without symptoms of portal obstruction (absence especially of ascites and gastro-intestinal hemorrhages), and occurring particularly in a non-alcoholic subject, points with some degree of certainty to hypertrophic cirrhosis of the liver. It must not be forgotten, however, that in portal cirrhosis severe jaundice may exceptionally be found, and that ascites and gastro-intestinal hemorrhages may likewise exceptionally occur in biliary cirrhosis; consequently in some cases it may be impossible, because of the inability to distinguish which of the two varieties lies before us, or whether both forms do not exist together, to say more than that a cirrhosis of the liver is present.

Treatment—1. *Medical Treatment.* We will consider the two varieties of cirrhosis together. We must in the first place endeavor to ascertain and treat the cause of the cirrhosis. In all cases alcohol must be strictly avoided, as well as irritating articles of food, such as spices, coffee, etc. A malarial patient should be removed to a non-malarial climate. The syphilitic patient should be given inunctions of mercury and large doses of potassium iodide. Secondly, we have to treat whatever produces impairment of health and a tendency to death, so far as lies in our power. The diet should be plain, simple, but ample for the maintenance of strength. Chronic gastritis is to be treated in the usual way, *i.e.*, by prescribing a milk diet for a time if the disturbance of the digestion be severe, then a careful regulation of the diet, all forms of starchy and saccharine food being reduced to a minimum to avoid fermentation and flatulence. Green vegetables and fruit (potatoes being avoided), beans, peas, eggs, boiled fish, and fresh lean meat may be eaten if well borne; so also may stale bread, especially Graham bread. Lavage of the stomach will be found indispensable in most of the cases. Plenty of fresh air and moderate exercise should be secured. The patient should avoid nervous anxiety and fatigue and be carefully protected from cold and wet.

Certain drugs may be found useful. Bitter tonics and acids may stimulate the appetite, and the milder preparations of iron may be used to overcome anæmia. Potassium iodide may be given with the slender hope of preventing further formation of connective tissue. Chloroform water, bismuth, and magnesia are of use for the nausea and vomiting induced by the catarrh of the stomach, while bismuth, salol, thymol, and peppermint water are to be recommended for the relief of flatulence. The bowels are to be regulated by salines, rhubarb, aloes, or calomel. Except in the later stages of the disease, or when hemorrhage takes place, diarrhœa, if not too profuse, need not be checked by medicine. If this be necessary, bismuth subnitrate, the mineral acids, krameria, catechu are perhaps the most efficient. These patients do not bear opium well. Hemorrhage from the stomach or intestines is best relieved by ice, morphine, and absolute rest. Ergot is of doubtful value. It may also, in some cases, be necessary to give drugs which will increase the production of bile. Ascites is the condition for which effective treatment is eventually demanded. At its onset or to prevent re-accumulation after tapping, we may use cathartics (magnesium sulphate, compound jalap powder), diaphoretics (heat), diuretics (bitartrate of potash, diuretin, or a combination of calomel, digitalis, and squill), abdominal electricity and massage. Tapping eventually becomes necessary to overcome the abdominal distention, and it ought not to be delayed. It should be repeated as often as is necessary to relieve distress from pressure. In some cases on record the ascites has not returned after a few tappings, but this result we may not ordinarily expect, since in most cases it is to be regarded merely as a means of adding to the immediate comfort of the patient, because the fluid usually quickly returns. The quantity of fluid removed at one time may be as high as ten quarts. Pleuritic effusion will require aspiration, and that possibly repeatedly. Toward the end of life alcohol may be necessary as a stimulant, and its use should be advised without question.

2. *Surgical Treatment.* Additional proof has been produced during the past two years to the effect that the establishment of a collateral circulation by operation will greatly benefit the patient suffering from cirrhosis of the liver with ascites. The operative procedure, which is to be carried out with the strictest aseptic precautions, is described in detail in the article entitled *Liver, Diseases of: Surgical Treatment.* The reader is therefore referred to this article for further information on the subject. Although up to the present time the mortality of such surgical treatment has been somewhat heavy, it is fair to assume that when the patients are turned over to the surgeon for operation at an earlier stage, *i.e.*, before they have reached the last stages of toxæmia, the results will make a better showing.

Clarence Arthur McWilliams.

LIVER, DISEASES OF: FATTY INFILTRATION AND FATTY DEGENERATION.—There are two varieties of fatty liver: (1) fatty infiltration, (2) fatty degeneration. During life it is impossible to be absolutely certain which variety exists. However, clinical experience makes us fairly certain of our diagnosis, since we know that a history of phosphorus poisoning indicates degeneration, while the history of over-indulgence in food and drink with resulting obesity suggests very strongly infiltration. It is possible for fatty infiltration and degeneration to be present in the same case. These two conditions are distinguished by microscopical and chemical means. In most tissues fatty degeneration shows itself by the occurrence of small drops of fat which show no tendency to run together, while in infiltration large drops are usually formed. There are exceptions to this rule, however, as large-sized drops are found in fatty degeneration of the renal epithelium and in liver cells in phosphorus poisoning and in acute yellow atrophy of the liver.

It is only in tissues like those of the liver, which normally act as storehouses of fat and also frequently undergo degeneration, that much difficulty is encountered. It is often difficult to decide how much fat has been produced on the spot by disintegration of the cell albumen, and how much has been deposited from the blood.

According to Perls, chemical analysis shows that in fatty infiltration there is a much greater quantity of fat than in fatty degeneration. It proves that in fatty degeneration there is a diminution of the solid (non-fatty) matter, that is, of the albumen, while the amount of water in the liver remains normal. In fatty infiltration, on the other hand, the fat is increased mainly at the expense of the water, the watery constituents of the liver falling from 77 to 50 per cent., while the amount of non-fatty solid matter, albumen, remains about the same. William Hunter, in vol. iv. of Allbutt's system, states that in phosphorus poisoning the percentage of fat is as high as 30, water 60, and non-fatty tissue 10 per cent., while in acute yellow atrophy the fat per cent. is only 4.2, water 80.5, non-fatty substance 15.3. The normal liver, according to him, contains about 3 per cent. of fat, 76 per cent. of water, and 21 per cent. of non-fatty substance. On post-mortem examination, if the other fat-containing tissues of the body, such as the epicardium, mesentery, epiploic appendages and subcutaneous connective tissue, show an abnormal amount of fat, it is very likely that the liver will show an infiltration.

Fatty liver may be classified as *dietetic, cachectic, infectious*, and *toxic*.

Dietetic.—It is well known that the liver is normally one of the fat repositories of the body; physiologically there is a certain amount of fat in the liver cells. This does not interfere at all with the function of the liver. When this infiltration becomes excessive, it is pathological. It is difficult to draw the line between physiological and pathological fat infiltration. The latter may occur in gluttons and drunkards and those leading a sedentary life. Infants who are overfed, especially with a food containing an excess of sugar, show a fatty liver.

Cachectic.—It is rather a paradoxical fact that the various cachexias—in which there is marked emaciation—show a fatty liver. Among these cachexias the tubercu-

lous affections of lungs, bones, joints, and lymphatics are especially to be mentioned; also carcinomata of various organs, chronic suppurations, such as bone disease, old bedsores, chronic dysentery, and chronic anæmia, including leukæmia and chlorosis.

Infectious.—In the various acute infectious fevers fatty liver is found.

Toxic.—Fatty degeneration is found in poisoning by phosphorus, arsenic, mercury, camphor, chloroform, and certain vegetable fungi. Phosphorus poisoning is the best example of this variety. Since the enactment of stringent measures, enforcing the use of a more insoluble and less poisonous form of phosphorus in the manufacture of lucifer matches in the United States and England, this form of poisoning is much less common than formerly. It is still occasionally seen in Austria.

Fatty degeneration is observed in the liver as a result of venous congestion ("fatty nutmeg liver"), and it is also observed in the neighborhood of carcinomatous abscesses and in cirrhosis.

Fatty degeneration of the new-born, so-called Buhl's disease, has been described; so also has an acute fatty degeneration occurring in puerperal women.

Explanation of Infiltration and Degeneration.—The dietetic form is due to an under-combustion of the fat—which may or may not be taken into the system in excess. Indigestion, with a sluggish flow of bile (which normally carries fat from the liver), helps to explain this fact. If large quantities of alcohol are consumed, the oxygen is used in burning this up, leaving the fat to be stored up in the liver.

In the *cachectic* form, of which that occurring in pulmonary tuberculosis is the most striking example, there is deficiency of oxygen, due primarily to the lung disease, and secondarily to the fact that the functional capacity of the tissues and organs has suffered in proportion to the disease. An anæmia is also present, so that less oxygen is carried in the blood stream. The fat present in the blood of consumptives may come partly from the food, partly from the breaking up of the albumen of the organism, and in part is taken up from the disappearing adipose tissue. The combustion of fat is incomplete and it is stored up in the liver.

Toxic.—When present in the fluids of the body phosphorus produces the gravest changes in the metamorphosis of tissues: the consumption of oxygen and the excretion of carbonic acid are diminished; at the same time an increase in the decomposition of albumen occurs. The fat arises from the decomposition of albumen, and lies in the liver uncombusted, because of a lack of oxygen.

Infectious.—This fatty degeneration is attributable to the poisons of the infectious diseases, as well as to the high temperature associated with them.

In fatty nutmeg liver of heart disease, there is an under-combustion of the fat produced in the liver cells, by reason of lack of oxygen.

Morbid Anatomy.—The liver is usually enlarged; it may be twice the size of the normal organ. It may occasionally be smaller than normal, and is then usually associated with Laënnec's cirrhosis. It is pale yellow, soft, and doughy when warm, showing a permanent pitting of the surface after pressure. The edge is usually rounded and blunt. On section, a film of grease adheres to the blade. In the degeneration of phosphorus poisoning the outlines of the liver cells are indistinct, the nuclei do not stain, and a finely granular detritus or large fat drops occupy the cells.

Symptoms and Signs.—There are no characteristic symptoms of this condition. There may be a feeling of fulness and pressure in the region of the liver and the stomach. In some, especially tuberculous cases, the distress caused by the liver may be severe. The area of liver dulness is usually enlarged, and on palpation the blunt edge of the liver can be felt below the margin of the ribs. In cases of infiltration the liver is not sensitive to pressure, but in the degeneration of phosphorus poisoning it may be exquisitely tender.

Prognosis.—This depends upon the prognosis of the fundamental disease. In itself fatty liver does not tend to shorten life.

Treatment.—It is directed to the causative disease, with no especial reference to the condition of the liver. If the fundamental disease—as, for instance, obesity—is benefited, the liver will improve with the rest of the tissues of the body.

James Rae Arneill.

LIVER, DISEASES OF: GALL STONES. See *Gall Bladder and Gall Ducts, etc.*, and *Concretions.*

LIVER, DISEASES OF: HYPERÆMIA.—(Congestion of the Liver.) A more or less persistent increase in the volume of the blood in the liver constitutes hyperæmia, and is pathological, in contradistinction to that temporary physiological increase which occurs during the period of digestion.

When the hyperæmia is due to an increased afflux of blood, it is termed a *fluxion*; when to a diminished efflux, a *congestion*. An *active* hyperæmia signifies a sudden determination of blood to the organ, or a fluxion, made manifest by definite acute symptoms; it usually subsides without structural changes in the viscus. A repetition of such an hyperæmia, and especially with an increased volume of blood in the hepatic artery, leads to the gradual development of interstitial hepatitis or cirrhosis. A *passive* hyperæmia, diminished efflux, is developed slowly, with few accompanying symptoms until after structural, changes which usually occur, have taken place.

An *active* hyperæmia is essentially acute; a passive, chronic. Some passive hyperæmias, however, occur in a short space of time, as in the form that accompanies a large pleural effusion.

Mechanical hyperæmia, active or passive, is caused by an obstruction to the outlet of blood, and this obstruction may be seated in the vena cava, the heart, or the pulmonary circulation.

It is, however, wellnigh impossible to separate the various forms of hyperæmia by hard-and-fast lines, and clinically it suffices to consider hyperæmias as either active or passive—which terms are practically synonymous with acute and chronic congestion of the organ. The difficulty of accurate subdivision arises from the fact that the various forms blend. Three sets of vessels are involved in the occurrence of congestion. The congestion due to over-repletion in the hepatic veins differs in form and behavior from that due to portal fulness. The two forms are readily distinguished by a consideration of the primary causal factors. To distinguish the hyperæmia which is due to an excess in the portal system of veins, from that which is dependent upon a fulness of the hepatic artery, is clinically impossible and scarcely practicable.

Etiology.—Active hyperæmia, acute congestion. The ingestion of *stimulating food* and *drink* causes active hyperæmia. When alcohol, fermented liquors, spices, and rich foods are taken frequently or in excess, especially by delicate persons who lead a sedentary life, the normal congestion associated with digestion becomes excessive and more or less permanent. Musgrove (*Medical Record*, 1902) considers the over-indulgence in highly seasoned articles of diet as seen in the East, and the consequent hyperæmia of the liver, to be directly contributory to the frequency of hepatic abscess. Residents in hot climates, with or without such indulgence, are particularly prone to attacks of hepatic congestion; hence *high temperature* has been considered a causal factor. Sudden checking of the *perspiration*, a severe and sudden, or a prolonged, *chill* or repetition of chills, is followed quite frequently by active hyperæmia. Persons living in a *malarious* region are liable to attacks of hepatic, as well as of splenic, congestion. During the *seasons* when malaria is rife, or when the changes in temperature are sudden and extreme, the disease is most liable to occur. Congestion of the liver is found, too, in many *infectious diseases*, as relapsing fever, yellow fever, epidemic cerebrospinal meningitis, and scarlet and typhus fevers.

After traumatism, a determination of blood to the injured area of hepatic tissue is always observed. It is said, also, that hyperæmia of the liver occurs in scurvy.

The suppression of customary discharges, especially of blood, causes fluxion to the liver. Cessation of the menses at the menopause, or by cold or nervous influence, and arrest of the hemorrhages of uterine disease from various causes, are not uncommonly followed by attacks of hepatic congestion. So, also, congestion of this organ not infrequently follows the stanching of hemorrhoidal bleeding.

It is said that habitual constipation induces passive congestion of the liver. It is difficult to prove, yet it can readily be surmised as possible that paralysis of the sympathetic nerve causes torpor of the hepatic circulation. It is true that we see in some diseases—as tuberculous peritonitis, in which the contents of the abdomen are jumbled into a mass and the sympathetic ganglia are degenerated—a hyperæmia of the liver; a condition, however, which could be explained by the assumption that the portal capillaries are obstructed. As Thierfelder suggests, the congestion associated with diabetes mellitus may be due to paresis of the abdominal sympathetic. The sluggishness of the circulation, which occurs in persons who exercise sparingly, is markedly seen in the stasis that occurs in the liver.

Active or acute congestion of the liver, therefore, is liable to occur in persons of sedentary habits, without muscular vigor, who are high livers and reside in hot climates. It should not be forgotten, however, that its occurrence in temperate climates, though of comparative infrequency, is yet not very rare. A chill, or sudden checking of perspiration, is a frequent exciting cause. Malaria is most commonly observed, of all diseases, to excite an attack. Hepatic congestion occurs most frequently in the autumn, and usually affects individuals of middle life.

The most important and frequent causes of hyperæmia of the liver, whether in temperate or in tropical climates, are mechanical. The forms of heart disease which cause backing of the blood into the venous system—such, for example, as cardiac dilatation and mitral and tricuspid valvular disease—are attended by hepatic congestion. In emphysema, interstitial pneumonia, and atelectasis, the venous flow is obstructed. The form of congestion is, under these circumstances, passive. Tumors of the mediastinum, aneurisms, thrombosis and tumors of the inferior vena cava, effusions in the pleura,[1] pressing upon or bending the vena cava, also cause hepatic congestion.

Symptomatology.—It is manifest that the symptoms of hyperæmia of the liver differ according to the cause. Moreover, they are intermingled with the symptoms that belong to the respective causal factors. In mechanical hyperæmia, for instance, the symptoms of obstructive heart or lung disease are present, along with those of hepatic congestion. In active hyperæmia the hepatic symptoms are less complicated, and serve as a type of the acute form.

After a chill, excesses in diet, or exposure to high temperature, complaints of pain in the liver, and of weight and fulness in the right hypochondrium, are made. The pain may extend to the right shoulder, is constant and associated with tenderness of the liver, and is excited by palpation along the margin of the ribs. Febrile reaction, not marked, attends the attack for two or three days, while general *malaise* is marked. At the same time a bad taste in the mouth, a tongue heavily coated with a yellowish material, nausea, thirst, anorexia, epigastric fulness, flatulency, and often vomiting, are present. The bowels, at first torpid, are relieved by diarrhœa. A gastro-intestinal catarrh usually accompanies the hepatic congestion, in which case vomiting and diarrhœa are more frequent. The ejecta from the stomach are composed of the food, an acid, glairy mucus, and bile-stained watery fluid. The stools are clay-colored and pasty, or watery, acid, and greenish, or dark-colored. Often there is some dyspnœa, and the so-called liver cough is present. Headache, usually frontal, accompanies the attack, and is associated with vertigo.

On physical examination the liver is found to be enlarged, extending two or three inches below the margin of the ribs. Its edge is rounded, elastic, and smooth on palpation.

In a day or two the conjunctivæ become yellowish, and even a light degree of general jaundice supervenes. The jaundice is due to associated catarrh of the gall ducts, or to pressure on them by the engorged vessels. Languor and debility continue for some time, while melancholy and hypochondriasis commonly occur. The countenance, at first flushed, or if the pain is severe, anxious and pinched, grows sallow and worn.

The urine is scanty and high-colored. It contains an excess of urates, some bile pigment if there is jaundice, and often small amounts of albumin and sugar (functional albuminuria and glycosuria). The presence of these ingredients may be intermittent, and may vary with the diet.

If the hyperæmia persists, the overloaded state of the portal vessels results in sluggish absorption of the products of digestion and over-repletion of the vessels of the gastro-intestinal mucous membrane. A true catarrh of the tract arises, and is all the more aggravated and persistent on account of the vascular stasis. Its symptoms replace largely those of congestion. In passive and mechanical hyperæmia, this catarrh is most marked. In these forms the liver is large, more firm, but less tender on palpation. Its size, in hyperæmia due to obstruction, is variable. Depletion by a purgative, or removal or relief of the cause (cardiac dilatation, etc.), would reduce it, and a recurrence of the cause would again be followed by enlargement. This temporary change characterizes mechanical hyperæmia. An enlarged, congested liver may be temporarily increased in size by sudden obstruction in some other area of the blood circuit. A pneumonia or a pleural effusion may thus aggravate the hepatic engorgement, and in turn be aggravated by the old liver disease.

The stasis in the hepatic circulation may be so extreme as to cause over-fulness in the portal vessels and secondary enlargement of the organs in that area. An ascites, out of proportion to the general anasarca usually present, will arise, the spleen becomes enlarged, and the hemorrhoidal vessels are dilated. The secondary phenomena are more probable if the changes incident to overgrowth of the cellular tissue of the liver ensue. In this class of cases the liver is diminished in size, and, if palpable, is very firm and hard.

Course, Duration, and Prognosis.—The course and duration are largely determined by the cause. An active congestion of the liver will subside usually in from two to three weeks. The subjective symptoms are often removed, while enlargement of the organ may continue for some time. Congestions of the liver which originate on the portal side of the hepatic circulation are more amenable to treatment, and last a shorter time, than the congestions on the hepatic side. The latter are due to chronic heart and lung disease, and in their varying course, now better, now worse, depend upon the primary causal agency. The prognosis, likewise, depends on the cause. When the liver becomes enlarged from congestion, and subsequently undergoes marked atrophy, the prognosis of the hepatic trouble is serious, and it in turn renders more grave, day by day, the primary disease. Care must be exercised not to mistake the initial congestion of true cirrhosis for simple hyperæmia. An active congestion of a liver, in which the anatomical structures are abnormal from previous disease, is serious and may fatally terminate a slow cirrhosis or a fatty liver.

Diagnosis.—The diagnosis is usually easy, but the condition cannot be clearly recognized unless the causal influences previously mentioned be evident. In addition to the cause, the shape and size of the liver, the gastro-intestinal catarrh, the condition of the urine, and the signs of secondary portal obstruction, must also be considered.

Morbid Anatomy and Pathology.—Before advanced secondary changes have taken place the liver is uniformly enlarged. The enlargement is most marked in the thickness of the organ. The surface is smooth, the capsule transparent, the edges are thick and rounded. On section, dark blood oozes freely from the vessels. The tissue is soft and darker in color than normal. The vessels are dilated; the enlargement of the hepatic vein in the acini is especially distinct in passive congestion, though a similar increase in size of the portal vessels occurs when the engorgement is limited to that side of the circulation. Throughout the substance of the organ numerous hemorrhages are seen. On microscopical examination, the vessels are seen to be dilated and their walls thickened by increased cell proliferation and the migration of leucocytes. Atrophy from compression of the hepatic cells is marked. A catarrh of the minute ducts is often present.

In the more advanced stages the atrophy of the cells is more marked, pigmentation and fatty degeneration of the cells in the portal zone are present, to a great degree, and the cell proliferation and the infiltration of leucocytes are not only seen in and about the vessel walls, but also between the hepatic cells of the individual acini. Newformed bile ducts are frequently to be seen in the new-formed connective tissue. This is due to an overgrowth of connective tissue, or to greater distinctness of the cells on account of destruction of the hepatic cells. A liver that presents such histological changes is called the *nutmeg liver*. It is normal in size or lessened, firm on section. The capsule is opaque, and the surface and interior are granular, the latter gorged with blood. The acini are distinct, the central vein much dilated and dark in color. The periphery of the lobule is yellow, the centre dark red. The term "nutmeg" is applied to the marbled appearance.

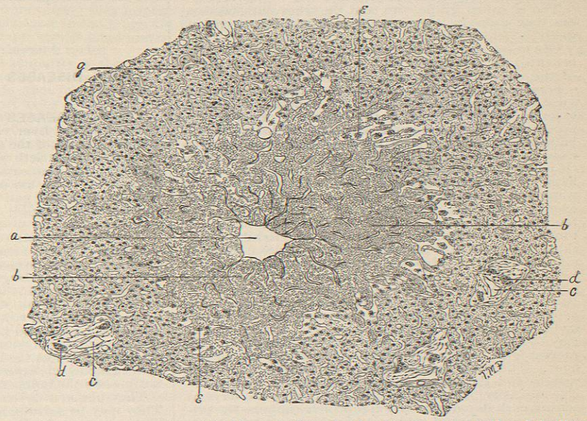

Fig. 3226.—Chronic Congestion of the Liver. (×300 and reduced.) Complete atrophy of the liver cells at the centre of a lobule. *a*, Dilated vena centralis; *b*, dilated capillaries filled with blood; *c*, portal vein surrounded by connective tissue; *d*, gall ducts; *e*, atrophied liver cells; *g*, nearly normal liver tissue. (From Delafield and Prudden's "Handbook of Pathological Anatomy and Histology.")

In more advanced stages the overgrowth of connective tissue is extreme, and leads to great atrophy and pigmentation of the gland elements. Crystals of hæmatin are observed free in the tissues. The liver is small, very firm, dark red. Such is the liver of *cyanotic atrophy*. The same macroscopical appearances are seen in *cyanotic induration*, but the increase of the connective tissue is greater, the cells are more degenerated, and the vessels are more engorged. These advanced changes are located in the region of the hepatic vein, while the interstitial overgrowth in cirrhosis begins in the portal veins.

Treatment.—The first indication to be met is the removal or amelioration of the cause. The methods to be pursued readily suggest themselves to the physician if he possesses a knowledge of the cause and mechanism of the congestion. Thus, the diet must be well selected, and bland, non-stimulating articles are to be given. Rich foods, pastries, condiments, sugars, and starches are to be avoided. Milk, eggs, beef-broth and beef-tea, lean meat, succulent vegetables, and acid drinks are required. Systematic exercise is essential, especially in sedentary subjects. Daily walks, horseback riding, sailing, are all good. Horseback riding is the best. A change of air, especially a residence by the sea, is often beneficial. Bathing, sponging, and douching is of much service.

The acute manifestations are best treated by local depletion, with morphine given internally to relieve the pain. Wet-cups over the liver, and leeches in the same area or around the anus, are to be used. If the pain is not so severe, mustard plasters are stimulating enough, and large poultices may be employed subsequently. Some few authorities assert that the engorgement which ensues is relieved by puncturing the liver through the abdominal walls with a long needle. Though long ago advocated, this practice has never been in vogue, but recently it has attracted attention by the advocacy of Dr. George Hurley.

To assist further in the depletion of the hepatic circulation, purgatives are indicated. The salines are of great value. Phosphate of soda, Rochelle salts, citrate of magnesia, and other salines may be used, preference being given to those first named. If there is much nausea or vomiting it may be relieved by small doses, from one-sixteenth to half a grain, of calomel frequently repeated, taken dry on the tongue. Some practitioners use large doses of the drug at once, and see evidences of liver congestion and torpor in every disease. The resinous cathartics are used by many, podophyllin being the favorite.

If the congestion of the liver be due to cold or checking of the perspiration, remedies to equalize the circulation must be administered. Saline diaphoretics or Dover's powder, a warm bath or a foot-bath, aconite or veratrum to quiet the circulation, and bromides to allay nervous excitement, are indicated. If there is much pain, small doses of Dover's powder are of great service, the ipecac which this preparation contains being of special utility. In malarious districts, large doses of quinine are used. In fact, quinine and calomel form the sheet-anchor combination for many practitioners. High temperature must be combated, and the usual remedies to relieve the cardiac and pulmonary affections that are the primary source of the congestion should be administered.

There are two drugs that have acquired a great reputation in the treatment of congestion of the liver, viz., muriate of ammonia and ipecacuanha. Their use origi-

nated in the East. The muriate of ammonia is given in doses of from gr. x. to gr. xxx. every four or six hours, and it speedily gives relief. Ipecacuanha is given in the same way as in the treatment of dysentery. The patient must be at rest, and the dose of ipecac should be preceded by a small dose of laudanum and by the application of a mustard plaster to the epigastrium. In this manner vomiting is often prevented. From thirty to sixty grains of the drug, every four or six hours, are given.

Alkalies are of great service in congestion of the liver. They must be administered well diluted, and on an empty stomach. It is advantageous to use the alkaline solution hot, and to sip it slowly. The natural mineral waters may be used. Those of Vichy, of Ems, and of Carlsbad, or the Hathorn and Congress waters of this country, are indicated.

The so-called grape-cure, and the whey-cure, were at one time quite in vogue in Germany, and both are, no doubt, of great value.

Of course, the patients who can afford it will be much benefited by a residence at the springs, taking a course of the waters and following the prescribed diet.

Some cases of chronic congestion of the liver refuse to yield to the system of treatment just indicated. In such, external applications over the surface of the liver are necessary. The compound ointment of iodine or the biniodide of mercury is of service. Care must be exercised to secure by the mercury only a slight ptyalism. Of more advantage than either is the nitric-acid pack or bath. An ounce of the acid is added to two gallons of water and cloths saturated with the solution are applied over the liver. If a mixture in this proportion be found too strong it must be further diluted with water. It causes extreme itching and burning or pricking of the skin. The acid bath is strongly recommended. Sir Ronald Martin directs that the bath should be composed of two ounces of strong hydrochloric acid and one of strong nitric in two gallons of water, at a temperature of 98° F. Both feet are to be placed in the bath; the abdomen, the hepatic region, the axilla, and the inner sides of the legs and thighs are to be sponged alternately, or the abdomen may be swathed in flannel saturated with the fluid. The process is to be repeated night and morning for half an hour. The fluid should be kept in wooden or earthen vessels, and the sponges and towels kept in cold water. The quantity of fluid may last for five or six days by adding each day a pint of water which contains the proportionate amount of acid. It should be well heated to raise the temperature of the entire bulk of fluid.

After a course of alkalies, or the use of the means suggested above, the patient is enfeebled and the digestion weak. Tonics are therefore now indicated. The mineral acids, quinine, and nux vomica are the best. With or without gastric disturbance, the chalybeate alkaline waters are of great value. Especially after a course at the "springs," an after-course should be taken at other springs, where a stimulating outdoor life and tonic waters would add to the vigor of the patient. In this period of convalescence the diet must be carefully selected, some mild wines employed, and particular attention directed to the regulation of the bowels by gentle saline laxatives.

ADDENDUM.

The writer has collected some interesting facts concerning hepatic disease in a study of all the cases of liver disease presented to the Pathological Society of Philadelphia, from 1857 to 1881, inclusive. The condition of the liver was recorded 430 times—184 times normal and 246 times diseased. In 10 instances the liver was congested, in cases in which death occurred from accidental causes. The subjoined table indicates the proportionate frequency of occurrence of congestion of the liver, simple and nutmeg, in 246 cases of liver disease. It is of interest to note that in 36 cases of cardiac disease the liver was healthy 5 times, the seat of *nutmeg congestion* 9, and of simple *congestion*, 4 times. Nine times it was fatty; 8 times cirrhotic; 1 the seat of red atrophy (due to congestion). Of 13 instances of nutmeg congestion, 9 attended cardiac disease; 1 chronic pleurisy; 2 carcinoma (heart weak); and 1 chronic diarrhœa. The liver was enlarged 8 times in 13. The spleen was enlarged 6 times; healthy, 2; cirrhotic, 1; not mentioned, 4 times in the 13 cases. The kidneys were congested or cirrhotic in 10 of the 13 cases. Eleven of the cases were males; 6 of them were over forty, 5 between twenty and forty, and 2 under twenty. In no instance were symptoms referable to the liver recorded.

TABLE OF RELATIVE FREQUENCY OF ALTERATIONS OF HEPATIC STRUCTURE. (TOTAL NUMBER OF CASES TWO HUNDRED AND FORTY-SIX.)

Variety.	No. of cases.
Fatty	80
Carcinoma	41
Cirrhotic	38
Congestion	24
Congestion, nutmeg	13
Abscess	13
Tuberculosis	9
Syphilitic gumma	5
Hydatid disease	4
Rupture of liver	3
Hemorrhage into liver	3
Pigment liver	2
Amyloid liver	2
"Diseased" liver	3
Leukæmic, "atrophied," red atrophy, chronic hepatitis, cavernous angioma, myeloid tumor, one each	6

John H. Musser.
Norman B. Gwyn.

[1] Bartels: Left Pleural Effusions.

LIVER, DISEASES OF: INJURIES. See *Abdomen. (Surgical).*

LIVER, DISEASES OF: NEW GROWTHS.—Of the tumors of the liver carcinoma is the most important. Fifty per cent. of the cases are said to occur between the fortieth and sixtieth years. It is found occasionally in children, and women seem to be attacked less frequently than men. An association between carcinoma of the bile passages and gall stones has been noted. Whether there is an etiological relation between the two has not been determined.

MORBID ANATOMY.—There may develop in the liver *adenomata* whose structure differs in certain respects from that of the normal organ. They are usually multiple, and about as large as a pea. They are not always sharply defined and ordinarily project from the cut surface. They consist of liver cells which commonly are arranged in double rows, and, in those cases in which the adenomata are found in otherwise normal livers, are connected with the liver cells in the neighborhood. They thus resemble less the true tumors than localized circumscribed areas of *hypertrophy* or *hyperplasia*.[1] They do not possess the independence that is involved in tumors.

They perhaps develop as a result of fœtal processes. They may be surrounded by a connective-tissue capsule. The vessels of the nodule are connected with the surrounding vessels. If there is no fibrous capsule, the capillaries as well as the rows of cells are continuous with those of the normal tissue. According to Orth,[2] Sabourin maintains that the nodules correspond to secretory lobules, having as a centre an interlobular portal vein and bile duct, and extending to the central veins of the surrounding acini; and if they are larger, they nevertheless correspond to the territory of distribution of a portal canal. He therefore seeks the etiology in changes in these portal or biliary vessels.

These hypertrophic adenomata must be distinguished from the true *tubular adenomata*. These occur as multiple nodules of various sizes that may bring about considerable enlargement of the liver. They may be encapsulated and consist of cylinders arranged in the form of gland tubules, which frequently have a tortuous course and possess distinct lumina that may contain fluid. The origin has been ascribed both to the liver cells and to the bile ducts. They may or may not be associated with cirrhosis. Metaplasia may result in the production of typical carcinoma.

In cirrhosis of the liver when large and especially spherical, tumor-like granules project from the surface, the possibility of a secondary enlargement, as a result of hypertrophy, or even the formation of adenomata must be considered.

The hypertrophic nodules contain enlarged liver cells often in the form of giant cells. They may be transformed partially or entirely into small-cell tubular adenomata. Proliferating rapidly they may push aside the

surrounding tissue or they may infiltrate it. In spite of the cirrhosis considerable enlargement of the organ may be brought about by these grayish-red or grayish-brown nodules. Metastases may result from the adenomata as such or they may first be transformed into true carcinomata. In these cases of *cancer with cirrhosis* the cirrhosis is apparently the primary process. On section of the liver one usually sees apparently multiple tumors, but, according to Ribbert,[6] more careful examination shows that he is dealing with cross sections of tumor cords. These new growths penetrate first into the capillaries, then into the larger vessels, the portal and hepatic veins. The reason for this will be more apparent if one recalls the close relation between the liver cells and the vascular system. In the portal veins the growth may reach the main trunk and extend from there peripherally into other branches. When these are cut transversely they appear like isolated tumor nodules. Metastases in the lungs are very common. Through the lymph vessels large metastases may be formed in the portal lymph nodes.

These tumors possess a certain similarity to the structure of the normal liver. The cells in the neighborhood of the liver cells are arranged in the form of anastomosing cords usually consisting of many layers. In the interstices are the blood-vessels with practically no connective tissue. Bile may be formed. Bile pigment appears in the epithelial cells or the secretion collects in small canals which resemble bile capillaries, but with a different arrangement, and which pass longitudinally among the cords. The same process may occur in the metastases.

Primary carcinomata are rare. They apparently may arise from the liver cells or from the smaller bile ducts. Some are periportal in distribution, the nodules following the periportal connective tissue and being smaller in direct proportion to their distance from the hilum. According to Ziegler,[5] these originate from the bile ducts. *Cystic adenomata* arising from the bile ducts may be in the form of single large cysts or large multilocular cysts. These are rare. Perhaps a portion of the congenital cysts belong in this class. The primary *nodular carcinomata* are often very large and most frequently occur in the right lobe. They may be single or they may appear in company with smaller, apparently secondary tumors.

Secondary carcinomata are much more frequent. Those that originate by direct extension from neighboring organs may have an arrangement similar to that of the nodular primary carcinomata. They may be much larger than the primary tumor and may be thus mistaken for primary tumors. One should therefore search carefully the neighboring organs, especially the gall bladder and the stomach, for a primary tumor. In the case of metastatic carcinomata a considerable number of isolated tumors of fairly uniform size is usually present. They originate from primary tumors in various organs. The primary tumors are most often in the stomach or some other organ in which the branches of the portal vein are distributed; but metastases in the liver may occur apparently in the case of a carcinoma in any part of the body. Microscopically the secondary tumors tend to present the same cellular and general structure as the primary. The structure may thus give a clew to the origin of the tumor.

All carcinomata of the liver tend to undergo central fatty degeneration and atrophy. In the case of the superficial nodules this is expressed by the presence of central umbilication, corresponding to the central atrophy resulting from the resorption of the fatty detritus. By complete fatty degeneration and softening of the central portions there may be formed spaces filled with more or less liquid material.

In the case of some secondary carcinomata, especially those originating from the stomach, one may demonstrate that they have developed apparently from embolic carcinomatous thrombi in the branches of the portal vein. In this way the network of the interlobular branches of the portal vein and the capillaries of the lobules in a considerable territory may be filled and distended with tumor masses. As a result the liver cells become compressed and distorted and finally undergo atrophy associated with an accumulation of brown pigment. At the periphery of the nodular secondary carcinomata the liver cells are often flattened and assume a concentric arrangement in a number of layers.

From the smaller vessels the growth occasionally extends into the hepatic veins, which then become filled with carcinomatous thrombi, that may extend into the vena cava. According to Orth,[2] it is this obstruction of the vessels, apparently, which leads to the cyanotic atrophy so common in the liver tissue between the tumor nodules.

According to Orth[2] the injection experiments of Frerichs have shown that the vessels of the carcinomata may be injected from the hepatic artery, but not from the portal vein. The branches of the hepatic artery become considerably enlarged.

Primary sarcomata and *fibromata* are very rare. *Secondary sarcoma* is more frequent. The metastatic secondary sarcomata, including the melanosarcomata, are identical with those which occur in other regions.

"The most important form is the melanosarcoma, which develops in the liver secondarily to sarcoma of the eye or of the skin. Very rarely melanosarcoma develops primarily in the liver. . . . In this form the liver is greatly enlarged, is either uniformly infiltrated . . . which gives the cut surface the appearance of dark granite, or there are large nodular masses of a deep black or marbled color."[5] The intravascular growth and embolic origin of secondary melanosarcoma are often very apparent. Extensive softening with cyst formation may occur. "There are usually extensive metastases, and in some instances every organ of the body is involved. Nodules of melanosarcoma of the skin may give a clew to the diagnosis."[5]

Rare forms of tumors have been described as myxosarcomata, gliosarcomata, leiomyomata, rhabdomyomata, etc.

Cavernous hæmangiomata are met with more often than the primary sarcomata. Varying greatly in size they may be just visible to the naked eye or as large as an apple. They are characterized by their dark red, sponge-like structure, the spaces being filled with blood. They may be multiple. The framework consists of connective tissue, sometimes containing smooth muscle fibres, or of liver cells. The larger forms are sometimes encapsulated and even pedunculated. Thrombosis followed by organization may lead to fibrous metamorphosis of the structure. Small *cysts* lined by simple or ciliated epithelium are to be regarded as congenital retention cysts resulting from adenomatous proliferation in the bile ducts. Occasionally they occur in large numbers, conspicuously in connection with cystic degeneration of the kidneys. Cysts originating after birth, likewise as a result of partial distention of bile ducts, are characterized, according to Orth,[2] by their firmer fibrous wall, and their contents, which, at least in the earlier stages, consist of bile-like material and later of cholesterin and less often of concretions. Both forms almost always occur immediately beneath the capsule.

Nodules of *adrenal tissue* in the liver have been described.

We may regard *lymphatic leukæmia* and *pseudoleukæmia* as closely analogous to tumor formation. The collections of lymphoid cells filling and dilating the capillaries of the liver and the masses of lymphoid tissue, which may cause considerable enlargement of the organ, may then be regarded as metastatic tumors.

SYMPTOMS.—"It is often impossible to differentiate primary and secondary cancer of the liver unless the primary seat of the disease is evident, as in the case of scirrhus of the breast, or cancer of the rectum, or of a tumor in the stomach, which can be felt. As a rule, cancer of the liver is associated with progressive enlargement."[5] "With the exception of the fibromyomata of the uterus cancer of the liver may constitute the largest tumor met with in the abdomen."[4]

"But there are cases of primary nodular cancer, and in the cancer with cirrhosis the organ may not be en-