recognised, its inevitable fatality, and that it is almost invariably set up by chronic alcoholic irritation.

2. In *hypertrophic cirrhosis*, described by Hanot as "biliary cirrhosis," the liver condition is stated to be characterised by increase in size and weight; the fibrosis which invades the interstitial tree affects the smaller twigs rather than the main branches, embracing single lobules and even single cells, instead of groups of lobules; and is accompanied, moreover, by a curious development of biliary canaliculi at the periphery of the lobule, due either to the formation of new ductules there, or to the exposure of existing ones, by denudation through the destruction of hepatic cells. In this form the liver is greatly enlarged; it is hard, smooth of surface, or only slightly granular. The clinical aspect, as laid down by authority, is characterised by features which contrast sharply with those of the atrophic form. Ascites is described as entirely absent, or, if present, is so slight as to be comparatively unimportant; while jaundice, on the contrary, is said to be an almost invariable symptom, and often intense and per-

sistent. Intermittent attacks of pain and pyrexia, enlargement of the spleen, and a liability to the supervention of acute symptoms, such as severe delirium, hæmorrhages, coma, and a chronic course of some years, are other signs which distinguish hypertrophic cirrhosis. Its cause is involved in obscurity; a doubtful factor, in some instances supposed to be malarial, in some alcoholic, in some obstruction to ducts is credited with setting up inflammation of the biliary canaliculi, which is regarded as the starting point of the pathological change.

3. *Syphilitic cirrhosis* is described as characterised by the presence of broad bands of fibrous tissue, separating isolated masses of liver tissue, and causing destruction of hepatic cells. The organ is often much deformed by the gradual contraction of these several fibrous bands, giving it a lobulated appearance, its surface marked by rounded bosses, often scarred and furrowed, or only slightly uneven; gummata may also be present in various stages—a hard liver, usually enlarged, sometimes contracted, with uneven, bossed, or slightly irregular surface. The clinical features of this kind of cirrhosis closely resemble one or other of the preceding forms—in the early stage of enlargement, the hypertrophic; in the latter, either the hypertrophic or atrophic alcoholic form. In one case jaundice, no ascites; in another both; in a third neither; but, as a rule, when advanced, ascites and perihepatitis. There is another form of syphilitic cirrhosis described as chiefly limited to the hereditary syphilis of childhood, a diffuse fibrosis characterised microscopically by its immature, partly cellular character penetrating the lobules, and even between the cells. The liver is greatly enlarged, clinically jaundice, ascites, and emaciation are amongst the chief symptoms.

FACTORS IN THE CAUSATION.

Atrophic cirrhosis of the contracted, hobnailed liver would appear to be almost invariably of alcoholic origin. It is said that malaria sometimes plays a part in its production, but the evidence of this is by no means conclusive. I have met with no instance of the kind; yet it is not intrinsically improbable that a disease like malarial fever, which sets up such marked fibroids of the spleen, should also set up an analogous fibrosis of the liver. In those cases attributed to malarious influences, however, when the cirrhosis is of the contracted, hobnailed variety, it is probable that alcohol, an agent by no means inactive in malarial districts, is largely concerned in its production.

THE QUESTION OF AN EARLY STAGE OF ENLARGEMENT IN THE ATROPHIC FORM.

Another question which has been much debated is, Is the liver enlarged in the early stage of atrophic cirrhosis? Is it first hypertrophied, and subsequently diminished in size as fibrotic contraction proceeds? The older writers generally held that this was so occasionally, but that it was exceptional. Murchison¹ expressed the opinion that it was a matter for further investigation whether, if a patient with enlarged liver survived long enough, the enlargement would be followed by cirrhotic contraction. Instances have been recorded by Dr. Frederick Taylor² and others in which the greatly-enlarged liver of alcoholic cirrhosis has been observed to undergo contraction. I have watched a number of these cases of cirrhosis with enlargement for considerable periods—one for nearly 12 years, one for 8 years, one for 2 years. In the first there has been some slight decrease in size, but great enlargement is a characteristic feature still, and in no case have I observed the condition change from an enlarged to a small contracted liver.

I think, however, that there is a state of engorgement, and congestive swelling in the atrophic cases, before fibrotic contraction is fairly in process. We are all of us familiar with the enlarged liver of the drinker, who has not developed any symptoms of actual cirrhosis. This, however, is quite distinct from the large hard liver of hypertrophic cirrhosis, which does not merge into the atrophic form. Such contraction as that described in Dr. Taylor's case must at least be extremely rare. The hypertrophied liver does not represent the early stage of the contracted form. The microscopic characters alone negative this; in the one the fibrosis is multilobular almost invariably; in the other, monolobular, or a

combination of the two. Hanot, indeed, recognised that hypertrophic cirrhosis was not merely an early stage of the atrophic variety, even when of alcoholic origin.³

The error of regarding the hypertrophic and atrophic condition as different stages of the same thing has probably arisen from its being observed that in some cases the liver was large, in others small; and since permanent hypertrophy was supposed to be limited to Hanot's biliary form, the inference was too hastily drawn that in alcoholic cirrhosis the large and small were connected, and merely different stages of the same affection. They are distinct varieties, rather than different stages of alcoholic cirrhosis.

ASCITES IN ATROPHIC CIRRHOSIS.

In the next place, is the occurrence of ascites a special feature of atrophic cirrhosis and practically limited to the final stage of it, and is it as rare in the hypertrophic form as it is common in the atrophic?

There can be no doubt as to the frequency of ascites in atrophic cirrhosis. In 23 consecutive cases of fatal cirrhosis at St. Mary's in which the liver was small (under 60 ounces) ascites was present in 17, absent in 6 only. Nor can there be any question of its grave significance in the majority of cases. It indicates, generally, that the disease has progressed towards its end, that the collateral circulation established has become inadequate to the full relief of the obstructed portal circulation, and that the destruction of liver tissue, advancing usually *pari passu*, has reached the limit of viability. As a rule, but by no means invariably, the patient has only a few months to live; now and again however the months are extended to a year or two. The supervention of marked jaundice in atrophic cirrhosis is of more immediately fatal augury than the appearance of ascites. There are cases of atrophic cirrhosis in which ascites supervenes before the final stage when sufficient glandular structure still remains intact and functionally effective to carry on life, and the obstruction to the portal system is not yet so great as to be past compensation by collateral channels. In these rarer instances partial recovery may take place for a time.

JAUNDICE IN ATROPHIC CIRRHOSIS.

In the standard descriptions of the symptoms pertaining to atrophic cirrhosis a point is made of the fact that, although a slight icteric tint may be present, marked jaundice is conspicuously absent, while it is almost invariably present in the hypertrophic form. Here, again, we have been misled by the assumption that hypertrophic cirrhosis usually corresponds with Hanot's special biliary form. The matter is of no great moment, but no such hard-and-fast line as to the occurrence of jaundice can be drawn. Jaundice of a marked character is not constantly absent in the small liver of alcoholism; and on the other hand, it is by no means constantly present in the hypertrophic form. Murchison⁴ observed that although decided jaundice is rarely met with in atrophic cirrhosis in the early stage, it may occur in advanced stages, and is then an ominous symptom. Dr. Foxwell,⁵ as the result of his investigation of 67 cases, concludes that jaundice and cerebral symptoms are as common when the liver is small as when it is large. In the 53 fatal cases of cirrhosis of all kinds at St. Mary's during the last ten years I find 8 in which marked jaundice was observed. In 4 of these cases the liver was small, and in 4 enlarged. The distinction between the two forms with regard to the occurrence of jaundice should be that whilst it is met with pretty equally in both forms, it occurs at an earlier stage in the hypertrophic than in the atrophic condition, in which latter it is often indicative of the approach of a fatal termination.

THE CHARACTER OF THE FIBROSIS IN ATROPHIC CIRRHOSIS.

Another question of some interest in connection with atrophic cirrhosis is whether the fibrosis is always of the coarse multilobular kind, affecting the main branches of the interstitial tree, or whether the finer interlobular variety, which is undoubtedly most characteristic of the hypertrophic form, is found in the small contracted liver also. In the 53 cases adduced before, the liver was examined microscopically in 18. In one only of the distinctly contracted specimens was the fibrosis diffuse, in the rest coarse and multilobular, with, in a few instances, a small amount of finer fibrosis invading the lobules.]

ATROPHIC CIRRHOSIS AND BRIGHT'S DISEASE.

A certain amount of interstitial change is found in the kidneys in a large proportion of cases (about one-half) of alcoholic cirrhosis. In most it is not extreme; and this fibrosis of the kidney is to be regarded as a result of alcoholism, just as is the fibrosis of the liver, the kidney being affected usually in minor degree. In some cases the interstitial nephritis is advanced, and, associated with the Bright's disease, a special form of cirrhotic atrophy is occasionally found, which has been more particularly described by Dr. Fagge and Dr. Hale White. In this the liver is greatly contracted, its substance atrophied, but the connective tissue is not always greatly increased. The capsule is much thickened, and there is also extensive perihepatitis associated with adhesive peritonitis, and ascites, which is attributed chiefly to the perihepatitis and chronic peritonitis.

OCCURRENCE OF PERIHEPATITIS AND PERITONITIS IN CIRRHOSIS.

In the 53 cases mentioned there are 15 in which extensive perihepatitis is recorded, usually with extension of peritonitis to neighbouring structures, and adhesions, notably of the diaphragm, stomach, and transverse colon.

Of these 15, 4 were syphilitic—in one of these parenchymatous nephritis was present—in the remaining 3 the kidneys were normal. Of the 11 others, in 4 the kidneys were more or less granular, in 3 enlarged, and in 5 normal.

In 22 cases the perihepatitis was slight or not recorded.

In 4 cases there was acute peritonitis. Of these the kidneys were fibrosed in 3; in 1 (which was syphilitic) there was cloudy swelling.

In 5 cases of the 53, there was some chronic peritonitis other than perihepatitis. Of these in 1 the kidneys were granular, in 2 large and tough, and in 2 not affected.

In 6 of the last cases examined, in which the condition was specially noted, it is stated positively that there was no peritonitis. In 5 of these the kidneys were cirrhotic; in 1 congested only.

Since, therefore, in these cases of cirrhosis with extensive perihepatitis and adhesive peritonitis of neighbouring structures the kidneys are not constantly affected, and, on the other hand, when the kidneys are granular there may be no peritonitis, the presumption is that the cirrhosis, perihepatitis, and Bright's disease are probably due to the same cause—chronic alcoholic poisoning.

HYPERTROPHIC CIRRHOSIS.

The first question which arises with respect to cirrhosis with enlargement of the liver is whether the affection described by Hanot and Charcot as a biliary cirrhosis of uncertain origin is to be accepted as the representative form of hypertrophic cirrhosis, or whether it even exists as a separate entity or is merely a modified form of alcoholic cirrhosis. Hypertrophic cirrhosis is described as having a special morbid anatomy and a distinct clinical history and course, which give it a marked individuality of its own.

I think we have been unduly biassed by the classical description of Hanot, as we were formerly by Laennec's description of the atrophic form, and have had no eyes to see obvious facts. Just as deference to Laennec's authority led to the too ready assumption that an enlarged fibrosed liver must be an early stage of the atrophic form, so, when the existence of a distinct hypertrophic condition, always hypertrophic and of special character, was demonstrated, it was assumed that every cirrhosis with enlargement must conform to this special variety. Subsequent research, however, has proved that many of the features which were considered to be distinctive of the so-called biliary cirrhosis are not peculiar to that exact type, but occur also in cirrhoses which in other points do not conform to Hanot's description.

THE CHARACTER OF THE FIBROSIS IN HYPERTROPHIC CIRRHOSIS.

There is now no question that a fine fibrosis, monolobular or intralobular in arrangement, occurs in conjunction with hypertrophy of the liver in cirrhosis due to alcohol; and this diffuse fibrosis appears also to be met with in the cirrhosis attributed to malaria which is usually hypertrophic.⁶ It is also established, I think, that the appearance of hyperplasia of bile ducts in the highly nucleated tissue between the lobules is not met with in cirrhosis with the clinical characters of Hanot's form alone. Dr. Saundby⁷ has shown that this appearance of new canaliculi occurs in cases of ordinary cirrhosis; and, also, that it is not always present in any marked degree in cases presenting the clinical characters of biliary cirrhosis. Dr. Frederick Taylor has produced evidence of a like nature.⁸ In a case of alcoholic cirrhosis with enlarged

liver recently under my care this appearance was found in conjunction with a coarse fibrosis, and this same formation of so-called biliary canaliculi is also seen in the simple fibrosis of cardiac disease. In another instance a fine fibrosis was observed in a case of alcoholic cirrhosis without jaundice in which the liver was small (53 ounces). The interstitial fibrosis most commonly met with in cirrhosis in which the liver is enlarged is a combination of coarse multilobular and fine monolobular development; the microscopic distinction, therefore, does not hold.

JAUNDICE IN HYPERTROPHIC CIRRHOSIS.

In the next place, the presence of jaundice is by no means a constant, nor, according to my experience, a very common symptom of hypertrophic cirrhosis.

In 17 consecutive fatal cases of cirrhosis with enlarged liver, varying in weight from 60 to 134 ounces, I find jaundice noted as a marked symptom in 4 only; in 6 it is described as slight, and in the remaining 8 none is recorded.

Again, out of 27 more cases of cirrhosis with enlarged liver, which have either recovered or been relieved, or in which, if fatal, no *post-mortem* examination was made, jaundice was absent in 19, and present only in 8.

In 10 cases of enlarged fibrous livers from different causes recorded by Murchison, jaundice is mentioned as a symptom in 4 only, and, as noted before, Dr. Foxwell's⁹ investigation of 67 cases of cirrhosis yielded the conclusion that jaundice was as common when the liver was contracted as when it was enlarged.

It is clear, therefore, that jaundice is by no means a constant or characteristic symptom of hypertrophic cirrhosis generally; and, on the other hand, as we have seen before, jaundice in marked degree is by no means rare in the atrophic form.

ASCITES IN HYPERTROPHIC CIRRHOSIS.

The absence of ascites in any important degree is described as being as distinctive a feature of the typical form of hypertrophic biliary cirrhosis as the presence of jaundice. This certainly does not hold good with regard to hypertrophic cirrhosis which has come under my observation.

In the 25 fatal cases of hypertrophic cirrhosis in which the liver weight varied between 60 and 134 ounces, ascites was present in 16. In 6, which recovered more or less perfectly, ascites was a prominent symptom in all but one.

In the 10 cases of hypertrophic cirrhosis given by Murchison, ascites was present in 7. Bristowe records one of alcoholic cirrhosis with great enlargement, in which the patient was tapped fourteen times for ascites.

In the 67 cases of alcoholic cirrhosis recorded by Dr. Foxwell, he found that ascites was as common when the liver was large as when it was small.

The absence of ascites, therefore, is not a constant and typical negative symptom of cirrhosis when the liver is enlarged, any more than the presence of jaundice is a reliable sign of distinctive symptom of the hypertrophic form. It is probable that this impression of the frequency of the occurrence of ascites in contracted liver, and its absence in the enlarged liver, has arisen partly from the fact that atrophic cirrhosis escapes notice until ascites arises to draw attention to it. A liver smaller than natural is not obtrusive. When the liver is enlarged, however, it challenges recognition, and is often discovered before ascites supervenes.

Enlargement of the spleen, again, is not an especial feature of hypertrophic cirrhosis. In the 53 cases I have instanced it was enlarged in the great majority of alcoholic cirrhoses, large or small, but the greatest size was reached in the syphilitic cases, in one of which it attained to 42½ ounces, or six times the normal weight.

PAIN, PYREXIA, DELIRIUM, COMA, THE TYPHOID STATE.

Another feature which is said to mark the course of biliary cirrhosis is the occurrence of attacks of pain and pyrexia. These symptoms are, however, met with in atrophic cirrhosis, in alcoholic hypertrophic cirrhosis without the characteristics of the biliary form, and notably in syphilitic cirrhosis.

Pain, tenderness, and pyrexia are, I imagine, merely an indication of the supervention of perihepatitis. Grave symptoms, such as delirium, the typhoid state, and coma, are not confined to hypertrophic cirrhosis of the so-called biliary type. They occur in hypertrophic cirrhosis of alcoholic origin, and in atrophic cirrhosis also. These symptoms cannot therefore be regarded as distinctive of special forms of hypertrophic cirrhosis.

CHRONIC COURSE OF HYPERTROPHIC CIRRHOSIS.

The course of the type form of hypertrophic cirrhosis is stated to be a chronic one of two, four, or six years, or more.

It must be allowed that the cases which run a protracted course are those in which the liver is enlarged; they are as a rule more benign. This is, however, not invariable; and Dr. Dreschfeld¹⁰ has recently published a case of alcoholic hypertrophic cirrhosis which, while agreeing with Hanot's affection in many respects, differed in the presence of ascites and in the short course of the disease. Chronicity, then, cannot be regarded as a constant character of hypertrophic cirrhosis.

It will be seen, therefore, that a cirrhosis in which the liver is greatly enlarged may fail to correspond, in one case or other in nearly every particular with the classical type, for which we all look, and so rarely find. Further, from the time of Murchison downwards, a number of cases of cirrhosis have been recorded by Saundby, Bristowe, Dreschfeld, Foxwell, and others in which although the liver was greatly enlarged, the symptoms have been those usually ascribed to the contracted liver, rather than those of so-called biliary cirrhosis. In the majority of instances the cause has been traced to alcoholic intoxication. A number of like cases have come under my own observation. The hypertrophic cirrhosis I have met with corresponds with the cirrhosis of Hanot chiefly in two respects: the enlargement of the liver is smooth and hard and the fibrosis is usually diffuse and monolobular and sometimes partly multilobular. In one instance, however, in which the liver weighed 152 ounces the cirrhosis was coarse and multilobular, with much fatty deposit.

Is it therefore to be inferred that no such special form of hypertrophic cirrhosis as that described by Hanot and recognised by Charcot actually exists? I think we are not warranted in assuming this absolutely. In addition to the original cases described so graphically by the French writers, other isolated instances have been observed now and again in this country and abroad, which agree closely with the so-called biliary type. For my part I have not, in spite of a close scrutiny for sixteen years, met with a case which has exhibited in full completeness the typical presentment of the French authorities. Although in view of the detailed description of such cases occurring in the experience of others it must be allowed that, clinically at all events, cirrhosis of that precise character does arise from time to time, it is certain that it is rare, at least in this country. It is, moreover, still doubtful whether these rare examples are entirely distinct from hypertrophic cirrhosis of alcoholic origin, or merely an exceptional variety of it. At all events, the representative fibrosis of the liver with enlargement is, in the vast majority of cases, a hypertrophic cirrhosis set up by alcohol equally with the atrophic form; in other instances by syphilis, and not the rare condition described by the French pathologists.

SYPHILITIC CIRRHOSIS.

The points at issue with regard to syphilitic cirrhosis are comparatively few. The ordinary form which results from acquired syphilis, in the tertiary stage, has usually well-marked characteristics when the liver is examined after death; although its identification during life is often extremely difficult, and mistakes in diagnosis are frequent with regard to it. In a typical instance the gummata in various stages, the massive wedges penetrating the liver substance at various points, the scarring and puckering, and lobulation, and the extensive perihepatitis are striking and unmistakable morbid appearances.

Microscopically may be seen the gummatous cellular growth developing into fibrous tissue or undergoing caseation. Endarteritis obliterans, a condition well known as the result of tertiary syphilis elsewhere, but, as far as I am aware, not observed directly in connection with hepatic syphilis, was discovered in 1 case by Mr. Plimmer, our able Pathologist at St. Mary's. In a paper by Hanot and Chauffard¹¹ this condition of arteritis is described as met with in hypertrophic cirrhosis with pigmentation and diabetes, to which latter the cirrhosis was regarded as secondary. In one case scarring is mentioned, which is very suggestive of syphilitic affection, but in the second case it is said that there was no history of syphilis, and the surface of the liver was flat and finely granular.

One point which is not sufficiently recognised and which I would emphasise is that the morbid changes in syphilitic cirrhosis may exist in every degree. Occasionally they are so slight as scarcely to alter the normal appearance of the organ;

a few small scars, some thickening of the hepatic capsule, or slight perihepatitis may be the only morbid features perceptible externally on *post-mortem* examination. This fact is of great importance in its bearing upon diagnosis. There may be no suspicious history, nothing whatever distinctive in the physical signs made out during life. The condition of the liver, as ascertained by examination of the abdomen may correspond exactly with that of an ordinary alcoholic cirrhosis.

Another feature of syphilitic cirrhosis is the degree of perihepatitis and local peritonitis in the region of the liver. This condition of general perihepatitis, with extension to neighbouring structures, is far more marked in connection with syphilitic disease than in any other condition.

The great development of perihepatitis and local peritonitis in many cases of syphilitic cirrhosis has been imperfectly recognised. Perihepatitis has been usually regarded as more especially associated with other forms. Murchison, however, observed that it was met with in marked degree in syphilitic cirrhosis, and in connection with long-standing disease of the heart. He described it as accompanied by fibrous bands extending from the capsule into the liver structure and causing atrophy.

There may be, as I have observed, no history of syphilis in these cases. In 10 cases of which I have notes—8 of which were fatal and the gummata found *post-mortem*—in 2 only was there any definite history of syphilis; in 2 others syphilitic scars were found during life; but distinctive physical signs in the shape of irregularities or bossing of the surface were found in 3 only. The condition usually disclosed by physical examination of the abdomen was a large liver, smooth and slightly irregular and hard; or sometimes simply that the liver was small and contracted.

The syphilitic liver, then, is liable to be mistaken—and, as a matter of fact is, I believe, frequently mistaken—for the alcoholic liver, an error which, in view of the remedial power of antisymphilitic remedies, may be disastrous.

Signs of perihepatitis, pain, and tenderness over the liver and pyrexia, when the degree is marked and persistent, afford a presumption in favour of the syphilitic nature of the affection. The temperature affords valuable indications of the existence of perihepatitis. Perihepatitis does occur in pure alcoholic cases, and in connection with Bright's disease; but, according to my observation, it is not so constant or so extreme as in syphilitic cirrhosis.

Another undetermined question is as to the specific cirrhosis which arises in young children as a result of congenital syphilis. I have not met with any instance of gummatous development such as is found in the acquired disease, although the condition has been recorded by Ziegler and others. The smooth liver greatly enlarged offers no points of distinction from a liver enlarged from other causes, and the diagnosis must depend upon the history or the existence of other signs of the disease. *Post-mortem* the microscope reveals a very distinct form of cellular infiltration and early fibrosis, which spreads through the lobules between the cells in a diffuse and characteristic manner. There may be no thickening of the capsule or external change perceptible.

In 8 cases of cirrhosis in children between 5 and 9 years of age, of which I have careful records, there was a reliable history of congenital syphilis in 2 only. In 2 there was one of alcoholism, and in the remaining 4 no cause could be made out. In connection with this subject of cirrhosis in children there are many interesting points—such as the effect of congenital obliteration or stenosis of the bile ducts, as recorded by Dr. John Thomson¹²; the question of hereditary tendency, as noted by Dr. Howard¹³ and Dr. Finlayson¹⁴; and the connection of this with nervous lesions, as noted by some observers. I think it is probable that congenital syphilis will furnish the key to the relation of these several conditions. With these matters, however, it is beside my present purpose to deal.

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ANTHRAX OF THE THIGH TREATED BY OPERATION: RECOVERY.

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THE occurrence of anthrax in the human subject in Ireland has been sufficiently rare to warrant the publication of the following brief notes, which are an abstract of a communication made to the North of Ireland Branch of the Association at a recent meeting. The patient and the excised morbid tissues were exhibited to the members. Some additional interest arises from the facts as to mode of infection and the bacteriological proofs being unusually clear in this instance.

The patient was a healthy labourer, aged 23, employed in a chemical manure manufactory. On December 22nd, 1899, while wheeling a barrow laden with bone manure he was accidentally pricked on the inner side of the thigh, in the lower third, by a splinter of wood on the barrow handle. Feeling something scratch him in this situation, he extracted a tiny chip of wood from his trousers and continued his work, taking no further notice of the occurrence as the spot was not painful. In three days a pimple formed and became a sore, which grew daily larger and more painful. Meantime his trousers, soiled by the bone manure, continued to rub against the affected part. On the tenth day, January 1st, he was obliged to abandon work, and sought advice at hospital. Free excision was recommended and carried out on the following day.

At this time, January 2nd, the characteristic signs were manifest—the central depressed black slough surrounded by a complete ring of vesicles. Inflammatory redness and swelling extended around for an area of 7 inches by 5, and very marked enlargement and tenderness of the femoral glands had recently supervened. It was this glandular implication which determined the operative treatment, as it appeared to point to a dissemination of the germs. The temperature and pulse up to this period remained undisturbed. The operation was begun by an incision in the groin, and the saphenous vein was ligatured first, lest the subsequent manipulations of the infected tissues should detach infective thrombi. The cluster of lymph glands was removed in one mass together with the surrounding fat. The wound was sutured and drained. Next a large oval patch surrounding the anthrax, and involving the tissues down to the vastus internus, was excised. The angles only were sutured in this wound and the centre left to granulate. The subsequent progress during the ensuing three weeks was satisfactory; systemic infection did not occur, and the patient made a good recovery.

Bacteriological investigations were made by Dr. Lorrain Smith; the chief interest, after demonstrating the nature of the disease, attaching to his report as to the presence or absence of anthrax bacilli in the swollen lymphatic glands in the groin. Subjoined is Dr. Lorrain Smith's report:

1. Fluid removed from blebs contains large numbers of bacillus anthracis.
2. Broth cultivation of this fluid for five hours contains a pure growth of these bacilli.
3. The fluid expressed from the central slough contains a few bacilli and multitudes of staphylococci.
4. The film made from the centre of a swollen lymph gland shows no anthrax bacilli. Two experiments were made to ensure accuracy.
5. Needle-prick inoculation of a mouse resulted in death of the animal in thirty-six hours, and microscopic examination of splenic pulp therefrom showed abundant anthrax bacilli.

From this report it appears that the inflammation of the lymphatic glands was not due to the arrival of anthrax bacilli in the glands, but was probably of a sapræmic nature.

FEMALE MEDICAL STUDENTS IN PARIS.—From the report of M. Petit de Julleville to the Minister of Public Instruction at a meeting of the Council of the University of Paris, it appears that the Faculty of Medicine has 129 female students. Of these 29 are French, while 100 belong to other nations; 91 being Russian, 5 Roumanian, 2 German, 1 Swiss, and 1 English. Twenty women are studying in the School of Pharmacy, of whom with one exception all are French. In the Faculty of Science there are 35 female students—21 French and 14 foreign.