mary fatty degeneration, or a general diffused hepatitis, the fatty change being secondary? The arguments mentioned above hold in a measure against the occurrence of diffused hepatitis as a primary affection; moreover, the degenerations in various other organs would point to a general origin of the disease, and not to a local diffused hepatitis. Then, too, the liver is a distensible organ, and its cells would not degenerate from the pressure exerted by the small amount of diffused new growth that is observed in acute yellow atrophy.

Symptoms.—The onset is gradual or sudden. In the former instance symptoms of gastro-duodenal catarrh may continue for two or three weeks, or even longer, before grave symptoms arise. In the latter, acute jaundice, accompanied by marked nervous phenomena, immediately ushers in an attack. The more protracted cases (the first classs) present two varieties of symptoms, viz., prodromal and toxæmic. The symptoms of the prodromal stage are those of acute gastro-duodenal catarrh—headache, nausea, vomiting, loss of appetite, a bitter taste in the mouth, and general malaise. Diarrhœa may also occur. At the end of forty-eight hours or frequently after the lapse of some days, slight icterus appears and gradually increases in intensity. Bamberger states that in very rapidly fatal cases, it may occasionally be completely absent. Fever does not usually attend these symptoms. On the other hand, the pulse and temperature, as in jaundice, are often subnormal. The fæces are colorless, grayish, or parti-colored; often, indeed, they are quite natural. The normal appearance of the fæces in acute atrophy has led to the expression that such stools furnish an unfavorable prognostic element in jaundice, while the presence of clay-colored stools is a favorable sign. The urine contains bile pigment, and does not indicate the serious changes that are to ensue. Enlargement of the liver may occur. While the occurrence of this change is uncertain in acute yellow atrophy, it is a more or less regular happening in poisoning by phosphorus.

The toxæmic stage succeeds these prodromal symptoms more or less suddenly, and after varying intervals of time. The prodromal stage may be very short or quite protracted—even as long as three months. In some cases the toxæmic stage is announced by a convulsion or sudden profound coma. Legg insists on careful observation of the pupil in cases of jaundice, and declares that dilatation of it is the most significant indication of approaching cerebral symptoms. In others the typhoid state gradually supervenes, and is characterized by stupor, low muttering delirium, subsultus tendinum, incontinence of urine and fæces, restlessness, hiccough, motor and sensory paresis, with dilated, often insensible, pupils. A dry and brown, or fissured and glazed, tongue is present, sordes collects in the mouth, and the vomiting of the prodromal stage continues, though the character of the ejecta changes. "Black vomit" now occurs instead of the vomiting of a clear acid or greenish-yellow fluid. The black vomit is due to the presence of blood which has oozed into the stomach. Dark tarry stools indicate its presence in the intestinal tract also. Constipation, however, may be present.

The typhoid state terminates in deep coma, death being preceded by irregular or Cheyne-Stokes breathing, an irregular pulse, involuntary discharges, and sometimes by recurring convulsions. In some instances the typhoid type is not assumed, but active maniacal delirium attends this stage, with or without convulsions; coma or exhaustion closing the scene. Again, there is no delirium, but frequent and prolonged general convulsions occur, with local spasms in the interval and coma vigil. It is believed that the cerebral symptoms are due largely to hemorrhagic extravasation in the brain and its membranes. The blood dyscrasia, however, is probably an important factor. The brain symptoms have been attributed to uræmia, but the theory of such origin is not upheld by clinical facts. Austin Flint, Jr., thought they were due to cholesteræmia, while others attributed them to the presence of bile acids in the blood. Frerichs ascribed them to the presence of leucin and tyrosin in the blood, but experiments do not confirm his notion.

It is in this period that hemorrhages occur from mucous surfaces, as the nares, mouth, pharynx, stomach, and intestines. They also occur in the skin and into the serous membranes. They are due to changes in the walls of the capillaries and small blood-vessels, or, as some assert, to the altered blood. The hemorrhages are passive, and in the skin they are observed as petechiæ or vibices; in the external mucous membranes they appear as sordes or small clots. They may be seen in the conjunctiva. Litten describes hemorrhages in the retina. The constant oozing from the nares may seriously threaten life, while in females an abortion is almost always attended by a most profuse hemorrhage. When hemorrhages into the stomach and intestines have taken place, the discharges present a dark appearance, as indicated above.

The urine undergoes marked change. It is passed involuntarily, and wetting of the person is prevented with difficulty. Albumin, hyaline, epithelial and granular casts are present, from associated tubal nephritis. The urea is much diminished in quantity or entirely absent; phosphates and chlorides are also diminished. The urea is replaced by leucin and tyrosin, sarcolactic acid, peptone, albumoses, indicating less advanced or perverted tissue metamorphosis. The presence of leucin and tyrosin is not pathognomonic, and they may be absent (Murchison); there is a marked increase in the output of nitrogen owing to destruction of cells of liver and other tissues. The urine is usually acid, dark in color, and contains bile pigment. Contrary to their course in the prodromal stage, the pulse and temperature are increased in the toxæmic period. This is especially true of the pulse. It is increased in frequency, often excessively rapid, and not uncommonly irregular. The temperature range varies. In some cases there is no rise; in others the ascent does not occur until the last day or two of life, and may attain the highest point after death. My friend, Dr. H. M. Wetherill, of the Pennsylvania Hospital for the Insane, of Philadelphia, kindly permitted me to study the histological appearance of the various organs removed from a patient of his, who died of acute yellow atrophy of the liver. The case presented many interesting features, none more so than the temperature range. The patient, fifty-one years of age, was admitted into the insane hospital, for acute mania with delirium, on March 12th, 1885. She did not improve, and on April 9th had a "bilious attack," followed in three days by jaundice and urticaria. After the jaundice the maniacal delirium gave way to a low, muttering form. The icterus appeared on the trunk and arms first, and then extended to the entire surface. She lived nineteen days, and during the course of the disease had diarrhœa with pale, loose stools, and hemorrhages from all the mucous surfaces, petechiæ and vibices. The liver dulness decreased in area, and the splenic increased from day to day. The urine became albuminous, contained hyaline and granular casts, blood and bile pigment, leucin, and tyrosin. The urea became diminished in amount. The temperature range is indicated below. During the three days preceding the jaundice, when the patient was "bilious," a rise of temperature occurred. It then fell to normal and remained low until four days before death, when a continuous ascent began, reaching the acme, 105⅘° F., thirty five minutes after death.

The pulse did not increase beyond 90. During the first, second, and third days of the illness it ranged from 82 to 90; from the fourth to the eighth day, inclusive, 76 to 82; and from the ninth to the sixteenth day the average daily range was 66. After the latter day it increased daily to the last day, when it was 86 (highest).

Along with the occurrence of cerebral symptoms and hemorrhages, changes take place in the liver and spleen. On physical examination the liver dulness is observed to decrease from day to day, and even may disappear anteriorly. One must be careful to remember that the flatulent distention of the intestines may cause apparent lessening in the area of hepatic dulness. It must also not be forgotten that the liver may be enlarged. The spleen, in a certain proportion of cases, is enlarged. Legg states that in one-third of the cases an enlargement is found at the autopsy. With the diminution in the size of the liver pain in the hepatic region is experienced. This may be extreme, entailing a great deal of suffering. It is most frequently seated in the epigastrium, and, as the changes are most marked in the left lobe of the liver, may be due to the atrophy. It is certainly a well-recognized fact that an agonizing pain attends acute yellow atrophy of the liver. Although attended by vomiting, the pain does not appear to have any relation to it. Marked tenderness in the epigastrium is associated with it in many instances.

Day of illness.	Degrees, Fahrenheit.		Day of illness.	Degrees, Fahrenheit.	
	A. M.	P. M.		A. M.	P. M.
First	99.0	101.0	Eleventh	96.8	97.0
Second	99.4	100.2	Twelfth	97.2	97.0
Third	100.4	100.0	Thirteenth	96.6	96.8
Fourth	99.4	98.4	Fourteenth	97.0	97.2
Fifth	98.8	98.0	Fifteenth	96.8	96.8
Sixth	98.0	99.2	Sixteenth	97.6	98.4
Seventh	99.0	98.4	Seventeenth	99.0	99.4
Eighth	97.6	97.6	Eighteenth	99.4	100.2
Ninth	97.0	97.4	Nineteenth	101.6	101.8
Tenth	97.4	97.6	Twentieth	104.6	104.8*

* At 5:20 P.M. death.

The course of acute yellow atrophy of the liver varies. It is sometimes extremely rapid. Fifty per cent. of cases are fatal between the fifth and the fourteenth days; before the fifth day death is rare and is usually to be seen only in pregnant cases (Thierfelder). Cases of death have been reported to occur twenty-four hours after the first seizure. The toxic stage is much shorter than the prodromal; the latter usually continues for from one to eight weeks. The disease runs a more rapid course in pregnant women.

The prognosis is unfavorable, but not necessarily fatal. Cases of recovery have been recorded. In Wilks' celebrated case the patient recovered, and died two months afterward of a relapse. The case of Marchand (*vide supra*) shows that true regeneration of liver substance—and therefore recovery—is a possibility.

Diagnosis.—The presence of the cardinal features of a case of acute yellow atrophy of the liver renders the diagnosis easy. The prodromal symptoms, the jaundice, the character of the urine, the nervous phenomena, the changes in the liver and spleen, and the hemorrhagic symptoms are to be kept in mind.

Care must be taken to exclude those forms of acute yellow atrophy which are due to phosphorus poisoning or to yellow fever, by attention to the antecedent circumstances and the history of the case.

Treatment.—The treatment can be stated in a few words. Some cures have been reported, and hence it is well to seek some methods of treatment. The indications that we can thus far discern are to relieve or cure the catarrhal symptoms as quickly as possible, and to allay the malignant symptoms of the second stage. The former is the treatment of gastro-duodenal catarrh or catarrhal jaundice. General principles guide us in the treatment of the latter.

John H. Musser.
Norman B. Gwyn.

[1] Revue de Médecine, 1886, vi., No. 4, 334-342.

LIVER, DISEASES OF: ANIMAL PARASITES.—The most important of the animal parasites of the liver is the echinococcus. Several others are occasionally found, but so seldom as not to justify the separate consideration of them. The round worm, for instance, migrates into the bile ducts and reaches the smaller tubes. The cysticercus cellulosæ is rarely found, and the same is true of the psorospermiæ. The pentastoma denticulatum and the so-called liver flukes, distoma hepaticum and distoma lanceolatum, are a little more frequent. The distoma hematobium is very rare except in North Africa. Other forms have been described under the names of distoma sinense and distoma conjunctum, but they may be regarded as curiosities.

The Echinococcus, or hydatid cyst, is a cystic disease of the liver due to the presence of the echinococcus polymorphus, the larval form of the tænia echinococcus; it causes a gradual enlargement and alteration of the form of the liver, and various functional disturbances both in it and in adjacent organs.

History.—In the works of Hippocrates,[1] Galen,[2] Aretæus,[3] and other ancient writers, references are made to large cysts of the liver containing water and, in some instances, numerous vesicles, which were undoubtedly hydatid in character; and in the literature of the sixteenth and seventeenth centuries we find many unequivocal references to the disease. The first accurate descriptions of the cyst are to be found in the "Sepulchretum" of Bonetus.[4] The parasitic nature of the disease was not known, however, until Pallas,[5] in 1766, discovered the parasite and showed its close relationship to the tapeworm. Götze, in 1782, determined that the scolices were the heads of embryonic tæniæ, and Bremser,[6] in 1821, described the disease as it occurs in man. The term echinococcus was introduced by Rudolphi, in 1801. The exact relationship of the echinococcus to the parent tapeworm, and the manner in which it invades the human body, remained hypothetical until Küchenmeister,[7] von Siebold,[8] and Leuckart[9] showed by direct experimentation that the hydatid is the larval state of the tænia echinococcus, which infests the alimentary canal of certain lower animals. The literature of the subject has been greatly added to by Davaine, Budd, Andral, Frerichs, Murchison, Heller, and Madelung.

Etiology.—Echinococcus of the liver is met with more frequently in Iceland than in any other part of the world. It has been estimated that every seventh person in that country harbors the parasite. Jonassen,[10] says, however, that this estimate is too high. No part of the world is exempt from the disease, but the statistics of its frequency in many parts are meagre. In the United States, and in most parts of Europe, it is comparatively rare. Lyon[11] was able to collect only 241 cases of hydatid disease in North America up to July 1st, 1901. Of these cases, 175 were definitely located in the liver.

The disease appears alike in both sexes, and at all ages, except during infancy. The wolf, fox, dog, and sheep are the most frequent hosts of the parasite. In Iceland it is generally attributed to the intimate relations which exist between the people and their dogs, the ova being conveyed no doubt in many instances by the tongue of the dog to the lips of his master; or they may be conveyed through contaminated drinking-water. In other countries the disease is probably more frequently acquired from infected meat or vegetables. Scolex-bearing cysts have been discovered in the livers and other tissues of the ox, sheep, hog, goat, deer, horse, squirrel, and many other animals. Richardson[12] attributes the frequent occurrence of the disease among the shepherds of Victoria, South Australia, to the eating of mutton, in the belief that the sheep have become infected from the shepherd dogs. Thomas[13] found at least forty per cent. of unregistered dogs, in various parts of Australia, infested. Heller[14] has suggested the possibility of autoinfection, on the supposition that the tænia may gain lodgment in the alimentary canal of man, although its presence there has not been demonstrated. Richardson's view is especially emphasized by Madelung in the Report of the Mecklenburg Physicians.[15] He shows that the dogs of Mecklenburg, where the disease is comparatively frequent, are not more numerous, do not come into closer contact with the people, and are not more generally infested by the tænia than in South Germany, where the disease is rarely encountered. Sheep, on the other hand, are more numerous than the people.

Pathology.—The tænia echinococcus is about 4 or 5 mm. (⅛ inch) in length, and consists of a head and three

segments (see p. 788 of vol. ii.). The head supports a rostellum bearing from twenty-eight to fifty-two rather blunt hooklets, arranged in two rows, as first recognized by Livois. The last segment is larger than the others combined, is endowed with both male and female generative organs, and, according to Leuckart, has the power to accomplish its own fecundation. This view is not, however, accepted by all investigators.

After fecundation has occurred the segment becomes filled with ova, estimated at about five thousand in number, each containing an embryo. As soon as the ova have become mature, the segment containing them becomes detached from the anterior portion of the worm, and is discharged from the intestinal canal of its host, either in its entirety or after it has been ruptured. Birth per vaginam is rendered improbable by the relatively small size of this channel. The ova reach the stomach singly, or in numbers, in the manner that has been described. Here the dense, sometimes calcified capsule is dissolved by the gastric juice, and the embryo is liberated. This is an oval, globular body, about three times as large as a human blood corpuscle, and armed at one extremity with six minute hooklets. From its point of liberation in the stomach or small intestine the echinococcus reaches the liver, either by boring directly through the intervening tissue, by penetrating a branch of the portal vein and being carried to the organ by the blood current, or by passing up the bile duct. Its migration, unlike that of the cysticercus or the trichina, is unattended by symptoms. Fortunately, the greater number of the parasites are destroyed before leaving the alimentary canal, and the cyst is usually single, but several cysts have occasionally been found in proximity. The cyst is usually located near one of the surfaces of the right lobe, but may be found in any part of the organ. When the embryo reaches the liver, it loses its hooks and acquires a vesicular form by the growth, from its caudal extremity, of a serous membrane which ultimately envelops it and becomes distended with fluid. It thus becomes the echinococcus cyst. The process by which this state is reached is in no sense pathological, but constitutes a period in the biological history of the parasite. The growth of the cyst is slow, years sometimes elapsing before it becomes large enough to create appreciable disturbances. When discovered, as in autopsy, it varies in size from that of a millet seed to that of the human head. The latter limit is rarely exceeded, but Leuckart records a case in which the cyst and its contents weighed thirty pounds.

The cyst is sometimes very delicate and of a gray color, sometimes thick and translucent, but usually it is only a little more dense than coagulated egg albumen. It is lamellated, consisting of a variable number of concentric layers, composed of a substance resembling chitin.[16] The inner layer, known as the parenchymatous or germinal membrane, is granular, and, according to Naunyn,[17] is provided on its inner surface with rapidly vibrating cilia. After a variable time, usually from two to five months, little mounds appear on the surface of the germinal layer, each of which has a small depression at its apex, which later becomes a vacuole-like cavity. These cavities then enlarge, and are known as daughter cysts. The process of budding may occur in the daughter cysts, giving rise to granddaughter cysts. Either generation may develop either endogenously or exogenously. The former method of growth is much the more common in man. The number of cysts formed varies from a few to several thousand. From the surface of the daughter or granddaughter cysts scolices, the heads of embryonic tæniæ, develop, either singly or in as great number as nine or more (Heller). These appear as conical projections, each having on its free extremity a rostellum, armed with a double row of hooklets and four suckers. The opposite extremity becomes constricted into a narrow pedicle, which later divides, liberating the scolex, thenceforth to float freely about in the interior of the capsule. The scolices and the brood capsules are endowed with the power of contraction, so that the heads may be protruded from the surface of the capsule or withdrawn; and after the head has become detached, it has also the ability to withdraw its anterior portion, with the rostellum and suckers, into the larger posterior part. Throughout the parenchyma of the scolex more or less numerous oval or spherical calcareous bodies are generally observed. Very rarely scolices develop directly from the germinal layer of the mother cyst. Küchenmeister[18] attributes this phenomenon to the invasion of what he designates the echinococcus scoliciparíens, having from twenty-eight to thirty-six hooklets; while he designates the parasite which produces daughter vesicles the echinococcus altriciparíens. This he describes as having from forty-six to fifty-two hooklets, and as sometimes present in the small intestine of man. His view has not, however, been generally accepted. Daughter cysts may develop also within the scolex, which then gradually becomes converted into a capsule.

In another variety of the disease the cysts remain sterile, no scolices being formed. These were first described by Laënnec, and were by him designated acephalocysts.

The multilocular echinococcus is a form of cyst which is encountered once in about one hundred and eighty cases of the disease. The sac, sometimes of very large size, is surrounded by an exceedingly dense fibrous capsule, firmly united to the surrounding tissue; is subdivided into numerous small cavities, and is filled with a thick, gelatinous, or colloid material, suggestive of cancer. Its real structure was demonstrated by Virchow.[19]

The echinococcus vesicles float in a limpid, usually clear, fluid, of neutral reaction, with a specific gravity of from 1.006 to 1.015. Sometimes, however, the fluid has a yellow or a pale green tint, and is slightly alkaline from admixture of bile, or is opalescent from the presence of fatty matter and other débris; or it may be a pale red from admixture of blood. Chemical analysis shows the presence of from 0.50 to 0.76 per cent. of sodium chloride, and small quantities of the earthy compounds of succinic acid, inosite, and grape sugar. Albumen is never present, except from the admixture of blood. Urea, creatin, and hæmatoidin[20] have occasionally been found, the last being considered peculiar to hepatic echinococci. Substances resembling toxalbumins and ptomaïns have been found by Maurton, Viron, and others. Cholesterin is found in cysts whose contents have undergone fatty degeneration.

As a result of the irritation produced by the echinococcus vesicle in the liver tissue, a firm fibrous wall is formed around it by a hyperplasia of the fibrous tissue of the organ, which extends also to the interlobular tissue for a variable distance around the cyst. This capsule is supplied with blood by vessels arising from the branches of the portal vein and hepatic artery. Old capsules frequently become more or less calcified (see Fig. 3221). As the cyst enlarges the parenchyma of the liver is, to a variable extent, destroyed.

The echinococcus sometimes dies, either spontaneously or as a result of accidents. Its growth is then arrested, and the cyst remains as a foreign body in the substance of the liver, its contents frequently undergoing retrograde changes and absorption, or calcification; or suppuration occurs, and the pus finds exit externally or through a neighboring viscus.

Symptoms, Course, and Termination.—Echinococci frequently exist in the liver for years without occasioning symptoms. The greater number of echinococci have been discovered post mortem. Only 7 out of 13 cysts, discovered in autopsy at Rostock, had been diagnosticated during life (Thierfelder); of 22 discovered at the Berlin Charité, 13 had been diagnosticated; and Madelung estimates that, outside of hospitals, only one-third of the cases are recognized during life.

In the majority of cases the first symptom to attract attention is the formation of a tumor, an apparent enlargement of the liver. The direction of greatest protrusion depends chiefly on the location of the tumor. If situated in the anterior portion of the right lobe, the hypogastrium is rendered prominent; if in the upper

portion of the lobe, it may push upward into the thorax, elevating the diaphragm, impeding respiration, often displacing the heart upward and to the left, and so compressing the right lung that the entire right side of the thorax yields a dull tone on percussion. It may also obliterate the intercostal furrows and even cause undue prominence of the chest wall. If situated in the lower portion of the lobe, the cyst may extend down to the pelvis; when it is in the left lobe, the epigastrium becomes prominent and the spleen may be displaced.

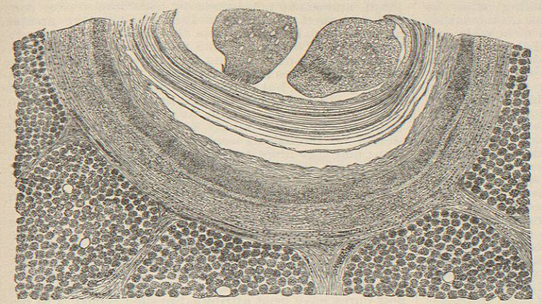

Fig. 3221.—Section of Echinococcus Cyst containing Two Scolices, in One of which the Rostellum is Protruded, in the Other Withdrawn. The firm capsule surrounding the vesicle shows a moderate degree of calcification. × 400. (From drawing of Dr. James M. French.)

If accessible to palpation, the cyst gives the impression of a smooth globular tumor, fluctuating as a rule, although not always, and frequently somewhat irregular in outline. If the tumor be of large size and contain many daughter vesicles, and if the abdominal wall be not too thick, we may, by grasping the tumor and exerting moderate compression with one hand, and striking a quick and pretty forcible blow upon it with the other, elicit a peculiar vibratory sensation—the hydatid purring of Briançon—which has been compared to the trembling of a bowl of jelly. By many this sign is considered pathognomonic, but it is absent in about half the cases, and has been elicited also in ovarian cysts and ascites.

Pain is an infrequent symptom. Bamberger states that some cases, up to a late stage, present no symptoms referable to the liver, while others are attended with more or less intense, undefined, pressing, or lancinating pains.

The pressure of a large echinococcus cyst upon adjacent organs produces symptoms which belong alike to all abdominal tumors of large size. Prominent among these are dyspnœa with cough, cardiac palpitation, indigestion, vomiting, constipation, and later ascites, œdema, and occasionally varicose veins. Unless the tumor be located in the immediate proximity of the portal vein or bile ducts, these vessels are not generally interfered with until late, when the growth has become large; jaundice is not, therefore, often present. There is usually no fever unless suppuration has occurred, and the nutrition of the individual, as a rule, is maintained. Suppuration of the cyst is announced by the development of pain and tenderness, and an elevation of temperature.

Rupture of the cyst occasions a new train of clinical manifestations, varied according to the direction in which the perforation occurs. Spontaneous recovery has followed evacuation through the abdominal wall, into the stomach or intestine, into the gall duct, the ureter, or the vagina. Rupture through the integument is generally preceded by "pointing"; rupture into the hollow viscera is followed by sudden intense local pain and the subsequent evacuation of a large quantity of fluid containing numerous vesicles. Rupture into the intestine or stomach produces a watery diarrhœa, and the tumor frequently becomes tympanitic from the admission of air. The entrance of bile into the sac causes death of the echinococcus and favors recovery. Rupture through the diaphragm into the pleural cavity causes severe pain and urgent dyspnœa. If death does not result promptly from shock or from violent pleuritic inflammation, the fluid may ultimately perforate a bronchial tube and thus find exit. The latter accident may prove fatal, either suddenly from strangulation, or in the course of weeks or months from prolonged suppuration, gangrene of the lung, and final exhaustion; or it may lead to recovery. It is signalized by a sudden expectoration of fluid, generally purulent or bloody in character, and containing echinococcus vesicles, entire or in fragments, and is commonly followed by a pneumothorax.

Perforation of the pericardium proves fatal immediately, or from acute pericarditis. After rupture into the peritoneal cavity death results from acute, generally violent, peritonitis.

In a few cases the inferior vena cava has been perforated, giving entrance to the vesicles and leading to instant death from embolism of the pulmonary artery. Perforation of the arterial system leads to embolism and its consequences.

The multilocular echinococcus is usually firm, rarely fluctuates, and is generally sensitive on pressure. It is frequently accompanied by enlargement of the spleen and ascites. Gastric disturbances are also more frequent than in the unilocular cyst, and jaundice is usually present. Gastric and intestinal hemorrhages and an effusion of blood into the subcutaneous tissues have occurred.

Diagnosis.—Echinococcus of the liver is to be differentiated from cancer, amyloid infiltration, syphilis, cirrhosis, and abscess of the liver, and occasionally from hydro- or pyothorax, from cystic disease of the retroperitoneal lymph glands, from enlargement of the gall bladder, and from aneurism of the abdominal aorta. In the early history of the disease its differentiation from other tumors of the liver or its neighborhood is often very difficult. A tumor with the history of slow, painless growth, with elasticity, fluctuation, and the peculiar hydatid fremitus, is in all probability an echinococcus.

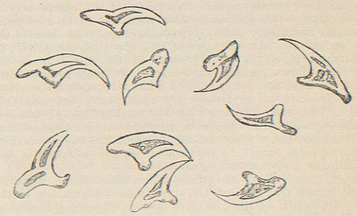

Fig. 3222.—Hooklets as found in Echinococcus Fluid. × about 750.

In every case, however, sufficient fluid should be withdrawn, through a small needle, to permit microscopical and chemical investigation. If the fluid be clear, limpid, free from albumin, of low specific gravity, and if it contain inosite, and succinic acid, there can be little doubt of

the hydatid character of the tumor. By the discovery of hooklets (Fig. 3222), and fragments of lamellated membrane, the diagnosis is established. But, unfortunately, many of these features are generally absent.

From cancer, the echinococcus may generally be distinguished by the absence of the lancinating pains, the tenderness, and the hard nodular surface of the latter, which is also frequently associated with a cachexia and cancer of other organs; from the medullary carcinoma, which has undergone softening, the distinction is often very difficult. The multilocular echinococcus can rarely be differentiated during life. From amyloid infiltration it is distinguished by the absence of the hardness, as well as of the history of prolonged suppuration and the wax-like hue of the skin that is characteristic of this affection. Syphilis must be excluded by the absence of evidences of present or past specific disease of the skin, mucous membranes, bones, and genitalia. In cirrhosis there are usually ascites and enlargement of the spleen, with the history of alcoholic excess and consequent gastric disturbances, which do not belong to echinococcus. Abscess of the liver usually follows an acute inflammation of the abdominal or pelvic organs, and is associated with acute symptoms, chills, fever, pain, and tenderness. An echinococcus which has elevated the diaphragm is distinguished from hydro- or pneumothorax by the course of the upper boundary of percussion dulness, which, in the echinococcus, is highest in the axillary line. Further, a pleuritic exudation is generally preceded by an acute attack of pain and dyspnœa, with fever and cough, and is frequently traceable to cardiac or renal disease, or to pulmonary tuberculosis; whereas echinococcus requires months, or even years, for its development, and by it the heart is pushed upward as well as to the left.

Cystic enlargement of the retroperitoneal lymph glands has rarely to be excluded; but in a case reported by Ransohoff[21] the liver was so compressed and its boundaries were so enlarged by a smooth, globular, fluctuating tumor that appeared to be a part of the liver, that the echinococcus could not be excluded until an exploratory incision had been made.

Enlargement of the gall bladder can be confounded only with a pendulous echinococcus cyst. Chemical and microscopic examination of the fluid is sufficient to establish the diagnosis.

Aortic aneurism can generally be excluded by its position and outline, as well as by its pulsation. Pean observed a case, however, in which an echinococcus cyst, as large as a child's head, rested directly upon the aorta in such a manner as to transmit a strong impulse.

An important point which arises in the differentiation of the echinococcus cyst, especially with reference to the adoption of operative procedures, is to determine whether the cyst is simple or compound, *i.e.*, whether the daughter vesicles have developed endogenously or exogenously. Occasionally the presence of two or more large and distinctly defined prominences will denote the presence of the latter variety, and the diagnosis will be established if, after withdrawal of the fluid from one cyst, the others remain distended. It may also be suspected in case the quantity of fluid which can be withdrawn from a large tumor by aspiration is relatively small. The case reported by Whittaker[22] is of interest in this connection. Here a large cyst protruded and was incised, the writer being present at the operation; the numerous smaller cysts found at the autopsy a few weeks later were not recognizable.

Prognosis.—The prognosis of echinococcus depends largely upon the size of the cyst and its location in the liver. If death of the parasite occurs, recovery is almost certain. If the cyst ruptures into a serous cavity, the prognosis is exceedingly grave; if into the vena cava, it is fatal; if into other vessels, it is usually so. Rupture into the intestinal canal, directly or through the bile duct, is, next to perforation of the abdominal wall, the most favorable route of exit. The prognosis of a living echinococcus in the parenchyma of the liver should always be considered sufficiently grave to warrant the adoption of measures for its destruction as soon as practicable after its discovery.

Prophylaxis.—This consists in the prevention of the contamination of food and drink by the ova of the tænia echinococcus. In Europe and America, where the domestic animals do not necessarily come into so close contact with their masters as in Iceland, and where greater attention is paid to cleanliness, the disease may be largely controlled. To this end, the public should be educated to the necessity of excluding dogs from those localities in which their fæces may contaminate the food and drink not only of man, but of sheep and cattle. Neither should dogs be allowed to eat the refuse or viscera of slaughtered animals, or, in fact, any uncooked flesh. All meats should be thoroughly inspected for the cysts, and should be thoroughly cooked before being eaten by man; and vegetables desired for raw consumption, as lettuce, celery, cabbage, and cresses, should be thoroughly cleansed and inspected before being placed on the table. Boiling is sufficient to destroy the vitality of the ova in either animal or vegetable food.

Medicinal Treatment.—The use of medicines in the treatment of this disease has been abandoned, for it is not believed that any drug can penetrate the dense capsule. When, however, we reflect that death of the echinococcus has occurred spontaneously, or as a result of the most trivial accident, we can readily comprehend how cures have been attributed to such remedies as potassium iodide, mercury, and various anthelmintic remedies or even to the application of salt solution.

Surgical Treatment.—Surgical measures are indicated, as a rule, as soon as the character of the growth has been recognized, for the chances of recovery are directly proportionate to the early destruction of the echinococcus. The methods employed are: (*a*) simple acupuncture; (*b*) puncture and aspiration; (*c*) electrolysis; and (*d*) free incision with subsequent drainage and irrigation.

Acupuncture has proved successful in a few cases. Aspiration has proved successful when only a small quantity of the fluid has been withdrawn, as well as after complete evacuation of the cyst. Its chief element of danger is the certainty with which suppuration follows its repetition. After the withdrawal of fluid by aspiration various substances have been injected for the purpose of destroying the parasite, but the practice is strongly condemned.

Electrolysis was originally proposed by Michon[23] and Althaus,[24] but was first extensively employed by Fagge and Durham,[25] who report eight successive recoveries from its use. The method consists in introducing into the most prominent part of the tumor two fine, gilded, steel needles, two inches apart, and both connected with the negative pole of a ten-cell battery capable of decomposing a saline solution. The positive pole—a moistened sponge electrode—is then placed on the surface and moved about over the hepatic region. The current is permitted to pass for about ten minutes. In the cases to which reference has been made, the immediate effect on the tumor was often a slight increase of size, owing to the disengagement of hydrogen gas. Constitutional disturbances were also produced in all but one case, the temperature ranging between 100° and 103° F. for from two to nineteen days. Later, however, the tumor diminished in size and finally disappeared, its absorption requiring from a few weeks to several months.

Incision of the echinococcus cyst may be performed by a continuous operation or at two sittings, inflammatory adhesion of the peritoneal surfaces being waited for in the interim. The method now generally adopted is that of first sewing the wall of the cyst to the abdominal wall and cutting between the sutures. The steps of the operation are the same as those adopted in opening a hepatic abscess and the dangers are no greater. Lihotzky[26] has reported twenty-five operations, four of which were under his own observation, with only four deaths, and three of these not attributable to the operation. Lihotzky's operations were all made by the two-sittings method, and

were all successful. Neisser[27] estimates the mortality at about one-third. Even if the latter estimate be correct, the operation is certainly indicated, at least after milder measures have failed.

Cases are sometimes encountered which require special methods of treatment, as when the echinococcus is attached to the liver by a pedicle, and has to be removed *en masse*.

The treatment of the multilocular echinococcus is wholly symptomatic. *James M. French.*

[1] Hippocrates: Aphorismi, vii., 55.
[2] Galen: Comment. in Aphorismos, vii., 54.
[3] Aretæus: De Causis et Sign. Diuturn. Morb., lib. ii.
[4] Bonetus: Sepulchretum, lib. iii., Sec. 21.
[5] Pallas: De Infestivis vivent. intra vivent., Diss. Inaug.
[6] Bremser: Journ. Complém., Paris, tom. xi., p. 282 (Davaine, p. 360).
[7] Küchenmeister: Pruger Vierteljahrsschrift, 1852.
[8] Von Siebold: Zeitschr. für Wissensch., 1853–54.
[9] Leuckart: Die Blasenbandwürmer u. ihre Entwickelung, 1856.
[10] Jonassen: Quoted by Madelung, *op. cit.*, p. 18.
[11] Lyon: Amer. Journ. of the Med. Sciences, vol. cxxiii., 1902, p. 154.
[12] Richardson: Edinburgh Med. Journal, 1867, p. 525.
[13] Thomas: Hydatid Diseases, with Spec. Ref. to Prevalence in Australia, Adelaide, 1884.
[14] Heller: Ziemssen, Cyclop. of Pract. of Med., Amer. edit., vol. iii., p. 579.
[15] Madelung: Beiträge Mecklenburg. Aerzte zur Lehre von der Echinococ.-Krankh., Stuttgart, 1885.
[16] Lücke: Virchow's Archiv, vol. ix., p. 189, 1860.
[17] Naunyn: Archiv für Anat., Physiol., etc., 1862, p. 615.
[18] Küchenmeister: Die in u. an d. Körper d. lebend. Mensch. vorkommenden Parasiten, vol. i.
[19] Virchow: Archiv für Anat., vol. xi., p. 80.
[20] Davaine: Traité des Entozoaires et d. Malad. Vermin., 2ème edit., p. 280, Paris, 1877.
[21] Ransohoff: Cincinnati Lancet and Clinic, vol. xi., p. 451, 1883.
[22] Whittaker: Medical News, Philadelphia, vol. xlix., p. 579, 1886.
[23] Michon: Cited by Davaine, *op. cit.*, p. 596.
[24] Althaus: On the Electrolytic Treatment of Tumors, etc., London, 1867.
[25] Medico-Chirurg. Transact., 1871, p. 1.
[26] Lihotzky: Deutsche Zeitschr. für Chirurg., Bd. xxiii., 1885, p. 114.
[27] Neisser: Die Echinokokkenkrankheit, Berlin, 1877 (quoted by Lihotzky).

LIVER, DISEASES OF: CIRRHOSIS. (I. PATHOLOGICAL).—Definition and Classification.—Cirrhosis of the liver is a chronic disease caused by a diffuse overgrowth of the connective-tissue framework of the organ. Proliferation of the stroma may take place around abscesses, echinococcus cysts, gummata, foreign bodies, etc., in the process of their encapsulation, but the term cirrhosis is not applicable to such localized conditions.

Although all parts of the liver are affected in cirrhosis, the alterations may be more marked in some regions than in others.

Until recently it has been understood that the increased tissue must attain some degree of maturity before a cirrhosis could be said to exist; however, the direction of investigation, both clinical and pathological, has been toward a more thorough comprehension of the early symptoms and changes, and at present discussions, under the caption of cirrhosis, of conditions unaccompanied by any considerable overgrowth of the stroma, are by no means rare.

Without doubt the tendency of work in this direction will not only add to our knowledge of the etiology of cirrhosis, but will also extend the present limitation of the term cirrhosis, so that precicatricial stages in its development will, in the course of time, be included and form essential parts of the disease.

Numerous descriptions of cirrhotic livers occur in the writings of the earliest medical works under such names as hepar durum, obstructio hepatis, scirrhus, etc. According to Frerichs, Morgagni especially had clear notions of cirrhosis. There is no doubt that many cases of carcinoma and other tumors were included in the same category with cirrhosis by these ancient writers. That Laënnec, to whom the name cirrhosis is generally accredited, mistook the yellow projections on the surface for new formations, is a fact frequently referred to.

The subject of the classification of the cirrhoses is a favorite one, and at meetings of physicians prolonged endeavors to attain a unanimity of opinion are not uncommon. Many facts bear witness that the last and final contribution to this topic has not as yet been written. The co-ordination between anatomic changes and clinical symptoms has not been completed; the conception of cirrhosis, as before stated, is constantly being extended to cover earlier and other conditions than it previously applied to; within recent years new forms of cirrhosis that are distinct entities, at least from the standpoints of morbid anatomy and etiology, have obtained recognition; and lastly, it should be remembered that the capacity for regeneration possessed by the liver is quite remarkable. In the light, therefore, of what has already happened it cannot be considered surprising that other combinations of regeneration and inflammation than those at present recognized in the liver should in the course of time procure the dignity of separate consideration.

There is generally found in most classifications a group of so-called "mixed cirrhoses" which includes cases not readily disposed of under more exact names. The difficulty of dispensing with this group and its constant recurrence even in the writings of the best authorities furnish sufficient proof of both the inexactness of present classifications and of the variety of processes that may contribute to produce a cirrhosis of the liver.

The ideal and scientific classification—the etiologic one—has not gained much prevalence on account of difficulties in adjusting it to groups of symptoms and morbid changes; likewise the subdivisions of pathologists seldom meet with the entire approval of clinicians. Thus it has happened that the forms of cirrhosis usually mentioned are derived from two perspectives: those of etiology and of morbid anatomy.

Atrophic and hypertrophic cirrhosis are the generally accepted forms; fatty cirrhosis, pigmentary cirrhosis, Glissonian cirrhosis, a cirrhosis due to calculi and long-standing biliary obstruction, a cirrhosis caused by passive hyperæmia and the cirrhoses of tuberculosis, malaria, and syphilis have also been described. They have, however, fewer clinical and anatomic features than the two first-mentioned forms, which allow of their ready recognition either at the bedside or in the necropsy room. And yet, although their permanent taxonomic position is dubious, certain recent contributions indicate that some of them are entitled to more recognition than they have heretofore received.

Etiology.—The factors generally recognized as causative are alcohol, arteriosclerosis, syphilis, tuberculosis, malaria, passive hyperæmia, pigmentation with intrinsic or extraneous pigments, biliary obstruction, and the acute infectious diseases.

Of all factors alcohol, in the form of the stronger spirituous beverages and when used daily for long periods, is entitled to the most important place. Although the experimental production of cirrhosis of the liver in animals with alcohol by various observers (Straus and Blocq, Lafitte, Sabourin, Afanassiew, and others) has not been attended with uniform results, they have on the whole confirmed clinical observations. It is extremely doubtful if any animal experiments will equal in value the verifying evidence furnished by the numerous cases recorded of atrophic cirrhosis in children and even infants, to whom misguided parents have given daily drinks of beer or stronger liquors. The exact manner in which alcohol produces cirrhosis of the liver is not known. It has been supposed by some that alcohol alone will not suffice, but that, following its ingestion, substances are formed in the alimentary canal which so act on the liver that cirrhosis is brought about.

The frequent observation of a cirrhosis of the liver in animals, accidentally encountered in the course of routine laboratory experimentation, is in direct accord with the fact that it has been purposely produced in animals by a great variety of substances; in fact, "there are few diseases," Siegenbeek van Heukelom wrote in 1896, "that are in process of production in such divers ways by investigators."

Among the substances used are arsenic, phosphorus, silver, lead, antimony and other metallic poisons, chloroform, paraffin, butyric, valerianic, acetic and other organic acids; croton oil and carbolic acid. The toxins of