REMARKS

ON

THE VARIETIES OF HEPATIC CIRRHOSIS.

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MR. PRESIDENT AND GENTLEMEN,—In opening the discussion upon this important subject, I desire, in the first place, to express my sense of the honour conferred upon me, and of my own slender qualifications for dealing in an adequate and satisfactory manner with the many aspects presented by it, or for placing before you more than an outline of the facts concerned in it. I must beg your kind indulgence for many shortcomings, and hope you will gather from those who will follow me in this discussion information on those points where my own knowledge proves wanting.

Pathological research has established the existence of no fewer than ten varieties of hepatic cirrhosis: 1, Alcoholic; 2, cardiac or cyanotic; 3, biliary; 4, diffuse syphilitic; 5, gummatous syphilitic; 6, tuberculous; 7, malarial; 8, scarlatinal; 9, rachitic; 10, diabetic. I have done my best to place before you specimens of livers showing these various conditions, and under the microscopes are sections illustrating their histological changes.

Alcoholic Cirrhosis.—This is the common type of hobnail or gin drinker's liver, to which the name cirrhosis was first applied by Laennec. It is caused by the abuse of alcoholic liquors, and occurs generally in adult males. These cases usually present themselves complaining of abdominal dropsy or hæmatemesis, and it is noteworthy that those who have abdominal dropsy usually escape hæmatemesis, and those who have hæmatemesis are generally free from dropsy of the abdomen. This division is, I believe, of considerable importance, as I shall try to show you. In both cases there are various dyspeptic symptoms due to catarrh of the stomach; there is often great flatulence; the skin is of a peculiar earthy tint, or even subicteric, rarely jaundiced. The urine is scanty, loaded with lithates, and high coloured from the presence of an abnormal urinary pigment, the so-called febrile urobilin; albumen in variable quantity may be present. The spleen is usually enlarged, while the liver dulness is notably diminished, especially in the epigastrium; but in the right vertical nipple line the dulness is reduced from four inches and a half to one and a half or two inches. Ascites is present in about two-thirds of the cases, and where it is absent the disease is very likely to be overlooked until a severe, and perhaps fatal, hæmatemesis suggests an examination of the hepatic region. Such cases are overlooked because they often present no marked disturbance of health. Some years ago, I examined the body of a man employed as a foreman engineer at a colliery, who had died suddenly from hæmatemesis. Although a hard drinker, he was a steady workman, and the books showed that he had not lost a day's work for two years. His liver was a most typical example of hobnail cirrhosis, and he died from the rupture of a varicose œsophageal vein.

The explanation of the absence of ascites is, there can be little doubt, the enlargement of other venous channels by which the blood from the portal system finds its way along the coats of the stomach and œsophagus to the superior vena cava, and I show you specimens of these enormously dilated veins. This adaptation, while preserving the patient from the misery of abdominal dropsy, is attended by the risk of fatal hæmorrhage, should the coats of a vein give way.

You now see the importance of the rough division I started by making of these cases into (a) those with ascites, and (b) those with hæmatemesis, and you can understand that to a considerable extent these two symptoms mutually exclude each other.

This is not the place to enter into detailed pathological descriptions. I will therefore only say that the liver in alcoholic cirrhosis is usually small, pale olive in colour, granular on the surface, with a thickened capsule, tough on section, its cut surface showing yellow bile-stained areas of hepatic tissue surrounded by

greyish translucent bands of newly formed fibrous material. This new formation grows in the portal canals surrounding groups of acini and invading them from their periphery. It is probable that the hepatic cells themselves take part in this process, and are actually transformed into fibrous tissue (Hamilton). Newly formed biliary canaliculi are also to be seen in the neighbourhood of the acini, running up to and continuous with rows of hepatic cells.

I am able to show you a patient who illustrates the chief points to which I wish to direct attention respecting the prognosis and treatment of this disease.

It is well known that it is an essentially chronic malady, but I do not think the general opinion of the profession is favourable to a prognosis of prolonged life when ascites is fully developed. Yet this man six years ago was in that condition. He was cured of his ascites by early and repeated tapping, and his condition now is relatively infinitely more satisfactory than could have been expected from any other plan of treatment. I consider that by tapping this man was allowed the opportunity to establish his collateral venous circulation, and is in the condition of those cirrhotic patients, who, I have shown, go on to the termination of their cases without marked symptoms of ill-health. I do not say this can always be done, because many of these patients are hopeless drunkards, and, in spite of all warning, continue to drink and relapse. Not long ago, in consultation with Mr. J. W. Moore, of Moseley Road, we effected a remarkable improvement in a young man, who was able to return to his ordinary life for about a year, but unfortunately he soon began to drink heavily and died. But the point I wish to emphasise, because it is a ray of hope in an otherwise hopeless condition, is that by early and repeated tapping a patient who will abstain from excessive use of alcohol may have his life prolonged many years. I should not like to conclude this part of my subject without reminding you that it was that great clinician, Dr. Charles Murchison, who first advocated this treatment, and to his writings I owe the practice I have followed with, I believe, great benefit to many patients.

Cardiac or Cyanotic Cirrhosis.—This condition need detain us a very short time. Where there is chronic congestion of the right side of the heart from permanent organic disease of the mitral valve or lungs (chronic bronchitis and emphysema) a cirrhotic process commences in the liver around the radicles of the hepatic vein. The liver is enlarged and tender, the peritoneum contains fluid, and there is often slight jaundice.

Biliary Cirrhosis.—This condition is caused by some permanent or protracted obstruction of the common bile duct. The patient's history always discloses that jaundice was the initial symptom, and it persists. The liver is generally enlarged, this form being the best known of what is called hypertrophic cirrhosis, but may be atrophied (Charcot); its surface is smooth, its tissue deeply bile stained, and traversed by fibrous trabeculae, which embrace single acini, and contain numerous capillary bile ducts of new formation. The course of these cases is very chronic, but the prognosis is very unfavourable, and they are liable to terminate with symptoms of acute yellow atrophy, or with so-called cholemic symptoms, muttering delirium, dry tongue, stupor.

Ascites is generally absent. The treatment is palliative, unless there is any hope that the obstruction can be removed by surgical means. The diet should consist of light animal and farinaceous food, with the use of some alkaline mineral water, such as Vichy water, taken with lemon juice or claret. The bowels should be regulated by the use of Carlsbad salt, or some analogous saline aperient.

Syphilitic Cirrhosis.—Diffuse syphilitic cirrhosis is met with in children who are the subjects of inherited syphilitic disease, and who usually present other signs of this cachexia, depressed nasal bones, atrophied incisor teeth, earthy complexion. The spleen is always enlarged as well. The enlargement of the liver is the only local sign of the affection. These livers are smooth, with opaque capsules, tough, and on section are seen to have undergone complete fibroid transformation in some parts. Under the microscope the lesion is seen to be diffuse, not restricted to any anatomical region as in the forms previously described; it invades the entire tissue, destroying the acini cell by cell. The treatment must consist of good food and fresh air, combined with one-twelfth of a grain of calomel two or three times daily.

Gummatous Cirrhosis.—This is a very bad name, and, indeed, these cases scarcely deserve to be called cirrhosis. In them the liver has been the seat of numerous gummata which have left fibrous tracts through the liver, puckering its capsule, deforming

its shape, and sometimes compressing the main bile ducts or veins. In most cases it is only recognised at the *post-mortem* table; but in the conditions last alluded to jaundice or ascites may have called attention to the liver, and its irregular shape may have been discovered by palpation.

The prognosis is only unfavourable in severe cases, and the only treatment can be that for constitutional syphilis—iodide of potassium in full doses, combined, in certain circumstances, with mercuric or mercurous salts.

Tuberculous Cirrhosis.—This condition is so little recognised that I fully expect to hear its existence questioned. It is, however, generally admitted by German pathologists. I have seen two cases. It occurs in phthisical or tuberculous patients without giving rise to any symptoms. The liver is enlarged, smooth, and of normal consistence, but on section a trabecular network is seen surrounding the lobules, and under the microscope this is seen to be of great extent, and accompanied by a most remarkable development of biliary canaliculi. It is certainly a rare condition, and it is doubtful if it could be diagnosed during life from other forms of hepatic enlargement. It is probable that the cirrhotic livers which have been described in connection with tuberculous peritonitis belong to this group.

Malarial Cirrhosis.—In the bodies of the persons who have been exposed to prolonged malarial infections the liver is often found enlarged, indurated, and pigmented. The symptoms are ill-defined. Jaundice is often present, and there is frequently a sense of weight in the hypochondrium, which leads to the discovery of the enlarged liver. It is no doubt the consequence of repeated attacks of malarial congestion. The new growth starts from the portal canals as in alcoholic cirrhosis, but it tends to invade, rather than circumscribe, the lobules. The newly formed connective tissue is loaded with pigment, caused by the excessive destruction of hæmoglobin.

The treatment consists in the administration of a course of arsenic combined with nitro-hydrochloric acid and a bitter infusion, the use of mercurial purgatives and saline and alkaline mineral waters, such as those of Carlsbad and Vichy. A course of treatment at Carlsbad is eminently suited for these cases.

Scarlatinal Cirrhosis.—This condition is known only to pathologists, who have found it in the bodies of persons dying from the sequelæ of scarlatina, but it is possible that it explains some of the not very uncommon cases of cirrhosis of the liver met with at the *post-mortem* table in young children. The liver presents no abnormal naked-eye appearances, but the microscope shows commencing new formation of fibrous tissue in the portal canals, fissures, and spaces.

Rachitic Cirrhosis.—This is a little known but not very uncommon form; it occurs in rickety children, and I believe an important feature in its etiology is chronic gastro-intestinal catarrh occurring in early childhood. The symptoms are those of intermittent gastro-intestinal catarrh. The child's appearance is cachectic, and the disease has a strong superficial resemblance to one of congenital syphilis, but there is no history of syphilis, and there are no specific signs.

The liver is enlarged, smooth, and of normal colour, or pale from fatty infiltration. On section it is tough, and traversed by trabeculae of fibrous tissue which surround single acini, and are accompanied by a considerable new formation of biliary canaliculi. The spleen is also enlarged.

The treatment should consist of calomel in gr. $\frac{1}{2}$ to $\frac{1}{3}$ three times a day, with syrup of the iodide of iron and cod-liver oil, fresh air, and careful diet, excluding all uncooked fruit and vegetables, oatmeal, brown bread, etc., using the lighter kinds of meat, fish, and fowl, eggs, milk, butter, cream, with very well-cooked and digestible vegetables, light custard puddings, etc.

Diabetic Cirrhosis.—This form of cirrhosis is not well known in

TABLE I.—Anatomical Changes in the Varieties of Hepatic Cirrhosis.

Varieties.	Size.	Colour.	Surface.	Capsule.	Consistence.	Bile Passages.	Position of New Growth.	Newly-formed Biliary Canaliculi.
Alcoholic... ..	Usually small	Pale olive	Granular	Thick	Tough	Nearly empty	Round groups of acini	Present
Cardiac or cyanotic ...	Usually large	Dark	Smooth	"	"	"	In centres of acini	Absent
Biliary	"	Olive or pale	"	Slightly thick	"	Dilated and full of bile	Around single acini	Abundant
Diffuse syphilitic ...	Large	Brown	"	Opaque	"	Normal or empty	Diffused	A few only
Gummatous	Irregular	Pale	Irregular	Thick and opaque in places	Cartilaginous in places	Normal	Irregular	Absent
Tuberculous	Large	Pale or normal	Smooth	Normal	Normal	"	Around single acini	Very abundant
Malarial	"	Dark pigmented.	"	"	Tough	"	Around single acini	Absent
Scarlatinal	Normal	Normal	"	"	Normal	"	Around single acini	"
Rachitic	Large	Pale	"	"	Tough	"	Around single acini	Present
Diabetic	"	Dark and pigmented or pale	Granular or smooth	Thick	"	"	Generally in centres of acini	Absent

TABLE II.—Clinical Features of the Varieties of Hepatic Cirrhosis.

Varieties.	Age.	Sex.	History.	Ascites.	Jaundice.	Hæmatemesis.	Size of Liver.
Alcoholic... ..	Adult	Usually male	Abuse of alcohol	Present in two-thirds	Absent until later stages. A subicteric tinge then only	May occur	Usually small
Cardiac or cyanotic ...	Any age	Either	Chronic heart disease	Not uncommon	Occasional late	Absent	Enlarged
Biliary	"	"	Early jaundice	Absent	Present and persistent	"	"
Diffuse syphilitic ...	Childhood	"	Hereditary syphilis	"	Absent	"	"
Gummatous	Adult	"	Acquired syphilis	May occur	Occasional	"	Irregular
Tuberculous	Any age	"	Tuberculous disease elsewhere	Absent	Absent	"	Enlarged
Malarial	"	"	Prolonged malarial infection	"	Frequently present	"	"
Scarlatinal	Usually in childhood	"	Scarlatina	"	Absent	"	Normal
Rachitic	Childhood	"	Rickets and chronic gastro-intestinal catarrh	"	Absent	"	Enlarged
Diabetic	Adult	Usually male	Diabetes	"	Absent, but skin may be bronzed	"	Generally enlarged

England, its recognition having been due to the labours of French pathologists, especially to Hanot, Letulle, and Brault. It is certainly rare, and I have not seen a case, though the liver of one of my patients was described by the pathologist as tough. It is sometimes but not always accompanied by bronzing of the skin. The liver is almost invariably large, its surface is covered by small granulations, and its consistence is tough. On section the increase of fibrous tissue is very apparent. According to some authorities the new formation of fibrous tissue commences around the hepatic radicles, but in some cases at least the growth commences equally in the portal canals. It is frequently, but not constantly, associated with an abundant deposit of pigment both in the liver cells and in the newly formed fibrous tissue. It does not appear to give rise to marked venous obstruction, and has not been observed to be accompanied in any case by ascites.

The disease is rare, curious, and therefore interesting, but its clinical relations, except its association with bronzing of the skin in certain instances, have still to be elucidated.

This concludes the brief outline of the varieties of hepatic cirrhosis which I have ventured to bring before you. I have not adopted any anatomical classification such as hypertrophic and atrophic cirrhosis, because these are accidents, the most typical atrophic form—the alcoholic—being sometimes large, and the most typical hypertrophic—the biliary—being sometimes small. Nor have I been able to follow Charcot in classifying these varieties according to the details of their microscopical structure as multilobular, monolobular, and monocellular, because these divisions are purely pathological, and have no clinical significance whatever. I am strongly of opinion that in the present state of medical science etiology is the best basis for classification, as that can be appreciated at the bedside.

In conclusion, gentlemen, I would ask for your opinions and experience first on the clinical symptoms and pathological characters of the various forms; and, secondly, as to their prognosis and treatment.

ŒSOPHAGEAL VARICES AS A CAUSE OF HÆMATEMESIS IN CIRRHOSIS OF THE LIVER.

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WITH ILLUSTRATIVE CASES BY

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I DESIRE in this paper to draw attention to a morbid condition of the veins of the œsophagus in cirrhosis of the liver which is not often described, but which, on account of the anatomical relationship of these veins, must often occur, and which has, moreover, a most important bearing upon the etiology of hæmatemesis in this disease.

I refer to the occurrence of a dilated and varicose condition of the veins at the lower end of the œsophagus. This dilatation is due to the development in this situation of a collateral circulation whereby blood from the portal vein can reach the heart without passing through the liver, thus relieving the portal engorgement which tends to occur in cirrhosis of the liver.

This collateral circulation may develop to such an extent as to prevent the occurrence of ascites in these conditions or to permit of its disappearance when it has once occurred, but the dilatation so occasioned renders these œsophageal veins liable to rupture and so to become the cause of hæmatemesis.

The nature of the circulation around the lower end of the œsophagus is such as to admit of an intercommunication between the blood in the portal vein on the one hand and that of the general systemic veins on the other, for we have here the veins of the cardiac end of the stomach belonging to the portal system while, those of the œsophagus belong to the azygos and inferior vena caval system of veins. Thus the capillaries in the mucous membrane at the cardiac orifice of the stomach are continuous with both systems. This can be proved by means of injections, either from the coronary vein of the stomach or from the œsophageal veins, and is well known. The particular channels whereby the anastomosis can take place may not be so well known, and I shall therefore describe them a little more in detail.

The blood from the cardiac end of the stomach is normally carried to the liver by means of the coronary vein, which runs from right to left along the lesser curvature and then turns back behind the stomach to join the portal vein just above its formation by the union of the splenic and superior mesenteric veins. A certain part of the blood is also taken by a smaller vein, which runs from left to right along the lesser curvature and may be described as the right or the small coronary vein, and which opens into the vena portæ near to the gastro-duodenal vein.

A third channel is that furnished by the vasa brevia of the splenic vein. The branches of these three veins intercommunicate to a greater or less extent, and those of the first and last named form a plexus which surrounds the lower end of the œsophagus and which forms the starting point of the anastomotic channel that opens up in cases of portal obstruction.

When the passage of portal blood through the liver is hindered, the blood finds an escape through the œsophageal veins, and there is therefore, to a greater or less extent a reversal of the normal flow in the veins mentioned, but mainly in the coronary vein

Fig. 1.—Diagram showing the course of the blood in the cases described. P.V., Portal vein with left coronary sending a branch up to the diaphragmatic and periesophageal veins; L.D.V., large diaphragmatic vein opening directly into inferior vena cava; L.I.P., left inferior phrenic vein opening into left suprarenal; P.P., periesophageal plexus in connection with the submucous and from which the intercostals take blood.

(that is, the left or great coronary vein). In cases where this vein is blocked by a thrombus the vasa brevia of the splenic vein take the main part in carrying the portal blood to the œsophageal veins.

Let us now consider the branches of the vena cava and azygos veins, whereby the portal blood finds its way to the heart under these abnormal circumstances. The plexus of veins before mentioned, which surrounds the cardiac orifice of the stomach, is more or less continuous with (1) the left inferior phrenic vein, which opens into the left renal, and so into the inferior vena cava; (2) it is continuous with a number of small veins which run in the substance and on the surface of the diaphragm, and open directly into the vena cava as it passes through this organ. These veins play an important part in this connection, owing to the short distance that the portal blood has to travel before reaching the main current in the inferior vena cava. In these cases of cirrhosis, etc., the whole surface of the diaphragm may be seen covered with a dense network of vessels, which coalesce to form four or five trunks of from one-tenth to one-sixteenth of an inch in diameter, and so empty themselves into the vena cava.

The plexus above mentioned is more or less continuous by