

or black granular pigment-matter in the blood of intermittent-fever patients is an attainment of pathological microscopy for which we are indebted to *Virchow* and *Meckel* (Melanæmia). This pigment is seldom found in the blood of children, because, for this purpose, it is necessary that the cachexia should be much prolonged, which, in children, is generally not the case, as they die of the disease too early. Moreover, intermittent fever is so easily recognized, and its treatment so rapid and effective, that, wherever physicians are consulted for it, it seldom advances to the development of the cachexia.

**Pathological Anatomy.**—In this country, a child rarely dies during the attack, but, in regions where pernicious fever prevails, death may occur even with the first paroxysm. The anatomico-pathological condition is then purely negative; a slight swelling of the spleen, with a superabundant amount of blood in the whole venous system, are the only abnormalities. But, in the bodies of infants who have perished from the cachexia of intermittent fever, various morbid changes are met with, such as anasarca and effusions into the serous cavities, lardaceous liver, large lardaceous spleen, with brown or black pigment-matter most profusely accumulated in the spleen, in the liver, in the brain, upon the mucous membrane, and in the kidneys, which are also frequently affected with Bright's disease. In such cases the urine found in the bladder is always albuminous, and casts may be seen, with the microscope, in its sediment.

**Treatment.**—The attack itself, in our milder forms of intermittent fever, requires a not very energetic but an expectant treatment. For the cold stage, a high temperature and mild cutaneous irritants are sufficient, such as friction with a woollen rag, with spirits of camphor, or other excitants; cool coverings, cold affusions to the head, the administration of plenty of cold water, and, when convulsions occur, one or two drops of laudanum, answer for the hot stage. The sweating that ensues should be encouraged in every manner possible. During the pyrexia the children must be kept upon a very strict diet, and in a uniform temperature. In sulphate of quinia, however, we have a very efficacious remedy for the prevention of the recurrence of the paroxysms. In children under one year, one grain, given in one dose, is generally sufficient to arrest the attack. To older children, two to three grains are given. The addition of sugar to this dose, thereby increasing its bulk, with the view of improving its taste, is practically useless, if not disadvantageous, for the taste is not improved in the least. In young children it is almost always easy to administer this remedy. But older children, who are not trained to swallow powders wrapped in wafers, are often, notwithstanding their best intentions, unable to swallow them, or refuse to attempt it. In

these cases, the employment of the remedy in the form of clysters may be practised; the method is very efficient.

A countryman, from a peat-moor, once brought his boy, five years old, to me, stating that he had suffered daily for six weeks from the fever, which the physician of his place was unable to cure, because it was totally impossible to administer quinine to the child. The boy was very anæmic, had a very large spleen, an indurated liver, marked ascites, traces of albumen in the urine, and slight œdema of the lower eyelids. I ordered him a ten-grain solution of quinine in one and a half ounces of water, and caused it to be administered in a clyster, in my presence, to which the child readily submitted. The man took him home to his moor again, and, although continually exposed to the malarial air, he had no further attacks, but, under a tonic treatment with *R. mart. pomat.*, meat diet, and beer, completely regained his former health and spirits.

When one dose of quinine is not sufficient, another must be administered during the next interval. The determination of the hour of administration, upon which great stress is laid by many physicians, seems to be less important in children than in adults. The main point is always for the quinine to be well and thoroughly absorbed, and therefore it is advisable not to give it directly before or after a meal, and immediately before the appearance of the chill, because during it the digestion is interrupted.

In our intermittent fever, quinine has never yet failed in my hands to perform its duty, and for that reason I have never had occasion to resort to arsenic in the treatment of this disease. Still I would not hesitate for a moment, in case of failure, to use it, since Fowler's solution is excellently well tolerated by children. If the cachexia is already developed, an after-treatment will be necessary. The best is the removal of the child from the malarial region to a dry, elevated residence. Where this is not practicable, we have to limit ourselves to iron and a meat diet, combined, in older children, with small quantities of beer.

#### G.—PERITONÆUM.

**PERITONITIS ACUTA AND CHRONICA.**—Peritonitis with sero-fibrinous exudation not unfrequently occurs in the new-born child, and even in the fetus. In older children, on the contrary, it is very seldom seen, without it is traumatic and tuberculous, because, in these, the principal causes of peritonitis—intestinal perforations, and diseases of the female sexual organs—rarely, if ever, occur.

**Etiology.**—All the forms of peritonitis that it has been customary to assume, in special pathology, are also observed in the new-born child. The idiopathic is the rarest form, and is almost exclusively seen in the foetus; the secondary is the most frequent, and the metastatic occurs in lying-in houses in which puerperal fever prevails. The latter forms cannot always be positively distinguished. The process takes its starting-point, in both, from the umbilical vessels, which are filled with ichor, but whether the inflammation is simply extended to the contiguous peritonæum, or whether this membrane, like other serous coats, becomes attacked by the pyæmic process, is often impossible to decide. This kind of peritonitis, depending upon an ichorous navel, is only liable to occur so long as the latter exists, six, or, at the utmost, eight weeks after birth. After that time, the traumatic form is only seen, such as that occurring after burns, and perforation of the stomach or bowels, or that resulting from an incarcerated hernia, or from intussusception. The tuberculous form may also be added.

**Symptoms.**—In peritonitis of the new-born child, pressure upon the abdomen is always painful, so much so, that the sufferers will utter loud but abrupt cries, even at the slightest touch. They are not capable of crying continuously, because the employment of the abdominal muscles for this purpose augments the pain. They are most quiet when they are completely uncovered, so that the abdomen is free on all sides; the legs are extended, and the thighs not drawn up against the belly, as is usually the case in colic, because the pains seem to become aggravated even by the contact of their own thighs. Older children suffering from peritonitis always maintain the dorsal decubitus, and cannot be induced to lie upon the one or the other side. A paretic state of the abdominal muscles is present in all cases, and a more marked tympanitis in consequence. In the new-born child fluctuation can never be felt, because (1) the exudation is a plastic membranosis, and (2) the tympanitic bowels press against the abdominal walls in such a manner that the fluid exudation when present is below and behind the bowels.

Vomiting is much less regularly observed in peritonitis of children than in that of adults, and diarrhoea is oftener present than constipation. The appetite is completely lost, but the thirst is great. If the peritonitis has reached the serous coat of the bladder, retention of urine will supervene, or a few drops only are discharged at a time, attended by severe pain. The febrile signs are always distinctly pronounced; the skin is hot and dry, the pulse frequent and small, and the breathing rapid and superficial. The respiration is distinguished from that of health by the circumstance that the diaphragm does not act at all, or but very little, while the pectoral muscles seek

to produce the utmost possible dilatation of the thorax. But, since the proper inflation of the lungs cannot by any means be accomplished by this kind of respiratory act, the children are compelled to execute one deep diaphragmatic inspiration for every ten to fifteen of those superficial ones, and this, being attended by pain, is accompanied by distortion of the face, and frequently by a pitiful cry. The color of the face is oftener pale than flushed; convulsions occur here less frequently than in pneumonia.

Peritonitis of the nursling, as a rule, terminates fatally after from one to three days. Tuberculous inflammation of the peritonæum of older children runs a longer course, and may even last for many months. But the lethal termination is also in this form almost unavoidable.

**Pathological Anatomy.**—The peritonæum exhibits at different places, especially on the contiguous surfaces of two intestinal coils, capillary injection and plastic exudation, by which complicated adhesions are produced. In peritonitis of the new-born child, which is due, almost invariably, to a phlebitis umbilicalis, the principal morbid alterations are found about the umbilical ring and on the concave surface of the liver, which, by plastic exudation, becomes agglutinated to the neighboring organs, the stomach, and large and small intestines. In the small pelvis some ounces of a purulent, sanious, or bloody fluid, are usually found. In the two cases of fetal peritonitis recorded by *Billard*, numerous tense bands and old adhesions were found present in the cadavers of the still-born infants. Lobular pneumonia is often present as a complication.

The treatment of peritonitis, as may be gathered from what has been hitherto said, is a most unsatisfactory one. That resulting from phlebitis umbilicalis seems almost invariably to be fatal. The treatment, therefore, which secures rest, cleanliness of the navel, and the best possible sustaining measures, seems the only one indicated. In tuberculous peritonitis, hectic fever, as a rule, is present, and, as its subjugation is altogether out of the question, we have to be content with trying to remove the febrile symptoms by the aid of small doses of quinine and morphine. For the peritonitic pains I use warm moist compresses, which are covered by a piece of gutta-percha cloth, and this by a dry cloth. They are much cleaner and more convenient to apply than cataplasms, which, especially at night, become cold and hard, and possess at no time any superiority to the application recommended. Traumatic peritonitis does, indeed, tolerate an antiphlogistic treatment to the extent of from three to twelve leeches, and, in case no diarrhoea be present, the addition of several doses of calomel, gr. ss. to j, during the day, till a green diarrhoea takes place. The

warm-water compresses above described should always form part of this antiphlogistic treatment, for they afford the greatest relief. If the pains are very persistent, opium is also indicated, as it is in most painful diseases.

(2.) ASCITES. HYDROPSICAL EFFUSION INTO THE PERITONEAL SAC.—Ascites is never a primary affection, but always a mere symptom of some other constitutional or circulatory disturbance. In young children it is inconsiderable as to quantity, often only a few tablespoonfuls of serum being found at the autopsy. Thus, children who die from hereditary syphilis, tuberculosis, marasmus, the effects of enteritis, from congenital malformation of the heart, or scleroma, have small serous effusions in the abdomen. Considerable, easily-demonstrable effusions occur only in children who are over one year of age, and generally as a result of scarlatina, or of intermittent fever, and occasionally, but less frequently, of abdominal typhus.

**Pathological Anatomy.**—In old children the quantity of the ascitic fluid may reach several pounds. The color of the serum is a wine yellow; sometimes a little coloring matter of the blood is also mixed with it. The reddish color thus produced may, however, also be due to one or another of the cutaneous veins having been cut at the opening of the abdomen, and their contents escaping into the peritoneal cavity. The chemical investigation gives a large percentage of albumen, and the salts as they are found in the serum of the blood. The peritonæum is either perfectly normal, or at some places displays white opacities, which are principally observed in protracted cases of ascites. Occasionally one of the intestinal coils or the liver is coated with a thin layer of exudation, so that we have to deal here with a transition into true peritonitis. In no autopsy should the mere finding of ascites content us, but its cause, one of the above-mentioned affections, should be sought for, in which the heart and kidneys, in particular, are to be subjected to the most scrutinizing investigation.

**Symptoms.**—The existence of ascites can only be satisfactorily proven by distinct fluctuation. Small effusions can never be detected in the dorsal decubitus; occasionally they may be discovered, by laying the children on the right side, and slightly elevating the pelvis, whereby all the serum then gathers into the right hypochondrium. The smallest quantities, however, may be detected by laying the child upon its belly, and then causing it to be raised up, so that the navel will form the most depending part of the whole abdomen. As in this position all the serum must gather round about the navel, it is then easily detected by percussion from below upward. Fluctuation is ascertained in this manner. The palmar surfaces of the fingers of one

hand are made to press against the abdominal parietes, while with the tips of the fingers of the other the abdomen is quickly and lightly struck at a point opposite to the pressing hand, or at least at a distance of a few inches from it. The undulation thus produced, in case fluid be present, between the pressing and the striking hands, communicates a peculiar thrill to the former. Besides being produced by the free fluid of ascites, fluctuation also originates from the presence of diarrhoeal contents of the intestines, from a bladder filled and mounting over the symphysis, and even from œdema of the abdominal walls, which, especially in nephritis after scarlet fever, is commonly very intense. The latter, however, is readily distinguished from true ascites, by the pitting from the pressure of the finger, and by the superficial character of the flaccidity. The distended bladder is easily emptied, and the intestinal catarrh readily relieved by a mucilaginous diet and small doses of opium, whereupon the true state of affairs will become manifest.

In mild grades of ascites, nothing can be discovered externally, and the circumference of the abdomen is not materially increased; but in the higher grades attention is attracted to the size of the belly, even at the first sight. The integument is glistening and tense; there is flatness on percussion of the lower part of the abdomen, the dulness extending over a large surface; the umbilical ring is distended, and the navel prominent. The pressure of the serum excites frequent inclinations to micturate, but only small quantities of urine are evacuated at a time.

The general phenomena, loss of appetite, fever, respiratory disturbances, etc., correspond with the conditions causing the ascites. The patients, as a rule, perspire but little, and pass very small quantities of urine. It is mostly dark-colored, and, in nephritis, contains albumen and fibrous casts. The stools are often diarrhoeal.

The prognosis does not depend upon the amount of the ascites, but upon its etiology. It may be regarded as unfavorable in almost all kinds of ascites, except in that originating after scarlatina, typhus fever, and hypertrophy of the spleen from febris intermittens.

**Treatment.**—This, of course, varies according to the cause. The conditions which give rise to ascites are of such a hopeless nature, that any special treatment, except a stimulating régime, will hardly be indicated. When caused by nephritis, after scarlatina, *roob\* juniperi*, which children usually take very readily unmixed and undiluted, is an excellent diuretic remedy; I direct one-half or a whole teaspoonful to be taken daily. It is also applicable in ascites

\* This is the succus spissatus juniperi of the European pharmacopœias, and is somewhat stronger than a fluid extract.—Tr.

after intermittent or typhus fever, but then a tonic treatment, consisting of a meat diet, beer, wine, and small doses of iron, is to be recommended in addition. The ascites which comes on after scarlatina subsides more rapidly than any other.

(3.) MORBID ALTERATIONS OF THE MESENTERIC GLANDS.—In all cases of enteritis folliculosa, the mesenteric glands become hypertrophied and indurated, and their impermeability most probably affects the atrophy that so frequently follows it, a detailed description of which has already been given in connection with that disease (p. 156). In addition, cheesy tubercles of the glands occur in older children; and, in those who perished by typhus fever, hypertrophy, or small abscesses of single glands, are sometimes met with.

The diseases of the mesenteric glands do not seem to give rise to any symptoms, but the nutrition, if a large number of the glands is involved in the hypertrophy, suffers very quickly. The glands, on the whole, are so small, and the bowels are always too tympanitic, to allow them to be felt.

### CHAPTER III.

#### DISEASES OF THE ORGANS OF CIRCULATION.

##### A.—HEART AND VASCULAR TRUNKS.

(1.) CONGENITAL ANOMALIES.—For the purpose of correctly understanding the congenital anomalies of the heart, this much of the embryology has to be premised: that the heart and roots of the vessels at the commencement of development are not hollow, but consist of a loose conglomerate mass of cells, without any chasm or channel, and without any cavities. At this period the heart still possesses the form of a straight cylinder, which above and below terminates in two prolongations; the two lower prolongations, the venæ omphalo-mesenterica, are the roots of the vessels which subsequently ramify in the germinal vesicle and conduct the blood from it to the heart; the two upper prolongations are the two future first aortic arches, which, in the embryo, carry the blood from the heart. The external upper surface, according to *Bischoff*, very gradually becomes firmer by the cells being deposited closer to each other, and thus the walls are formed, and a cavity is developed within, in which the fluid and cells, forming the first trace of the blood, accumulate. The cardiac cylinder then assumes an S-like shape, and begins to

contract and dilate in a slow rhythm, by which its fluid contents are propelled anteriorly and upwardly into the aortic arches, while that from the venous trunks, on the other hand, is sucked in from below and behind.

By-and-by this cardiac canal, by various curvings, dilations, and constrictions of single parts, becomes the heart proper, consisting of the aortic dilatation, *one* ventricle and *one* auricle. The septa do not become developed till a later period, by which the right and left ventricle and auricle are formed. Imperfect development or faulty insertions of these partition-walls are the most frequent causes of malformation of the heart.

Nevertheless, cases of malformation are also observed as the result of an embryonal inflammatory process of the muscle of the heart, and its consequent atrophy and cicatrizations.

The best compilations on the congenital anomalies of the heart are to be found in the text-books of *Rokitansky* and *Bamberger*, which have furnished the basis for the following summary:

(1.) *Absence of the heart* (acardia) occurs only in monstrosities, where the upper half of the trunk is at the same time wanting, and the nervous system consequently exists only in a rudimentary form. The converse of this is the duplex heart in double malformations (diplogensis); this occurs especially in doubling of the upper half of the body, where two completely-separated hearts either occupy each a separate pericardium or a common one.

(2.) *Abnormal situation of the heart*.—Here we may have the foetal heart occupying a central position in the thorax, or *transposed*, so that the cardiac impulse is felt at the right of the sternum. In this latter case we have generally an accompanying displacement of other organs, particularly the liver and stomach.

Again, the sternum may be absent and the integument wanting, and when this condition occurs we have the heart entirely exposed, or merely covered by the pericardium.

If a greater portion of the thoracic and abdominal walls is wanting, we have the condition called *eventration*, in which the abdominal organs lie without the body.

In very rare instances a defect or splitting of the diaphragm occurs, and the heart then penetrates through this opening into the abdominal cavity.

(3.) *Abnormal shape and size*.—Variations of the shape of the heart are often devoid of importance. It may be broad, cylindrical, or fissured at the apex. A pointed and a round heart may perform its functions naturally, whereas, on the contrary, abnormal bigness or smallness of the whole heart, or some of its parts, is complicated with