

## IV. CIRRHOSIS.

**Definition.**—A chronic disease of the liver, characterized by a gradual destruction of liver-cells and an overgrowth of connective-tissue elements, in consequence of which the organ becomes hard and usually small.

**Etiology.**—The disease occurs most frequently in middle-aged males. It has been regarded as rare in children, except in the syphilitic form, but Palmer Howard collected 63 cases, to which list Hatfield, in a further search of the literature, has been able to add 93, so that its occurrence in early life is more common than has been supposed.

The following are the recognized factors in inducing the disease: (a) *Alcohol.*—The abuse of spirits is the common cause. It is more frequent in countries in which strong spirits are taken than in those in which malt liquors and wines are used. The change results from the irritative effect of the strong solution of alcohol absorbed from the stomach. The fusel oil is thought to be the offending material. Similar effects are doubtless produced by other substances, such as rich, highly seasoned foods, or, as has been suggested, by ptomaines and other alkaloids.

(b) *Syphilis.*—We have already considered (under Syphilis) the forms of cirrhosis, diffuse and gummatous, produced by this poison.

(c) *Cyanotic Congestion.*—In cases of chronic disease of the heart and lungs the liver is in a condition of persistent venous hyperæmia, in consequence of which the central cells of the liver lobules atrophy and there is hyperplasia of the connective tissue.

(d) *Malaria.*—Sclerosis of the liver may follow prolonged malarial poisoning. In this country it is very rare.

(e) *Tuberculosis.*—We have already referred to the sclerotic changes in the liver produced by tuberculosis. It rarely, if ever, induces a condition which can be recognized clinically.

(f) *Scarlet Fever.*—The fact noted by Klein that in scarlet fever there was an infiltration with small cells, an acute interstitial hepatitis, gives a clue to the occurrence of some of the cases of cirrhosis of the liver in children. In other infectious diseases, too, such as typhoid, there are localized necrotic areas which must be replaced by connective tissue. In the cirrhosis of early life, excluding the alcoholic and syphilitic cases, the acute infectious diseases are probably the important antecedents.

(g) *Rickets.*—The enlargement of the liver in this disease is associated with increase in the connective tissue, which surrounds the individual lobules and produces changes in the bile-ducts (Hodgben).

(h) *Anthraxis.*—It occasionally happens in coal-miners that the carbon pigment reaches the liver in large quantities, is deposited in the connective tissue about the portal canal, and may lead to a variety of cirrhosis, which has been described by Welch.

In animals, artificial obstruction of the bile-passages results in cirrhosis, but in man there may be persistent stenosis of the common duct or ob-

struction without marked increase in the connective tissue. The causes which induce the cirrhosis which we meet at the bedside are alcohol and syphilis.

**Morbid Anatomy.**—Practically on the post-mortem table we see cirrhosis in four well-characterized forms:

(a) *The Atrophic Cirrhosis of Laennec.*—The organ is greatly reduced in size and may be deformed. The weight is sometimes not more than a pound or a pound and a half. It presents numerous granulations on the surface; is firm, hard, and cuts with great resistance. The substance is seen to be made up of greenish-yellow islands, surrounded by grayish-white connective tissue. This yellow appearance of the liver induced Laennec to give it the name of cirrhosis.

(b) *Fatty Cirrhosis.*—Even in the atrophic form the fat is increased, but in typical examples of this variety the organ is not reduced in size, but is enlarged, smooth or very slightly granular, anæmic, yellowish white in color, and resembles an ordinary fatty liver. It is, however, firm, cuts with resistance, and microscopically shows a great increase in the connective tissue. This form is quite as common in this country as the atrophic variety. It occurs most frequently in beer-drinkers.

(c) *Hypertrophic Cirrhosis.*—Enlargement of the liver occurs in the early stage of the ordinary atrophic cirrhosis, but the increase is moderate and largely due to hyperæmia. The fatty cirrhotic liver is also large, and may reach a hand's-breadth below the costal margin. The term hypertrophic cirrhosis should be restricted to the form described by French writers, which is also known as *biliary cirrhosis*. Unfortunately, this has been used by some writers to include as well the cases in which there has been permanent occlusion of the duct, either by stricture or a calculus; the induration, however, is slight under these circumstances and hypertrophy very rare. It seems best to limit the terms *biliary* and *hypertrophic* cirrhosis to the form characterized by permanent enlargement of the liver, a marked involvement of the smaller biliary canaliculi, and retention in an unusual degree, in comparison with atrophic cirrhosis, of the number and form of the liver-cells, in spite of the great increase of the lobular connective tissue. In this form the liver is greatly enlarged; in one of my cases it weighed seven pounds. The surface is smooth, it is exceedingly firm, resists cutting, and presents on section a deep greenish-yellow color. All of my cases have been in hard drinkers.

(d) *Perihepatitis; Glissonian Cirrhosis.*—In this form the liver is greatly reduced in size, much altered in shape, and everywhere surrounded by a firm grayish-white membrane, sometimes of semi-cartilaginous consistence, varying from 10 to 15 mm. in thickness. This fibrous investment can be stripped off readily, and the liver substance may look almost normal, but usually shows cirrhotic changes. The capsular thickening may be slight, and the portal connective tissue chiefly involved. The capsule of the spleen is, as a rule, similarly affected, and both processes are asso-



ciated with a proliferative peritonitis. The condition is most frequent as a result of alcohol, but occurs also in instances of cyanotic induration.

The two essential elements in cirrhosis are destruction of liver-cells and obstruction to the portal circulation.

In an autopsy on a case of atrophic cirrhosis the peritonæum is usually found to contain a large quantity of fluid, the membrane is opaque, and there is chronic catarrh of the stomach and of the small intestines. The kidneys are sometimes cirrhotic, the bases of the lungs may be much compressed by the ascitic fluid, the heart often shows marked degeneration, and arterio-sclerosis is usually present. A remarkable feature is the association of acute tuberculosis with cirrhosis. In seven cases of my series the patients died with either acute tuberculous peritonitis or acute tuberculous pleurisy. Pitt states that twenty-two and a half per cent of the cases of cirrhosis dying in Guy's Hospital during twelve years had acute tuberculosis.

The compensatory circulation is usually readily demonstrated. It is carried out by the following set of vessels: (1) The accessory portal system of Sappey, of which important branches pass in the round and suspensory ligaments and unite with the epigastric and mammary systems. These vessels are numerous and small. Occasionally a large single vein, which may attain the size of the little finger, passes from the hilus of the liver in the round ligament, and joins the epigastric veins at the navel. Although this has the position of the umbilical vein, it is usually, as Sappey showed, a para-umbilical vein—that is, an enlarged vein by the side of the obliterated umbilical vessel. There may be produced about the navel a large bunch of varices, the so-called caput Medusæ. Other branches of this system occur in the gastro-epiploic omentum, about the gall-bladder, and, most important of all, in the suspensory ligament. These latter form large branches, which anastomose freely with the diaphragmatic veins, and so unite with the vena azygos. (2) By the anastomosis between the œsophageal and gastric veins. The veins at the lower end of the œsophagus may be enormously enlarged, producing varices which project on the mucous membrane. (3) The communications between the hæmorrhoidal and the inferior mesenteric veins. The freedom of communication in this direction is very variable, and in some instances the hæmorrhoidal veins are not much enlarged. (4) The veins of Retzius, which unite the radicles of the portal branches in the intestines and mesentery with the inferior vena cava and its branches. To this system belong the whole group of retroperitoneal veins, which are in most instances enormously enlarged, particularly about the kidneys, and which serve to carry off a considerable proportion of the portal blood.

**Symptoms.**—(a) *Of the Atrophic Form.*—The most extreme grade of atrophic cirrhosis may exist without symptoms. So long as the compensatory circulation is maintained the patient may suffer little or no inconvenience. The remarkable efficiency of this collateral circulation is well

seen in those rare instances of permanent obliteration of the portal vein, which may exist for many years.

The symptoms may be divided into two groups—obstructive and toxic.

*Obstructive.*—The overfilling of the blood-vessels of the stomach and intestine leads to chronic catarrh, and the patients suffer with nausea and vomiting, particularly in the morning; the tongue is furred and the bowels are irregular. Hæmorrhage from the stomach may be an early symptom; it is often profuse and liable to recur. It seldom proves fatal. The amount vomited may be remarkable, as in a case already referred to, in which ten pounds were ejected in seven days. Following the hæmatemesis melæna is common. Enlargement of the spleen occurs from the chronic congestion. The organ can usually be felt. Evidences of the establishment of the collateral circulation are seen in the enlarged epigastric and mammary veins, more rarely in the presence of the caput Medusæ and in the development of hæmorrhoids. The distended venules in the lower thoracic zone along the line of attachment of the diaphragm are not specially marked in cirrhosis. The most striking feature of failure in the compensatory circulation is ascites, the effusion of serous fluid into the peritoneal cavity. The conditions under which this occurs are still obscure. The abdomen gradually distends, may reach a large size, and contain as much as 15 or 20 litres. Œdema of the feet may precede or develop with the ascites. The dropsy rarely becomes general.

Jaundice is usually slight, and was present in only 35 of 130 cases of cirrhosis reported by Fagge. The skin has frequently a sallow, slightly icteric tint. The urine is often reduced in amount, contains urates in abundance, often a slight amount of albumen, and, if jaundice is intense, tube-casts. The disease may be afebrile throughout, but in many cases, as shown by Carrington, there is slight fever, from 100° to 102·5°.

Examination in the early stage of the disease may show moderate enlargement of the liver, which may be painful on pressure. At this period the patient may come under observation for dyspepsia, hæmatemesis, slight jaundice, or nervous symptoms. Later in the disease, the patient has an unmistakable hepatic facies; he is thin, the eyes are sunken, the conjunctivæ watery, the nose and cheeks show distended venules, and the complexion is muddy or icteric. On the enlarged abdomen the vessels are distended, and a bunch of dilated veins may surround the navel. When much fluid is in the peritonæum it is impossible to make a satisfactory examination, but after withdrawal the area of liver dulness is found to be diminished, particularly in the middle line, and on deep pressure the edge of the liver can be detected, and occasionally the hard, firm, and even granular surface. The spleen can be felt in the left hypochondriac region. Examination of the anus may reveal the presence of hæmorrhoids.

*Toxic Symptoms.*—At any stage of atrophic cirrhosis the patient may develop cerebral symptoms, either a noisy, joyous delirium, or stupor, coma, or even convulsions. The condition is not infrequently mistaken for



uræmia. The nature of the toxic agent is not yet settled. The symptoms may develop without jaundice, and cannot be attributed to cholæmia, and they may come on in hospital when the patient has not had alcohol for weeks.

The fatty cirrhotic liver may produce symptoms similar to those of the atrophic form, but it more frequently is latent and is found accidentally in toppers who have died from various diseases. The greater number of the cases clinically diagnosed as cirrhosis with enlargement come in this division.

(b) *Hypertrophic* or biliary cirrhosis has a definite and distinctive symptomatology. The liver may be enlarged for months or even years. Jaundice persists for some time, on which point French writers lay great stress. It may, however, come on acutely with the other symptoms. It is intense, like an obstructive jaundice, but, as a rule, the stools are bile-stained. It may continue for a long time without the development of other symptoms; then delirium sets in and all the features of an acute febrile jaundice. The tongue is dry, the pulse rapid, the temperature ranges from 102° to 104°, and petechiæ occur on the skin. The patient may present every feature of acute yellow atrophy, including even the convulsive seizures. The attack in one of my cases proved fatal within ten days; in another it was prolonged for three weeks. Ascites does not develop. The enlargement of the liver may be the sole diagnostic criterion between these cases and acute yellow atrophy. I do not know, however, of the occurrence of leucin or tyrosin in the urine in this condition.

(c) The perihepatitis with cirrhosis cannot be distinguished from the ordinary atrophic form.

**Diagnosis.**—With ascites, a well-marked history of alcoholism, the hepatic facies, and hæmorrhage from the stomach or bowels, the diagnosis is rarely doubtful. If, after withdrawal of the fluid, the spleen is found to be enlarged and the liver either not palpable or, if it is enlarged, hard and regular, the probabilities in favor of cirrhosis are very great. In the early stages of the disease, when the liver is increased in size, it may be impossible to say whether it is a cirrhotic or a fatty liver. The differential diagnosis between common and syphilitic cirrhosis can sometimes be made. A marked history of syphilis or the existence of other syphilitic lesions, with great irregularity in the surface or at the edge of the liver, are the points in favor of the latter. Thrombosis or obliteration of the portal vein can rarely be differentiated. In the case of fibroid transformation of the portal vein which came under my observation, the collateral circulation had been established for years, and the symptoms were simply those of extreme portal obstruction, such as occur in cirrhosis. Thrombosis of the portal vein is frequent in cirrhosis and may be characterized by a rapidly developing ascites.

**Prognosis.**—The prognosis is, as a rule, bad. When the collateral circulation is fully established the patient may have no symptoms what-

ever. Three cases of advanced atrophic cirrhosis have died under my observation of other affections without presenting during life any symptoms pointing to disease of the liver. There are instances, too, of enlargement of the liver, slight jaundice, cerebral symptoms, and even hæmatemesis, in which the liver becomes reduced in size, the symptoms disappear, and the patient may live in comparative comfort for many years. There are many cases, too, in which, after one or two tapplings, the symptoms have disappeared and the patients have apparently recovered.

**Treatment.**—Ordinary cirrhosis of the liver is an incurable disease. Many writers, speaking of the curability of certain forms, show a lack of appreciation of the essential conditions upon which the symptoms depend. So far as we have any knowledge, no remedies at our disposal can alter or remove the cicatricial connective tissue which constitutes the *materia peccans* in ordinary cirrhosis. On the other hand, we know that extreme grades of contraction of the liver may persist for years without symptoms when the compensatory circulation exists. The so-called cure of cirrhosis means the re-establishment of this compensation; and it would be as unreasonable to speak of healing a chronic valvular lesion when with digitalis we have restored the circulatory balance as it is to speak of curing cirrhosis of the liver when by tapping and other measures the compensation has in some way been restored.

The patient should abstain entirely from alcohol, and, if possible, should take a milk diet, which has been highly recommended by Semmola. In any case, the diet should be nutritious, but not too rich. Measures should be employed to reduce the gastro-intestinal catarrh, and the patient should lead a quiet, out-of-door life and keep the skin active, the bowels regular, and the urine abundant. In non-syphilitic cases it is useless to give either mercury or iodide of potassium. When a well-marked history of syphilis exists these remedies should be used, but neither of them has any more influence upon the development of a new growth of connective tissue in the liver than it has upon the progressive development of a scar tissue in a keloid or in an ordinary developing cicatrix. The ascites should be tapped early, and the operation may be repeated so soon as the distention becomes distressing. The continuous drainage with a Southey's tube may be employed. It is much better to resort to tapping early if after a few days' trial the fluid does not subside rapidly under the use of saline purges. From half an ounce to an ounce and a half of sulphate of magnesia may be given in as little water as possible half an hour before breakfast. Elate-rium, the compound jalap powder, or the bitartrate of potash may also be employed. Digitalis and squills are often useful. In the syphilitic cases or when syphilis is suspected iodide of potassium may be given in doses of from fifteen to thirty drops of the saturated solution three times a day, and mercury, which is conveniently given with squills and digitalis in the form of Addison's or Niemeyer's pill. A case of well-marked syphilitic cirrhosis with recurring ascites, in which tapping was resorted to on eight



or ten occasions, took this pill at intervals for a year with the greatest benefit, and subsequently had four years of tolerably good health.

## V. ABSCESS OF THE LIVER.

**Etiology.**—Suppuration within the liver, either in the parenchyma or in the blood or bile passages, occurs under the following conditions:

(1) The tropical abscess. In hot climates this form may develop idiosyncratically, but more commonly follows dysentery. It frequently occurs among Europeans in India, particularly those who drink alcohol freely and are exposed to great heat. The relation of this form of abscess to dysentery is still under discussion, and Anglo-Indian practitioners are by no means unanimous on the subject. Certainly cases may develop without a history of previous dysentery, and there have been fatal cases without any affection of the large bowel. In this country the large solitary tropical abscess also occurs, oftenest in the Southern States. In Baltimore it is not very infrequent, as may be judged from the fact that during two years there have been at my clinic five cases, and I know of the occurrence of three or four additional cases during this time in the city.

The relation of this form of abscess to the *amœba coli* has been carefully studied by Kartulis and exhaustively considered in a monograph by Councilman and Lafleur. The descriptions and illustrations of these authors are most convincing as to the direct etiological association of this organism with liver abscess. Clinically the patient may have *amœba coli* in the stools and well-marked signs of liver abscess without marked symptoms of dysentery and even with the feces well formed.

(2) Traumatism is an occasional cause. The injury is generally in the hepatic region. Two instances have come under my notice of it in brakemen who were injured while coupling cars. Injury of the head is not infrequently followed by liver abscess.

(3) Embolic or pyæmic abscesses are the most numerous, and may develop in a general pyæmia from any cause or follow foci of suppuration in the territory of the portal vessels. The infective agents may reach the liver through the hepatic artery, as in those cases in which the original focus of infection is in the area of the systemic circulation; though it may happen occasionally that the infective agent, instead of passing through the lungs, reaches the liver through the inferior vena cava and the hepatic veins. A remarkable instance of multiple abscesses of arterial origin was afforded by the case of aneurism of the hepatic artery reported by Ross and myself. Infection through the portal vein is much more common. It results from dysentery and other ulcerative affections of the bowels, appendicitis, occasionally after typhoid fever, in rectal affections, and in abscesses in the pelvis. In these cases the abscesses are multiple and, as a rule, within the branches of the portal vein—suppurative pylephlebitis.

(4) A not uncommon cause of suppuration is inflammation of the bile-passages caused by gall-stones, more rarely by parasites—suppurative cholangitis.

In some instances of tuberculosis of the liver the affection is chiefly of the bile-ducts, with the formation of multiple tuberculous abscesses containing a bile-stained pus.

(5) Foreign bodies and parasites. In rare instances foreign bodies, such as a needle, may pass from the stomach or gullet, lodge in the liver, and excite an abscess, or, as in several instances which have been reported, a foreign body, such as a needle or a fish-bone, may perforate a branch of the portal vein itself and induce extensive pylephlebitis. Echinococcus cysts frequently cause suppuration; the penetration of round worms into the liver less commonly; and most rarely of all the liver-fluke.

**Morbid Anatomy.**—(a) *Of the Solitary or Tropical Abscess.*—This is not always single; there may be two or even more large abscess cavities, ranging in size from an orange to a child's head. The largest-sized abscess may contain from three to six litres of pus and involve more than three fourths of the entire organ. In Waring's statistics, sixty-two per cent of the cases were single. The abscess in nearly seventy per cent of the cases was in the right lobe, more toward the convexity than the concave side. In long-standing cases the abscess-wall may be firm and thick, but, as a rule, the cavity possesses no definite limiting membrane, and section of the wall shows an internal layer, grayish in color, shreddy, and made up of necrotic liver substance, pus-cells, and amœbæ; a middle layer, brownish red in color; and an external zone of hyperæmic liver tissue. The pus is often reddish brown in color, closely resembling anchovy sauce. In other instances it is grayish white, mucoid, and may be quite creamy. The odor is at times very peculiar. In one instance it had the sour smell of chyme, though no connection with the stomach was found. In a recent case of amœbic dysentery there were multiple miliary abscesses in the liver, all of which contained amœbæ.

The bacteriological examination of the contents show that as a rule the pus is sterile (Kartulis). The termination of this form of abscess may be as follows, as noted in Waring's 300 cases: Remained intact, fifty-six per cent; opened by operation, sixteen per cent; perforated the right pleura, nearly five per cent; ruptured into the right lung, nine per cent; ruptured into the peritonæum, five per cent; ruptured into the colon, nearly three per cent; and there were in addition instances which ruptured into the hepatic and bile-vessels and into the gall-bladder.

(b) *Of Septic and Pyæmic Abscesses.*—These are always multiple, though occasionally, following injury, there may be a large solitary collection of pus.

In suppurative pylephlebitis the liver is uniformly enlarged. The capsule may be smooth and the external surface of the organ of normal appearance. In other instances, numerous yellowish-white points appear