

The average weight of the liver of a new-born child is 100 grammes, but that of a child one and three-quarter years old is 250 grammes, so that any actual decrease of the structure of this organ is altogether out of the question.

The physical examination of the liver in children is conducted very much upon the same principle as in the adult. It is first percussed in the axillary line (a perpendicular line drawn from the axillary cavity downward), then in the mammillary line (a perpendicular one drawn from the nipple downward), and finally in the sternal line (a perpendicular one from the sternum outward). By the first two lines, the diameter of the right lobe, by the last, that of the left lobe, of the liver is ascertained.

This percussion is rendered very difficult by the restlessness of small children, and, in the examination of the position of the liver, allowance must especially be made for the very important influence of the respiratory acts. Older children, from the third year upward, may, by friendly coaxing, be induced to allow themselves to be examined as quietly as adults.

Diseases of the liver are extremely rare in young children. As we have already described icterus neonatorum in the chapter on the diseases originating directly after the delivery, there remain only the syphilitic and fatty livers, and the congenital anomalies of the liver, to speak of. The other hepatic diseases, cirrhosis, carcinoma, and echinococcus, are very seldom met with in children, and, when they do occur, their symptoms differ in no respect from those of the adult. Acute hepatic atrophy, so far as I am aware, has never been observed in children.

(1.) **SYPHILITIC INFLAMMATION OF THE LIVER.**—*Rayer* and *Ricord* were the first who pointed out the connection between a peculiar morbid alteration of the liver and secondary syphilis, but *Dittrich* established it satisfactorily by a number of more accurate investigations. In general, however, the disease is very rare. I have dissected many children with hereditary syphilis (perhaps fifteen), and always carefully examined the liver, but only once found this morbid condition.

Pathological Anatomy.—At the autopsy of children with congenital syphilis, almost all of whom, as is well known, perish, a peculiar kind of inflammation, which attacks the glandular substance of the liver, is sometimes found. The exudation of this inflammatory process is partly plastic, and at a later period becomes transformed into cicatricial tissue. It, however, rarely reaches this condition in children, for death usually ensues too early. This exudation is composed of sero-plastic material, and therefore absorbable, and of a grayish or

yellowish mass, which exhibits, microscopically, elementary granules, oil-globules, and only a few liver-cells. This yellowish-gray material, when the process has lasted long enough, is found in masses of the size of a hemp-seed, or of a pea, and circumscribed by the plastic substance, which subsequently becomes cicatricial. When these morbid changes occur at many points of the liver, it thereby acquires an uneven, roughened appearance, and the peritoneal covering, if the morbid alteration takes place on its superficial surface, becomes indurated and callous. The free spaces of the liver in children, as a rule, are normal; in adults, cirrhosis, carcinoma, and nutmeg or fatty liver, may occur along with the affection under consideration.

In young children, the form of the liver is rarely changed by this disease; usually nothing is found but a few spots, which, on section, prove to be firmer than the normal tissues, have a pale color, and are composed of granules, oil-globules, and only a few liver-cells.

Symptoms and Therapeutics.—The special symptoms belonging to liver-disease are slightly marked and difficult to recognize. These unfortunate children, who usually are two or three months old, bear upon them the signs of hereditary syphilis, such as condylomatous excrescences around the anus and at the angles of the mouth, ulcers about the mouth, ozæna and a syphilitic exanthema. The nutrition is very imperfect, and the emaciation extreme. The upper surface of the liver may possibly be found irregular or nodular, and its free border in some places slightly thickened and more rounded than in health.

The minor alterations of the liver are, of course, totally indistinguishable, and cannot be diagnosticated. As a complication, we may mention fibrous degeneration of the kidney, followed by anasarca. The most remarkable feature about this disease of the liver is, that icterus never occurs with it, but a grayish earthy hue of the skin supervenes on approaching dissolution.

No special treatment can be recommended for this condition. All those children who are not fed at the breast of their own mothers, and, on account of their liability to inoculate any other woman, cannot get a wet-nurse, almost invariably perish, no matter whether their livers are affected at the same time or not. The inunction treatment, where the state of the skin allows it, offers the best means thus far known for a recovery. This subject will be found more fully treated under *Therapeutics of Syphilis*.

(2.) **THE FATTY LIVER (*Hepar Adiposum*).**—By fatty liver we understand a greater quantity of fat in the hepatic cells than is normally present, and always in such amount that distinct alterations of

color of the parenchyma take place. This change of color is an important feature, because, if observed and regarded, we avoid the error of supposing that a few liver-cells containing oil, such as may be found in every *post-mortem* examination, must be considered fatty degeneration. In the infantile organism, fatty liver is a tolerably frequent accompaniment of consumptive diseases, especially of pulmonary tuberculosis and of protracted intestinal catarrh.

An attempt has been made to explain its origin in tuberculosis in various ways, and most authors are inclined to the view that in this instance it is produced by the embarrassed respiration, in consequence of which the oxidation of the hydro-carbons and fat cannot properly progress. But *Frerichs* very correctly observes that very many more decided disturbances of the respiration, pulmonary emphysema, for instance, do not induce fatty livers, and that, on the other hand, in other tuberculous diseases, such as tuberculosis of the bones, in which the lungs may be totally unimpaired, the fatty liver may also be found present.

He believes, therefore, that here the cause must be sought in the *altered state of the blood* that supervenes during the process of emaciation, and which consists in its becoming overloaded with fat, which in the progressive emaciation is absorbed. Hence the fatty liver is more marked in pulmonary tuberculosis than in other consumptive diseases, because in unimpaired lungs a greater consumption of oxygen takes place, and consequently a more rapid transposition of the excess of fat in the blood is effected.

Pathological Anatomy.—A well-marked fatty liver has a larger surface than the normal organ, is flattened, and its margins are thickened and rounded. Its upper surface is smooth, glistening, whitened, and has a doughy feel; the pressure of the finger leaves an impress. In color it is yellowish red or pale yellow, and a dry, slightly-warmed blade of a knife becomes coated with fat when drawn through its substance. The quantity of fat, as ascertained by analysis, is very considerable. In one case *Frerichs* found 78 per cent. in the dried substance of the liver; in the fresh state, the same liver contained 43.84 fat, 43.84 water, and 12.32 tissues, cells, vessels, etc. Simultaneously, the normal quantity of the water of the fatty liver becomes diminished, and may fall from 76 per cent. down to 43 per cent. The fat consists of olein, margarin, and traces of cholesterin.

In less-marked cases these pathological characteristics are also less distinct. As the hepatic lobules always become affected in such a manner that the cells lying on the periphery of each lobule first undergo fatty degeneration, while the centre of the lobule, about the hepatic veins still remains free and of a normal color, a reticulated ap-

pearance of the incised parenchymatous structure is produced, called the nutmeg-liver.

The brownish-red and pale-yellow substances alternate in such a manner, that the first forms small islands, which are surrounded by the latter like a bright-yellow zone. The form of the brown islands depends upon the direction in which the lobules have been cut through. Where the central veins have been cut through transversely, they are round; where the incision falls parallel with the central veins, they will represent oblong or leaf-like figures.

The deposition of fat does not always take place in all parts of the liver alike, hence the islands differ in size, especially those near the surface of the liver, where they may be scarcely changed from the normal appearance.

The microscopic appearance is very characteristic. The morbid change is limited to the liver-cells only, and no free fat is ever found in the intercellular spaces of the parenchyma. The hepatic cells, which, in the normal condition, are but slightly granular, exhibit at first fine, minute oil-drops within their walls, which, increasing in number, at length become confluent, and obscure the primitive cell-granules. The normal constituents of the cell thus obscured may be again rendered visible by removing the newly-deposited fat by the addition of oil of turpentine. The form of these fat-loaded cells is generally roundish, their angles having disappeared.

As regards the remaining functions of the liver, it is remarkable how little they are disturbed. The sugar-generating function of the liver, a modern discovery, but now well understood and appreciated, does not become deranged, nor does the bile it secretes deviate in quality or quantity from the composition of the normal fluid.

It is very difficult to define the boundaries between the physiological and pathological fatty liver, as it is met with in almost all nurslings, from whatever disease they may have died. All young animals who are still nursing usually furnish the same condition.

The disappearance of the fatty liver, as the age of the animal increases, is proof positive that the fat-infiltrated cells do not become destroyed, but that the liver becomes normal by a disappearance of the fat, and that therefore the fatty liver, under certain circumstances, may also be *curable*. The most frequent pathological conditions with which fatty liver associates are: tuberculosis, next rachitis, then hereditary syphilis; and, lastly, those diseases which lead to atrophy, such as enteritis folliculosa, diphtheritis, acute exanthemata, and typhus fever.

Symptoms and Treatment.—It may have been concluded, from what has been already said, that the symptoms of this condition are

very uncertain and unreliable. An enlargement of the dulness in the hepatic region is the first cardinal point in the diagnosis, although absence of this sign has no conclusive significance whatever; for in many instances a decidedly fatty liver is found without the least increase in volume. The fatty liver, however, does frequently become enlarged, flattened, and pushed forward against the abdominal walls, where it may be discovered by palpation and percussion. Such cases also present the condition called abdominal plethora, which consists in abdominal gaseous development, giving rise to flatulence and disposition to diarrhoea. The diagnosis becomes most probable when the above-mentioned diseases, tuberculosis, etc., have existed or still exist.

The fatty liver of children will hardly ever become the object of direct treatment. When it is possible to remove the condition which caused it, it will disappear spontaneously; if not, there is no remedy that will exercise a direct influence upon the fat of the liver.

(3.) CONGENITAL ANOMALIES.—The malformations of the liver are either very insignificant, simply affecting its form, or they may affect its organization so seriously as to compromise the life of the infant. As regards the deviations from the normal form, we have a quadrangular, triangular, flat, or round form; the division of the lobes may either be absent altogether or multiplied. As regards the great anomalies, we may mention, first of all, the total absence of the liver in monsters, especially acephalia. In diplogenetic monsters a double organ is also present. Similar malformations are observed in the gall-bladder and ductus choledochus. In congenital fissure of the diaphragm, which, on the whole, very rarely occurs on the right side, the liver may mount up into the right pleural cavity, and in congenital rupture of the umbilical cord (*vide p. 63*) it may lie exposed through the abdominal fissure.

In congenital transposition of the viscera the liver lies in the left hypochondrium, and with this a total or partial transposition of the rest of the abdominal and thoracic viscera is always combined. The spleen, as a rule, is cut up into small spleens, which lie in the right hypochondrium; the cardiac orifice of the stomach on the right, the pyloric on the left side, and the heart in the right thorax.

Hyrŕl has seen this transposition of the liver and of the rest of the viscera only four times. I have never yet met with it. The diagnosis cannot by any means be difficult, if a due amount of judgment and care be exercised in the examination.

F.—SPLEEN.

Idiopathic, primary affections of the spleen probably never occur in children, but in some acute diseases a secondary enlargement of this organ originates, such as has been more minutely described in typhus abdominalis. It is not as easy to detect an enlargement of the spleen in the child as in the adult, because children, up to the third year, are generally much averse to a protracted examination of the splenic region. It is best to examine them in the dorsal or right lateral decubitus. A normal spleen can never be discovered by palpation; and even the decidedly hypertrophied spleens are often so movable or so soft, especially in typhus fever, that they frequently escape detection. The hard, indurated splenic tumor, found in advanced rachitis and in chronic intermittent fever, may be discovered by palpation, and, in subjects much emaciated, may even be seen pushing out the thin abdominal covering. It moves downward with every inspiration, and upward with every expiration.

In moderate hypertrophy the spleen retains its oblique direction downward and forward; but, in chronic and more marked indurations, it will assume a more vertical position in line with the body, as the ligament pericolicum, which in the normal condition gives it an oblique direction, gradually becomes elongated. The form of the spleen, wherever it can be felt, is very characteristic. It is an oblong oval, with blunt borders, and an indentation on the internal aspect at about the middle of the tumor.

In the examination of the spleen by percussion, very gentle blows should be given, particularly when the intestines are tympanically distended, because, by heavy blows, the tympanitic resonance of stomach and intestines is developed, and the splenic dulness is lost. This examination should be practised with the patients always in the same position, because variations of the posture alone produce decided changes in the dulness. In general, all those dull sounds of the splenic region, which in children under one year extend under the pleximeter, beyond the normal boundary, are to be considered pathological. In marked ascites, and in serous effusions into the left pleural sac, the dulness of the spleen cannot be detected by percussion at all; it also disappears in intense tympanitis, so that, even when the hypertrophy of the spleen is considerable, a perfectly sonorous tympanitic percussion-sound may be heard over the corresponding space.

Besides the occurrence of the splenic tumor in typhus fever, it is in-

variably present in intermittent fever, and a description of the latter may therefore very properly follow here.

INTERMITTENT FEVER (*Febris Intermittens*).—Intermittent fever occurs just as often in young children as in adults. Cases are even recorded of infants being born with enlarged spleens, and suffering febrile paroxysms at the same hour in which the mothers had their paroxysms during pregnancy. I have never met such a case; the youngest child that I have had to treat for intermittent fever was eight weeks old. The symptoms to be presently described have reference only to children under two years; in older children the whole course is so characteristic, that every one who has once seen an adult suffering from a paroxysm of intermittent fever will immediately recognize it.

The etiology of infantile intermittent is naturally the same at all ages. The fever is confined to certain locations, and is never observed in children who have not lived, or at least stopped for some time, in malarial regions. The most exhaustive treatise on the etiology of malarial affections is to be found in *Griesinger's "Infecting Diseases"* (*Virchow's "Hand-book of Special Pathology"*), in which the circumstances of the quantity of the water of the earth, of the temperature, of the climate, etc., are discussed in detail.

Symptoms.—In children, the quotidian type is the most frequent; still the tertian, and even the quartan, also occur. The hour in which the attack comes on is not always the same; the paroxysm, however, is always confined to a certain period of the day; the morning, afternoon, or evening. As regards the attack itself, it is usually not fully developed, but rudimentary in one or more of its phenomena. Actual chills, it is true, occur, in which the children are seized with shivering, low moaning, collapse, and have blue lips and nails; immediately after they become decidedly hot, have a dry skin, great thirst, and restlessness, and finally break out in a general perspiration, thus completing a perfect picture of an intermittent febrile paroxysm. As a rule, however, the symptoms are not so conspicuous, and often leave the malarial character to be divined by their recurrence every day, or every other day, at the same hour. The shivering chills are often totally absent, and a remarkable paleness of the skin, blueness of the nails and lips, discoloration around the eyes, cold extremities, and low whimperings, or slight convulsive twitchings of the facial muscles, are only present. The pulse does not become very much accelerated, but very small. Respiration is normal, as to frequency; the breath does not become cool, nor does the temperature of the mouth sink in the least. During the various symptoms, representing the cold stage,

children rarely pass any urine; but, if they have partaken of much milk shortly before, they generally throw it up. This stage never lasts longer than one, or, at the most, one and a half hours. During it the child presents a most critical appearance, and the physician may seriously compromise his professional reputation if he should give a prognosis immediately after first beholding the child. I myself once committed such an error in the early course of my professional career.

In the second stage the vessels of the skin become turgid, the face is flushed, the pulse harder and quicker than in the cold stage, the cardiac impulse stronger, and felt over a larger area. There is very great restlessness, the child cries aloud, and is frequently attacked by convulsions, conjointly with which the pupils become remarkably dilated. The urine and stools are retained.

This condition may last from two to three hours, and is much more correctly observed and described by the mothers than the cold stage. But it is also often transient, lasting barely one-quarter of an hour. It is followed by a slight perspiration, the third stage.

While the patients are awake the perspiration is not very well marked; the skin, it is true, feels moist, but drops of sweat are very seldom seen upon it; free perspiration takes place only when sleep ensues. The heat and redness of the face then diminish, the thirst decreases, and the urine is discharged tolerably dark in color. The pulse assumes again its normal condition, and the patients once more present all the signs of general good health. But, in the intervals of freedom from fever, they are seldom perfectly well; are generally very restless, have less appetite, and a sluggish, irregular digestion. The febrile paroxysms are frequently so little pronounced that an observation of several days is necessary in order to confirm the diagnosis.

The intermittent-fever cachexia, in small children, is very soon established, as early as after one to two weeks, and is characterized by the following symptoms:

Anæmia appears very rapidly, the color of the skin becomes perfectly white, or turns to a slight grayish tint, the lips and mucous membrane become pale, the emaciation progresses and becomes marked, slight œdematous swellings form under the eyes, the enlarged spleen is easily felt, and, when the emaciation increases, may even be seen. The liver also swells up, and its parenchyma becomes indurated (lardaceous liver). The intestines are tympanitic with gas, the stools are mostly diarrhoeal, and, in the last days of life, sometimes mixed with blood. The important discovery of the presence of brown