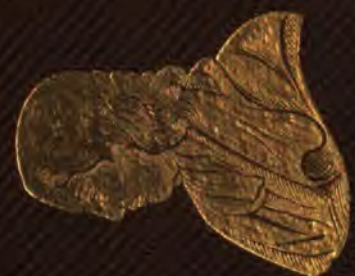

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# LECTURES

ON

# CHILDREN'S DISEASES.

## A HANDBOOK

FOR

## PRACTITIONERS AND STUDENTS

BY

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### DISEASES OF THE DIGESTIVE ORGANS.

### I. *Inflammatory Affections of the Mucous Membrane of the Mouth.*

The simplest form of this disease (stomatitis), which is very common in childhood, is characterised by a general or local bright or dark redness of the mucous membrane, especially of the gum, which at the same time is swollen and tender to touch. An unusual amount of saliva trickles from the half-opened lips, and they also are often somewhat swollen and of a bright red colour. On attempting to take the breast the infant lets go the nipple on account of pain and begins to cry; and when he is sucking, the mother often observes that the mouth is considerably hotter than usual, even before any other symptoms are noticed. The increase of the salivary secretion gives an unusually glazed and shining appearance to the mouth, and the dorsum of the tongue is often covered, either all over or in streaks, by a whitish-grey coating, consisting for the most part of a large quantity of desquamated epithelium. The region of the lower jaw is at the same time not uncommonly œdematous, and seems fuller than usual, partly for this reason and partly owing to the concomitant enlargement of its lymphatic glands. We may also have slight elevations of temperature, restlessness, and sleeplessness.

This form of stomatitis is oftenest seen during teething (vol. i., p. 158). In older children, it often appears as the result of acute infectious diseases, especially measles and scarlet fever; and in these cases it frequently passes on to a more severe form with a fibrinous exudation. But to this I shall return in speaking of these diseases. Simple cases require no special treatment, as the stomatitis passes off of itself whenever a group of teeth comes through, or when the exanthem

disappears. You have, therefore, nothing to do beyond seeing that the mucous membrane of the mouth is not subjected to any severe irritation.

The second variety has a much more characteristic aspect. Like the first, it is usually called "thrush" by the public; and by medical men it is called "aphthous stomatitis."[1] Most of the children whom I have seen suffering from this disease were at the age of dentition, *i.e.*, between the 7th month and the middle of the 3rd year. One child of 14 months had had as many as five attacks since he was 4 months old. These always came on with the eruption of a group of teeth, and then at once disappeared. It is less common for this affection to occur either before or after dentition, and in these cases we must assume some other unknown influence besides the irritation of the teeth. Besides the symptoms which have been described under simple stomatitis (which, however, are generally more severe in those cases), we observe greyish-yellow or greyish-white spots from the size of a pin's head to that of a pea and larger, situated on the margin, tip, and dorsum of the tongue—less commonly on its lower surface and near the frænum. These are surrounded by a narrow red border; they are round and occasionally somewhat indented in outline. Between these there are also patches of an elongated form, either so few in number that they may be easily counted, or perhaps very numerous, sometimes lying close together and here and there coalescing so as to form greyish-white plaques or sinuous figures. In a few cases I have seen greyish-white patches of a firm consistence about the size of a sixpence, rising above the level of the mucous membrane, on which their edges lay loosely like those of a fungus. We often also see plaques of a varying size on the mucous membrane of the cheeks, the hard and soft palate and even on the tonsils, also on the lips which are reddened and much swollen so as to project like a snout. When we examine these patches, or when the children touch them, they are apt to bleed and then they become covered with dark-brown or blackish crusts of dried blood. Groups of herpetic vesicles on the margins of the lips and whitish-grey excoriations at the angles of the mouth are not uncommon accompaniments; and, owing to the enlargement of the submaxillary lymphatic glands, which can

[1] Synonym: "stomatitis fibrinosa, follicularis."

generally be felt, there may be a distinct swelling of the lower part of the face in these severe cases; and this is increased by the slight œdema of the connective tissue surrounding the glands. The tongue is often covered, with the exception of its edges, with a thick greyish-yellow greasy coating, and the secretion of saliva is increased to such an extent that it keeps trickling out of the half-opened mouth. The gum is dark-red and swollen, and covered here and there with a delicate whitish epithelial membrane. Only in very rare cases have I observed the plaques confined to the mucous membrane of the cheek and palate, while the tongue was quite unaffected. It is invariably accompanied by a high temperature—especially in the evening—irritability and restlessness; but, above all, by pain, which makes eating and drinking very difficult.

In some of the cases there is an unpleasant smell from the mouth in addition to the symptoms I have described, and we then generally find on careful examination that the gum is hyperæmic and bleeds readily, and that the portion of it immediately surrounding the teeth is generally in process of breaking down into a greyish-yellow *débris* which readily comes away in little pieces on the spatula.

Although this disease is so common, its pathogenesis is still obscure. I have never been able to discover that the plaques are formed from vesicles as used to be assumed; for in all the children who came to me the patches were already fully formed. And even in the cases where new ones formed during the course of treatment, I could never make out an initial vesicular stage. We must not be misled by the fact that among the ordinary perfectly flat plaques we occasionally find a few which are more or less raised above the surface. These are by no means to be regarded as vesicular elevations of the epidermis, but as solid laminated exudations; for they are composed of fibrinous lymph under the epithelium. One can never simply pick off a plaque with the forceps; for as Robin showed, they are firmly united to the mucous membrane by fibres and by an amorphous material. They have consequently a certain resemblance to diphtheritic products, but the resemblance is only external. The disease is never anything more than a local inflammation of the mucous membrane of the mouth, with quite superficial fibrinous exudation, and, as far as my experience goes, it always

takes a favourable course. The affection is usually cured in 8—10 days when properly treated. The increased salivary secretion first diminishes, any fœtor of the mouth that may be present disappears, and then the plaques diminish in size from the periphery towards the centre so rapidly that after a few days a patch, which was of the size of a pea, is now no bigger than a pin's head. During the process of cure, very thick, whitish-grey patches becomes steadily yellower and more transparent, lose the surrounding red border, and finally disappear entirely without leaving a trace of scar or any other alteration, except perhaps a patch of darker redness at the affected spot.

When the gangrenous ulceration of the border of the gum mentioned on p. 3 is added to the fibrinous stomatitis, the process becomes more serious. I have, indeed, seen such cases heal just as quickly as the ordinary ones. Still, we must always remember that this constitutes also the commencement of a worse form, which we shall become acquainted with under the name "stomatitis ulcerosa," or "stomacace."

It has been formerly maintained (Taupin) that the disease is contagious, and as a matter of fact I have myself occasionally met with cases where several members of one family had taken ill simultaneously after using the same spoon or glass. One boy took stomatitis after repeatedly biting an apple which another child was eating who was suffering from this disease. But such cases are exceptional, and are not sufficient to justify the assumption of a parasitic origin. Whether the large number of cases observed at the polyclinic at the same time was accidental or not I shall not decide.

In the treatment, I recommend specially the internal use of chlorate of potash, which in these cases has a specific action, and indeed I find that it acts most promptly in the very cases in which there is also fœtor of the mouth and implication of the margins of the gums. In very young children we can scarcely ever give chlorate of potash in the form of a mouth-wash or gargle, for they have not the sense to use it in this way. The slight pain which the medicine causes on contact with the diseased mucous membrane is not worth consideration, compared to the disappearance of the fœtor and salivation. Many cases recover under this treatment in 5 or 6 days. Less commonly you meet with obstinate cases which resist even the con-

tinued use of chlorate of potash or even become worse under it, without your being able to find any reason for this peculiarity. Under such circumstances I have usually got good results from the use of sulphate of zinc (grs. xxiv. to ʒ i.), or sulphate of copper (grs. xii. to ʒ i.); with these the affected parts were painted 2 or 3 times a day.

I have repeatedly observed a form of fibrinous stomatitis following acute infectious diseases, especially scarlet fever and less commonly measles. Externally it is very similar to the ordinary form, but it is usually more extensive and severe. This form may occasion copious hæmorrhage from the tongue and lips; I shall return to it in speaking of scarlet fever. Stomatitis appears occasionally along with simple catarrhal sore-throat in children. In one boy of 5 who was suffering from sore-throat, I found the soft palate above the tonsils studded over with numerous round yellowish patches of the size of half a lentil-seed. All of these had a hole in the centre, indicating a relationship to the mucous follicles such as cannot be made out in aphthous stomatitis.

Less commonly than the disease just described, we meet with another which is termed "stomatitis ulcerosa," or "stomacace." In this form the fibrinous patches are either absent or are only of secondary importance; but the affection of the gums calls for the chief attention of the physician. They are dark or bluish red, swollen, slightly bleeding, and they break down gradually to a greyish-yellow pulp commencing at the edge surrounding the teeth, so that the crowns of the teeth are exposed and finally loosened. Purulent matter flows from between the teeth on pressure, a fœtid smell comes from the mouth and adjacent soft parts, and the cheeks and submaxillary connective tissue become œdematous. This swelling and the increasing pallor of the child often disfigure the face to such an extent as to cause anxiety, and even to make one fear that noma is about to appear. In the next few days, however, the slow advance of the process and the continued softness of the swelling show this fear to have been unfounded. The impossibility of taking a sufficient amount of nourishment owing to the pain is all the more serious as the disease has generally a protracted course and may last for many weeks. Under these circumstances we sometimes see the process impli-

cating the periosteum of the jaw, passing down into the alveoli and causing the teeth to drop out, and in the end giving rise to localised necrosis of the jaw. In one of these cases there was for many days a trismus-like contraction of the masticatory muscles on both sides, which could only be explained by reflex action and which rendered the opening of the jaws impossible, thus allowing only fluid food to be taken.

Child of 1¾ years. During the previous 6 weeks, fœtor oris, salivation, enlargement of the submaxillary lymphatic glands, hæmorrhages from the gums. Since that time 14 teeth had fallen out, and several deep cavities, covered with grey pulp, could be made out in the alveolar processes of both jaws. Improvement under chlorate of potash. Termination unknown.

Child of 1½ years. Fœtor oris, considerable swelling of the right cheek and of the submaxillary region, collapse, pallor, fever, inability to eat. The whole gum, especially on the right side beneath, red and swollen, separated from the teeth, which are surrounded by pus. The alveolar margin on the left side below much swollen and painful, and contained two loose teeth which were extracted at once. Often-repeated syringing of the mouth with ¼ per cent. solution of permanganate of potash; internally the continuous use of decoction of cinchona with chlorate of potash. After 3 months, considerable improvement, but necrosis of the margin of the right lower jaw, from which a few sequestra were removed. Swelling considerably less, suppuration diminishing. Further course unknown.

When the course of the disease is very chronic, fatal collapse may be occasioned at last by the continued suppuration and deficient nutrition. The comparatively small number of such severe cases that I have had under treatment may perhaps explain the fact that I have not yet met with this sad termination. With the exception of the two which I have just given, all the cases examined by me were slighter, and yielded to the steady use of decoction of cinchona with chlorate of potash (Form. 27) and antiseptic syringing with permanganate of potash, carbolic or salicylic acid. Pure country air is to be strongly recommended for those whose circumstances admit of it. The removal of loose teeth or sequestra, when this can be done without too great violence, accelerates recovery.

In older children, from 5—8 years—*i.e.*, during the period of the second dentition—we see ulcerative stomatitis not uncommonly affecting the gum and causing redness, swelling, and

gangrenous ulceration of its margin, but not penetrating deeper. The chief change, however, appears in the mucous membrane of the tongue, cheeks, and lips. In these cases we have the formation of large uneven ulcers covered with a greyish yellow pulp, having abrupt swollen borders of mucous membrane which bleed readily on being touched and cause an extremely fœtid odour from the mouth. If the ulcer is situated on the margin of the tongue, you almost always find another quite similar one on a corresponding part of the inner surface of the cheek, so that we cannot doubt that transmission takes place from contact of the two surfaces. I have observed the same thing on the surfaces of the lower lip and lower gum where they touch one another. Moreover, the marked swelling of the surrounding mucous membrane makes the ulcer appear much deeper than it really is. For, after the final throwing off of the greyish-yellow membrane formed of *débris*, there usually only remains a superficial loss of substance which readily heals. I am not in a position to tell you anything definite about the etiology of this disease, which is associated with necrosis of the mucous membrane, or even of the bone.[1] The children whom I have observed, at least, presented no trace of any special dyscrasia, but had previously been perfectly healthy. Only a few showed the symptoms of "cachexia pauperum." It seems to me to be beyond doubt that both the first and second dentition may have something to do with this, for hyperæmia, hæmorrhages, small abscesses of the gum and salivation occur often about these periods, and may readily reach higher degrees of inflammation.

In one girl of 7 years, all the milk-teeth that remained were loosened, while the permanent teeth appeared very rapidly behind and above them. The whole gum was dark-red, swollen, bleeding readily, and extremely tender. After the complete eruption of a few new teeth, spontaneous recovery occurred within 4 weeks.

In a boy of 7½ years, an elongated ulcer formed after the

[1] A case which Grandidier published as one of "phosphorus-necrosis" (*Journ. f. Kinderheilk.*, 1861, H. 5 und 6) appears to me as more than doubtful. I have several times had the opportunity of observing in children (during the second dentition) who were otherwise quite healthy and had nothing wrong with their mouths, a copious flow of saliva (especially during the night) which occasionally was somewhat tinged with blood and wetted the pillow. The parents were very anxious about it; but I never saw any bad results from this condition, even when it lasted for weeks and repeatedly recurred.

extraction of one of the lower posterior molars on a corresponding part of the mucous membrane of the cheek. From this the hyperæmic swelling of the readily-bleeding gum spread further forwards and occasioned enlargement of the lymphatic glands, fœtor oris and œdematous infiltration of the cheeks. Recovery after 10 days under chlorate of potash and painting with sulphate of copper (2 per cent. solution).

This case at the same time illustrates the treatment, which is just the same as that which I recommended (p. 4) for stomatitis aphthosa.

## II. *Cancrum Oris.* (Noma.)

This fearful disease is peculiar to childhood, and especially to the period between the 3rd and 8th year. Fortunately, it is so rare that even physicians in very large practice only meet with it at long intervals.

As a rule, on first examining the patient, we find one half of the face considerably swollen, especially the cheek and half of the upper lip, sometimes also the lower lip and the chin. Occasionally the swelling spreads upwards as far as the lower eyelid, so that the eye is half closed, and the whole face is disfigured. The swelling is colourless and pale, and usually has an oily appearance, owing to increased secretion of sebaceous matter. There is little or no tenderness to touch, but the swelling is very tense, so that it scarcely pits at all on pressure with the finger. On careful palpation we feel a more or less extensive ill-defined hard area situated deeply near the most prominent part of the swelling.

Although, as I have mentioned above (p. 5), ulcerative stomatitis may also be accompanied by a similar swelling of the soft parts, still we must always bear in mind the possibility of noma under these circumstances, especially if the patient is cachectic or extremely poor, or has recently recovered from a severe disease—more particularly pneumonia, typhoid, or one of the acute exanthemata. On examination of the cavity of the mouth, we then find not merely a fœtid but a distinctly gangrenous odour which steadily increases, and may be so strong as to become positively injurious to those in immediate attendance on the child. This is not always the case, however. In two children I found the odour so slight, even up to the time

of death, that I had to put my nose quite close to the child's lips in order to make sure of it. The swollen and tense condition of the cheek renders it extremely difficult to open the mouth or to press down the tongue with the spatula. If we succeed, in spite of these difficulties, in getting a glance at the interior of the mouth, we see on the mucous membrane of the swollen cheek, less commonly on the upper or lower lip, a large ulcer (as large as a shilling at least) of a brownish, greenish, or dirty grey colour. In the neighbourhood of this ulcer the mucous membrane is swollen and œdematous, and projects over its margins. In a child of one year, I saw a gangrenous ulcer commence on the gum of the lower jaw near the incisor teeth, which rapidly destroyed the frænum, and in a few days perforated the chin. For the gangrenous destruction of the mucous membrane spreads so rapidly, that even after a very few days a large part of it is changed into a greyish-brown, shreddy, offensive pulp, and the gum and the mucous membrane of the palate soon suffer the same fate also. With the spatula or forceps we can detach friable and ragged portions of the gangrenous *débris*. Still, as a general rule, the masses adhere more firmly to their base than one would at first expect. Fœtid saliva usually trickles from the mouth, the submaxillary lymphatic glands are swollen, and the swelling may extend downwards more or less deeply over the affected side of the neck, owing to œdematous infiltration of the surrounding connective tissue.

One would suppose that such an extensive gangrenous lesion would necessarily cause an active sympathetic affection of the whole system. And yet the extent to which this happens is by no means what one would expect. Only in the cases where noma follows immediately on an exhausting local or general disease is there marked collapse from the very first. The general health may remain surprisingly good for days. We find the children sitting up in bed, even playing, and eating with a good appetite, although there cannot be any doubt that portions of gangrenous *débris* are being swallowed with the saliva. Attacks of diarrhœa also frequently occur, and are very difficult to check. These are not explained by the post-mortem, and are probably caused by the septic decomposition of the intestinal contents, set up by the putrid matters which have been swallowed. In spite of the appearance of general good health, however, there is

nearly always some fever; the temperature in some of my cases varied between 102·9° and 104°F., the pulse and respirations being correspondingly rapid.

Even at this stage fatal collapse may suddenly take place, before the destructive process has yet implicated the entire thickness of the soft parts. Oftener, however, life still lasts after not only the gum and periosteum of the jaw have become gangrenous and the bones bare and some of the teeth have fallen out of the necrosed alveolus, but even after the gangrene has affected the corresponding side of the tongue and palate, and the perforation of the cheek or lip is completed. The most prominent part of the swelling takes at first a rosy colour, it then becomes hard, and soon acquires a blackish tinge. The further development is generally rapid. In a child of 2 in whom noma came on three weeks after scarlet fever, there was no spot to be seen on admission at 6 p.m. At 9 o'clock there was a black spot of the size of a farthing at the left angle of the mouth, and next morning it had increased to the size of a florin. There soon appears a black, dry gangrenous patch, implicating the whole thickness of the soft parts, which spreads rapidly, and may finally extend rapidly upwards to the eye and downwards to the neck. A pale red flush surrounding this patch indicates the line of demarcation of the gangrene, at which a portion of the dead tissue either separates spontaneously, or may be detached by the physician. A correspondingly large aperture, with sharp borders is then left, which permits one to see freely into the mouth where the destructive process has taken place. From this opening we also often have liquids running out. Large portions of the cheek, lip, and eyelid may be entirely lost in this way, and still the unfortunate child scarcely ever shows any signs of pain. Indeed, we sometimes see the patient sitting up and asking for food even at this stage. Increasing loss of strength, persistent diarrhœa, or extensive broncho-pneumonia (which is certainly due in part to the inspiration of gangrenous fluids into the air passages) finally bring the lamentable condition to an end, after it has lasted, perhaps, 2—3 weeks. Further, sudden death is also reported to have occurred owing to the entrance of air into the veins of the gangrenous tissue. I have usually found that consciousness lasts till death.

Only a few cases take a favourable turn, and this may even

occur in the last stage after the perforation of the soft parts is already complete. When the gangrenous destruction is arrested, there is a return of strength, if the nourishment is sufficient; and after all the dead tissue has separated the cicatrisation begins, the soft parts being drawn together as by bands. In these cases, which are very rare, there always remain cicatricial deformities—ectropion of the lower eyelid, adhesion of the cheek to the jaw, contraction of the cavity of the mouth—which may, however, be partially remedied by plastic operations. We must not, however, forget that the name "noma" is often misapplied. Children are frequently brought to me who are said to have recovered from this disease, and in proof of this a piece of necrosed bone from the alveolar margin is shown which has separated, or been removed, leaving a considerable cavity in the bone. These cases belong, however, to the class of ulcerative stomatitis described above, and not to that of real noma. For the latter condition can only be assumed when the soft parts of the cheek or lips have been more or less extensively destroyed by gangrene, and when there is, therefore, disfiguration and deformity.

At post-mortem examinations of children who have died of cancrum oris, we find very little beyond what we found on examination during life. But in the dead body we can discover more exactly how far back into the pharynx the gangrenous destruction extends. There are sometimes analogous gangrenous changes in the internal organs, especially putrid bronchitis, broncho-pneumonia or gangrene of the lungs, and catarrh of the intestinal mucous membrane. The latter, however, is absent in many cases where death has resulted from severe diarrhœa. No significance can as yet be attached to Jordan's[1] observations on the presence of moving micro-organisms in the blood.

A very important cause of noma is the "cachexia pauperum" due to very poor circumstances, bad nourishment, want of cleanliness, and damp unhealthy dwellings. Under these circumstances I have seen cancrum oris appear after an attack of bronchitis, pneumonia, or dysentery, likewise in tubercular children. It is especially apt to occur if the loss of strength caused by these diseases has been increased by a debilitating line of treatment. Whether mercury, especially

[1] *Lancet*, 1877, ii., 538.—Ranke, *Jahrb. f. Kinderheilk.*, xxvii., S. 309.

calomel, is able to cause cancrum oris after long use is a matter of controversy in regard to which physicians are still at variance. In children this remedy excites even stomatitis and salivation far less commonly than in adults, although it is much oftener used in practice among the former. I think when we bear this in mind we will agree with those who deny that calomel has any action in producing cancrum oris. My own experience, certainly, goes to support this view; and I believe every physician of experience will agree with me. It is of course evident that we must not use calomel for weeks together in sickly, exhausted children; and, indeed, such treatment would be quite unsuitable in other respects also. On the other hand, the infectious diseases—scarlet fever, measles, typhoid—are just as likely to be followed by cancrum oris as by gangrenous affections of the skin and vulva. But that, under very unfavourable circumstances, it is possible for an attack of ulcerative stomatitis to pass into noma, may, I think, be inferred from the following cases which have come under my own observation.

Albert P., 4 years old, showed cerebral symptoms (drowsiness, tremor, chewing movements, &c.) during convalescence from a severe attack of bronchitis. The treatment consisted at first of ice-compresses to the head, and later of inunction of tartar emetic ointment. This was applied so unskilfully that a gangrenous sore formed on the occiput, and, after the skin had partially separated, the bone was laid bare. About this time dirty ulcers formed on the gum and on the tongue, accompanied by salivation and fœtor oris; and about 6 days later a greyish-green ragged ulcer the size of a shilling on the mucous membrane of the left cheek. To this was soon added the characteristic swelling of the cheek, with central hardness and redness. The rapidly advancing gangrene poisoned the atmosphere of the ward. Death from rapid collapse, even before the perforation was complete.

Girl of 3 years, with general tuberculosis and fatty degeneration of the liver (*P.-M.* on March 28, 1881). She had taken ill 3 weeks before with stomatitis, and it was soon accompanied by ulceration of the mucous membrane of the cheek, bloody crusts on the lips and fœtor oris. Gradually a fœtid discharge from the mouth appeared, and on the 26th a hard, shining, bluish-red swelling of the whole right cheek. On painting the inner surface, the mucous membrane came away in fragments. Death on 27th.

There can be no doubt that noma appears in almost all cases at first as a gangrenous ulcer of the mucous membrane, and from this position penetrates the whole thickness of the soft

parts, so that one is almost certain to find the gangrene already far advanced inside by the time that redness on the outer skin and induration have appeared. I cannot, however, deny that the view that the gangrene may occasionally set in from the very beginning on the outer part of the cheek without the mucous membrane being implicated, is sometimes justified. However, I have only observed this occur in one case; and in it the noma developed from a phlegmon on the cheek.

In June, 1875, I was consulted about a child of 7 months, who, although of robust constitution, had suffered from 100 abscesses of various sizes on the most different parts of the body. Finally a large abscess formed in the left cheek, with extremely hard infiltration of the surrounding very adipose connective tissue. This was opened, and normal pus evacuated. We had often previously examined the interior of the mouth, but had always found the mucous membrane of the cheeks quite unaffected. We were therefore quite unprepared to find the abscess change into a gangrenous cavity, which gradually caused necrosis of half the cheek. The mucous membrane, however, still remained unaffected and of a normal colour; until, finally, the process penetrated inwards and destroyed the whole thickness of the soft parts, producing a perforation of the cheek the size of a half-crown. The spread of the gangrene now became arrested for the first time, healthy granulations formed all over the edges, the fever ceased, and—under treatment with a camphor dressing, and regular syringing with a ½ per cent. carbolic lotion—the healing of this immense defect was almost completed on the 13th July, when the child was carried off during an epidemic of cholera infantum.

The treatment of noma must be strengthening as far as possible (decoction of cinchona, wine, beef-tea, yolk of egg). Should the process spread in such a manner as to interfere with the swallowing of nourishment, we must have recourse to nutritive enemata (of yolk of egg, beef-tea, milk, peptone). To limit the spread of the gangrene many recommend the use of caustic substances—especially painting with fuming nitric acid—while others apply liquor ferri perchloridi or a concentrated solution of carbolic acid. Others again recommend the application of a thick paste composed of camphor to the gangrenous parts. I have seen no result from any of these remedies; and indeed we can expect to succeed only where the gangrene is confined to the mucous membrane of the mouth. If the soft parts are already entirely converted into a gangrenous mass, the only thing which can do any good is the actual cautery. I therefore advise you

in every case to use it at the very first, as soon as you discover that noma has begun on the mucous membrane. The best way of applying this is to use Pacquelin's "thermocautery." By means of this instrument, owing to its having blades of different forms, we are enabled to reach very readily different parts of the mouth which would otherwise be hard to reach. When the patch of gangrene is fully formed, we may cut away the whole of the destroyed part with one of the knife-shaped blades just as with an ordinary knife. At the same time we must be careful to remove all the gangrene by cutting well within the normal tissue. In one exceptional case, where severe bleeding resulted, the submaxillary artery had to be tied. But even when the gangrene becomes arrested after the operation, we are by no means always certain of success. On the other hand, I have seen two cases in which, although the edges of the wounds looked well and the granulations were healthy, the children died 4 or 5 days afterwards with symptoms of sudden collapse or as the result of a complication (diarrhœa, broncho-pneumonia). We must not neglect to wash out the mouth with a solution of boracic acid, salicylic acid, thymol or chlorinated lime (1½ per cent.), and to cover the mouth with a compress dipped in camphorated wine or iodoform.

### III. *The Inflammatory Affections of the Pharynx.*

Children over four years are affected almost oftener than adults with catarrhal sore-throat, especially the slighter forms of it. On the other hand, infants in the first years of life are much less commonly affected by it. As a rule the symptoms do not differ from those in later life, and I may therefore confine myself here to a few remarks.

The first thing that attracts our attention is the condition of the temperature. In most cases the local condition, the pain on swallowing—of which adults usually complain from the first—is either not a prominent symptom or is quite absent, while the fever sets in with a severity that reminds one of a serious acute disease. The sore-throat begins generally with fretfulness and lassitude. The children want to go to bed, they refuse nourishment, and usually vomit once or oftener. Shivering followed by fever, or the latter alone, now sets in, the temperature rises

rapidly to 103·2°—104° F. and over, so that the physician on examining the soft palate and tonsils finds them much reddened, and therefore is led to believe that he has here the prodromata of scarlet fever. I have even seen epileptiform convulsions set in as a result of this initial rise of temperature (vol. i., p. 173).

In a boy of 4 years, who had never previously suffered from convulsive attacks, violent epileptic fits suddenly set in on 9th April, 1885, with a temperature of 103·1° F. These lasted until evening, with intervals of coma, and then disappeared, and were followed on the 10th by catarrhal sore-throat. Recovery in a few days.

As a rule the temperature falls considerably on the second day, and may even reach the normal; and the children then remain either quite free from fever or only present slight elevations in the evening. Meanwhile, however, the local symptoms in the throat begin to appear distinctly for the first time. Many children have such a degree of nervous irritability that under these circumstances they have a pulse-rate of 136—144, which is apt to occasion anxiety; but after 24—36 hours it subsides considerably. Whether the high initial fever is sufficient to stamp catarrhal sore-throat as an infectious disease, as many would have it, is doubtful. Enlargement of the spleen, which Friedreich has observed, I have never been able to make out with certainty.

The resemblance of certain forms of sore-throat to diphtheria is especially important in practice. Very often, on the second day of the disease yellowish-white or greyish-yellow spots, of the size of a pin's head or larger, appear on the reddened and more or less swollen tonsils. These spots are somewhat rounded and vary in number, being sometimes isolated but often scattered pretty thickly, and in places coalescing so that the tonsils are covered with uneven grey or yellowish-white patches. This appearance may arouse the anxiety not only of the parents, but also of the physician. Usually the character of these spots admits of no doubt as to their innocent nature. The appearance is due to a purulent secretion which oozes out from the folds of the mucous membrane of the tonsils,[1] lies only loosely on the surface, and can be wiped off with the spatula. This, however, may cause a little blood to appear from small abrasions of the

[1] The name "angina follicularis" is therefore not justified. That of "angina lacunaris" is to be preferred.

mucous membrane. The yellowish colour of the spots also differs essentially from the greyish-white or pure white colour of the diphtheritic membrane. Still cases are not uncommon in which one has to suspend one's judgment for 24—36 hours, if one does not wish to be exposed to unpleasant reproaches. The larger confluent patches are especially apt to cause doubt. These may be pretty firmly adherent to the tonsils, and the high initial fever as well as the accompanying enlargement of the submaxillary lymphatic glands seem to support the view that the case is one of diphtheria. Even the most experienced may be doubtful as to the diagnosis here; and I must admit that I have myself been sometimes deceived in the first days of the disease. Fortunately the doubt does not last long, for while simple catarrhal sore-throat is arrested within 24—36 hours, or has even commenced to improve by that time (*i.e.* the yellow patches are being thrown off), diphtheria shows a steady increase in the extent and thickness of the membrane. I attach no special importance to the fever, nor to the enlargement of the lymphatic glands, since these features are common to both infections. Nor can one expect to arrive at a decision from a microscopic examination of the matter detached; for in either case you may find bacteria. You must also remember that white or grey fragments of false membrane consisting of amorphous fibrin, pus corpuscles and epithelium occasionally occur on the tonsils or on the arch of the palate even in simple sore-throat, rendering the diagnosis still more doubtful. In several children suffering from this kind of sore-throat, I have also seen the apex or one border of the uvula covered with a greyish-white coating. These are cases of regular croup of the mucous membrane of the pharynx. They have nothing in common with diphtheria excepting the mere superficial appearance, and are of purely inflammatory origin (Trousseau's "angine couenneuse"), and—as I have frequently observed to be the case in adults—they may be combined with abscesses in the tonsils.[1] Under these circumstances you will always do well to defer your judgment as to the nature of the disease for 24—36 hours, to isolate the patient for a time from other children, and to observe the further course of the case carefully. At any rate very many cases are put down off-hand by superficial observers as diphtheria which are nothing

[1] *Cf.* E. Wagner, *Jahrb. f. Kinderheilk.*, xxiii., S. 407, 409.

but severe cases of catarrhal sore-throat. The wonderful results of many physicians who say they have cured almost every case of diphtheria with chlorate of potash and other drugs are really to be explained in this way.

It cannot be denied, however, that slight cases of real diphtheria are met with, among those doubtful ones, which end favourably in 6—8 days after the separation of all the membranes. The cases where several children in one family have taken ill in the same way and at the same time, or in succession, have appeared to me especially suspicious when the patches which were originally confined to the tonsils have made their appearance also on the margin of the soft palate or the uvula, but particularly when there is much discharge from the nasal mucous membrane. Although the presence of these features in a case does not absolutely prove that it is one of infectious diphtheria, yet under the circumstances one is bound to treat it as such. If we then, as not uncommonly happens, discover afterwards that the patients are affected frequently, perhaps every year, by a bad attack of this ambiguous kind of sore-throat—it is better to acknowledge that we have been mistaken than run the risk of falling into the conscious or unconscious error of those who boast of repeated recoveries from diphtheria in the same child. Finally, I should mention that we sometimes find in children tolerably extensive and even pretty deep greyish-yellow uneven ulcerations of another kind on one tonsil (very seldom, as far as my experience goes, on both tonsils at once). These ulcerations, judging from the yellow points of pus which are here and there visible in the neighbourhood, have arisen from the coalescence of little abscesses lying close together. They, therefore, have nothing whatever to do with diphtheria, nor yet with syphilis, as has sometimes been wrongly assumed, and they almost always get well spontaneously after 8—14 days.

In every case of catarrhal sore-throat, whether the degree of fever with which it sets in be high or not, I advise you to have the children kept in bed for some days, and only to give them a mild purgative (mist. sennæ co., confect. sennæ, Form. 7, 28) if their bowels are not sufficiently open. I do not expect any great advantage from the use of chlorate of potash, which is often ordered in these cases, and gargling with decoction of

elderberries or mallow can only be used in the case of children who are old enough to understand how to do it. When there is a great tendency to these sore-throats we may try to avert their frequent recurrence by painting the tonsils daily with a solution of nitrate of silver (grs. xxii. to ℥i.). Still, this does not invariably succeed, and it is only as the child gets older that the tendency to recurrence disappears.

As the result of the frequent recurrence of these attacks of inflammation, the tonsils may in time become hypertrophied. As a rule, however, I have seen this hypertrophy in children who had not suffered at all, or, at least, very seldom from sore-throat. I have also been able to ascertain, by definite symptoms, the presence of scrofula as a predisposing cause only in a few of the cases, although its presence is often assumed without sufficient reason. The development of the hypertrophy of the tonsils is so slow that the symptoms do not usually appear during the earliest years of life, and we rarely have an opportunity of observing the affection as early as the first or second year. There are three symptoms in particular which should direct your attention to the tonsils:—an unusual snoring of the children during sleep, or even noisy breathing during the day; an altered and nasal tone of the voice; difficulty of hearing, caused by the obstruction of the Eustachian tube. On examination, we find both tonsils extremely large (and in rarer cases one only) so that the entrance to the pharynx appears more or less narrowed, and in extreme cases almost entirely closed, by the contact of the tonsils with the uvula. We then see the two tonsils almost touching one another, and the uvula with the soft palate pressed backwards and upwards, or even forwards. At the same time deglutition may be quite uninterfered with, and the only pain and difficulty in swallowing under these circumstances is that due to the frequently occurring attacks of sore-throat. The interference with the breathing may also cause sudden wakening from sleep and screaming, and cases have been reported in which there were regular attacks of pavor nocturnus, so that in children suffering from attacks of this latter disease it is always well to examine the pharynx. I must also here remind you that neither in cases of sore-throat, nor even where the tonsils are considerably hypertrophied, can we feel the enlargement from without in the submaxillary region, as not

merely the laity but also many medical men have asserted. I have frequently endeavoured to press the enlarged tonsil outwards with the finger introduced into the mouth, so as to make it possible to feel it from outside, but I have never succeeded, and in fact this is an anatomical impossibility on account of the number of structures which intervene (superior constrictor of the pharynx, bucco-pharyngeal fascia, platysma, parotid gland, skin). Therefore, what you do feel under these circumstances are either enlarged lymphatic glands or infiltration in the connecting tissue, and not the tonsils.

When a considerable hypertrophy of the tonsils takes place in very early childhood, a malformation of the thorax similar to that in rickets (the so-called pigeon-breast) may be produced owing to the interference with the free entrance of air into the lungs (Dupuytren, Shaw, &c.). In this condition the external atmospheric pressure exceeds the internal owing to the deficient filling of the lungs, and the yielding costal cartilages are pressed inward so that the thorax is flattened laterally and the sternum projects more than usual. Narrowing of the nostrils and arrested growth of the upper jaw with considerable concavity of the palate and crowding of the teeth have been observed in such children. Only in exceptional cases, however, has the interference with breathing reached such a degree that tracheotomy had to be resorted to.

The only remedy to be recommended is excision, or at least partial resection of the enlarged tonsils.

## IV. *Contagious Parotitis.*

(Cynanche Parotidea—Mumps.)

In the region in front of the ear, and under the angle of the jaw, there occur in children various swellings which may be readily confounded with one another by the inexperienced. In this region you often find during the first dentition, but also much later, a diffuse swelling generally confined to one side; this is at first doughy in consistence and of a normal colour, gradually becomes harder and red, and finally presents fluctuation, after which a large amount of pus is evacuated, either spontaneously or on incision. I should not have mentioned these common connec-

tive-tissue abscesses (which appear to arise from adenitis of the lymphatic glands) at all, were it not that I have repeatedly found that in the first stage they are taken for mumps. The same may be said of the œdematous swellings which occur as the result of stomatitis or alveolar periostitis. The swelling of contagious parotitis is distinguished both by its consistence and its course from that of all the other forms. In the majority of cases the children, who are generally between 3 and 8 years, are brought to you with the disease already developed. You then find a diffuse, more or less extensive swelling on one or on both sides, in front of the ear. This swelling is generally soft, but in many cases somewhat resistent. It extends downwards, over the angle of the jaw, and ends behind it in a rounded point—the lower end of the parotid gland, which can often be distinctly felt. If both sides are simultaneously affected, it may happen in severe cases that the diffuse swellings on the two sides meet under the jaw, and the whole submaxillary region is swollen like a sausage. Usually, however, only the parts in front of the ear, and those behind the lower jaw, are prominent, and they give the face an appearance of striking breadth when seen from the front. The swelling may even extend downwards over the neck, as far as the outer end of the clavicles, and in this case, when both sides are affected, the head has an odd pear-shaped appearance. A few enlarged lymphatic glands can often be felt under the jaw. The skin over the swelling is generally of a normal colour, less often slightly red, and scarcely tender at all even on pressure; although on eating, chewing hard substances, or attempting to open the mouth wide, many of the patients complain of pain. I have occasionally found the veins over the right temple and those near the eye on the affected side much distended, probably owing to the pressure which the enlarged parotid was exerting on the facial vein. Like most other medical writers, I have failed to find changes in the salivary secretion; but I have often found a slight degree of tonsillitis. In many cases the child feels in other respects perfectly well. Although almost always we find, on using the thermometer, a slight rise of temperature (100·4°—101·3° F.), at any rate at the very commencement or during the first day; only rarely have I seen children in whom the fever was pretty high (102·2°—104° F), and there was violent headache and vomiting.

The disease lasts, on an average, 5—7 days. During this time the swelling increases for two or three days, remains stationary for about 48 hours, and then disappears gradually. Still, I have seen the course prolonged to 10—14 days, owing to the two sides being successively affected. The metastasis to the testicles, which is occasionally met with in adults, I have never seen in children,[1] nor yet the ending in suppuration nor the permanent induration of the parotid gland. In all my cases complete resolution occurred.

Very recently various writers have observed serious disturbances of hearing—sudden and incurable deafness—after mumps. This was explained partly by implication of the mucous membrane of the pharynx and extension of the process through the Eustachian tube to the middle ear and labyrinth, and partly by the Glasserian fissure of the petrous bone having become affected.[2] I have never myself observed this complication, although I have seen one case (to which I shall return later on) where nephritis followed.

The fact that it almost never attacks the same individual more than once, its undeniable contagiousness, its stage of incubation of about 14 days, and its frequent occurrence as an epidemic—all render the infectious nature of this disease a matter of certainty. We must assume that the infective material (the nature of which is still unknown to us[3]) enters the parotid gland by the orifice of Steno's duct, and there gives rise to a state of irritation with parenchymatous swelling of the gland. I have never been able to discover the enlargement of the spleen and of many of the lymphatic glands, which some say they have found in these cases, and which is held to prove their infectious nature. It is, moreover, still doubtful whether the parotid gland alone has the property of taking up and retaining the contagium, or whether it shares this with the sublingual and submaxillary glands. Some cases which

[1] A few cases have been reported of this metastasis in boys of 12—14 years, even ending in atrophy of the affected testicle.

[2] Seeligsohn, *Klin. Wochenschr.*, 1883, No. 13 u. 18.—Roosa, *Centralbl.*, 1883, No. 41.—Moos, *Klin. Wochenschr.*, 1884, No. 3.—Gruber, *Wiener allgem. med. Zeit.*, 1884, Nos. 4—6.—Pierce, *Arch. f. Kinderheilk.*, vi., S. 373.

[3] The bacteria which French writers have described as occurring in the saliva, urine, and blood of these patients, require further confirmation. *Cf.* Capitan and Charrin, *Soc. de biologie*, 28 Mai, 1881.—Ollivier, *Revue mens.*, Juillet, 1885. —Boinet, *Lyon. méd.*, 1885, 9.

Penzoldt[1] has published, and also the observations of Soltmann[2] are in favour of the latter view, and I have myself treated one adult in whom both submaxillary glands were swollen after the occurrence of gastric and febrile premonitory symptoms, and in whom a metastasis to the right testicle finally took place, although the parotid had not become affected. The period of incubation of the disease varies between 14 and 22 days.

Considering the invariably favourable prognosis, it is unnecessary to isolate a child suffering from this form of parotitis from other children. I advise you, however, in any case to keep the child in bed for the first few days, and after that to keep him confined to one room at any rate. The swollen parts should be covered with wadding. I have tried the application of an ice-bag, but this does not shorten the course of the disease, and is therefore unnecessary.

### V. *Inflammation of the Floor of the Mouth.*

The disease which I describe under this name is on the whole a rare one, and is mentioned by only a few authors. Holthouse[3] describes a case of this kind by the name of "subglossitis," and I shall now give you three other cases which have come under my own observation.

A pale girl of 9 years, admitted into the ward in May, 1878, with otorrhœa of both ears, but otherwise healthy, complained on 1st October of pain in the neck, although nothing abnormal was found on examination. On the next day, however, the whole floor of the mouth was much swollen and tender, the mucous membrane pale all over. Opening the mouth and every movement of the tongue painful and difficult. Copious secretion of saliva. Temp., m. 102·9°, ev. 103·6° F. In spite of a powerful purgative and the application of ice-bag under the jaw, the swelling was even larger on the 3rd, and could also be made out externally under the jaw. It was doughy in consistence and œdematous. The tongue was pressed upwards by it and completely immovable. The upper and lower jaw were about $\frac{2}{3}$ inches apart, the latter being immovable. Temperature to 100·9° F. As the symptoms had not decreased by the 4th and the salivation was even more marked and the temperature had risen to 104° F., I ordered 5 leeches to be applied under the jaw, and chlorate of potash to be taken internally. Even

[1] *Deutsche med. Wochenschr.*, iv., 19, October, 1878.
[2] *Jahrb. f. Kinderheilk.*, xii., S. 409.
[3] Hirsch-Virchow, *Jahresber. f.* 1871, ii., S. 505.

by the evening there was relief of the symptoms and diminution of the swelling. On the following day the temperature also fell, and on the 6th it was only m. 100° and ev. 101·8° F. Pain and swelling distinctly less, lower jaw now movable, mucous membrane nowhere reddened, tongue not swollen, its surface covered with a whitish coating. This showed under the microscope nothing save the ordinary fungi and a large amount of fattily-degenerated epithelium. During the next few days there was steady improvement and rapid recovery; so that the whole process lasted about a week. After an interval of 3 weeks, however, a relapse occurred without any evident cause, the symptoms being quite similar. This second attack again lasted 6—7 days, and recovered under the same treatment. Since then the child has remained healthy, except for a slight otorrhœa.

A girl of 7 years, admitted on 27th Feb., 1883, had taken ill 4 days previously with fever and slight nocturnal delirium. There was a diffuse swelling, extending from the posterior border of the right sterno-mastoid up to the middle line of the neck, and from the mastoid process to the commencement of the larynx. This swelling was soft, hot and reddened. Mouth half-open, excessive discharge of saliva, thickly coated tongue, swallowing interfered with. Owing to the difficulty of opening the mouth wide, the pharynx could scarcely be examined. An ice-bag was applied, and fragments of ice given internally. On the 28th the tongue was pressed forwards and upwards, tender, immovable; the speech very indistinct. The floor of the mouth was much swollen, and here and there there was a dirty-grey coating on it. Just under the tongue, to the right of the frænum there was a small opening, out of which bloody serum oozed. T. 102·7°. Gargling with solution of thymol; 5 leeches applied to the swollen submental region. On the 1st, condition unchanged. On the 2nd, during the night, a copious discharge of pus from several places under the tongue. On pressure over the submaxillary region, pus oozed out as from a sponge. The general health seemed good, and the child could drink well. T. 99° F. During the next few days the swelling subsided, and there was less discharge of pus. The tongue returned to its normal position, and the mouth could be readily opened and shut. On the 6th, recovery, only a few of the cervical glands remaining slightly enlarged.

A girl of 7 years, admitted on 27th May, 1884, with a fluctuating abscess in the right submaxillary region. 1½ tablespoonfuls of offensive pus evacuated by incision. The whole floor of the mouth much swollen, pressing the tongue upwards against the palate, and discharging offensive pus from several openings. T. ev. 102·6° F. Gargling with lukewarm solution of thymol, antiseptic dressing to the wound. On the 6th June discharged cured.

In these cases we had a phlegmonous inflammation of the

connective tissue under the mouth, which spread through the mylo-hyoid muscle to the adjacent connective tissue and, after causing high fever and severe local distress (extreme swelling and pushing upwards of the tongue, salivation, &c.), ended with a discharge of pus under the tongue, or even externally (in the third case). A peculiar feature of the first case was the relapse which took place three weeks afterwards. I am still quite in the dark as to the causes of this affection. In a child of two months who recovered from it and afterwards died of convulsions Tordeus[1] found the submaxillary gland destroyed by suppuration; but in my cases none of the salivary glands were affected. In a case described by Du Prè,[2] the disease was said to have been caused by the child's chewing a blade of grass. There is as yet no ground for assuming an infectious origin.

## VI. *Stricture of the Œsophagus.*

In extremely rare cases, stricture of the œsophagus appears as a congenital condition, and then, from the very first day, any milk which is taken is at once rejected through the mouth and nose. In one case of this kind, that of a boy of four years, the swallowing was alternately better and worse, so that we were obliged to assume that the congenital stricture which we had found on examination was temporarily aggravated by the swelling of the mucous membrane.

Stricture due to compression by neighbouring organs or by tumours, or even to carcinomatous degeneration of the walls of the gullet, is almost as rare in childhood as the congenital form. On the other hand, cases of stricture caused by the application of caustics to the œsophagus are much commoner than in adults. These cases are usually due to drinking a solution of caustic soda, which is used for washing and scouring, and is often mistaken by the children for "Weissbier." I have often known this accident happen to children between 2 and 12 years, and on one occasion to a boy as young as 15 months. If some days have passed since the drinking of the alkali, you still find distinct traces of its caustic action on the mucous membrane of the mouth and pharynx, with great difficulty of

[1] *Deux cas de l'angine sous-maxillaire*, &c.: Bruxelles, 1885.
[2] *Journ. de méd. de Bruxelles*, Déc., 1886.

swallowing and the retching up of mucus, sometimes mixed with blood. The youngest of these children suffered also from aphonia, which was due to the action of the caustic and to swelling of the glottis, and was afterwards replaced by extreme hoarseness. Since older children find out their mistake whenever they begin to swallow, and then spit out part of the fluid, the injurious effect may in their case be limited to the mouth and pharynx, or a small quantity may get into the œsophagus, but no further. This fact explains the comparatively rare occurrence of inflammatory stomach-symptoms in such cases. Nevertheless I have several times observed symptoms of gastritis in recent cases; there were persistent violent pain in the region of the stomach, vomiting of all food and drink and of large quantities of mucus, constipation, fever, great tenderness of the whole epigastric region, and, in one case, discharge of blackish blood from the anus without vomiting of blood. In the majority of cases, however, the children come under treatment for the first time several weeks after the accident has taken place, *i.e.*, at a time when the traces of burning in the mouth and throat have long disappeared, and the symptoms of stricture of the œsophagus from cicatricial contraction of the ulcers are already well-marked. In these cases the main symptom is always the vomiting, or rather retching up of any food that is taken, along with tough masses of mucus, immediately after swallowing—even before the meal is finished. At first fluids can still be swallowed, but later on they cannot; I have seen cases where not even a single teaspoonful of water could be taken without violent retching at once setting in. Many children indicate exactly with the finger the place behind the sternum to which the food and drink are able to reach. The keen appetite which remains, and which can in no way be satisfied, makes the case the more distressing; and even within a few months the children fall victims to regular starvation. Increasing emaciation, which at last reduces the child to a skeleton, an earthy complexion, sharpened features, and the most extreme weakness—are all necessary consequences of this condition. In a boy of 10 who was brought to my ward in this wretched state, and who had a subnormal temperature and a cyanotic tinge of the skin and mucous membranes, I found the pulse only 44, and so small as to be scarcely perceptible. This

was probably due to the heart-muscle's participating in the general atrophy. Under these circumstances death from increasing exhaustion is inevitable, unless we can manage to improve the nutrition, either by dilating the stricture or in some other way.

In order to discover the situation and degree of the stricture, we introduce the œsophageal sound, and if this—as usually happens—fails to go through the contraction, we try a gum-elastic catheter or a whalebone probang fitted with a small olive shaped end of steel. The extreme degree of contraction in these cases is often surprising, and obliges us to use thinner instruments until at last we find one that can get through the stricture, which may be situated either in the upper or lower part of the œsophagus. Sometimes we have been able distinctly to make out two strictures, one of which could be more easily overcome than the other. The statements of children who are old enough to be intelligent, and who of themselves indicate the situation of the obstruction, are generally confirmed on examination with the probang. The only means of cure is the gradual dilatation of the narrowed place by means of bougies or probangs fitted with an ivory or metal olive-shaped end introduced every day. This proceeding, however, demands infinite patience and caution. We must be very careful to avoid a forcible introduction of the instrument, as this would cause a danger of perforation; and we must, therefore, always begin with a probang or bougie sufficiently small to go right through. We may, indeed, be obliged to begin with a piece of catgut. If we leave the instrument in daily for about 5 or 6 minutes, and after a while for longer, we are often able to introduce a larger one within a few days. When we succeed in this, the dysphagia always diminishes and small quantities of fluid get into the stomach without retching taking place. Gradually the child's strength and the state of its nutrition improves, and I have often been astonished to observe how rapidly under these circumstances the cheeks can fill out again and the colour improve. Thus it happened, for instance, in the case of the boy mentioned above; the slow shabby pulse returned completely to its normal condition within a few weeks, and the bluish tinge of his cheeks gave place to a normal colour. In these cases as much patience and perseverance is required of the mothers as on the part of the physician. For, since the cicatricial connective tissue which by its contraction produces the

stricture still possesses the tendency always to contract again after its artificial dilatation, the success of the treatment is always only temporary unless the dilatation is repeated daily for many weeks or even months. In practice among the poor, however, and in the polyclinic, this perseverance is very often wanting; all the more so because the introduction of the instruments generally occasions violent screaming and retching, and is very distressing to the mothers. Only in one case at the polyclinic[1] have I as yet been able to obtain complete recovery; while in the others there was only a degree of improvement, and the patients ceased to attend. On the other hand, treatment in the hospitals promises better results; and I myself have notes of several cases which were discharged cured. But even in such cases as these the recovery is not always permanent.

A boy who had acquired a stricture of the œsophagus in his 4th year by drinking an alkali was discharged from the ward in a satisfactory condition after dilatation had been continued for 5 months. He was able to swallow soup, bread-and-milk and soft vegetables easily, but no meat. When he returned to the ward in his 12th year, he was no longer able to swallow even fluids. He was extremely emaciated, and only the finest bougies could be passed through the stricture, which was situated above the cardiac orifice. After treatment continued for 6 weeks, he was discharged from the hospital apparently cured. The further course of the case, however, has remained unknown.

The local treatment is rendered more difficult in a number of the cases by the fact (which has been proved by post-mortem examination) that the stricture may not only be very tight and rigid, but also of considerable length. When the cases have lasted for some time, also, a dilatation of the œsophagus takes place above the stricture, and the instrument is apt to get into the pouch thus formed. In such incurable cases the only remedy that remains is gastrotomy, which in recent times has been repeatedly performed with success. During the treatment the nourishment is to be supplemented by enemata of yolk of egg, beef-tea, wine, or peptone.

## VII. *The Diseases of the Stomach.*

The stomach, like the œsophagus, is much less frequently affected by serious disease in children than in adults. I have

[1] *Beiträge zur Kinderheilk. N. F.*, S. 275.

never observed cases of acute inflammation either of the mucous membrane or of the submucous tissue apart from those which I have just mentioned as having been caused by drinking a caustic fluid. Chronic catarrh of the mucous membrane of the stomach and round ulcers of it are rare, and carcinoma quite exceptional. Although a few cases of round ulcer of the stomach have been described,[1] and although I have frequently had older children under treatment whose symptoms (cardialgia and hæmatemesis) almost put this diagnosis beyond a doubt,[2] still, I think that I need not dwell upon this subject here as I have nothing to add to the symptoms so well-known in adults. The tubercular ulcers which are occasionally observed, the small ulcerations in new-born children (vol. i., p. 66), the diphtheritic processes in the mucous membrane which I have found in the stomach of children who had died of diphtheria, the extravasations of blood, &c.—have, all of them, a merely pathological interest and cannot be diagnosed. This being so, I shall confine myself here to the consideration of a few morbid conditions which do not occur, indeed, exclusively in childhood, but which, when they are found, present peculiarities which are important for practice.

### *Gastric Dyspepsia.*

This is the most important of these conditions on account of the frequency of its occurrence. You will remember the account which I endeavoured to give you of the dyspepsia of new-born children and of older infants (vol. i., pp. 73, 125). I shall therefore speak here only of the dyspepsia of older children, which is one of the commonest diseases a practitioner meets with. Every time the stomach is overloaded with food, and especially when the food is of a kind difficult of digestion and unsuited to the child's stomach, a more or less speedy natural cure may take place, at this as at any other age, by the onset of spontaneous vomiting; or even by copious offensive evacuations. When this does not take place, the "gastric attack" developes into a "status gastricus sive saburralis," a condition of whose nature we know practically nothing in spite of its frequency. Whether acute catarrh of the mucous membrane, or a chemical alteration of the

[1] *Cf.* Chvostek, *Archiv f. Kinderheilk.*, iii., S. 267.

[2] *Beitr. zur Kinderheilk.*, 1861, S. 89 and *N. F.* 1868, S. 278.—Wertheimber, *Jahrb. f. Kinderheilk.*, 1882, xix., S. 79.

digestive juices, or distension of the stomach-wall with gas, or whether—as I believe—a combination of these conditions exists, we do not know. We must, therefore, content ourselves in the meanwhile with studying the symptoms. The children have no appetite, the tongue is more or less covered with a white or yellowish coating, and looks as if spread over with thick felt, and there is an offensive smell from the mouth, and an alteration of temper. Many suffer from nausea and quickly vomit up again everything they take. At the same time the children are fretful, languid, dull-eyed, complain of headache, and cannot hold their head up for long. Fever is often but not always present, and it is sometimes very high (102·2°—104° F.) with a very rapid pulse (120—144 and over), great thirst, redness of the cheeks, rise of temperature in the evening and nocturnal delirium, which is also, though less commonly, present during the day. The bowels are usually confined, less frequently the motions are loose and copious, and in many cases the epigastric region is somewhat distended, tense, and tender on pressure. Many also complain spontaneously of pressure or pain in the region of the stomach. Under these circumstances the beginner is apt to be perplexed, and even the experienced practitioner is not always able at once to give a definite opinion with absolute certainty. We think, of course, at once of indigestion, but the certainty of our diagnosis is interfered with by the haunting fear that typhoid fever or tubercular meningitis may be in process of development. I have already (vol. i., p. 319) spoken of the diagnosis of the latter, and I shall only add that the thick whitish-yellow fur on the tongue and the foul breath are in favour of the presence of dyspepsia. Moreover, in doubtful cases we need not be afraid to use an emetic, which even in the case of a mistaken diagnosis could not have any prejudicial or even any important action in the first stage of meningitis, or of commencing typhoid. For acute dyspepsia, however, if it has recently arisen, or even if it is some days old, there is certainly no better remedy; and I believe that the present school of treatment, made timorous by the former abuse of emetics, is far too prone to neglect the use of this class of remedies. This tendency has gone so far that physicians have asked me, in perfect earnest, whether I ever use emetics! After the emetic has acted properly (Form. 6), the whole group of apparently serious symptoms often vanishes as if by magic, and

if the medicine has not at the same time acted on the bowels, all that we now need is a mild purgative (Form. 7, 28). Should the loss of appetite and the fur on the tongue persist, small doses of hydrochloric acid (Form. 3) are sufficient to remove the morbid condition in a few days. A very important matter, however, is the diet, which, even in those who are doing very well, must consist for several days exclusively of light food (thin porridge with mucilaginous drinks, thin broth, biscuit, and such things). The timidity about using emetics which is now so common, has generally for its Nemesis a longer continuance of the condition. If it is six or seven days since the illness began, we need not expect any rapid action from the emetic. In these protracted cases I have the children confined and put upon the strictest diet and hydrochloric acid; and to this, when there is constipation, I add compound senna mixture or infusion of rhubarb with tartrate of potash (Form. 7). When the appetite is long of returning, I should recommend the use of tincture of rhubarb, 2—3 teaspoonfuls daily, continued for several days.

I have already repeatedly pointed out to you that nervous symptoms, epileptiform convulsions (vol. i., p. 167), clouding of the intellect (vol. i., p. 170), aphasia and retardation of the pulse (vol. i., p. 319) may arise reflexly from irritation of the nerves of the stomach due to dyspepsia, and may readily mislead the physician. Through the same reflex mechanism, a series of symptoms may arise confined principally to the respiratory system, which I have designated "asthma dyspepticum."[1]

> On the 10th May, 1875, a girl 9 years of age came to my polyclinic with anxious sunken features, and slight cyanosis of the face and of the nasal and buccal mucous membranes. The respiratory movements of the thorax very superficial, 70 in the minute. The alæ nasi and the accessory muscles of respiration working, noisy expiration. Pulse very small, about 108. Very great weakness, so that the mother's statement that the child had come on foot to the hospital (about 10 minutes' walk) was received with surprise. The thoracic organs normal in every respect. The child complained continuously of great dyspnœa and weakness, and soon afterwards also of headache and tenderness in the region of the stomach. This region seemed, when the child lay on her back, to be somewhat distended, gave a tympanitic note, and was very tender on pressure. As to the history, we were only

[1] *Berliner klin. Wochenschr.*, 1876, No. 18.

able to find out that the child had been well until the previous evening, but had then begun to complain of pains in the region of the stomach. She had passed a very restless night, and cyanosis and dyspnœa had appeared in the morning. As the case seemed to be serious and the cause obscure, I did not venture any decided treatment, but ordered small doses of morphia. There was, however, no occasion to carry out this treatment; for the child had scarcely reached home when she began to complain of severe nausea, and from that time till evening frequently vomited up fragments of food, including large undigested pieces of hard egg, which we now discovered to have been eaten very hastily on the previous day. When these masses were got rid of, peaceful sleep, and a feeling of good health at once followed. On repeated examination in the ward on the following day, the child seemed perfectly well, so that nothing remained to be done but to regulate the diet.

In this case we have a combination of apparently serious asthmatic symptoms due to irritation caused by undigested food, and vanishing as if by magic when the irritating matters were got rid of. Very great dyspnœa, cyanosis, extreme smallness of the pulse and coldness of the hands—all the symptoms being present without the slightest abnormality of the lungs or heart. Nor could we assume compression of the thoracic organs by a dilated stomach, for no abnormally high position of the diaphragm could be made out. Another case had an entirely similar course.

Boy of 9 years, brought 9th January, 1876. Pains, for 6 days previously, in the region of the stomach, which was distended and tender. Respiration superficial, 50 in the minute; pulse small, 120 and more; face and mucous membranes cyanotic. On examination we found incompetence of the mitral valve, with moderate dilatation of the right ventricle. Nothing abnormal in the lungs. There was also a thick fur on the tongue and an offensive smell of the breath. I ordered an emetic at once, the success of which was apparent. On the very next day the respirations had fallen to 32, the pulse was normal, and the cyanosis quite gone. On the 11th the child was perfectly well, apart from the objective signs of the old heart-complaint.

These two cases will suffice to illustrate to you the symptoms of "asthma dyspepticum." In my work above referred to you will find yet another striking case, that of a child of 9 months just weaned who was suffering from dyspeptic vomiting. In the case of this infant as in the others such violent dyspnœa set in,

with small, almost uncountable pulse, cyanosis and apathy, that we might have assumed the presence of a serious affection of the thoracic organs, although on repeated examination we failed to find the slightest abnormality of the heart or lungs. This case also ended in complete recovery within a week. Some quite similar cases were subsequently published by Silbermann,[1] and I have myself had under treatment another boy of 12 who presented dangerous symptoms of collapse (coldness of the extremities, extremely rapid small pulse, quick breathing, slight cyanosis) along with dyspeptic diarrhœa, and who finally recovered under treatment with hydrochloric acid. These processes are not sufficiently explained by the experiments of S. Mayer and Pribram,[2] who observed an increase of pressure in the arterial system and a retardation of the pulse on the application of various kinds of irritation to the stomach in dogs and cats. These experiments can, at most, only explain to us such cases as those mentioned in vol. i. at p. 320, in which the pulse-rate was distinctly retarded by reflex irritation of the inhibitory fibres of the vagus in dyspepsia. In our cases of "asthma dyspepticum" the pulse was not slowed, but on the contrary very much accelerated. I shall not decide whether, as Silbermann thinks, we have at first a paralysis of those fibres of the vagus which control the heart's action. I would remind you at the same time of the peculiar feeling of oppression which not unfrequently occurs in cases of dyspepsia, with or without flatulence. This consists in the patient's often having an inclination to inspire as deeply as possible and not being able to do so. As soon as he does succeed in doing so, the troublesome necessity for taking deep breaths disappears for a time but soon returns, and the attack ends with a succession of rapidly repeated spasmodic yawns. Here also we have probably to do with a reflex action in connection with the vagus, which may also under similar circumstances excite an intermittent action of the heart. I have seen this kind of asthma, not only in adults, but also repeatedly in children of 6—12 years of age who were brought to me by their parents with the idea that they were suffering from disease of the heart or lungs. In two or three of the cases this condition had already existed for weeks with varying intensity, and was so

[1] *Berliner klin. Wochenschr.*, 1882, No. 23.
[2] *Sitzungsber. d. Wiener Acad.*, Juli, 1872.

striking that during one minute several deep, and yet not altogether satisfactory, inspirations were taken in which the shoulder-muscles largely participated. In these cases, also, we could almost always ascertain that dyspepsia was present or that the large intestine was full of fæcal accumulation, and treatment directed against these conditions was generally successful. The nervous nature of this asthma was also shown by the fact that when the children's attention was in any way distracted—*e.g.*, by their being examined with the stethoscope—the asthma ceased at once, but returned as soon as the examination was at an end.

Chronic dyspepsia in children is, as I have already remarked, far less commonly the result of stomach-disease than of some other important chronic disease, either general (tuberculosis) or local. I may also mention here that loss of appetite, distension of the stomach, nausea and constipation very often occur in anæmic children, and disappear when the condition of the blood is improved. You will also often have children brought to you because their mothers are anxious about their loss of appetite, and on examination they present absolutely nothing abnormal. Closer examination shows further that the children are eating just as much as they need, although they are not satisfying the exaggerated wishes of their parents in the matter. In these cases you will often find that the children are very fastidious in their eating, and have a decided aversion to many kinds of food, for example to beef-tea, meat, or even milk. All those things must of course be taken into account before we rashly assume the presence of morbid anorexia. Equal caution is required in judging of the various forms of "stomach-ache" in children, which I shall discuss under the name of cardialgia.

### *Cardialgia.*

The significance of these stomach-pains—which occur from about the 6th year and upwards, and are common enough—is much harder to make out than is the case in adults; and this is owing to the inadequacy of the child's description of the seat and nature of the pain. I have often found on examination that the so-called stomach-pains were not situated in the stomach at all, but in the colon transversum, and that they began in the epigastrium or in either hypochondrium, and radiated from hence

towards the umbilicus or descending colon. They were therefore not "cardialgia," but "colic"—due to over distension of the large intestine with gas or fæces. I have only rarely observed actual stomach-pains as the result of indigestion, and when they did occur they were always accompanied by other dyspeptic symptoms such as I have already described. In these cases, also, an emetic brought speedy relief; and I therefore advise you not to be deterred from using this remedy in acute dyspepsia merely owing to apprehension of there being an inflammatory condition of the stomach—unless, indeed, you can find a definite cause which might have produced such an affection. Thus in one child who had eaten some boiling-hot turnips a few days previously, I found persistent pain in the epigastrium, especially after eating, so that every form of nourishment was refused. In this case we were certainly compelled to assume an injury to the mucous membrane; and in fact the pains were completely relieved in a few days by taking nothing but iced milk and an oleaginous emulsion.

Regular paroxysms of cardialgia, apart from the cases in which one can assume the presence of a round gastric ulcer, I have only observed in older chlorotic girls of 10—16 years of age; and they resembled entirely those occurring in adults. The swollen and tense condition of the epigastrium during these attacks, which compels the patients to put off all confining articles of clothing, belts, &c., points to a spasmodic contraction of the gastric orifices. This prevents the gases contained in that organ from escaping and thus produces an excessive and painful distension of its walls, which rapidly disappears after eructation and the passage of flatus. In several girls who were approaching puberty, or had already menstruated two or three times, and also in one boy, I have had an opportunity of observing dilatation of the stomach; and to this condition I now desire to direct your attention. The chief symptom in these cases was an unusual fulness or globular projection of the epigastrium which, according to the degree of the flatulent distension, was either tolerably soft or extremely tense. The percussion-note, which was generally dull in the upright position, became loud and tympanitic when the child was lying down, and in that position the distension and tenderness were generally distinctly less. When a soda powder was given, however, the swelling and tension at once re-appeared,

and the contour of the dilated stomach could often be distinctly recognised. The degree of distension did not always increase perceptibly after eating or drinking, and as cardialgia and other dyspeptic symptoms were almost always absent, the only things complained of were a feeling of tension in the region of the stomach, eructation or transitory nausea, but, above all, shortness of breath on movement or after eating. In one case, with very extreme dilatation, there was an upward displacement of the heart to the extent of one intercostal space. On the left side of the thorax the note was loud and tympanitic in front, from the costal margin up to the 4th rib, just as in pneumothorax. The apex-beat was found between the 3rd and 4th ribs, and, accordingly, the cardiac dulness could be most distinctly made out in that region. The degree of dilatation, moreover, was never constant but varied from time to time; and these variations could certainly not always be explained by eructation of gas.

The state of our knowledge of the etiology of these cases leaves much to be desired. In one girl of 12 the condition was referred by the mother to an attack of varioloid from which the patient had recovered 7 weeks previously. In another child of the same age it was supposed to be due to an attack of typhoid fever. In most of the cases, however, we find the dilatation of the stomach either preceded or accompanied by hysterical symptoms—fits of screaming or weeping, cardialgia, attacks of somnambulism and ecstatic symptoms such as I have already described (vol. i., p. 230). Anæmia is found sometimes, but not always; indeed some girls appear particularly healthy in this respect. In one case the attack was accompanied by regular epileptic fits. I consider that the symptoms in most of these cases are occasioned by a spasmodic contraction of the openings into the stomach, which need not be regarded as of great importance. As a matter of fact, when the trouble has lasted for some weeks or months with varying severity, it either disappears spontaneously or is replaced by other hysterical symptoms. I cannot from my own experience determine whether the onset of menstruation has a favourable effect or not; but judging from the analogy of other hysterical symptoms occurring about the time of puberty, I am inclined to think that it probably has. Of the remedies which I have tried, only one (namely, the faradic current) has had any effect; and that merely tem-

porary. When we apply one electrode to the vertebral column, and the other to the distended epigastrium, the latter always collapses at once, even although no eructation takes place. It is doubtful whether this result is due to the action of the abdominal muscles or to an independent contraction of the muscular walls of the stomach. Unfortunately this successful result is always merely temporary. It sometimes lasts only for a few hours, and at most only two or three days. Even in the few cases in which the electric treatment was continued steadily for 3 or 4 weeks, we could not boast of any lasting result. I therefore recommend the use of electricity as a palliative measure only in extreme cases.[1]

I have never as yet had occasion to observe in childhood that cause of dilatation of the stomach which is commonest in adults, namely, stricture of the pylorus or duodenum; and, further, I have known only one instance of dilatation caused by accumulation of an enormous amount of food in the stomach. This was in the case of a girl who sought to appease her appetite, which was very large, by eating enormous quantities of potatoes.[2] In such cases, after the stomach and intestine have been emptied we must order a strict dietary from which all vegetables are excluded; and at the same time we must endeavour to remove the atony of the stomach-wall which the over-distension has caused, by the use of an ice-bag, nux vomica, and electricity.[3] To this class also belong the cases of dilatation of the stomach due to fermentative dyspepsia, which, as I have already mentioned (vol. i., p. 128), occur even in early infancy. Prominence in the epigastric region, a tympanitic note, and splashing sounds on percussion or when the child is moved—are the main symptoms in these cases and they are combined with dyspeptic phenomena.[4] Occasionally there is vomiting of large quantities of sour frothy fluid containing numerous fermentative fungi. I have also frequently met with these symptoms in children as old as 14 years;[5] but these cases differed in no way from those

[1] Machon (*Contribution à l'étude de la dilatation de l'estomac chez les enfants*: Genève, 1887, p. 17) describes, on the other hand, a case of this kind said to have been cured by the interrupted current.

[2] *Vide* a case of Machon's (*loc. cit.*, p. 28), in which an enormous dilatation of the stomach from ingesta was found at the post-mortem.

[3] *Beitr. zur Kinderheilk., N. F.*, S. 282.

[4] Comby, *Arch. gén.*, Août, 1884.—Moncorvo, *Revue mens.*, Juillet, 1885.

[5] *Beitr. zur Kinderheilk., N. F.*, S. 314.

observed in later life. The use of the stomach-pump gives good results in these children also, as I have frequently found.

We must not confuse this vomiting due to fermentative dyspepsia with another form which I have often seen in older children, and which also has certainly nothing to do with any serious disease of the stomach. This form of vomiting occurs particularly in delicate "nervous" children, and especially in the earlier part of the day when the children have eaten their breakfast or dinner hurriedly, just before going to school. In one boy who was very nervous, and in a very excitable girl of 8 years, the vomiting occurred through the day, always after emotional causes—*e.g.* when the children had been scolded by their father. It remained away for days and then recurred and lasted with varying intervals for weeks or even months, without having any further result. I think that these are really cases of "nervous vomiting"—*i.e.* of hyperæsthesia of the mucous membrane of the stomach, which rapidly excites a reflex contraction of abdominal muscles, and consequently vomiting of everything that has been taken. In the cases I have met with, complete recovery always took place, either under a tonic line of treatment or even without any treatment at all.

## VIII. *Cholera Infantum.*

Although this dangerous disease (cholera nostras) attacks children of all ages, and even adults, it occurs with far the greatest frequency during the first and second years of life; and it is within that period, also, that it exerts its most fatal effects. We may gather from this that the nature of the feeding (especially when the children are hand-fed) and the change from the breast at weaning are important factors in these cases. Infants who are getting good breast-milk from their mothers or wet-nurses are affected very rarely indeed, compared with the hand-fed babies one meets with among the poor. So far we have to deal with facts; but all beyond this is hypothetical. Nevertheless we have exceedingly good grounds for taking an infectious factor into account in such cases. This view is strengthened by the fact that this disease occurs as an epidemic during the hot summer months of June, July, and August—and particularly in large cities. Among these in Germany, Berlin and

Munich are notorious. Its prevalence during these months is so invariable, that the disease has been appropriately called "cholera æstiva" (the "summer-complaint" of the Americans). Every practitioner knows that when the very first warm days of early summer set in, cases of this kind at once begin to crop up. The number increases every week until they are so widely distributed that they amount to an epidemic. The number of deaths from it is very great, and especially among poor patients. At last, in September, the cases become fewer; but a few straggling ones are found even into October. In spite of the most diligent research, the nature of the supposed infectious material is still unknown to us. Definite forms of bacteria to which we could with good reason ascribe an infective property have not yet been found, although the motions contain an immense number of different varieties of these organisms. At the same time we must not overlook the fact that micrococci—especially fermentative fungi—are found in all motions, and are especially abundant in those which have an acid reaction.[1] When, however, we consider the cases of intestinal mycosis, descriptions of which have been published,[2] we may expect that careful research in this direction will, if persevered in, yield satisfactory results. According to these investigations, rapidly fatal, cholera-like affections seem to have been caused by filamentous fungi like those of anthrax, which were found not only in the contents of the intestine, but also in the epithelium and in the submucous tissue of the intestinal canal. From thence they had found their way into the lacteals and mesenteric glands. The comma-bacilli, also, found by Finkler and Prior in the intestinal contents of adults in cases of cholera nostras, and which in recent times have given rise to so much discussion, should also be mentioned here. Still, we must in the meantime regard the origin of cholera infantum from micro-organisms as merely a hypothesis. It is an undoubted fact that a heightened atmospheric temperature considerably increases the tendency to fermentative dyspepsia which is always present, all the year through, in young children who are unsuitably fed (vol. i., p. 131); and that it causes the disease to

[1] Nothnagel, *Zeitschr. fur klin. Med.*, iii., S. 205.

[2] Burkart, *Klin. Wochenschr.*, 1873, No. 13.—Demme, 18. *Bericht des Jenner'schen Kinderspitals* pro 1880, S. 26.

appear not only as an epidemic, but also in an extremely acute and fatal form. We may, indeed, conclude from this fact that the germs of infection are developed in enormous numbers by the heat, and that when they have found their way into the stomach along with the food, they exert their injurious influence there. Under certain circumstances the disease may appear endemically in crowded rooms, even during winter.[1]

Both the clinical and pathological features of cholera nostras in little children are extremely like those of acute dyspepsia in infants (vol. i., p. 131). In both cases we find very varying degrees of severity, from a more or less profuse diarrhœa to the most severe and rapidly fatal cholera infantum. The first symptom is the occurrence of loose motions, which are, to begin with, of a brownish-yellow or greenish colour, and which rapidly succeed one another. Either there is no pain whatever in those cases, or it is so slight that even older children scarcely complain of it. Apart from anorexia and great thirst, the general health may remain good, and with careful nursing the diarrhœa passes off either spontaneously or as a result of proper treatment, after 24—48 hours, as soon as the fermenting contents of the intestine have been removed from the body by the increased peristalsis. For this reason, also, it is not advisable to give an astringent medicine at the beginning. On the contrary, the suitable diet and treatment for such cases is just that which I have recommended for acute dyspepsia (vol. i., p. 137); hydrochloric acid or small doses of calomel are especially useful. In older children abstinence (mucilaginous drinks) and rest in bed are to be strongly recommended. In another series of cases the disease sets in suddenly with very violent symptoms. Occasionally the attack begins with very high fever, as in infectious diseases,[2] while more generally we only find a slight rise of temperature, if any. Profuse watery motions and vomiting rapidly succeed one another. The severity of the latter varies greatly; it sometimes takes place only at long intervals, sometimes very frequently—every time fluid is taken; and there are even cases in which the vomiting is the chief symptom, and in which only very few loose motions occur in the course of a day. The rapid failure of strength, however, is common to all; and it is more rapid and

[1] Epstein, *Prager med. Wochenschr.*, 1881, No. 33.

[2] Demme and Epstein have observed temperatures of 104°—107·6° F.

more severe the younger the patient is. We find it, however, also in older children and even in adults. Great lassitude, pallor of the skin, sinking of the eyes into their orbits, coldness of the cheeks, hands and feet, increasing rapidity and smallness of the pulse, weak and almost inaudible voice, slight cyanosis of the face and mucous membrane—all indicate the failure of the heart's energy. The restlessness and jactatation which are present at first soon pass into a drowsy apathetic condition. The tongue and mouth are dry, the thirst extreme, the abdomen generally little if at all distended, and not tender on pressure, and the discharge of urine considerably diminished, owing to the great loss of fluid through the stomach and intestine.

In these very acute cases the motions, which at first retained their fæcal colour and offensive smell, soon become watery and bright yellow, and finally colourless. There is generally neither mucus nor blood mixed with them; and if traces of blood do occur, they come from the lowest part of the rectum or from the neighbourhood of the anus, which has been abraded by the copious discharges. I have frequently made a microscopic examination of such stools, and I have never been able to discover any formed elements, except a large quantity of desquamated epithelial cells and bacteria, such as we also find in the motions in other forms of diarrhœa. The chemical character of these stools has not yet been sufficiently investigated.

While a certain proportion of these cases recover under suitable treatment, the violent symptoms gradually subsiding, other cases end fatally, especially those where the patient is very young and in poor circumstances. The tables of mortality for the summer months furnish a terrible proof of the fury with which this disease decimates the population in our large towns. The immediate cause of death in these cases is always the rapid exhaustion which is accompanied by symptoms of collapse and hydrocephaloid (vol. i., p. 314)—cadaverous pallor, cyanosis, constant drowsiness with half-closed eyes, finally complete coma, depression of the open fontanelle, displacement of the frontal and occipital bones under the edges of the parietal bone (in cases where the sutures are not yet closed), coldness of the extremities and imperceptibility of the pulse. Almost invariably there is a dark shadow round the sunken eyes in the last stage, especially marked on the lower lids. This is caused by the orbital ridges

projecting beyond the sunken eye-balls, and by the venous congestion of the eyelids caused by the cardiac debility. We can often recognise severe cases of cholera infantum at first sight, from this appearance alone. I have also nearly always found the leashes of enlarged conjunctival blood-vessels, and the little fragments of mucus on the surface of the conjunctiva, of which I have already spoken (vol. i., p. 322). We also often find dimming of portions of the cornea, especially of that part of it which is not covered by the half-closed eyelids. In this disease, as in tubercular meningitis, these changes in the appearance of the eyes have always seemed to me an almost certainly fatal sign; and I have only found myself mistaken in this matter in two cases. One of these children recovered, although his cornea was already dimmed by the characteristic threads and fragments of mucus; but it must be mentioned that there had been a slight amount of catarrhal conjunctivitis before the cholera had set in. In the second case, which was that of a boy of 11 years with a violent attack of cholera following indigestion, the patient was in a state of collapse, and the lower halves of both corneæ (which were not covered by the half-closed eyelids) were dim as if sprinkled over with dust. By the next day (when the collapse had ceased and the eyelids were completely closed) the corneæ had completely recovered their normal lustre. Occasionally in this last stage the vomiting and purging suddenly cease, to the great joy of the parents, who once more begin to entertain the highest hopes. I warn you, however, to guard against rashly over-estimating this symptom, unless you find along with it an increase of the child's strength and an improvement in the general condition. I have often seen the hydrocephaloid go on developing and end fatally, in spite of the fact that neither the vomiting nor the purging were by any means very severe, or might even both have entirely ceased. In some of these cases, delusive hopes had been aroused by the temporary return of warmth and improvement of the pulse, due to the administration of stimulants.

At the post-mortem examination of such cases we find nothing at all that is characteristic, as almost every writer admits, and as I have myself very often found. We generally observe nothing but an abnormal pallor of the whole digestive mucous membrane, or perhaps slight enlargement of the solitary

glands and Peyer's patches, in other cases streaks of redness and swelling of the mucous membrane of the stomach and intestine. General anæmia, atelectasis of portions of the lung-tissue, venous hyperæmia of the brain and pia mater, recent thrombosis of the sinuses and of other veins (*e.g.*, of the renal veins) are often found, and are to be attributed to the extreme weakness and the lowering of the heart's energy. I think, therefore, that we are not justified in regarding the disease as merely a catarrhal condition, although on microscopic examination proliferation of round cells has been discovered under the mucous membrane. I still think that an abnormal chemical action in the contents of the stomach and intestine set up by unknown influences, is to be regarded as the primary process. This, when it goes on for days, may certainly bring about a catarrhal condition secondarily by the constant irritation which it causes. It is thus that I explain the fact that many children, after recovery from cholera infantum, suffer from ordinary intestinal catarrh.

The very dangerous nature of the disease readily accounts for the large number of drugs which physicians have had recourse to in treating it. You will excuse me if I do not enumerate all of them here, or criticise them individually. Many physicians have in the course of their own practice formulated a method for the treatment of cholera infantum to which they adhere, even although the results are not entirely satisfactory. Others, again, are always experimenting, and eagerly take up every new drug recommended by hasty and inexperienced observers, use it for a time, and then give it up. Every summer we have members of the profession again asking what is the best remedy for use in general or in special cases during the prevailing epidemic of cholera infantum. Everything goes to confirm the sad fact that no specific exists which is certain to destroy the germs of this disease when they have found their way into the intestine. Neither quinine, nor carbolic acid, nor salicylic acid has proved of any value, although I have often enough made trial of them; and chloral hydrate has only a moderating effect, at most, on the vomiting. But neither this drug, nor the recently recommended resorcin, can in severe cases prevent a fatal termination. As it is impossible to destroy the actual germs of the disease, nothing remains but to combat their effects—that is, to treat the fermentative processes in the

stomach and intestine which they have given rise to. In all cases where the germs of infection have not entered the intestine in too great number, and where their action is, consequently, not too violent, we may succeed in obtaining recovery when all the toxic and fermenting matters have been got rid of. In other cases, however, even the strongest stimulants are unable to avert the collapse due to the continuous discharges.

It follows, then, that the only remedies at our disposal for the treatment of cholera nostras are those which I have already recommended for fermentative dyspepsia (vol. i., p. 137), and that the practice of giving opium at once, which only serves to keep the injurious matter longer in the intestinal canal, is as unsuitable in the former as it is in the latter disease. In recent cases—that is to say, in those seen within the first two or three days—we are often successful with small doses of calomel (Form. 2) and hydrochloric acid (Form. 3), and, if those fail, with creasote (Form. 4). When signs of debility set in, you should at once order a warm (95° F.) camomile or mustard bath[1] 1—3 times a day, in which the children should be left 5—15 minutes. And you must give port, sherry, or Hungarian wine (20 drops—a teaspoonful, according to the child's age). The wine is often retained while other nourishment (milk, beef-tea) and the medicines are speedily rejected. As to milk, I should advise you to give it only iced, and from a teaspoon (vol. i., p. 136). If, in spite of this precaution, the child still continues to reject it, you should try rice-water or barley-water, concentrated beef-tea or white-of-egg (one to about a pint of water, according to Epstein and Demme). Should the attack continue in spite of the treatment, or should the patient only have come under treatment after the disease has lasted some days, I then no longer avoid the use of opium, for we may now safely assume that the infectious germs have been got rid of, so that we have not now to dread their retention. In such cases I add 5—15 drops of tincture of opium to the hydrochloric acid mixture, and also give, twice or thrice daily, a starch enema, containing 1½—3 drops of laudanum. It is desirable in the utmost degree that the sick-room should be as large as possible and that the linen should be kept scrupulously clean; but unfortunately these advantages are only available in

[1] About 2 oz. of mustard made into a paste with cold water, enclosed in a linen bag and put in the bath.

a minority of the cases. When the hydrocephaloid condition becomes very marked we must try injections of sulphuric ether and camphor (Form. 14), iced champagne (1—2 teaspoonfuls), mustard baths, wet packs and douches to the whole body. By these means we endeavour, on the one hand, to cause a strong determination of the blood towards the skin, and on the other, to stimulate the heart—often, alas! in vain. The almost unquenchable thirst of the little patients is indicated by the eager way in which they open their mouths at the sight of a cup or spoon; and it is best satisfied by giving cold milk or iced water. Should a condition of catarrhal diarrhœa remain behind, you should make use of the remedies which I shall presently mention in treating of that condition.

Such is the treatment which I have found most effective for cholera infantum. After trying many other remedies I have always to return to these; and I therefore consider myself justified in recommending you to use them in preference to any others. As to the washing-out of the stomach and intestine in cholera with a 2—2½ per cent. solution of boracic acid (Demme), I have no sufficient experience of it; nor yet of the water injections recommended by Meinert.[1] Subcutaneous injections of a physiological solution of common salt (6 : 1000; up to 2 oz. and more daily) which we have tried this summer for the first time in a series of cases, has often a favourable effect on the collapse. I must, however, reserve any definite opinion as to their value.

## IX. *Catarrhal Diarrhœa.*

An attack of catarrh of the intestinal mucous membrane may, as I have already mentioned, be caused by the continued action of abnormal chemical contents, especially by the masses of fæces in process of acid-fermentation which are found in cases of dyspepsia and cholera (vol. i., p. 130). I have frequently observed that other irritants also—especially foreign bodies—may have the same effect. A girl of 2½ years was seized with vomiting and obstinate diarrhœa owing to having swallowed some bits of chalk and egg-shells which she had picked up in the court. Similarly, the child of a barber suffered from an extremely obstinate

[1] *Verhandl. d. 4. Congresses für innere Medicin.*

diarrhœa owing to having swallowed some bits of hair ; and she did not recover till a few doses of castor oil were given owing to the hair having been noticed in the motions. Besides such forms of direct irritation of the mucous membrane, we may, as in adults, have attacks of intestinal catarrh caused by injurious atmospheric influences (chills, wetting). In a third series of cases it sets in as a sequela of one of the infectious diseases, especially of measles. As a rule, the colon is more frequently and more severely affected than the small intestine.

We diagnose catarrhal diarrhœa generally from the more or less plentiful admixture of shreds and fragments of mucous in the motions, which are often, in addition, spotted and streaked with blood. We very often observe tenesmus in these cases, the straining taking place repeatedly after the completion of the defæcation ; and a small portion of dark-red rectal mucous membrane may be forced out along with the fæces. This straining and pressing downwards is seen especially in those cases in which the catarrh is situated in the lower part of the colon, including the rectum ; while, on the other hand, when the disease is higher up, the fæces are liquid and are forcibly ejected with much noise. Occasionally, also, I have seen a large number of living thread-worms passed in the motions, which had been dislodged from the folds of the bowel by the violent peristaltic movements, and the strong current of the fluid. Fever may be quite absent in these cases, and when it does occur it is slight and of a remittent type (morning temperature quite normal or nearly so ; evening temperature, 100·4°—101·3° F.). While the thirst is great, the appetite may be normal or even lessened, and the tongue covered to a moderate extent with a grey fur. Painful colic often precedes and accompanies defæcation ; but it may be entirely absent.

Almost all these cases of diarrhœa end favourably if well nursed and treated. It must be very rare that cases are met with which are so severe that they can be described as acute enteritis, for I have seen very few such. Let me briefly mention one of the worst of these.[1]

A delicate boy of 2 years, who had often before suffered from diarrhœa, took ill suddenly, 12 days before my first visit, with violent epileptiform fits, high fever, and profuse diarrhœa.

[1] *Beitr. zur Kinderheilk., N. F.*, S. 304.

The two latter symptoms had lasted continuously ever since, and had obstinately resisted all medicine (hydrochloric acid, ipecacuanha, opium, calumba, tannin, nitrate of silver internally and in the form of enemata); they had indeed considerably increased during the 3 previous days. There were generally 12—15 quite loose, spinach-green motions mixed with much mucus, preceded and accompanied by violent crying and restless tossing about. There was high fever, with coldness of the extremities. During the next few days the collapse increased, and the child became apathetic, with half-closed eyes, small and very rapid pulse, also slight flatulent distension. Finally, the diarrhœa ceased, the flatulent distension increased considerably, and coma and death ensued on the 17th day of the disease. *P.-M.*—General anæmia, fatty liver, extreme follicular enteritis reaching from the middle of the small intestine to the sigmoid flexure. Extensive hyperæmia and swelling of the mucous membrane; many of the follicles enlarged and burst. In many places there are very small circular ulcers, surrounded by a hyperæmic ring scattered thickly over the mucous membrane, like the holes in a sieve. The Peyer's patches were reddened and retiform.

The commencement of this case with violent fever and reflex convulsions was in itself an indication of the severity of the disease. We had here a condition of acute and extensive follicular enteritis, which is probably to be regarded as due to a sudden aggravation, from some undiscovered cause (error in diet?), of a previously existing chronic intestinal catarrh. We much oftener find that simple intestinal catarrh is neglected by the parents, especially if they are poor, and attributed to "the teeth," and the physician is not consulted until it has gone on for many weeks, or even months. When the disease becomes chronic in this way, it is always as a result of neglect, as I have already mentioned to be the case with dyspepsia (vol. i., p. 129). The diarrhœa lasts for a number of weeks with varying severity, the motions sometimes occurring seldom, sometimes as often as 10—12 times in the day. In the meantime the form of the abdomen may remain normal, and there may be no colic at all; while in other cases the children complain of pain before defæcation and of tenesmus, and the abdomen is somewhat distended with gas. The fæces are more or less liquid, their quantity and appearance varying much. They may be greenish-brown, blackish or even pretty light in colour, containing much mucus, and very offensive. We not uncommonly find small quantities of blood in them. When the diarrhœa is not very profuse, and the

children are strong, months may pass without the appetite and nutrition being noticeably impaired. In the majority of cases, however, we soon notice emaciation, shrivelling of the skin and wasting of the muscles (especially of the adductors of the thighs) and a pale complexion. These symptoms become more marked from week to week if the diarrhœa is not arrested, until we have finally an extreme degree of emaciation and atrophy. Prolapse of the rectum often occurs during defæcation, the anus finally becomes paralysed and the fæces are constantly escaping. This sad condition is nearly always accompanied by a remittent type of fever (100·4°—102·2° F. in the evening), and often ends fatally from the occurrence of broncho-pneumonia with increasing symptoms of collapse. Towards the end we often find the formation of thrush in the mouth and throat, and œdematous swelling of the feet, hands, and face, which must generally be regarded as due to the lowering of the heart's energy and the consequent venous congestion. In a smaller number of cases we find the cause of this in thrombosis of the larger veins or concomitant nephritis.

Even in these apparently very extreme cases the severity and extent of the anatomical changes can never be determined before death. I have often enough convinced myself of the truth of Rilliet and Barthez's statement, that in the post-mortem of such cases we may find a state of things entirely at variance with the symptoms observed during life. The hyperæmia and swelling of the mucous membrane, which in those chronic cases usually presents a brownish or somewhat greyish-red tinge, may affect a greater or smaller extent of it, and may or may not be accompanied by enlargement of the intestinal villi. There may either be extremely few small ulcerations in the neighbourhood of the ileo-cæcal valve, or else a great number of follicular ulcers in the small or large intestine, without there necessarily being any relation between the amount of this pathological change and the severity or relative slightness of the symptoms, especially of the diarrhœa. We must be particularly on our guard against too hastily assuming the presence of extensive follicular ulceration wherever we find copious diarrhœa, increasing atrophy, and remittent fever. I have often been much surprised to find under such circumstances nothing after all but a moderate amount of intestinal catarrh and two or three

isolated follicular ulcers near the ileo-cæcal valve or in the colon. But we must never neglect to examine the mucous membrane of the rectum in examining such cases post-mortem: for the catarrh and ulceration may be most marked in this situation, while lesser changes are found in the upper portion of the intestine. On the other hand, we have often found the entire mucous membrane dark-red or slate-grey in colour, and studded all over with numerous follicular ulcers from the lower end of the ileum down to the rectum. The fatty degeneration of the liver which has been already mentioned by Legendre is also often found. The organ is not always much enlarged, but is of a pale-yellow colour and somewhat pulpy in consistence and under the microscope it presents the characters of the fatty liver.

Cases of catarrhal diarrhœa in childhood must, as follows from what I have already said, be regarded as serious from the very first; and as the danger is greater in children than in adults there is all the more need to urge strongly on the parents the importance of carrying out the physician's instructions. This greater danger is due on the one hand to the greater tendency of the intestinal follicles to hypertrophy and ulceration; and on the other hand, to the great liability of the mesenteric glands in children to become enlarged and to caseate as the result of repeated or long-continued irritation of the intestinal mucous membrane—just as we see enlarged bronchial glands resulting from bronchial catarrh or broncho-pneumonia. I need not again allude to the further possibility of general miliary tuberculosis developing.

In considering the treatment of a recent case of catarrhal diarrhœa, we have first to consider the question as to whether constipation had existed before it set in; and, again, whether an attack of indigestion had been the proximate cause of the trouble. Under these circumstances it is best to commence the treatment by giving a mild purgative—a dessert-spoonful of castor oil or a few doses of calomel (grs. $\frac{1}{4}$—$\frac{3}{4}$)—especially when there is tenesmus, and when the motions are small in quantity and spotted or streaked with blood. After these stagnating or chemically irritating matters have been got rid of by this means, we often see the diarrhœa disappear in a few days. Since, then, most cases of primary intestinal catarrh in childhood especially during the first years of life, are originally of a dyspeptic nature—we may

in almost every recent case commence with a purgative, even though we cannot make sure of indigestion having been the cause. If, however, there have been copious loose motions for days, or if we can discover with certainty that the cause has been a chill, or the abuse of purgatives or tartar emetic, I advise you to put the child to bed and to have him kept very warm, only allowing a mucilaginous and farinaceous diet, and ordering a mixture containing ipecacuanha with a small addition of tincture of opium (Form. 29). I prefer giving these drugs in this form rather than in that of Dover's powder, because I have found that the latter even in small doses causes nausea, which is only exceptionally occasioned by the use of the former. Instead of the tincture, we may use the extract of opium (gr. $\frac{1}{3}$—$\frac{1}{13}$). The dread with which many medical men regard the use of opium in children is not justified when the drug is given under due supervision and in proper doses. Should the diarrhœa resist this treatment and continue for a week or longer, I should recommend you in the next place to give subnitrate of bismuth in large doses (for a child in its first year, gr. iss.; if older, as much as grs. ivss. every 2 hours: Form. 30). Of the efficacy of this remedy I have convinced myself in a very large number of cases during many years. Under its use the motions become of a firmer consistence—often within a few days—and assume a greyish-green colour; but in order to guard against relapses it is necessary to continue giving it for a pretty long period, 10—14 days at least. In obstinate cases it is advisable to add extract of opium (grs. $\frac{1}{21}$—$\frac{1}{13}$) to each powder. We often hear complaints about the inefficiency of bismuth; but I am quite sure that this is due to the fact that many physicians give it in much too small doses, and not nearly often enough. You will occasionally, however, meet with cases which resist this remedy also and pass into a chronic condition. We then have recourse to astringents: and among these infusion of calumba or of cascarilla with small doses of opium (Form. 31, 32) are often very effectual, although it is difficult to get the children to take them, owing to their bitter taste. Tannic acid also (grs. i—iss.), has often done good service in my hands, especially in combination with tincture of nux vomica (Form. 33); but it may certainly interfere with the appetite, already poor. Among the metallic remedies, the most reliable is nitrate of silver

(gr. $\frac{1}{30}$—$\frac{1}{20}$, Form. 34); and if this has been used for about a week without effect, acetate of lead (gr. $\frac{1}{7}$—$\frac{1}{4}$), thrice daily, with small doses of opium (Form. 35). Even in apparently hopeless cases I have occasionally seen good results from the use of the latter, and never any injurious or toxic action.

You must, however, be prepared to find that all these remedies are often without effect, and that they may even do harm by causing loss of appetite, nausea or even vomiting. In such cases you may resort to enemata and especially to irrigation of the intestine with various medicated fluids; and the latter proceeding, especially if persevered in, may be of much service. For this, we use an ordinary irrigator or a glass funnel to which is attached a long india-rubber tube with an end-piece of horn or ivory. You must take care that the end-piece when introduced lies free in the cavity of the rectum, and is not pressed against its walls; for this would block the opening of the tube and prevent the fluid from passing into the bowel. The occurrence of such an accident is at once indicated by the level of the fluid in the funnel or irrigator remaining unaltered; the end-piece must then be withdrawn a little and then carefully re-introduced. The irrigation is best carried out in the knee-elbow position, but can also be effected with the patient lying on his right side. We generally employ a solution of acetate of lead (5 : 1000), less frequently alum or tannin (20 : 1000); and of this 10—18 oz. are used each time.[1] When the mucous membrane is very sensitive, some of the fluid is often returned during the process of irrigation; but otherwise it generally remains in the intestine 5—10 minutes and often much longer. One of the first cases I treated in this way was surprisingly successful.[2]

> Girl of 2 years, admitted 9th April, 1874, with chronic catarrh of the colon, which had lasted for months. Profuse mucous diarrhœa; flatulent distension; extreme emaciation. After internal remedies (calumba, opium, lead, &c.), had been tried to no purpose, irrigations with a solution of acetate of lead were begun on the 29th, and administered once daily; later on alum and tannin solutions were used alternately with the lead; all internal remedies were stopped. The number of the motions, which had been 5—6 daily, at once fell to 2—3. This treatment was continued

[1] Nitrate of silver, which is so much used in enemata (gr. 1—1½ to 2 oz. distilled water) I have never yet tried for irrigation.

[2] *Charité-Annalen*, Bd. i., S. 613.

till 11th July, *i.e.*, for nearly 2 months; at which time the motions occurred 2—3 times a day, and were quite normal, the flatulent distension had disappeared, and the child's nutrition was so much improved that she scarcely looked like the same child. In the beginning of August, complete recovery.

My further experience, however, has not justified the high expectations which this case led me to entertain. Although I have from time to time met with cases in which the very first irrigations had a surprisingly good effect, yet in many others the treatment was quite unsuccessful or the success was merely temporary. The method, however, is always worth trying in obstinate cases which resist all internal treatment; but it requires perseverance, for no favourable result can be expected in the first few days.

It is especially difficult, in children, to adhere to a suitable diet; and, without this, recovery is out of the question. The diet must be limited to meat, soups, good milk, red wine, mucilaginous drinks, eggs, rice, fine flour, and pounded meat. Everything that tends to fermentation must be forbidden—vegetables, fruit (cooked or raw), leguminous food (peas, beans, &c.), and so on. There is only one objection to the pounded raw meat so much used, namely, that it may give rise to tape-worm. Whether milk will suit the child, is a point that can only be settled by actually making trial of it. I never shrink from trying it, and I have often seen the motions which had previously been quite thin, at once become more firm in consistence under a milk diet. When we consider the bland nature of milk, and how very successful it often is in irritated conditions of the alimentary mucous membrane in adults—surely the dread of its use in chronic intestinal catarrh in children is exaggerated. For older children I would also recommend the use of dried whortleberries (vaccinium myrtillus) made into a thick compot, of which the patient may take one or two saucerfuls in the day. This old popular remedy has done me very good service, and that very speedily, in cases which, though perhaps not severe enough to cause anxiety, had yet resisted the action of many drugs; and most children are very fond of it. I have seen black motions of a firm consistence resulting from its use even within 24 hours, and recovery taking place without any other remedy.

I must now add a few words on that form of intestinal catarrh

which comes on secondarily as a consequence of other morbid processes. I refer to its occurrence not merely as a complication—*e.g.*, with bronchial catarrh, which indeed is extremely common—but as an important link in the chain of symptoms of a general morbid condition. The most striking instances of this are the infectious diseases, especially measles and typhoid fever. In the latter, as a result of the enlargement of the glands of the intestine, some degree of the catarrh of the mucous membrane is almost always found, although it is often of but limited extent. The presence of this catarrh is not always revealed by diarrhœa; and, indeed, cases of typhoid fever with persistent constipation or with almost normal motions are by no means rare in children. In measles, intestinal catarrh is a common symptom during the period of eruption; and many epidemics are distinguished by the prevalence of obstinate diarrhœa, which may retard the convalescence. Less frequently we find the same in scarlet fever; and then, I have found, it has generally an unfavourable significance, especially when it occurs in the first stage or is very severe. Most cases in which I have observed copious diarrhœa from the first were malignant in their nature and proved rapidly fatal.

I have, however, not uncommonly discovered after death the presence of a more or less extensive intestinal catarrh, with or without enlargement of the follicles, in children who had died of the most diverse diseases, and who had suffered little if at all from diarrhœa during life, so that nobody had thought the presence of such a condition possible. Indeed, this latent disease of the mucous membrane occasionally reaches a degree far exceeding the limits of catarrh; and it even presents a condition of severe hæmorrhagic enteritis, with here and there a croupous or diphtheritic character, even although no serious intestinal symptoms had been observed during life. I found this condition most markedly in two cases of chronic nephritis.

Otto W., 9 years old, admitted in the beginning of January, 1874, with eczema and chronic nephritis (œdema, &c.). On the 14th, sudden pains in the stomach and complete loss of appetite; tongue greyish-white; epigastrium somewhat distended and tender on pressure. Two loose brown motions said to have been passed. During the next few days this condition continued without there being any fever. On the 16th, vomiting occurred once; the motions continued to be normal. But on the 18th there

was also pain in the lower part of the abdomen on the right side. Temp. never above 99·5° F. Increasing debility; sudden death of the child. We found at the post-mortem well-marked chronic nephritis, and much redness and swelling of the mucous membrane in the fundus and along the greater curvature of the stomach, which was covered by a layer of tenacious blood-stained mucus. The mucous membrane of the intestine vascular throughout, marked hyperæmia in the ileum and ascending colon, along with numerous hæmorrhages and considerable swelling of Peyer's patches and the solitary glands. Some of the mesenteric glands enlarged to the size of a cherry, hard, and reddish-grey on section.

How trifling were the symptoms in this case (cardialgia, one fit of vomiting, only two loose motions during the whole illness, and entire absence of fever) when contrasted with the inflammatory and hæmorrhagic condition of the mucous membrane of the stomach and bowel found after death! This contrast was even more striking in the following case:—

Girl of 11, who was admitted into the hospital with caries of the petrous bone and chronic nephritis, and who died with uræmic symptoms after a few days, without any striking intestinal symptom having been observed during life. At the post-mortem we found the mucous membrane of the whole lower third of the ileum, not only dark-red from hyperæmia and hæmorrhagic infiltration, but also covered in places by a coherent fibrinous membrane, which could be picked off like that of croup.

We were unable to discover the cause of the extremely irritated condition of the alimentary mucous membrane in these cases. Perhaps the irritation caused by the urea, which was eliminated from it and was then decomposed, had something to do with it. This is supposed by Treitz to be a cause of intestinal catarrh and ulceration of the mucous membrane of the intestine in cases of chronic nephritis. From a clinical point of view, the fact that such a severe affection remained latent is extremely striking, and must perhaps be explained, in the second of those cases, by the symptoms of uræmia which during the patient's last days obscured all the others.

## X. *Dysentery.*

The gradual development of dysentery from catarrhal diarrhœa is oftener observed in children than in adults. In many cases—

especially in the first two years of life—the physician may be in doubt whether the case is one of real infectious dysentery or merely of non-infectious acute catarrh of the large intestine. As I have already mentioned, mucus and small quantities of blood may occur in the motions in any case of diarrhœa. And even the tenesmus, which is indicated by the constant groaning and pressing down during and after defæcation, and by the unwillingness of the children to quit the night-stool, is no rare symptom. It is only when defæcation is unusually frequent, and when nothing but a little mucus, or a very small quantity of fæcal matter mixed with mucus and blood is passed, that one describes the case as "dysenteric." I do not, however, mean to express the opinion that this condition is really caused by specific infection,[1] and is therefore "dysentery" in the true sense of the word. Both pathologically and clinically the cases are to be regarded as colitis, and they can only be supposed to be of an infectious nature when they occur as an epidemic at certain seasons, especially in the months of August and September, or at least when the disease is met with in several members of the same family simultaneously. The infectious nature of sporadic cases of colitis is always a matter for doubt.

The symptoms of this disease in childhood differ in no essential particular from those in adults. The attack generally commences with diarrhœa, and it is only after 24—48 hours that the regular dysenteric motions set in. These consist of small quantities of tenacious, glassy, brownish mucus streaked with blood, which is passed with severe tenesmus very frequently—sometimes 5 or 6 times, and even oftener, in an hour. We also often find, along with this, colic, tenderness and distension of the abdomen, and repeated vomiting. The appetite is lost, and there is great thirst. Fever may be entirely absent or only moderate in degree; thus the temperature may reach 101·3°—102·2° F. only in the afternoon and evening. But in severer cases there may be a continuous remittent type with morning remissions (m. 100·4°, ev. 103·1° F. and over). This condition may gradually diminish and come to an end after 8—10 days; but it may also last much longer. In a few children I have seen the fever, tenesmus, and frequent defæcation (16—20 daily) last 3 weeks before recovery took place. Even in the slightest cases

[1] Prior, *Centralbl. f. klin. Med.*, 1883, No. 17.

the weakness which is indicated by the pulse and still more by the child's whole aspect, is far greater than in a case of simple catarrh of equal duration. The anæmic pallor left behind is also more extreme and persistent. In severe cases, however, the debility advances rapidly—owing to the numerous painful and always bloody motions, and the high fever—to the stage of dangerous collapse. Coldness of the extremities, thready pulse, great apathy and drowsiness, which is only interrupted by restlessness when the tenesmus and violent colic recur—subnormal temperature (96·8°—98·6° F.), finally paralysis of the sphincter ani, which remains permanently patent and discharges continuously a quantity of offensive brownish mucus, often containing fragments of membrane and clots of blood—all these symptoms gradually lead up to the fatal result, which is immediately due to extreme cardiac debility. In some cases the above-mentioned paralysis of the sphincter has been so complete that I have been able by separating the nates to dilate the anus sufficiently to obtain a good view of the lower end of the rectum without using a speculum. Under these circumstances we may always expect that there will be very extensive ulcers of the mucous membrane of the colon, generally due to diphtheritic gangrene; but of these we may often find only rudiments left.

Max M., 7 years old, admitted on 13th July, 1877. He was said to have had a severe attack of diarrhœa 5 days before, after eating a large quantity of cherries (6 motions, containing undigested cherries). On the following day the motions contained nothing but mucus and blood, and they occurred almost every quarter of an hour, and were accompanied by violent colic and tenesmus. The boy at once passed into a dazed apathetic state, and became feverish. No source of infection could be discovered. On admission, great pallor and exhaustion, eyelids half-closed, T. 102·9°, P. 132 small. Complete loss of appetite, little thirst. Tongue rather dry, covered with a thick greyish-yellow fur. Abdomen much depressed, scarcely tender, flaccid. Constant tenesmus, colic, every hour 6—8 scanty motions, which consisted of nothing but brownish-green bloody mucus. Treatment: A dessertspoonful of castor oil, an ice-bag to the hypogastrium, sherry. In the evening an irrigation of the intestine with a solution of acetate of lead (5:1000) and morphia (p. 50). On the following day the intestinal symptoms continued and the extremities were cold; T. 101·1°, P. 132, thready; sighing and groaning; death during the night of the 14th.

*P.-M.*—In the lower part of the ileum there was marked redness

of the mucous membrane beginning ⅔ inches above the ileo-cæcal valve, and, a little higher up, a diphtheritic membrane. In the ascending and transverse portions of the colon there were many ulcers left by the separation of this membrane; in the descending colon these were even more numerous, and here there was, in addition, diphtheritic membrane of recent formation. This membrane extended to the upper third of the rectum, while the lower two-thirds remained unaffected. All other organs normal.

This extremely acute case—lasting scarcely a week—can hardly be explained by errors in diet. In spite of the very rapid course deep gangrenous changes had already taken place in the mucous membrane. In the following cases this was even more striking.

Richard S., 8 years old, admitted on 29th July, 1878. He had taken ill suddenly, 5 days previously, with severe diarrhœa, for which no cause could be assigned. After it had lasted 36 hours there were bloody motions and tenesmus, and these during the last few days recurred almost every 15—30 minutes. Motions very small in quantity, consisting simply of mucus and blood. Abdomen not distended, hardly any tenderness; tongue covered with a thick greyish-yellow fur, loss of appetite, great weakness, P. 132 small, T. 97·7° F. After a spoonful of castor oil the motions twice or thrice contained little masses of fæcal matter; but this did not last. No improvement, in spite of irrigations (twice daily) of solution of acetate of lead and the internal administration of ipecacuanha and opium, and, later on, of nitrate of silver, both internally and by rectum. Violent pains, persistent bloody and mucous stools, increasing debility with pulse of varying quality, ranging from 104 to 136; Temp. always subnormal (97·2°—99° F.). Death on 4th August, after a duration of 12 days.

*P.-M.*—Colon much contracted, its serous membrane congested. Small intestine unaffected, with the exception of slight catarrh and enlargement of the follicles. In the cæcum the mucus membrane commenced to be intensely red, and from the hepatic flexure onwards there were ulcers with jagged outline, some in process of cicatrisation, and some with a diphtheritic membrane. This deposit also extended on to the mucous membrane surrounding the ulcers, which was much reddened and swollen, in the form of a discoloured friable false membrane, which could be easily scraped off. Further down this change was more extensive, and the mucous membrane hæmorrhagic. Beyond the splenic flexure there are only little isolated areas of the mucous membrane left; and these finally disappeared altogether, so that the internal surface of the much thickened intestine was formed entirely of diphtheritic membrane. All other organs unaffected.

In a boy of 5 years, admitted 22nd June, 1882, the disease only lasted for six days, and the symptoms were extremely violent, yet nevertheless we found at the post-mortem a thickening of the muscular coat, increasing steadily from the ascending colon to the rectum (about 2 lines thick below) along with diphtheritic ulcerations of the mucous membrane, which in the cæcum had been entirely destroyed in some places so as to lay bare the muscular coat.

In other cases the disease lasts much longer—for weeks and even months (dysenteria chronica). During this time the symptoms vary in severity; a temporary fæcal character of the motions especially, may give rise to delusive hopes. In a girl of 6, admitted on 19th September, 1876, the disease lasted in this way for almost 8 weeks; the morning temperature was normal or even sub-normal, while that of the evening always rose to 102·2°—103·5° F. In this case the alternation of motions consisting of pure blood and mucus with others of a firm consistence or even consisting of scybala, was especially striking, but yet in spite of all our attempts at treatment we were unable to arrest the steady progress of the debility and emaciation. At the post-mortem we found almost the same condition as in the case of Richard S. just given. It is just in those protracted cases that —even after the first threatening danger has been escaped— stricture of the colon or rectum caused by the cicatrisation of the necrosed areas may be left behind (just as in adults) and become the cause of death. Even in cases of moderate severity we must be prepared for such an occurrence.

In several children I have observed, as a result of a not very severe attack of dysentery, symptoms which had caused great anxiety to the parents and the physician. While defæcation was otherwise normal and the general health seemed perfect, blood-streaked masses mixed with mucus and fragments of membrane were passed from time to time without pain or tenesmus. Motions of this character were occasionally passed daily, in varying quantity, for one or several weeks together. Then there were intervals of weeks, or even months, during which in spite of the most careful daily examination of the fæces, nothing suspicious could be discovered. If the fæcal matter is placed in water, it forms delicate, blood-stained, floating fragments, which under the microscope appear mainly structureless, but partly composed of fibres with some blood and pus-corpuscles

scattered among them. I have seen motions of this character recur from time to time during a period of years, after recovery from an attack of dysentery; but I have only in two cases been able to satisfy myself that complete recovery had really occurred. If no abnormality is observed on examination of the rectum with the finger and speculum, the position of the disease must be looked for further up where it is beyond reach. We should naturally assume in such cases that the dysentery had left behind it ulcerations or localised inflammatory processes of the mucous membrane of the colon, and that these heal for a time and then break out again under the influence of any irritation, *e.g.*, retention of fæces. I have never hitherto been able to procure recovery in any of these cases by the use of internal astringents, or by irrigation of the intestine. In two cases the patients did recover spontaneously after the disease had lasted, with intervals, for years.

I was more fortunate in the case of a child of 2 who was brought to the polyclinic on 30th Jan., 1877. In the beginning of December, 1876, he had had an attack of colitis, with bloody stools and tenesmus, following measles; and this, after lasting for 3 weeks, had left behind the following condition. There were still 4—6 motions daily; of these some were pulpy and fæculent, others mixed with blood and purulent mucus, or consisted only of small quantities of mucus and blood. Tenesmus and prolapsus ani were almost invariably present. The child was pale and thin, but otherwise looked healthy; and examination of the rectum yielded no results. On the supposition that there had been an attack of colitis which had left behind some ulceration, I ordered an enema of nitrate of silver (gr. $\frac{3}{4}$ to $\frac{3}{5}$ i.) to be given daily; and, after 5 days, enemata of alum (a teaspoonful to a cup of water). Under this treatment all symptoms disappeared within 14 days.

The treatment of dysenteric conditions is the same at all periods of life. In cases which are recent—of a few days duration at most—I begin by administering a mild purgative, a dessertspoonful of castor oil, or a pretty large dose of calomel (grs. iss.—ivss.), and then for some days I give an emulsion of castor oil (Form. 36) or small doses of calomel (gr. $\frac{1}{4}$—$\frac{1}{2}$) every three hours. When the motions have become fæculent, and there is no longer a possibility of any considerable amount of fæcal accumulation existing in the bowel, I have recourse to ipecacuanha with addition of tinct. opii (Form. 29). When the abdomen is much distended and very tender we may apply an

ice-bag, and allow no food except iced milk, or at most a little oatmeal-water and a little beef-tea. This treatment is generally successful in the less severe cases (the so-called catarrhal dysentery). If it does not succeed I should recommend you to use the already mentioned (p. 50) irrigations of tannin, alum, and especially those of acetate of lead. Previous to making these injections we must always wash out the large intestine with warm water, or with a solution of salicylic acid (1 : 1000). These irrigations may be given twice daily.

Hedwig H., 11 years old, admitted on July 2nd, 1878. Said to have had an attack of violent colic and diarrhœa 3 days previously, after having eaten large quantities of red-currants. On the next day severe tenesmus, persistent pain in the abdomen; the motions increasing in frequency, and consisting only of blood and mucus. On admission the bowels were moved at least 15—20 times daily; abdomen painful on pressure, moderately distended; great thirst, tongue covered with a greyish-yellow fur. T. 101·1° F., P. 120. After a spoonful of castor oil, the motions became fæculent, but soon resumed their dysenteric character. The same effect followed a second dose of castor oil; and also calomel (gr. ½ along with extract of opium, gr. ½ every 2 hours), had failed to produce any real effect by the 6th. The abdomen was distended and tender, vomiting had also occurred on one occasion. T. m. 101·1°, e. 102·2° F. On the 8th, after an emulsion of castor oil had proved unsuccessful, irrigations with a solution of acetate of lead (5 : 1000) were given twice daily and continued till the 12th; all other remedies were abandoned. During these days the number of the motions diminished, they began to be always fæculent, although they also from time to time contained some blood and mucus. The temp. was low (98·4°—99·7° F.), pulse 104—108. After the 12th, subnitrate of bismuth (grs. iii. every 2 hours) was given instead of the irrigations. On the 15th completely firm fæculent motions; child otherwise well. Discharged on the 24th.

The cases previously given (p. 56) show that in very severe cases these irrigations are of no effect. In any case, however, they certainly deserve to be preferred to emollient and astringent enemata. These were formerly held in high favour, but they certainly never reach beyond the rectum, and afford no prospect of success except in cases where we have reason to assume that the main changes are in that part of the bowel. But any one who has had a few opportunities of seeing on the post-mortem table the frightful destruction of the intestinal canal which this disease produces will readily understand why in very severe cases

of dysentery neither irrigations nor the most highly-praised medicines (ipecacuanha, nux vomica, nitrate of silver, subnitrate of bismuth, &c.) can do any good whatsoever.

## XI. *Obstruction of the Bowels.*

You will often be called upon to treat very young children, even those at the breast, for constipation. There are many children who, unless artificially aided, remain constipated for days. Sometimes it is only with the greatest exertion, during which they get red in the face, that they are able to pass their motions, which take the form of hardened scybala, which fall into the night-stool with a noise like that of a stone, and by their mechanical action cause little erosions of the anus, giving rise to hæmorrhage. Owing to this we often observe traces of blood on these hard fæcal masses. We are generally struck also by the very pale yellow or whitish-grey colour of the fæces, which sometimes have just the same appearance as in jaundice, without there being a trace of jaundice present and without the general health being disturbed in any other way. This may therefore be due either to deficient secretion of bile or to its pigment being paler than usual; but on this subject, so far as I am aware, nothing very definite is known. It is also possible that an increase in the amount of calcareous matter in the fæces may be the cause of their hardness. When infants at the breast have suffered from a certain amount of constipation ever since birth, this condition may be removed by a change of nurse or by weaning. In many cases, however, it continues to later childhood, and is not got rid of until the use of a mixed diet and active muscular movements bring about a gradual improvement. It is worth mentioning that defæcation may suddenly begin to take place spontaneously (and continue doing so for days or weeks) without any recognisable cause for the change, and then give place to the previous condition of constipation. The mothers generally try to treat the children themselves by giving Gregory's mixture, compound liquorice powder, castor oil, tincture of rhubarb, tamarinds, &c., before calling in the doctor; but to have any effect, pretty large doses of these medicines need to be given. It is best, in such cases, to use no internal remedies,

but to confine oneself to ordering the daily use of a small piece of soap, or an enema of cold water; to the latter, in very obstinate cases, may be added a pinch of common salt. With this treatment, and especially with patience, you will nearly always succeed. I have also often made use of massage of the abdomen, but not with invariable success.

This simple form of obstruction scarcely ever gives rise to any morbid appearances. I have, however, seen (in two boys of 7—9 years) an extreme distension of the whole abdomen occur as the result of great accumulation of fæces in the large intestine. The globular form of the abdomen, its great distension and tenderness to pressure in places, caused serious anxiety not only to the parents but also to the physician in attendance, and at the first glance I myself could not help suspecting the presence of chronic peritonitis. Both patients were the children of well-to-do and very indulgent parents, and had been fed with all manner of unsuitable delicacies (oysters, pastry, &c.) without any one noticing whether defæcation was correspondingly free during this "polyphagia." Thus had arisen gradually the enormous distension of the intestinal canal by fæcal masses, and by the gas developed from them. It was not until purgatives had been administered daily for a week, and the diet restricted (meat, compot, all starchy and leguminous foods being excluded) that we were able gradually to lessen the distension of the abdomen, and finally to get rid of it. The enormous quantities of scybala and soft fæces, extremely dark in colour and offensive, which were daily passed, were simply astonishing. As a purgative in such cases, I should recommend especially confection of senna either alone in doses of 1—2 teaspoonfuls daily, or in the form given in Form. 28. Sometimes, but comparatively rarely, this causes such severe colic that it has to be discontinued; generally it can be given for several weeks together with good results.

In a few cases (affecting exclusively children in the 1st and 2nd years of life) the constipation was evidently due to pain at the anus. At the moment of defæcation a spasmodic and painful contraction of the sphincter took place, making the evacuation of the fæces impossible, even although the children repeatedly sought it. Every attempt immediately caused loud screaming, and was at once given up, and sometimes several days passed

of dysentery neither ... medicines (ipecacuanha, ... of bismuth, &c.) can ...

XI. ...

You will often ... even those at th... children who, ... days. Some ... which they ge... motions, whi... into the ni... their ... rise to i... blood ... by th... sph... ou... g... t...

...ne anus in such cases. ...ssures in the folds sur- ...t where the skin passes ...e extremely tender to the ...echanical action of hard ...ainful contractions of the ...n. are due to reflex irritation ...n, along with the hard masses ... few drops of pure blood, are ...es higher up above the anus in ...n that case only be recognised by ...ium. I was unable to determine ... an internal fissure of this kind, ... the sphincter ani. In a child ...n spite of careful examination ...o find any fissure, although the ... so strong that the attempt to ...ne anus had always to overcome ...ver. I had forced an entrance, and ... part of the wall tearing, the ...nt. The pain and contraction ... had been given for a few days, ... and gave no further trouble. This ...ing or tearing the sphincter, is not, ...e to do with fissures. In such ... the condition by touching with ... an ointment of tannin (1 : 20), ... time. In order to lessen the ... may try painting with a solution ... the inunction of an ointment of ... no result, you must undertake ... and the adjacent muscular ... the treatment is to be, it ...; for otherwise the masses ... up irritation and cause per- ... secretion of offensive mucus.

... 13 months with fissure in whom ... intervals of 8—10 days; left-sided ... previously.

... from eczema of the anus

Much more serious than any of the causes of constipation which I have already mentioned are, of course, those which are due to impermeability of the intestinal canal at any part of its course, giving rise to symptoms of ileus. This may occur immediately after birth, and then we are justified in diagnosing congenital stricture, or even atresia, of part of the intestinal canal. The duodenum and the ileo-cæcal region are the situations in which these congenital malformations most frequently occur. Still, as the following case shows, they may be present at any part of the intestinal canal.

> Child of 3 days, admitted 7th January, 1881, with hands and feet in a very rudimentary state (fingers and toes almost quite absent); otherwise well developed. No motion of the bowels since birth, but everything taken had been vomited along with blackish-green masses. Anus and rectum appeared normal; abdomen of moderate tension, scarcely distended at all. No fever. Diagnosis: atresia of a portion of the small intestine (because there was no flatulent distension, which would certainly have been present had the atresia been situated lower down). Hardly any change up till the 18th, the day of death; gradual collapse. *P.-M.*—Duodenum and jejunum dilated, for a length of 31½ in., to the thickness of a large thumb, and ending in a blind sac 8 inches long and 2 inches in diameter. Immediately below this there was a solid band 2¾ in. long, of the thickness of a knitting-needle (the obliterated intestine), and then came the remaining portion of the intestine, empty and collapsed.

This combination of the fœtal obliteration of the intestine, with absence of fingers and toes, is remarkable, and the diagnosis arrived at was strengthened considerably by the presence of the latter malformation.[1] Imperforate anus, which is a much commoner condition, and which the physician soon discovers, owing to no meconium being passed, need not detain us here, as it is a malformation, the consideration of which belongs to surgery rather than medicine. I now turn to those morbid conditions which generally cause symptoms of ileus in the bowel of a normally developed child.

As a general rule we find the same causes acting in children

[1] *Cf.* Gärtner, *Jahrb. f. Kinderheilk.*, xx., 1883, S. 403.—Tobeitz, *Arch. f. Kinderh.*, vii., S. 117.—Hirschsprung, (*Jahrb. f. Kinderheilk.*, xxvii., S. 1) describes two cases of extreme congenital dilatation of the sigmoid flexure, and of the transverse colon in children of 7 and 11 months respectively. There was marked thickening of the intestinal walls and numerous deep ulcerations, which he referred to an attack of phlegmonous enteritis.

as in adults, only their relative frequency is not the same. Thus, *e.g.*, strangulated hernia, which in adults takes the first place among the causes of ileus, is of rare occurrence in childhood. Cases have, however, occurred in children in the first weeks of life, and some of these were successfully operated on.[1] So that even in children we must never forget the possibility of this condition occurring; however young the child may be, we must never neglect, in cases of ileus, to examine the well known seats of hernia. Neither this form of ileus, nor those due to volvulus, stenosis of the intestinal canal, or obstructing material in its lumen (*e.g.*, fæcal concretions), differ at all from the cases of the same condition occurring in adults. The only form of this almost quite peculiar to childhood is the very rare condition in which the lumen of the intestine is obstructed by a mass of round-worms knotted together; but in these cases we can only have a suspicion, and cannot form a real diagnosis. The only form of ileus which presents characteristic symptoms, and the one which is commonest in childhood is intussusception—to which we now pass.

### *Intussusception.*

I do not here speak of the numerous little invaginations in the course of the small intestine, which one often finds post-mortem in children who have died of the most widely different diseases. The absence of all symptoms, the extremely small portion of intestine affected, and the ease with which the invaginated gut can be drawn out by a slight touch of the hand, prove that these invaginations can only have taken place shortly before death—during the death-agony. Intussusception, with which we have here to do, always affects a much larger portion of the intestine. We generally find that the lower end of the ileum, along with the cæcum, becomes invaginated in the ascending colon, and, as the case progresses, draws the latter along with it into the transverse, or descending colon. In many cases, indeed, the invagination is smaller, and only attains a length of from 2½ to 3 inches. But it may grow much larger, and cases have often been observed in which the lower end of

[1] *Berliner klin. Wochenschr.*, 1879, S. 488 u. 677.—Demme's *Jahresber. f.* 1878, S. 58.—*Arch. f. Kinderheilk.*, iii., 1882, S. 203, *et seq.*

the ileum, and the ascending and transverse colons, were invaginated in the descending colon, and the extremity of the intussusception thus formed was felt in the rectum, or was even extruded beyond it.

The vast majority of the cases of intussusception which occur in childhood, occur during the first year. The fact is striking, but the cause of it is unknown. The great amount of passive movement to which infants are subjected (especially the swinging up and down, and to and fro), has been blamed by many writers; but it can scarcely be regarded as having such a very injurious influence when we compare it with the violent active movements of older children, among whom, nevertheless, cases of intussusception are far less common. A preceding diarrhœa has been assigned as the cause, but there is often no history of such an attack. The diagnosis rests mainly on the presence of three symptoms:—constipation, vomiting, and passage of blood from the anus. As a rule the disease begins quite suddenly, in the midst of perfect health, with violent screaming, great restlessness, frequent vomiting, and constipation. Purgatives and enemata have no effect, the latter being at once returned. Often there occurs also (sometimes on the very first day, but always at some stage of the case) a discharge of a varying quantity of blood from the anus, at first mixed with fragments of fæces, afterwards with mucus and serous fluid; or it may even be discharged pure and partly coagulated. As a rule the quantity is overstated by the relatives, but there is sometimes 1—1½ tablespoonfuls, or even more, discharged at a time. Tenesmus is rarely absent, and there are 5, 6, or even more, of these evacuations in the day. Drinks, which the children eagerly ask for, nearly always excite vomiting. The abdomen may retain its normal form and softness for the first 24—48 hours, but then generally becomes tense, distended with flatus, and tender. Whenever this takes place, we are no longer able to make out distinctly by palpation the swelling in the course of the colon which is caused by the intussusception. It is proved by experience, however, that this may possibly be done in children just as well as in adults, so long as the abdomen is soft, and its walls flaccid. I have never myself been able to make out with certainty the presence of a tumour in any case which I have examined, either by palpation or by percussion, because the intussusception was

quite covered by coils of small intestine much distended with gas. I therefore attach no particular diagnostic significance to the discovery of a tumour in these cases. I have twice been able to feel the rounded end of the intussusception distinctly by introducing the finger well into the rectum, and to determine its size. The end of the intussuscepted gut had a certain resemblance to the vaginal portion of the uterus, and also like it, presented in the centre, or more or less towards the side, a rounded or linear opening into which the finger could be passed for some distance. This opening represented the compressed and swollen lumen of the intussuscepted portion of intestine. In such cases, of course, the diagnosis is beyond a doubt. Very rarely, the intussusception is extruded from the anus for an inch or so by violent pressing down, and then has the appearance of a dark-red, bloody tumour, with a central opening.

When we can neither feel the intussusception in the rectum, nor see it externally, the diagnosis cannot be made with absolute certainty. It may, however, be made with great probability because, as I have already mentioned, all other causes of acute ileus in early childhood are much less common, and because the combination of these three symptoms—complete obstruction, vomiting, and bleeding from the mucous membrane of the intussuscepted portion of the bowel—are almost conclusive, according to the experience of all medical authorities. The further course is just the same as in adults. In the cases which end fatally (and these unfortunately are the great majority), we have—increase of the flatulent distension, constant complaining and screaming as if in pain, which presently gives place to complete apathy, cold cheeks and extremities, extremely rapid and small almost imperceptible pulse, occasionally unilateral or bilateral convulsions, finally fatal collapse after the disease has lasted 4—8 days. In cases which end in recovery there is an undoing of the invagination with passage of flatus and faeculent motions; or there may occur separation and discharge of the gangrenous portion of the affected portion of bowel, the normal lumen being restored and the intestine shortened to a corresponding extent. It is worthy of note that, according to the experience of all writers, diffuse peritonitis following intussusception is much rarer in children than in adults.

Child C., 1 year old, had always suffered from constipation, but during the last few days had had quite normal motions. On 15th October, 1873, the child seemed perfectly well till late in the evening. During the night, great restlessness, no sleep; one motion, consisting only of mucus and blood; vomiting of milk towards morning. Castor oil had no effect, nor had several enemata, these being at once returned. Vomiting of everything taken, and another motion of pure blood. Abdomen normal, no fever. On the 17th, drowsiness, progressing collapse, no motion. In the evening a large enema of iced water; an hour afterwards there was a loose brown motion, in which the castor oil taken on the 16th could be distinctly recognised. During the night, several more motions. On the 18th, everything normal with the exception of weakness. Permanent recovery.

Child of 4 months, healthy. On 27th January, 1884, sudden violent screaming, restlessness, repeated vomiting and passage of 3 motions, consisting of nothing but pure blood. No fæces or flatus passed. On 28th, vomiting after everything taken; abdomen distended with gas; repeated passage of blood, but no motion, and no flatus passed. Constant screaming, commencing collapse. On 29th, brought to the polyclinic in the same condition. Nothing abnormal was felt per rectum. A somewhat loose motion took place immediately after the rectum was examined, and during the course of the day a large amount of flatus was passed on several occasions. The swelling of the abdomen rapidly subsided, and no more vomiting occurred. Complete recovery.

Child Ph., 1 year old, always previously healthy, suddenly took ill on evening of 27th February, 1875, with violent screaming and vomiting, for which no cause could be assigned. On the following morning a large amount of pure blood was passed per anum; moderate amount of flatulent distension. These symptoms continued to increase in severity till 2nd March, on which day I saw the child for the first time. The vomiting had now ceased, but no fæcal motion could be obtained. I found collapse had already set in. Repeated enemata of iced water had no effect. When I now examined the rectum with my finger I felt the intussusception distinctly about the middle of it, with its central opening; and I was able to make out the size of it with the point of the finger as if it had been the vaginal portion of a uterus. Immediately on my finger being withdrawn there occurred a discharge of dirty-brown fluid from the anus, and along with it a piece of gangrenous intestine of about 2½ inches in length was passed. I found on examination that it was part of the colon. The symptoms of ileus now came to an end, and during the next 3 days numerous loose fæculent motions took place. On the 6th, however, constipation again appeared, the flatulent distension returned, and the presence of severe colic was indicated by constant screaming. Nevertheless the child seemed considerably

better, and the pulse was stronger; vomiting did not occur, and small quantities of beef-tea, wine, and iced milk were taken. The abdomen was covered with an ice-bag, and a mucilaginous mixture (℥ iv.) with tinct. opii (gtt. iv.) given in order to ease the violent pain. Under this treatment after the 8th, there occurred daily 8—10 very large loose brown motions; these had a gangrenous smell, but contained no more fragments of intestine. The flatulent distension gradually disappeared, the appetite improved, the pains were less frequent, and the appearance better, so that the ice-bag was dispensed with. The diarrhœa persisted in spite of the use of opium, and on the 20th there were still as many as 15 very loose, bright yellow, slimy motions. It was only on the use of tannin with tincture of nux vomica (Form. 33) and of starch enemata that diminution of the diarrhœa took place; and by the 30th there was complete recovery.

In these cases there must have been an invagination of a small portion of bowel which in the first and second cases suddenly became undone, and in the third was discharged in a gangrenous state after the condition had lasted only an extremely short time (3—4 days). In this case we did not have complete recovery taking place at once, but severe intestinal catarrh was set up by the fragments of gangrenous tissue which were left behind; this kept us anxious for weeks and endangered the child's life. Since, however, we very rarely find cases of intussusception recovering thus spontaneously, the physician will hardly be satisfied with an expectant line of treatment. The same objections to an active line of treatment apply here as in later life. As soon as the diagnosis of intussusception has been established, we must desist from the use of purgatives, which can only do harm by strongly increasing peristalsis. Enemata are also avoided by many, because it is supposed that they return without effect and, like purgatives, favour a further increase of the intussusception. This supposition is, however, by no means well-established; for in our first and second cases the fact that a discharge of fæces took place—in the one case within an hour of the administration of an enema of iced water, in the other immediately after rectal examination—appears to indicate that when peristalsis is set up from the rectum it may really exert a favourable influence. I should therefore, even though the proceeding is not invariably successful, always recommend the trial of iced water enemata every 1 or 2 hours. To this treatment I ascribe the recovery in the follow-

ing case, which cannot but be regarded as one of intussusception in spite of the fact that no blood was passed per rectum.

Child of 6 months, examined for first time on 12th February, 1881. During the previous 3 days, constipation, which had resisted calomel, castor oil, and several enemata; also no flatus passed. Frequent vomiting of a yellow offensive fluid. Abdomen distended, nothing wrong with the rectum. Features sunken, extremities cold. Treatment: every two hours an enema of iced water, iced milk in teaspoonfuls, and wine. During the following night there was still frequent vomiting of a brown offensive fluid. After o'clock the child became quiet. Soon after, rumbling noise in th bowels, followed by 2 loose, brownish-yellow, very offensive motions. On the 13th, the abdomen softer, the patient seemed better, P. 140. Enema of iced water every 3 hours. Steady improvement. Several loose motions. On the morning of the 14th, a pulpy green motion passed for the first time. Child seemed well. In this case, also, feverish symptoms with diarrhœa occurred during the next few days, similar to those in child Ph. (p. 67).

Instead of enemata we may try the injection of a large quantity of iced water by means of an irrigator, and the mechanical pressure of the fluid may have the same effect as we try to produce by other mechanical methods of reduction—I refer to inflation of the bowel by means of bellows and the introduction of a whalebone probang with a sponge at the end of it, with which one endeavours to push the invaginated portion of bowel directly upwards when it can be felt in the rectum. These methods of treatment are sometimes successful, as is proved by a small series of cases of recovery (Nissen, Senator, Herz[1], and others); and there is nothing to be said against their employment, provided we proceed with caution and promptly desist if no result is produced. The danger lies in the fact that we can never know beforehand whether the intussusception is still reducible, or is already fixed by adhesion of the two serous layers (the intussuscepted portion of bowel and its so-called sheath). In the latter case any forcible attempt at reduction might result in the adhesions and even the serous membrane itself giving way —in which case the consequences would of course be fatal. I should therefore only undertake attempts at mechanical reduction by the introduction of a probang when the invaginated portion can be felt from the rectum, and can readily be pushed upward, although it always comes down again. In such cases we may

[1] *Arch. f. Kinderheilk.*, viii., S. 31.

without danger repeat the attempt frequently, and it may at last lead to complete recovery. Otherwise I think it best not to have recourse to such a forcible proceeding, which may readily result in rupture and peritonitis. In the cases (and they are far commoner) in which we cannot feel anything in the rectum, but only diagnose the intussusception from the other symptoms, we may likewise try inflation with air, the patient being under chloroform; or should this not succeed, limit ourselves to irrigation with large quantities of iced water, applying an ice-bag to the abdomen and endeavouring to alleviate colic, if it is severe, by the use of opium or morphia (Form. 10). Further, recent experience in cases of ileus in adults goes to show that washing-out of the stomach is worthy of trial. No food must ever be given except small quantities of wine and iced milk (from a spoon). In rare cases reduction is said to have been produced by hanging up the child by the legs, and by massage of the abdomen (the patient being under chloroform) especially at the place where a tumour is felt. In desperate cases laparotomy—with reduction of the intussusception and formation of an artificial anus — has been often performed, but only very rarely with success. The reason for this want of success is, that even when the intussusception was found, it was nearly always impossible to draw the invaginated portion out of its sheath.[1] As it is impossible to establish any definite indication as to the exact time when laparotomy should be performed, the physician who risks this operation must himself incur responsibility for the result. Braun[2] has advised operating on the first or second day of the disease if possible, because reduction may probably still be effected at that time; but the one objection to this is, that the diagnosis is generally very uncertain in this early stage.

[1] Bell, Marsh and Hutchinson, *Jahrb. f. Kinderheilk.*, x., S. 427 *et seq.*—Gnändinger, (*Jahrb. f. Kinderheilk.*, xvii., S. 304) describes a fatal case in which laparotomy was performed, and the gangrenous portion of intestine resected. —Godlee, *Arch. f. Kinderheilk.*, iv., S. 310.—Lauds, *Jarhb. f. Kinderheilk.*, xx., S. 149.—Herz, *Arch. f. Kinderheilk.*, v., S. 386.

[2] *Arch. f. Chir.*, Bd. 33, H. 2.

## XII. *Rectal Polypus.*

We have already found that a discharge of blood from the intestinal canal occurs in melæna neonatorum (vol. i., p. 65), in intussusception, and also in colitis, dysentery and its sequelæ, and that the blood in the latter cases is always more or less mixed with mucus or fæcal matter. Other causes of hæmorrhage in the bowel, especially gastric ulcers and intestinal cancer, are extremely rare in childhood. I have several times met with piles in children, *e.g.*, in a boy of 6, who sometimes complained of pain on defæcation and had, just above the anus, three or four bluish-red venous hæmorrhoids of the size of a pea or larger, which were extruded from the anus on pressing down. Regular hæmorrhage from piles I have not as yet met with. We also occasionally meet with cases of passage of blood from the intestinal canal which, in spite of the most careful investigation, remain a mystery so far as diagnosis is concerned. Example—

> In March, 1868, I was consulted about a child of 1½ years, who had suffered for 8 days previously from gastric symptoms, loss of appetite, tendency to constipation, and furred tongue. Vomiting suddenly set in, and recurred several times in the course of a few hours, large quantities of food and mucus being brought up along with some blood. An hour afterwards, copious discharge, from the anus, of dark blood mixed with fæces; this recurred 4 times in the course of the night. The whole amount of blood passed would be about a cupful. At the same time no fever and no collapse. On the following morning the child vomited several times, and showed signs of colic. Then followed a few normal stools, and the recovery was permanent.

I must leave it to yourselves to form a diagnosis of the above case. There is a class of cases in which the hæmorrhage is caused by polypi in the rectum which occur pretty frequently between the ages of 3 and 12; and the diagnosis is then much simpler. Polypoid growths on the mucous membrane of the large intestine, varying from the size of a pea to that of a bean and larger, are by no means uncommon in children. I have sometimes found them post-mortem in large number in children who had died of the most diverse diseases, and had presented no particular intestinal symptoms. The rectum, however, and especially the part of it an inch or two above the

sphincter, is the most usual seat of polypi. They arise from an overgrowth of the mucous membrane and its mucous glands (adenoma), and they may reach the size of a cherry or even that of a small plum, and finally retain their connection with the point from which they originally sprang merely by means of a long narrow pedicle.

The first and indeed the only symptom of rectal polypi is hæmorrhage; this is rarely spontaneous, and almost always occurs only during or immediately after defæcation, the blood oozing from the anus in drops, or in somewhat larger quantity, so that the child's clothes are found to be stained by it. The blood is never thoroughly mixed with the fæces but lies on the surface, being passed towards the end of defæcation. In every case of this kind you ought at once to suspect polypus of the rectum, and on no account to treat the matter as of no importance. For, even although the amount of blood lost each time is small, yet the daily repetition of the hæmorrhage makes the children weak and anæmic. The trouble very often lasts for many months or even longer before the parents think of consulting a doctor. I have, indeed, known it to be mistaken, in the case of little girls, for premature menstruation and entirely disregarded. On closer enquiry we often learn that whenever the child's bowels are moved, "something comes down" out of the anus; and if we either happen to be present when defæcation takes place or (in the case of older patients) if we ask the children to press down strongly, we may sometimes catch sight of a dark-red rounded swelling, from the size of a bean to that of a cherry or larger, which bleeds readily on the surface. This is extruded from the anus, and drawn back again when defæcation is over. I should, however, particularly impress upon you that it is quite a matter of chance whether you make this out or not. I have very often sought to discover such a tumour extruding from the anus but have never succeeded in seeing it, although the patients themselves said that something came out from the anus when their bowels were moved and then went back again. The digital examination of the rectum is, then, always indispensable; and it is best to perform it with the children in the knee-elbow position. In doing so you can often feel the polypus distinctly just above the sphincter. But we must not rely too much on this examination. Should the polypus be situated somewhat high up it may

escape the finger by slipping out of reach at the point where the rectum widens out, and then the examination reveals nothing. In such cases also we may have difficulty in detecting the polypus by means of the anal speculum, because when the speculum is introduced it is apt to push the polypus out of the way.

In June, 1877, a girl of 6 years was brought to my consulting-room, who was said by her mother to have suffered from prolapse of the rectum. The frequent occurrence of hæmorrhage during defæcation was, however, rather in favour of the presence of a polypus; and I therefore had the child taken into my ward in order that the matter might be more thoroughly examined. In spite of the most careful examination on several occasions, neither I myself nor the resident medical officers were able to feel a polypus in the rectum, although the mother stoutly maintained that a tumour had been extruded from the anus only a quarter of an hour before. The patient was made to sit on the stool and press downwards strongly, and when this was unavailing I discharged the child without having come to any definite decision about the case. A very few minutes afterwards, however, the mother came back with the child, and now actually showed us a bluish-red polypus, of the size of a plum, projecting from the anus, connected with the mucus membrane of the rectum by a narrow pedicle more than ¾ inches in length. I at once seized the pedicle with my two fingers in order to fix it and cut it off; but before I was able to do so the child got frightened, and made a violent movement: the pedicle was torn, and the polypus remained in my hand. From this moment the hæmorrhage ceased and did not return.

You may infer from this case that rectal polypi with long narrow pedicles may get torn off during the passage of hard fæcal masses, and as a fact this mode of spontaneous separation is by no means uncommon, although the polypus is not always found. The only treatment is that by operation. We seize the polypus when it appears externally with a pair of dressing forceps, draw it still further out and cut the pedicle through with scissors. It is a more difficult matter when the polypus is not to be got at outside the anus, and we have to try to catch hold of it within the rectum; but in any case division of the pedicle is preferable to ligature as being the quicker method, and it is not at all dangerous.

### *Prolapse of the Rectum.*

Prolapsus ani is a much commoner condition than polypus in children, and, as its occurrence in later life is very rare, we may almost look upon it as an affection peculiar to childhood. It is most frequently found in the first two years of life, although it occurs often enough also between the ages of 2 and 6.

This condition is really an invagination of the rectum in miniature, but as it is extruded from the anus it has no sheath. We must here bear in mind the fact that above the lowest, very muscular, portion of the rectum, there is another which is somewhat wider and laxer, and that this again passes above into another which is encircled by a strong muscular coat. Now it is the latter that, by means of its contraction, may cause the middle lax portion to become invaginated within the lower and to be extruded from the anus in the form of a tense, glistening, red swelling. Sometimes nothing but mucous membrane is extruded; but this only produces a small prolapse, and larger ones may be held to be always due to an invagination of all the coats. The length of the prolapsed bowel varies greatly, being on an average 1¼—1½ inches, in a few cases a little more. The surface, that is to say the extroverted mucous membrane, often bleeds, and I have occasionally seen it more or less covered with living thread-worms. Like polypus, prolapsus—especially when small in size—is generally only visible during defæcation, and is afterwards retracted spontaneously into the rectum. But when the prolapse is larger it sometimes remains outside the anus; and in such cases the mother should return it each time after defæcation is completed—but she cannot always do so. Every practitioner knows that such attempts at reposition may be exceedingly difficult, not on account of contraction of the sphincter—which in these cases is likely to be very weak—but because the child at once presses down strongly whenever the finger is withdrawn after reposition, and thus forces the prolapse out again. One child of 3 (whose two brothers also suffered from polypus ani), was able to retract the prolapse by taking a deep inspiration when his mother told him to "draw up."

In many cases, especially in very young children, I have found it impossible to discover the cause of the prolapse (which had often already lasted many months) in spite of the most careful

investigation. The children were in other respects perfectly healthy, and had never suffered from any distinctly intestinal symptoms. Occasionally I have found as a cause a habitual state of constipation which always necessitated a constant pressing down; oftener, it was to be referred to there being or having been an obstinate attack of diarrhœa or dysentery. In these cases the cartarrhal swelling of the mucous membrane and the increased laxness of the middle portion of the rectum are to be regarded as predisposing factors. Prolapsus ani may arise from excessive intra-abdominal pressure during a paroxysm of whooping cough or during violent screaming, although I have but seldom seen this happen, Whether atony of the internal sphincter may not play some part in the causation of prolapsus ani, is a question as yet undecided. That it has some influence, seems to be indicated by the fact that in most of these cases we can pass the finger into the rectum very easily, and do not meet with the very strong resistance which is generally experienced. Moreover, we find that in such cases the most efficacious remedies are those which have the power of increasing muscular contraction.

The constant recurrence of severe pressing down is further proved to be of importance in producing prolapse, by the fact that stone in the bladder is apt to give rise to it. I advise you therefore, particularly in boys suffering from prolapse who have passed the second dentition, always to bear in mind vesical calculus, especially when other suspicious symptoms are likewise present, such as dribbling of urine, frequent micturition and unusual length of the penis. In two cases of this kind, where prolapse of the rectum accompanied every defæcation and even micturition, a calculus was found on examination, and was afterwards removed by operation in the surgical wards. There can be no doubt that a constant pressing down during micturition affects the muscles of the rectum by sympathy; and when this action is constantly renewed, it finally causes prolapse. The oftener the bowel comes down, the more is the sphincter stretched and relaxed; and this atony, in turn, favours the occurrence of the prolapse. It is asserted that oxyurides in the rectum may also produce it, by the irritation which they produce exciting violent reflex pressing down, *i.e.*, in the same way as in a case of colitis or dysentery implicating the rectum, the

tenesmus is occasionally accompanied by prolapsus ani. As I have already mentioned, I have myself seen a few cases in which the prolapsed and reddened mucous membrane was covered in places with thread-worms: but as I soon lost sight of the patients I am still in doubt whether there was here any causal relation or merely a chance complication.

Prolapsus ani is almost always a chronic condition lasting many months or even years. Temporary improvement and unexpected relapses are not uncommon. It is therefore always advisable to commence treating the malady as soon as possible, and not to leave it to nature. Occasionally recovery takes place with wonderful rapidity. Thus, I remember a very few cases in which re-position on a single occasion (the finger being left in the rectum for a few minutes) was sufficient to cure the prolapse permanently. One of these was that of a girl of 9 who was admitted into the hospital on March 25th, 1874, with severe prolapse which recurred at once after every reposition, and was cured permanently by a subcutaneous injection of strychnia (gr. $\frac{1}{33}$) and a pledget of lint introduced into the rectum and fixed with a T-bandage—even although the pledget had to be removed next day on account of diarrhœa. Such cases as this, however, are exceptional and very difficult to explain. In other cases, reposition and even the application of a pad are only palliative means as they fail to prevent the recurrence of the prolapse and must constantly be re-applied. I must here mention that when the prolapse is being returned (which is best effected in the knee-elbow position) its central part, as being the portion which last came down, must always be first pushed in. You insert two fingers of the right hand, covered with an oiled rag, into the central opening of the prolapse, and then gently press inwards. In children who scream and press down violently while this is being done, the administration of chloroform may be necessary. In order to prevent the immediate recurrence of the prolapse we apply a thick compress or a sponge to the anal orifice, and then draw the nates tightly together by a firmly applied bandage or by broad strips of plaster.

As the chief indication is to give tone to the sphincter ani, those medicines are to be recommended which favour its contraction, especially nux vomica and strychnia. The former I have very frequently used (Form. 37); but its success

has been so uncertain that I have now very little faith in it. Strychnia has been given by French physicians either in the form of powder applied locally to the prolapsed bowel, or by subcutaneous injection; but, judging from my experience, I do not consider it at all a trustworthy remedy. I have obtained much better results from subcutaneous injections of ergotin into the perineum and immediate neighbourhood of the anus.[1] In children of 1½—3 years I inject ergotin, gr. ⅓—1⅓ once daily (Form. 38), and as a rule a distinct improvement takes place within the next 8 days. First, the prolapse no longer accompanies every evacuation, but only occurs at intervals, sometimes at intervals of days; then, after a few weeks, it disappears entirely. Although I have repeatedly verified the value of ergotin (which never has any injurious local effect) in these cases, I am still far from regarding its action as absolutely certain. As a fact you will often meet with very obstinate cases which resist the action of this remedy and require other methods of treatment. I have only seen temporary benefit result from the insertion in the rectum of pretty large fragments of ice, and nearly as little from enemata of a solution of tannin, or alum, or astringent decoctions (krameria, oak-bark). I have sometimes seen rapid success result from repeated painting of the prolapsed mucous membrane with nitrate of silver (grs. xvii. to ℥i). When these methods fail, nothing remains but surgical treatment—either the excision of a few folds of skin round the anus, or better the application of Paquelin's thermocautery, in the form of points and lines, in the immediate neighbourhood of the anus.

Whatever treatment is tried, we must see to it that the children give over the violent pressing down. Sometimes the prolapse ceases to recur whenever the patients pass their fæces while lying in bed instead of sitting on the night-stool, because then the intra-abdominal pressure does not tell nearly so much. I therefore repeat once more the advice which I have been in the habit of giving for years—that you should not allow such patients to sit on the stool in the usual way with their feet pressing against the ground, but should place the vessel on a steady chair or table and hold the child firmly on it with his legs hanging free; for in this position the amount of pressing down is much lessened.

[1] *Vide Charité-Annalen*, Jahrgang i., 1874, S. 614.

When there is constipation, defæcation must be rendered easier by the use of laxatives; while in cases of diarrhœa and dysentery, the prolapse may be cured by the successful treatment of these affections.

## XIV. *Intestinal Parasites.*

Helminthiasis (worm-disease) formerly bulked largely in the pathology of childhood, but is now relegated to an extremely minor position. But although medical opinion does not now attach such undue importance to internal parasites as it once did, they are still regarded by the public as of very great importance. This opinion is not confined to the lower classes; and a physician often finds it somewhat difficult to refrain from making this diagnosis, which is forced upon him by mothers who ascribe the most diverse ailments of their children to the presence of worms; and he has even to guard against being intentionally deceived. We often enough find mothers asserting that they have seen worms in their children's stools, and afterwards acknowledging that the statement was untrue. In all these cases, however, we must beware of going too far in making light of the presence of worms—as is now the fashion owing to a reaction from the old ideas—and of regarding them under all circumstances as quite harmless inmates of the child's alimentary canal. Although cases do now and then occur, they are comparatively rare, in which the influence of these guests in causing definite symptoms is indisputable, and in which recovery ensues from treatment directed against the parasites.

Into the natural history of intestinal worms I shall not enter here, and for it I refer you to the classic works of Davaine[1] and Leuckart.[2] The following will suffice, I think, for our purposes, although three groups only of internal parasites are referred to—oxyuris vermicularis, ascaris lumbricoides, and tænia.

1. Oxyuris vermicularis (thread-worm) is a little white worm about $\frac{2}{5}$ in. long and $\frac{1}{50}$ in. broad; it is spindle-shaped, and ends posteriorly in a sharp point. It inhabits the colon in large numbers, but shows a greater preference for the rectum, where it is nourished by the fæces. In former times only the

[1] *Traité des entozoaires*, 2. édit.: Paris, 1877.
[2] *Die menschlichen Parasiten u. s.w.*: Leipzig, 1868.

females were known; the males, which were first discovered during this century by Bremser and Sömmering, are much less numerous than the females (being in the ratio of 1 to 9), are much smaller ($\frac{1}{10}$—$\frac{1}{8}$ in.) and end in a spiral tail. The oxyurides (which are also called "ascarides" when they occur in masses) may be discharged with the fæces, on the surface of which they are visible as innumerable moving worms which look like little pieces of thin white thread. At other times they wander out from the anus of their own accord independently of defæcation, generally in the evening when the children are going to bed, and by their movements excite a lively irritation which causes the children to scratch the neighbouring parts; and on the strength of this itching the parents usually form their diagnosis of "worms." Careful examination even at this time often reveals a number of thread-worms around the anus, and the mothers often collect a considerable number and bring them to the hospital. In many cases, however, we not only find itching, but there may be actual pain in the anus, generally at bedtime, which gives rise to crying. I remember one boy especially who suffered in this way: he was extremely restless, threw himself on the ground, screamed passionately and pressed the anus against the ground with all his might in the attempt to find relief. The almost periodic regularity with which such scenes are repeated every evening makes them likely to be mistaken for convulsions, unless they are very carefully observed; and as a fact inexperienced practitioners have often been misled into the error of diagnosing incipient intermittent fever and ordering quinine, and have recognised their mistake afterwards on discovering the presence of thread-worms. Sometimes the itching occurs by day also, or in the middle of the night; and in that case we almost always find worms outside the anal aperture. As the worms are only capable of movement when lying on a moist surface (such as the mucous membrane), and when they get outside on the dry surface of the skin, very soon become motionless (for they have no springing power), we can hardly suppose that they can ever get further of themselves. Although it cannot be denied that oxyurides are found in the vulva in little girls, where by their irritation they give rise to hyperæmia, mucous discharge and a tendency to masturbation—still, this happens far less commonly than is usually supposed. Only on two or three occasions have mothers

told me of having found worms in this situation; but I have never had ocular proof of it. And surely it seems more probable that in these cases a direct transmission of the worms and their eggs has taken place by the medium of the child's fingers while scratching, rather than that the parasites have of their own accord found their way into the vulva. The same remark applies to the cases—common enough—in which not only several children of the same family, but the mother also is suffering from thread-worms. Under such circumstances one is apt to assume off-hand that the worms have travelled from one person to another, without considering that for the worms to travel from the anus of one individual to that of another is a sheer impossibility; for in going such a distance they would inevitably get dried up. In these cases also, then, we must assume a direct transmission of the oxyurides or their eggs by the fingers, by sponges, &c. It is also established that the majority of the worms which find their way out of the anus, or are discharged in large numbers with the fæces are pregnant females, and that the patient's fæces always contain a quantity of mature ova. By means of the fingers which have been used in scratching and also owing to the drying-up and pulverisation of fæcal matter, the eggs may readily (especially in small uncleanly rooms) get into the stomachs of other persons, and there their capsule is dissolved by the gastric juice and the embryo liberated. This also accounts for the great difficulty of successfully treating those parasites (which in some families it takes many years to extirpate), and likewise their occurrence in idiots of dirty habits, in whom we sometimes find the colon covered by a thick layer of oxyurides like a fur (Vix). Transmission of mature ova or of hatched embryos in this manner may certainly be held to explain the rare cases in which thread-worms have been found in other situations far removed from the colon, *e g.*, on a moist eczema of the inguinal folds (Michelson[1]), or even in the mouth (Seeligsohn[2]). The genuineness of such cases, however, seems to me doubtful owing to the fact that in these regions the fæcal matter which is necessary to the worms is altogether absent.

2. The Round-Worm (Ascaris lumbricoïdes). These are cylindrical annelids, resembling earth worms, of a brown or

[1] *Berl. klin. Wochenschr.*, 1877, No. 33.
*Ibid.*, 1878, No. 40.

reddish-grey colour, and of considerable size. The female grows to nearly 15½ in. in length, the male rarely to more than 10 in.; their greatest diameter being ¼ and ⅛ in. respectively. The body grows at both ends, especially at the anterior end; the mouth is surrounded by three lips furnished with extremely fine teeth; the caudal extremity is short and conical. The posterior part of the body in the male is hook-shaped and curled upon the abdomen, and the penis, which is club-shaped, is often seen projecting from the pouting cloacal aperture. The vulva lies just behind the anterior third of the body, or more towards the centre.

Round-worms are occasionally present in the child's small intestine, in almost incredible numbers. I remember one child who while taking an electuary of santonica passed, during many days in succession, such a large number of round-worms of different sizes that they almost filled the chamber-vessel; and yet there had been no symptoms of the presence of such enormous numbers in the intestine.[1] You will now readily understand how, in such cases, the worms may get entangled together into large balls so as actually to obstruct the lumen of the intestine and cause symptoms of ileus (p. 64), and even give rise to a tumour, which can be felt from without through the abdominal wall.[2] In the great majority of cases, however, the number of worms present is much smaller; and in the many post-mortems of children which I have seen, it has always struck me as remarkable that we so seldom find large numbers of round-worms in the intestinal canal. I have often found only a single specimen. If then, as is shown by the case above, there may be no striking symptom in spite of the presence of enormous masses of worms, we may readily infer that a similar absence of symptoms will occur much oftener when there are only a few worms in the intestine. In fact, I can mention to you only one thing from which you may infer the presence of round-worms with certainty: namely, that some have been passed. At a certain stage of its development the worm sets about quitting its habitation, and then it wanders from the small intestine downwards to the colon and upwards into the duodenum and stomach. From the former it is discharged with the motions, either dead or alive,

[1] Fauconneau-Dufresne (*Union méd.*, 1880, p. 62) publishes the case of a boy of 12 who in the course of 3 years passed more than 5,000 round worms, partly per anum and partly by vomiting.

[2] *Jahrb. f. Kinderkrankh.*, 1876, x., S. 298.

or it may creep out of the anus (apart from defæcation); and then it is usually found lying in the child's bed, curled up upon itself. From the stomach it is expelled by vomiting, or else by its own movements it finds its way up the œsophagus into the pharynx, or even further. Cases are not extremely rare of the worm having crept out of the child's mouth during sleep, and being found dead, in the morning, on the pillow. Apart from the microscopic discovery of ova in the fæces, this expulsion of the round-worms is (as in the case of thread-worms) the only indubitable sign of their presence. All the other symptoms to which the laity, and even many practitioners attach importance—the pale complexion, the dark rings round the eyes, the foul breath, the itching of the tip of the nose, and the frequent colic—can do nothing beyond rousing suspicion and so justifying the use of anthelminthics. Nothing is really conclusive except the discovery of worms or their ova in the discharges. Where this proof is wanting, you must never be content to explain morbid symptoms by assuming the presence of worms; you must rather bear in mind that the symptoms may possibly be due to some disease which is quite different and much more serious in its nature, and this possibility can only be excluded by repeated and careful examination.

We may here touch on the much discussed question as to whether round-worms are capable of exerting any local action on the portion of intestine which they inhabit. We should not be justified in denying this *à priori*; for the worms when present in very large numbers in the rectum are certainly capable of setting up a catarrhal condition there. The character, also, of the parasite's mouth (the lips being furnished with teeth) is in favour of the possibility of such influence. As a matter of fact it is stated that hyperæmia of the mucous membrane of the small intestine, and even diarrhœa, may arise from the irritation of round worms; but in my own practice I have never met with an undoubted case of this. We must always bear in mind that in an ordinary attack of catarrhal diarrhœa the round-worms which happen to be present in the intestine, and are on their way to the colon, may be discharged along with the fæces just as the same thing may occur in cases of dysentery or typhoid fever. Some writers, however, go much further, and think it possible for the round-worm, by dint of pressing strongly with its head

against the mucous membrane, to force apart the fibres of the mucous and other coats of the intestine (even without using its teeth) and slip into the peritoneal cavity through the opening thus made. Those who hold this view point to cases in which one or more of these worms were found free in the peritoneal cavity, although no aperture could be discovered at any part of the intestinal canal through which it was possible for the parasites to have passed. I cannot believe in this theory, according to which the worms slip through the coats of the intestine by pushing aside their tissue elements, the hole so formed becoming hermetically closed; nor yet in the supposition that the round-worm is capable of "eating its way through" the intestinal wall by means of its "toothed" lips. I think it more probable that in any case where round-worms have been found in the peritoneal cavity they have crept through some ulcerated aperture already existing in the intestine. This aperture may have been already in communication with a localised peritonitic deposit, or have been still covered only by the delicate gauze-like serous coat which gave way when the worm pressed on it. Least of all can I imagine that the worms after having found their way by their own exertions into the peritoneal cavity are capable of exciting a circumscribed peritonitis ending in a discharge of pus externally. Such an abscess—which is called a "worm-abscess" on account of worms coming out of it—I have never myself met with. I have, however, in two cases of chronic tubercular peritonitis observed spontaneous rupture take place through the umbilicus or in its neighbourhood, and after the evacuation of some fæculent matter a few round-worms also were discharged. I believe that all cases of "worm-abscesses" are to be similarly explained, *i.e.* the worms avail themselves of some aperture which happens to be in the wall of the intestine owing to follicular or tubercular ulceration, in the neighbourhood of which a circumscribed peritonitic deposit has already been formed, to make their way out of the bowel; they then find themselves in an abscess, to the origin of which they have in no way contributed.[1] In

[1] The case which Marcus publishes (*Deutsches Arch. f. klin. Med.*, Bd. xxix., H. 5 u. 6) of "perforation" of the duodenum by round-worms seems to me as inconclusive as the case of Wischnewsky (*Arch. f. Kinderheilk.*, vi., 207). In my opinion the former was a case of perforating ulcer of the duodenum, the latter one of a similar condition in the small intestine. See also Weihe, *Klinische Wochenschr.*, 1883, S. 131.

an experiment I ordered santonine grs. iii. with castor oil ℥ iss. (a teaspoonful 4 times daily). On the following day 2 living round worms were passed; whereupon the pains at once ceased and did not return. This child was brought to the hospital on 31st October, 1887.

In spite of my scepticism, I cannot deny the connection of the nervous symptoms in these cases with the irritation of worms; and I must also grant the possibility of the reflex symptoms taking a convulsive form.[1] There is consequently no objection to our trying anthelminthics in such nervous conditions, so long as we do not neglect to search carefully for any other possible source of the disease. Should no worms be then passed, there still remains the microscopic examination of the fæces in which you will find a more or less large number of worms' ova in almost every case of helminthiasis. When you have once become acquainted with the appearance of these ova, which are granular discs—of a round or oval shape according to the species of the parasite—you can readily distinguish them from any other similar objects which may be present; and when you find them, you may take the presence of worms as proved. This examination, which is always rather distasteful and to some, indeed, exceedingly repulsive, may therefore lead to a diagnosis of worms, even in a case where no worms are actually to be found.[2] In no case will anthelminthic treatment given at random (*i.e.*, without being preceded by microscopic examination) do the children any harm; unless, indeed, there is some positive contra-indication.

The treatment consists principally in the administration of santonin. Of this drug it is usual to give ¾—1½ grs. three or four times a day, in the form of powder or lozenges, during two or three successive days, and to give a purgative about the third day (castor oil, mist. sennæ co.), so as to hurry the worms rapidly through the intestinal canal—they having been rendered incapable of movement by the action of the santonin. Santonin has one great advantage over santonica (from which it is

[1] According to a case reported by Guermonprez (*Gaz. méd.*, 1880, p. 34) it seems that a series of hysterical symptoms may entirely disappear after the discharge of a quantity of tape-worms. See also Wischnewsky, *Arch. f. Kinderheilk.*, vi., 206.

[2] *Cf.* Banik (*Münch. med. Wochenschr.*, 1886, No. 26) who in 315 children found worms' ova in 38·8 per cent., those of oxyuris vermicularis being far the commonest. In 60 children under one year no ova at all were found.

prepared, and which was formerly given); namely, that children take it much more readily. Still, I hardly think that we can attribute to it any more favourable results than those which were formerly obtained by the latter. At any rate, I can remember cases in which I procured the evacuation of a much larger number of worms by the use, during several consecutive days, of Störk's "worm-electuary," or the electuarium anthelminthicum (Ph. Paup.), than I can effect now by using santonin. The reason may, perhaps, have been that in these electuaries the santonica was combined with a purgative (jalap). It is therefore best to give santonin also in combination with a purgative, *e.g.*, with calomel (grs. ¾—1½), or castor oil (gr. ½ of santonin in a teaspoonful)[1], and in this way also we shall best avoid the symptoms of poisoning (vomiting, coma, retention of urine, &c.) which have occasionally been observed after moderate doses of santonin (grs. 1½). It is a well-known fact that santonin imparts a yellow colour to the urine, and may even in exceptional cases cause yellow vision (xanthopsia). Since, however, other toxic symptoms have also been observed, such as urticaria, vomiting and even epileptiform convulsions (especially after larger doses, of 3—4½ grs.), it seems advisable to combine the drug with a purgative, if merely for the sake of hastening as much as possible its discharge from the body. For round-worms, we give either santonica or santonin, internally only; but in a case of oxyurides we must use, in addition, enemata or irrigations of the rectum, *e.g.*, with a solution of perchloride of mercury (gr. ½), or of santonin (gr. ½—3). These enemata or, better still, injections of a larger quantity of fluid by means of an irrigator—should be given towards evening and should be allowed to remain as long as possible in the rectum. Decoction of leeks or of hard soap, which are much used, have often failed in my hands; and indeed all the remedies in use, external and internal, prove often enough inefficacious. When the pruritus ani is very severe I order a little blue ointment to be rubbed into the neighbourhood of the anus several times a day,

[1] According to Lewin's experiments, the administration of santonin suspended in castor oil is further to be recommended, because when given in the form of powder or lozenges it is almost if not entirely dissolved in the stomach, but when combined with oil it is carried undissolved into the intestine, and can there exert its parasiticide action. (Caspari, "Ueber das Verhalten des Santonins im Thierkörper." *Diss.:* Berlin, 1883).—Lewin, *Klinische Wochenschr.*, 12, 1883.

or a suppository (ung. hydrarg. 2, with ol. theobrom. 4, or with pulv. saponis 3) to be inserted. In many cases I have seen a good effect from the use of suppositories of santonin (grs. 1½ with ol. theobrom. grs. 15), introduced in the evening and washed out in the morning by a simple enema.[1] I may remark in passing that pruritus ani occurs in children quite apart from worms, though only occasionally; but in these cases the child suffers more during the day than at night. Thus I have seen one such case in a healthy boy of 11 years, who was suffering from obstinate constipation. A few wine-glassfuls of "Ofener Bitterwasser" was sufficient on several occasions to relieve the pruritus by curing the constipation.

3. Tænia,—Tape-worm. Although these occur far less frequently than round-worms or thread-worms, still we find them in children as often—according to Monti,[2] even oftener—than in adults. I formerly published[3] 33 cases, and since then I have met with three times as many; not a month passes in which two or three children with tape-worm do not turn up at the polyclinic. The age of the patients varies greatly. Although I have not hitherto met with a case of tænia in a fœtus,[4] or in a child of 5 days,[5] still, I have seen several children just a year old who had frequently passed portions of tape-worms. The great majority of the patients, however, are between 2 and 12 years of age. When we consider the well-known connection between cysticercus and tænia, it is difficult to see how tape-worms could possibly occur in a fœtus or new-born child; since we cannot assume the possibility of an infection of the fœtus through the blood of the mother. In such cases the only form of tape-worm which could occur would be tænia "elliptica" vel cucumerina, which, according to Leuckart, may arise from the child having got into its mouth a dog-louse (tricodectes canis), a parasite found among the hair in dogs and cats. A few cases of this kind have been published.[6] In all cases of tænia which

[1] Lime water, quinine (¼—1 p. c. solution), larch turpentine (with gum acacia, ana grs. xiv., infus. camomile, ʒiv.) have also been recommended for use as an injection.

[2] *Arch. f. Kinderheilk.*, iv., S. 175.

[3] *Beitr. zur Kinderheilk.*, S. 133; *ibid., N.F.*, S. 327.

[4] Barrier, *Maladies des enfants*, ii., p. 98.

[5] *Oesterr. Jahrb.*, 1873, i., Analecta, S. 103.—*Jahrb. f. Kinderheilk.*, v., S. 444.—Hirsch u. Virchow, *Jahresber.*, 1872, ii., S. 701.

[6] *Cf.* A. Hoffmann, *Jahrb. f. Kinderheilk.*, xxvi., H. 3 u. 4.

I have met with in infants and children under 2 years, I have found that the patients had been given raw meat and sausages, in addition to their milk. Ever since Leuckart discovered in the flesh of oxen a cysticercus, which developes into tænia mediocanellata, just as the cysticercus in pork does into tænia solium, we have been able to understand how tænia may occur after the patient has partaken of pounded raw meat; but when, about 40 years ago, Weisse, of St. Petersburg, observed the occurrence of tænia in infants who were taking pounded meat for diarrhœa, no one could understand how the worms arose. According to Stein[1] tænia mediocanellata is much commoner than tænia solium (out of 221 cases there were 176 of the former and only 45 of the latter); but this calculation, as Monti rightly holds, applies only to children under 2 years of age, as it is only to them that raw meat is usually given.

The majority of children affected with tape-worm who have come under my treatment, presented no morbid symptoms whatever. The only thing which the mothers noticed was the passage from time to time of a few mature and still-moving segments (proglottides) or larger pieces, sometimes an ell in length. These were passed either with the motions or quite apart from them, and, in the latter case, they were found among the child's clothes or in its bed. It was only in comparatively rare cases that the children complained of anything, but there were occasionally pains in the stomach or bowels, nausea, regurgitation of water into the mouth, tenesmus, sometimes also a feeling of formication or numbness in the legs, or even difficulty in walking. More serious symptoms—such as chorea, epilepsy, catalepsy, &c.—which other authors have attributed to tænia, I have never as yet been able to refer with certainty to such a cause, and therefore I do not regard tape-worm as more injurious to the child's health than round-worms or thread-worms, and these, by the way, are often found in a child simultaneously with tape-worm. In children, as in adults, the eating of herrings and wild strawberries often results in the spontaneous discharge of proglottides or of large portions of the worm and at the same time, especially in little children, fretfulness, colic, and restlessness during sleep are observed. I have also several times observed portions of tænia passed along with

[1] *Entwickelungsgeschichte u. Parasitismus der menschl. Cestoden:* Lahr, 1882.

the motions in the course of an attack of acute chronic diarrhœa, and on one occasion with diarrhœa during the course of a case of typhoid. Occasionally the tænia which is partially extruded from the anus during defæcation, is torn in two by the mothers in their attempt to pull it all out. We should, therefore, refrain from pulling at the extruded portion of the tape-worm, and rather fix it outside the anus by means of sticking-plaster, or (as Monti has seen successfully attempted) have it carefully wound upon a piece of wood, and then try to obtain its complete discharge by the use of purgatives and enemata. My own experience does not enable me to decide whether the injection of chloroform-vapour into the rectum, which has been recommended for these cases, really favours the discharge of the remainder of the worm.

I have long regarded kousso as the best remedy for tape-worm in children. It always seems to me to be the most certain in its action, and we have therefore treated all our hospital cases with this drug to begin with. According to the patient's age I give in the morning 120—160 grains in two divisions (in coffee or milk), one dose half an hour after the other. This may indeed cause nausea or even vomiting. If we give a spoonful of castor oil an hour later, the loose motions which result in the course of the day very often—although not always—contain large masses of tape-worm, or even the entire chain of segments along with their apparatus for holding on, which we are wont to call the "head." In many cases, indeed, this "head" is not found, but only the extremely delicate portion near it, and in these I usually repeat the treatment 2 days later. It is also advisable to give castor oil the day before the remedy is administered, in order to clear out the intestine, and it is also well to give the patient herrings to eat in the evening. On the day of the treatment, however, when the purgative action has commenced, we should give an enema of cold water every 2 hours at least, so as not to allow the bowel to rest or the tape-worm, which has been rendered torpid by the kousso, to resume its hold on the wall of the intestine. We have indeed to record a very considerable number of cases in which this treatment was unsuccessful, but we have obtained no better results from the use of other well-known remedies. Of these I may specially mention pomegranate-

root and male-fern root, which I have generally given together (ext. filicis maris grs. 38—45, syr. aurant ʒv., decoct. granati rad. ad ℥vii., to be taken in 3 portions at intervals of half an hour).[1] After the lapse of an hour we give a teaspoonful of castor oil, and when it begins to act we administer the cold-water injections which I have just recommended. Recently I have tried muriate of pelletierin (the alkaloid of pomegranate-root bark) in doses of grs. iv., but upon the whole it has failed oftener than it has succeeded.

## XV. *Acute and Chronic Peritonitis.*

You will meet with acute peritonitis[2] less frequently in children than in adults, except that form of it which occurs in new-born children in connection with pyæmic and septicæmic symptoms. In these cases, however, the special symptoms—distension, tension, and tenderness of the abdomen, and vomiting—are usually so complicated by those of the general condition, or so masked by them, that we do not obtain a distinct clinical picture of the disease. In older children I have repeatedly seen acute peritonitis following scarlatinal nephritis, but especially perityphlitis—those inflammatory processes which take place in the cæcum and its immediate neighbourhood, especially in the vermiform appendix. Whether those forms of inflammation arise from excessive distension of the cæcum by fæcal matter or the irritation of a fæcal concretion in the vermiform appendix, they always present the same symptoms as in later life :—the pain, at first confined to the ileo-cæcal region, but soon spreading over the greater part of the peritoneum, the frequent formation of a tumour caused by the inflammatory exudation, and the repeated relapses. I shall, therefore, confine myself here to a few remarks on treatment, especially with regard to the use of opium in peritonitis. My experience is strongly in favour of this method of treatment, even in children.

[1] Monti (*l. c.*, S. 204) gives pomegranate-root in much larger doses, R. granati rad. cort. ʒiii., aquæ dest. ʒvi., macera per 48 hor. : decanta. Of this infusion he gives the child ʒiii.—v., and he maintains that he has been most successful with this; but he finally acknowledges that no remedy is absolutely sure to act, that one cannot say definitely beforehand what remedy will be most successful, and that in exceptional cases one must make use of all three remedies.

[2] Pott, *Jahrb. f. Kinderheilk.*, iv., S. 157, 1879.

I keep the intestine completely at rest, and do not give castor oil or calomel unless I am convinced of the presence, from the first, of large masses of fæces in the cæcum—because there has been constipation for some time, or because they can be made out on palpation. When there is no accumulation, I should advise you to abstain from giving purgatives, and if the ileo-cæcal region is very tender to apply 4—8 leeches, according to the age (without after-bleeding), and to keep an ice-bag steadily applied to this region. I have seen a few cases in which cold could not be borne, and in such cases the ice-bag must be replaced by warm poultices. Internally, I give extract of opium, gr. $\frac{1}{10}$—$\frac{1}{5}$ along with some syrup in an oily emulsion, every two hours until the spontaneous pain ceases and the tenderness on pressure diminishes. As soon as this occurs, defæcation generally recommences spontaneously, or after the use of an enema or a spoonful of castor oil. Constipation lasting for 6 to 7 days is of no serious significance. By this method of treatment, when commenced betimes, I have been able in almost all cases to bring about recovery, and, in those where a tumour had been formed by the exudation, to prevent the occurrence of suppuration—even in children who in the course of a few years were frequently readmitted into the hospital on account of relapses. I have only seldom found opium fail, *e.g.* in a girl of 5 years who, during the whole course of an attack, suffered from violent colic, diarrhœa and tenesmus. In this case, castor oil (a teaspoonful every hour) had to be substituted for the opium; and this brought about recovery within a few days, during which there was a persistent evacuation of small scybala which had been retained.

The exudation which takes place in the neighbourhood of the cæcum forms a hard swelling, which can be distinctly made out by palpation and by its dull note on percussion. It may extend beyond the mesial line, and upwards as far as the level of the umbilicus. Should it not be re-absorbed under the steady use of an ice-bag, but increase in size and tenderness, while the fever persists with evening exacerbations, we must order hot poultices continued day and night; and in such cases we sometimes have spontaneous discharge of the pus, either externally, into the intestine (which I have twice seen), the bladder or vagina. Should this termination by rupture be delayed, and should the persistent hectic threaten to exhaust the strength, we must set

about opening the abscess by surgical means. This operation has lost a great part of its danger since the introduction of antiseptic methods.[1] Such peritoneal abscesses or "abdominal empyemata" may, moreover, form quite independently of perityphlitis in other situations of the abdomen, either without evident cause or as the result of an injury.

A girl of 10 years, who came to my polyclinic on November 11th, 1879, had been badly maltreated in August by a big dog, which had thrown her down and trodden upon her abdomen. This was followed by acute peritonitis, which terminated in the middle of September with a discharge of pus through the umbilicus. In November there was still, in the place where the umbilicus had been, a red sore of the size of a sixpence, covered with granulations, from which there was a continuous discharge of pus.

Boy of 4 years, admitted on 4th January, 1883. Shortly before Christmas he had fallen on a piece of paling, landing right on his umbilicus. In the beginning of January, great swelling of the umbilicus and infiltration round about it. Treated in the polyclinic by painting with iodoform-collodion. On the 10th, spontaneous rupture and discharge of a cupful of pus. On the 13th, discharge of fragments of potato and shells of peas from the abscess-opening at the navel. The action of the abdominal pressure caused the evacuation of much fluid fæcal matter, which, however, had not a fæcal odour. Motions normal, no fever, no pain. Careful antiseptic dressing. Discharge on 21st. Further treatment as an out-patient. Complete recovery.

Boy of 2 years, admitted on 29th October, 1883. Said to have had a fall on the belly in May, which was followed by swelling of the umbilicus. This gradually increased, and in October there was an extensive phlegmon. From the umbilicus, which lay in the middle of this, there was a discharge of thin brown masses without a fæcal odour, and these became of a lighter colour when the child was restricted to a milk-diet. Appetite and motions normal. Temp. 97·7° F. Discharged on November 5, unrelieved.

M. L., 10 years old, examined by me for first time on December 18th, 1876. Symptoms of acute peritonitis for past 14 days, which had begun without discoverable cause in the left iliac region (application of leeches on two occasions, ice-bag). I found a diffuse swelling, very tender, and dull on percussion, occupying the lower part of the left half of the abdomen, and extending to above the level of the umbilicus, while the right side was quite free. There was also pain during defæcation and micturition, remittent fever,

[1] Domme (21. *Jahresber. d. Jenner'schen Kinderspit.*, 1884) reports 2 cases in which perityphlitis and abscess occurred from foreign bodies having been swallowed (glass beads, buttons) and which were cured by operation.

great debility. In the last few days attacks of very violent colicky pains, with loud screaming and collapsed appearance, the intervals being quite free from symptoms. Treatment: warm poultices to the abdomen, oily emulsion with extract of opium. On the 20th, repeated discharge of mucus and purulent fluid from the rectum, and during the night 4 or 5 very copious loose purulent motions, of the colour of café au lait. On the 21st, swelling and pain entirely gone. Continuation of the purulent discharge, mixed with little fæcal masses. Complete recovery a few days afterwards. I afterwards learned that 2 small relapses occurred in the same situation during the next 2 years; they did not, indeed, result in suppuration, but still they excited some suspicion that there must still exist some unknown local cause for the peritonitis.

In these cases we find the peritoneal abscesses discharging either through the umbilicus or into the rectum. When we consider that the umbilicus is the part of the abdominal wall which yields most readily, that the fascia is absent in this situation, and that the abdominal cavity is here closed only by the cicatrix of the skin, the fat and the peritoneum—we can readily understand how in all distended states of the abdomen, whether due to pregnancy, solid tumours, or fluid, thinning and hernial protrusion of the umbilicus are apt to occur. This is especially so in childhood, when the umbilicus is even less capable of resistance than in adults. Although some authors[1] have maintained that most cases where there is a discharge of pus from the umbilicus are not really cases of peritonitis, but that the suppuration has almost always arisen outside the peritoneum, or in the so-called subperitoneal tissue, yet I am of opinion that they carry their contention too far. At the same time, my own experience forces me to admit that abscesses do occur in the abdominal wall, which generally arise from an injury, and are readily mistaken for peritonitis. I shall soon have an opportunity of giving you some cases of chronic peritonitis (confirmed by post-mortem examination) in which there was a discharge of pus (and, in one case, of ascitic fluid also) through the umbilicus; and I see no reason why an accumulation of pus taking place acutely might not give rise to the same occurrence. At any rate, the communication existing between the umbilical abscess and a

[1] Vaussy, *Des phlegmons sous-péritonéaux de la paroi abdominale antérieure*: Paris, 1875.—Gauderon, *De la péritonite idiopathique aiguë des enfants*: Paris, 1876.

coil of small intestine (discharge of food and fluid fæces without any fæcal odour) which was found in the second and third of my cases, proves that here we really had had a circumscribed abscess in the abdominal cavity itself, which had discharged on the one hand through the umbilicus and on the other through the intestine.

In another case, that of a girl of 12 years, as well as in that given above, the left iliac region was the original seat of the peritonitis, and in that situation there had been a considerable inflammatory deposit (January 1881). The peculiar feature in this case was the fact that after the acute inflammation had been relieved by ice-bags, opium, &c. (although the tumours caused by the exudation remained) there occurred in the course of the next few weeks 4 or 5 attacks which might be regarded as ileus (obstinate constipation, frequent vomiting, severe pain in the abdomen, moderate fever, collapsed expression of the face). These attacks usually lasted 12—18 hours, and terminated in the discharge of a hard plug of fæcal matter, followed by a large number of scybalous masses. Castor oil and enemata were sufficient in the first attack only, in the later ones we had to administer irrigations of iced water every 2 hours in order to effect the discharge of the fæcal plug. Medicine was generally vomited, and we therefore ceased giving it; the pain was relieved by subcutaneous injections of morphia. The tumours disappeared by the end of February, and after that the child remained perfectly well. The condition was evidently due to the lower part of the colon having been compressed or bent upon itself by the surrounding exudation, so that the fæces went on accumulating and became compressed into a hard plug.

Acute peritonitis due to perforation of one of the abdominal organs is very rare in children, apart from the cases above-mentioned of ulceration of the vermiform process.[1] This is to be explained by the great rarity at this age, of round gastric ulcers and other diseases of the abdominal organs ending in perforation. Even typhoid fever, as we shall see later on, produces it only in very exceptional cases. I have, however, several times seen

[1] In a boy of 11 years who died after 6 days' illness with symptoms of acute peritonitis, the cause was found at the post mortem (16 June, 1884) to be perforation of the vermiform process by a fæcal concretion, the centre of which was formed by an orange pip.

diffuse purulent peritonitis following scarlet fever, especially secondary nephritis; but I shall return to this in considering that disease.

We now pass on to chronic peritonitis. Apart from the very common tubercular form, this is to be regarded as a rare affection. Adhesions of the abdominal viscera, especially of the internal genital organs which are found so often in adults, and particularly in women, are very rare in childhood; while cases presenting characteristic symptoms of non-tubercular chronic peritonitis are so rare that some writers utterly deny that they ever occur. This opinion, however, is not justified, and the following case[1] affords incontestable proof of the fact.

Anna S., 4 years old, admitted into the hospital on 14th November, 1873; said to have been quite healthy till 8 days previously (?). Since that date distension of the abdomen had been noticed. On examination we found an extreme degree of ascites, flattening out of the umbilicus, and distinct fluctuation. Absolutely no pain or tenderness of the abdomen. Liver-dulness reaches to the 5th rib. Resp. 28—40, some dyspnœa. On the right side, dulness and weak breathing below the scapula. Slight œdema of the feet; urine normal; no fever. On the 16th, the abdomen was punctured with an exploratory trocar, and 6 pints of a greenish, very albuminous fluid were removed, containing numerous pus-corpuscles, fibrinous coagula, and a few flocculi, which were found under the microscope to consist of a fibrous network filled with cells, and which therefore led us to suspect that there was a sarcomatous growth in the abdomen. On palpation, however, we found nothing abnormal except that the lower margin of the liver extended downwards 1½—2 inches; no tumour anywhere. But as the ascites returned within 8 days to the same condition as before the operation, the puncture was repeated on the 24th with the same result as before, but this time nothing was found that seemed to indicate a new growth. Until 13th December—*i.e.* for about 20 days—the condition remained pretty much the same; temp. often rising to 101·5° F. in the evening, pulse 96—140, general condition evidently getting worse, emaciation increasing, repeated vomiting and occasionally diarrhœa. Under expectant treatment the ascites diminished remarkably, and on 13th December we could feel distinctly in the umbilical region hard bodies (some nodules, some like bands) through the abdominal wall, which had now become flaccid. These ran together at the border of the hypogastrium into a large tumour, which could be almost entirely surrounded by the hands on palpation;

[1] *Berl. klin. Wochenschr.*, 1874, No. 10.

and when the abdominal wall was moved over its surface, as on palpation, there was occasionally a distinct feeling of friction. Copious fæcal evacuations caused by castor oil produced no change, so that the diagnosis of a new growth seemed to be confirmed. The condition remained almost unchanged till death, which took place, with increasing symptoms of collapse, on the 21st, and on the last day the following condition was noted: "abdomen soft and yielding. Lower edge of the liver felt $\frac{2}{5}$—$\frac{3}{5}$ in. below the costal margin. To the right of the linea alba, extending from the margin of the liver downwards into the right iliac fossa there is a tumour of the breadth of one's hand, consisting of several sausage-shaped swellings adhering together, which is seen to project above the surface of the abdomen. The other tumours which were formerly made out are now less distinctly felt."

*P.-M.*—About 17½ oz. of turbid fluid in the abdomen. Both the visceral and parietal layers of the peritoneum presented all over tolerably broad and long streaks of recent fibrinous greyish-yellow lymph. The convolutions of the small intestine firmly adherent to one another all over by extremely short and dense peritoneal adhesions, so that they could only be separated by the knife. The serous coat of the intestine was extremely thickened throughout, friable and readily torn, opaque and lustreless, and in very many places transformed, along with the sub-serous tissue and the superjacent lymph, into a bluish-white semi-transparent fibrous tissue $\frac{1}{5}$—$\frac{2}{5}$ in. in thickness, which grated under the knife. Mesentery, and greater and lesser omenta, much shrivelled. The whole intestinal canal strikingly shortened, mucous membrane pale; liver somewhat enlarged, a layer of blood on its convex surface (perihepatitis hæmorrhagica). Right pleurisy with effusion.

Here, then, we have a case of very extensive chronic peritonitis without a trace of tuberculosis; for, as we afterwards discovered, the onset of the disease was to be attributed to an injury, namely, a kick on the region of the liver which the child had received a few weeks before from her brutal father. The disease probably began with the hæmorrhagic inflammation of the serous coat of the liver, which was found at the post-mortem. The process had gradually spread over the whole peritoneum, and had also affected the right pleura. From this, then, we see that injuries to the abdomen may be followed, not only by acute, but even by chronic peritonitis with considerable adhesion of the coils of the intestine to one another, and a large amount of serous effusion; and this may take place so slowly and insidiously that the increasing ascites is the first thing

to attract notice. You will also remark the very slight tenderness of the abdomen, and the fact that defæcation was generally normal in spite of the intestinal coils being closely matted together; and you will find the same thing in the tubercular form.

But in this case the great general fibrous thickening of the intestinal wall deserves especial attention. This assumed the form of tumours during life and so led me to the diagnosis of a sarcomatous growth in the abdomen, and that all the more strongly because the microscopic examination after the first puncture, and the palpable friction over the false tumour seemed to support this assumption. We may therefore conclude from this important case that fibrous thickening of the intestinal wall occurring in the course of chronic peritonitis may in places reach such a degree as to occasion a deceptive feeling of tumours (sarcomata). We may further conclude that the microscopic examination of the ascitic fluid in such cases may reveal bodies which by their alveolar structure suggest the idea that they are possibly fragments of a tumour, while in reality they are only fragments of fibrin with pus-corpuscles entangled in their meshes.

This is the only case of chronic non-tubercular peritonitis in childhood in which I have been able to confirm the diagnosis by post-mortem examination. I have, however, met with several cases which presented the symptoms of incipient or even far-advanced chronic tubercular peritonitis, which yet to my surprise recovered completely. Some of these children (who were almost all girls) had been formerly healthy, a few had suffered from osteomyelitis or other scrofulous symptoms. Are these to be regarded as cases of recovery from tubercular peritonitis or merely cases of simple chronic inflammation? I rather incline to the latter view, and I see no reason why the peritoneum may not become affected by a form of chronic inflammation accompanied by serous effusion quite apart from tuberculosis, just as well as the pleura. The chief symptom in these cases—sometimes indeed the only one—is ascites, for which, in spite of the most careful examination and inquiry, one can discover no cause; in particular, we must exclude any liver-affection. The general health may in the meantime be almost unimpaired, even pain or tenderness on pressure is not present in all cases—at least there are times

when it is entirely absent. In a girl of 11 who was in my ward for a long time and who recovered, the disease had arisen soon after measles; and a similar case was published by Fiedler.[1] The treatment which I have employed has consisted in painting the abdomen with iodoform-collodion and applying wet compresses. But the best results were got from early puncture, which was repeated thrice in the girl just mentioned, a large quantity of ascitic fluid being removed on each occasion.

## XVI. *Abdominal Tuberculosis.*

In treating of meningeal and pulmonary tuberculosis, I have already pointed out to you the frequent occurrence of caseous products in the abdominal organs. You very often find tubercles in the spleen and liver, sometimes in enormous numbers, and occasionally they are so small as to be scarcely discernible by the naked eye. Tubercles in the spleen and liver, however, may attain even the size of a pea or larger, and in the latter organ we frequently find in their centre a cavity filled with yellowish-green fluid—the lumen of a divided bile-duct surrounded by masses of tubercle. Very often we also find numerous miliary nodules in the peritoneum, large omentum, serous covering of the liver and spleen, diaphragm, bowel, &c.; and they are also to be found in the kidneys, and even in the internal genital organs in little girls. In these cases the mesenteric and other abdominal lymphatic glands are often more or less enlarged, and some or all of them change into caseous masses. But all these changes are only found accidentally at the post-mortem; we may indeed suspect their presence with probability in children who are known to be tubercular, but we cannot diagnose them with certainty.

On the other hand we may certainly make a diagnosis in cases where the tubercle is confined either mainly or altogether to the abdominal organs, even though the contents of the thoracic and cranial cavities are affected with tuberculosis very slightly, or just at the end of life. Although tubercle of the liver and spleen could not be recognised with certainty during life in any of the cases I have observed, and even the caseous degeneration of the mesenteric glands could only exceptionally be established

[1] Fiedler, *Jahresber. d. Gesellsch. f. Natur- u. Heilkunde zu Dresden*, 1885 u. 1886.—Vierordt, *Die einfache chron. Exsudativperitonitis*: Tubingen, 1884.—Stiller, *Deutsches Arch.*, 1875, xvi., S. 412.

*intra vitam*, yet the diagnosis of tubercular peritonitis is—as we shall soon see—usually less difficult. Before going further into this, however, let me first say a few words as to the affections of the mesenteric glands referred to above.

The time is now long gone by when the degeneration of these glands figured so largely that nearly every atrophic condition in childhood was attributed to an enlargement of them, hindering or even quite obstructing the flow of the chyle. "Atrophia meseraica" ("glands in the belly," as the common people call it) turns out in most cases to be really tuberculosis, which is more or less general but concentrated mainly on the abdomen, and in which the mesenteric glands are only affected secondarily to the peritoneum or intestinal mucous membrane. These glands may indeed become hypertrophied—even in children who are otherwise healthy, when they suffer from often-recurring intestinal catarrh; and then, under unfavourable circumstances, they are just as likely to become caseous as are the bronchial glands from chronic bronchitis and whooping cough. Much oftener, however, the affection of the mesenteric glands arises from tuberculosis of the intestine or peritoneum, the infection being transmitted along the lymphatic vessels or lacteals. I have frequently seen a few lymphatic vessels, passing from tubercular portions of the bowel and traceable through the mesentery, which were distinctly studded by miliary tubercles. In most cases the enlargement and hardness of the mesenteric glands only attains a moderate degree, and cannot be made out by palpation of the abdomen. Indeed, pretty considerable enlargement (I have seen some as large as a plum) or even masses of glands can often not be felt through the abdominal wall because of the flatulent distension of the superjacent bowel and the consequent tense condition of the abdomen. For this reason, in one girl of 5 years who suffered from chronic tubercular peritonitis, I was never able to make out a tumour, although at the post-mortem we found a mass as large as a child's head, consisting of nothing but an agglomeration of tubercular mesenteric glands. But wherever the flatulent distension is absent or diminished temporarily we may often be able to feel enlarged glands, as movable rounded nodules of various sizes, through the relaxed abdominal walls; but we must always bear in mind the possibility of mistaking fæcal nodules for enlarged glands.

Tuberculosis of the peritoneum is the only form of abdominal tuberculosis which presents a characteristic clinical picture; and it only does so when we have not simply a mere miliary tuberculosis, but also a state of inflammation superadded—just as in the case of tubercle of the pia mater. In the great majority of cases the inflammation takes an insidious and chronic course; still we must always be prepared occasionally to meet with such a rapid onset that under certain circumstances—when there is little or no history with the case, and especially in a hospital—the symptoms may readily be mistaken for those of acute peritonitis. As a matter of fact we really have in these cases a condition of acute peritonitis which has been added to abdominal tuberculosis of some standing, just as basilar meningitis is added to tuberculosis of the brain and pia mater, or pericarditis and pleurisy to that of the pericardium and pleura. Cases of this kind, however, seem to be rare on the whole.

I myself only remember one boy of 5 years, who was admitted into the hospital on 10th January, 1879, without any history. He was tolerably well-nourished, and on examining the thorax we found nothing but weak breathing in the upper part of the lungs. The symptoms of inflammation in the abdomen—great tenderness, distension and tension, vomiting and fever—which were found on admission, were referred to acute peritonitis, the cause of which could not be discovered. The child became collapsed and died on 18th, and at the post-mortem we found the following conditions:—

The abdomen was much distended, and contained about 2½ pints of somewhat discoloured purulent fluid; both layers of the peritoneum covered with fibrino-purulent exudation, all the coils of the small intestine adherent to one another. Numerous miliary and submiliary tubercles in the serous coat, partly also in the deeper layers of the intestinal wall; the lower end of the ileum as if sown all over with them, so that in this situation the peritoneum seemed much thickened. In other situations the tubercles occurred in very thick clusters, with hæmorrhages in their neighbourhood. A few tubercles in the omentum. In the mucous membrane of the small intestine, many solitary tubercles the size of a millet-seed, and tubercular ulcers the size of a sixpence extending down to the muscular coat, which were more numerous further down. Near these also a few caseous follicular ulcers. Just before the cæcum the whole intestinal wall was changed into an ulcerated surface, on which caseous tubercles and recent grey nodules were visible together. The ileo-cæcal valve almost entirely destroyed by the

ulceration. The vermiform appendix dilated to about thrice its normal size, its lumen slightly contracted where it opened into the intestine; the mucous membrane of the dilated portion studded with deep tubercular ulcers. In the colon also very numerous ulcers as far as the sigmoid flexure. Fatty degeneration of the liver. Upper lobes of both lungs slate-grey, more or less destroyed by cicatricial contraction, with a few caseous nodules.

One is naturally inclined in this case to attribute the peritonitis to perforation of one of the numerous tubercular ulcers. Still no such perforation could be found anywhere. The case was really one of recent miliary tuberculosis of the peritoneum, following upon old tubercular intestinal phthisis and combined with acute inflammatory symptoms. The following case shows that under these circumstances the acute inflammatory symptoms may not appear prominently, and that the clinical picture may assume a typhoid character.

Johann S., 2¼ years old, admitted on 23rd January, 1878. Said to have been formerly healthy, and to have taken ill 3 weeks previously with loss of appetite, diarrhœa, great lassitude and fever. Steadily increasing flabbiness, pallor and wasting. Temp. 100·8° F. Lips and tongue dry, and covered with crusts; intense thirst; loose, clay-coloured, very offensive motions. Spleen could not be felt nor percussed on account of the extreme flatulent distension. Abdomen abnormally tense, nowhere specially painful, tympanitic. No free fluid to be made out in the abdomen. Catarrhal sounds in the lower lobe of the left lung; nothing else abnormal. During the next 6 days, continued fever (m. 100·8°, ev. 103·3° F.), pulse 120, always becoming smaller, increasing flatulent distension, but no increased tenderness of the abdomen. The child kept scratching its mouth and nose, causing erosions of these parts. Steady collapse; death on 29th.

*P.-M.*—In the distended abdomen about 3½ oz. of turbid brown fluid, with tough fibrinous flocculi. Intestinal coils much distended with gas and adherent to one another in many places by loose fibrinous adhesions. The serous coat reddened in these situations. Omentum much shrunken. Numerous miliary tubercles on the surface of the intestine and on the parietal peritoneum. The heart and lungs normal, with the exception of bronchial catarrh and a few patches of atelectasis. Bronchial and mesenteric glands likewise normal; typical fatty degeneration of the liver. Mucous membrane of the intestine unaltered.

The exclusive limitation of the tuberculosis to the peritoneum constituted the peculiar feature of this case. No other organs were affected, not even any of the bronchial and

mesenteric glands, and only the liver showed the fatty degeneration which is so common in tubercular patients. Further, in spite of the most careful examination we were not able to discover a caseous deposit anywhere from which the miliary tuberculosis of the peritoneum might have arisen. The comparatively rapid course of the disease (about 4 weeks)—the symptoms being far more suggestive of typhoid than of tubercular peritonitis—is of significance from a clinical point of view.[1] In particular I would draw your attention to the very slight amount of tenderness of the flatulent abdomen, which was remarkable considering the condition we found at the post-mortem.

Cases, however, such as that just mentioned are much rarer than those having a chronic course and presenting the classic symptoms of tubercular peritonitis. The most striking feature in this clinical picture is the very gradual increase in the size of the abdomen, which is at first regarded as due to simple flatulent distension, and is therefore not attended to, but in time arouses the parents' anxiety, and causes them to seek medical aid. Of the many children whom I have had under treatment for this disease the youngest was 2½ years old, while the majority were between 3 and 8 years. A single glance at the abdomen is sufficient to arouse the suspicion of any experienced practitioner. For after the distension has gone on steadily increasing for some months the abdomen presents a noticably elliptical form. The abdominal walls are extremely tense, even so much so that the skin becomes quite glossy, and the epigastric veins are dilated and shine through it like blue cords. In very extreme cases the umbilicus is flattened or even —as I have occasionally seen—bulged outwards like a bladder. The appetite suffers, the children become thin and flabby, and when the disease is far advanced the prominent globular belly and the wasted limbs always seem to me very characteristic. Many patients also complain, from the beginning, of colicky pains and tenderness of the abdomen; but I have far oftener found both spontaneous pain and that caused by pressure either entirely absent or else limited to certain regions of the abdomen. When the abdomen is extremely distended we often discover on percussion and palpation an accumulation of free fluid

[1] *Cf.* Demme, 20. *Jahresber. d. Jenner'schen Kinderspitals f.* 1882, S. 33.

—the note changing with every change of the patient's posture (just as in any form of ascites) and the feeling of fluctuation being obtainable. This, however, is by no means constant, as there is often only a little fluid in the pelvis; and then the abdominal enlargement is mainly due to the great distension of the intestine with gas. This flatulent distension may press up the diaphragm and make the percussion-note tympanitic, not only all over the abdomen, but also over the sides of the thorax as high up as the 5th rib. Occasionally some parts of the thorax give a dull, and others a tympanitic note, which is not affected by the position of the patient. This is to be explained by the fluid effusion having become encapsuled by peritonitic adhesions. We can also occasionally make out on palpation hard bands caused by thickened and adherent coils of intestine, and less commonly, larger masses, which may be mistaken for new-formations or enlargements of viscera, *e.g.* of the spleen; but we find post-mortem that they are caused by loculated peritoneal abscesses. I have myself seen two such cases.

The abdominal distension is so characteristic of this disease that I have even seen it last till death in some cases of chronic peritonitis which ended fatally from tubercular meningitis; although, as a rule, there is usually a marked falling in of the abdomen before death in that disease. Nevertheless there are exceptions to this also. I have myself several times observed unusual flatness, or even retraction, of the abdomen, with or without tenderness, during the whole course of the disease; and when this is the case we always find at the post-mortem complete absence of fluid effusion, emptiness and contraction of the whole intestine, and adhesion of the coils to one another and to the parietal peritoneum. In such cases, indeed, the peritonitis merely formed one link in the great chain of general tuberculosis; and the symptoms of the latter condition were so marked that no diagnosis of the peritoneal affection could be made during life on account of the absence of the characteristic abdominal enlargement. Nevertheless, even in the cases in which chronic tubercular peritonitis forms the most prominent lesion, there may be no enlargement. In a girl of 6, in whose case I suspected tuberculosis, and who, after long illness with remittent rises of temperature, died at last of diphtheria, I found chronic peritonitis with complete adhesion of the coils of

the intestine to one another and to the abdominal wall, with innumerable miliary tubercles in the peritoneum and newly-formed adhesions—although no trace of tubercle was found in any other part whatever, except in a few of the lumbar glands. The abdomen in this case had been unusually flat, hard, and insensitive during the whole course of the disease, so that the condition found after death occasioned great surprise. I repeat, however, that even when the peritoneal cavity has been completely obliterated by general adhesions, the abdomen may be considerably enlarged by gaseous distension of the adherent bowel.

Cases like the one just mentioned, in which the tuberculosis is exclusively limited to the peritoneum and the abdominal organs, affecting at most the bronchial glands also, are by no means rare.

Child of 2½ years. The intestinal folds adherent all over to one another and to the abdominal wall. Accumulation of a light chocolate-coloured fluid in the free spaces. Spleen and liver surrounded by a firm fibrinous coating, and closely adherent to the neighbouring parts (diaphragm, abdominal wall, &c.). Numerous miliary tubercles in the parietal peritoneum, in the serous covering of the abdominal viscera, and in the omentum. Both lungs, the bronchial glands, and the parenchyma of the liver and spleen, entirely free from tubercle.

Child of 5 years. No fluid in the abdominal cavity. All the intestinal coils adherent to one another and to the abdominal wall; likewise the large omentum, which was transformed into a hard mass ⅝ in. thick. Between all these parts there was a deposit of numerous friable caseous masses. A large number of miliary tubercles on the free surface of the peritoneum. Spleen small, very hard, enveloped in a tenacious coating and adherent in all directions; its tissue, like that of the liver and spleen, was free from tubercle. Bronchial glands caseous.

Otto T., 4¼ years, admitted on 6th May, 1879, with emaciation, globular distension of the abdomen, palpable enlargement of the liver below the costal margin (confirmed also under chloroform). Disease had lasted already for 6 months without fever and without any symptom of the other organs having become affected. Death on 19th, from collapse and œdema of the lungs. *P.-M.*—Intestinal coils adherent in many places to the walls and to one another. On separating the adhesions one noticed a large number of greyish-yellow miliary tubercles in the false membrane, which was dark-red and very vascular; others also visible in the omentum and on the lower surface of the diaphragm. Tubercular

ulcers at many points in the intestine, with grey nodules on the corresponding serous membrane. Fatty degeneration of the liver; spleen normal; lungs and pleura likewise free from tubercle. Œdema of the lungs. Caseous degeneration of the bronchial and mesenteric glands.

In all these cases, with the exception of the two in which the bronchial glands were caseous, you find that the thoracic organs were quite unaffected, and only the peritoneum, and perhaps the mucous membrane of the intestine and the mesenteric glands, tubercular. This peculiarity of abdominal tuberculosis, which has been pointed out by former writers,[1] also explains the fact that the patients suffering from it usually present no other symptom during the whole course of their illness (which may last 6—12 months), except those above mentioned: namely, an extremely distended and globular state of the abdomen, which is covered by large veins, and may or may not be painful, anorexia, increasing debility and emaciation, generally accompanied by irregular rises of temperature in the evening (up to 103° F.)—the morning temperature remaining normal or subnormal (98°—96·4° F.). Death occurs either from a chance complication or from utter exhaustion; and before it takes place œdema of the lower extremities and scrotum may set in, as a result of the increasing cardiac debility.

In many cases diarrhœa is also added to the symptoms just given, and either keeps returning at short intervals, or persists in spite of all remedies; it is due to tubercular ulcers of the intestine. The more extensive the tuberculosis is, the more complicated are the symptoms; and the physical examination of the lungs, the constant cough, and the remittent temperature along with the local signs of chronic peritonitis, give us the same clinical picture which I have already endeavoured to describe to you in speaking of pulmonary tuberculosis (vol. i., p. 434). To the enlargement of the inguinal glands, which has in a few cases appeared to me of diagnostic significance, I now no longer attach any importance, because this condition is so exceedingly common in children, and because I have found it absent, or at least very slightly marked, in some cases of tubercular peritonitis.

You will excuse me if I do not enter into the anatomical appearances, in consideration of having already given you an

[1] *Cf.* Seyffert, "Ueber die primäre Bauchfelltuberculose," *Diss.*: Halle, 1887.

account of the post-mortem conditions in certain cases (p. 105), which afford a sufficiently clear idea of it. I shall only further mention that I have hardly ever failed to find more or less advanced fatty degeneration of the liver. I have frequently also seen a moderate degree of cirrhosis of that organ, which is to be explained by the extension of the inflammatory process from the porta hepatis along the connective tissue sheaths of the portal vein, or else by the irritation due to the presence of miliary tubercles in the liver-substance. Parenchymatous nephritis has also been observed as a complication.

It sometimes happens that in the last stage of this disease external rupture takes place, owing to the bursting of an abscess which has formed in the abdominal wall. I have observed this occurrence in 5 cases, and the opening is always through the umbilicus, out of which pus, serum and yellow liquid fæces from the small intestine, are discharged in gushes; and, as already mentioned (p. 83), round-worms may come out along with the fæces. In one case which was examined after death a communication was found between the external abscess and a perforated coil of small intestine adherent to the umbilicus, along with all the appearances of advanced peritonitis. In two other cases the perforated coil of intestine was not adherent to the umbilicus, but opened into a large abscess, situated behind the umbilicus, circumscribed by adhesions and filled with fæculent pus, which had burst externally. In one child who had a large quantity of free fluid in the abdominal cavity, there suddenly occurred, a few days before death, a copious evacuation of purulent-looking matter, which was followed by a rapid diminution in the size and tenderness of the abdomen. At the post-mortem we found no fluid in the abdominal cavity; but in the posterior wall of the peritoneum (corresponding to the right iliac fossa), there was an ulcerated aperture, 3 lines in diameter, through which the probe passed into a sinuous canal leading towards the rectum. Although no direct communication with the latter could be distinctly made out, we cannot doubt that in this case the fluid had ruptured into the rectum. Further, in two other cases I observed this rupture into the intestinal canal; and in these its occurrence was announced by copious diarrhœa and an extremely rapid falling-in of the abdomen, which had previously been much swollen.

Abdominal tuberculosis, like every other form of that disease, may end in a fatal attack of meningitis. As an example, I may give the following, chosen from a number of cases.

A boy of 8, treated in hospital for pericarditis in 1878 (vol. i., p. 489), was re-admitted on 3rd October. On admission he presented an extreme degree of ascites, for which the abdomen had to be punctured in order to relieve the dyspnœa, and about 3¼ pints of a turbid, greenish, very albuminous fluid were removed. On examination we found a marked prominence of the liver below the costal margin, and its sharp edge could be distinctly felt. As nothing was found wrong with the heart or kidneys, and there was not the slightest œdema anywhere, I felt all the more justified in my suspicion that some disease of the liver was the cause of the ascites in this case. Urine always free from albumen, scanty (12¼—14 oz. in the day), with a copious uric-acid sediment. The enlargement of the abdomen soon began to return after the puncture, and by 13th October had reached its former degree. The cutaneous veins continued to enlarge, but there never seemed to be any pain either spontaneous or on pressure. By a second puncture, on 11th November, nearly 5½ pints of viscid albuminous fluid were removed, but re-accumulation speedily took place. The boy became steadily more wasted and anæmic, but was still able in spite of the enormous amount of ascites to walk about the room, had a good appetite, and was quite free from fever. Thus a few months passed. But towards the end of February, 1879, the umbilicus was protruded like a bladder, and from time to time clear serum began to trickle from it. This recurred frequently, and could be increased by pressure on the abdomen. At the end of March a pointed swelling appeared in the umbilical region and became red, so that we expected a rupture presently, which, however, did not take place. The trickling of serum from the umbilicus still continued, and the tension of the abdomen became markedly diminished. After 16th April the temperature rose (ev. as high as 103·1° F.) without evident cause. The lungs appeared normal on examination. The onset of cerebral symptoms (apathy, drowsiness, vomiting, headache) soon cleared matters up; and on 7th May, death occurred after repeated convulsions.

*P.-M.*—Both the parietal and visceral layers of the peritoneum, but especially the latter, were thickly covered over with greyish-white transparent nodules, which were almost all surrounded by an exfoliating area. On the surface of the liver, and also on the mesentery, these tubercles had run together into clusters the size of a bean, forming irregular projections. The omentum appeared as a broad swelling about 1¼ in. thick, partly lying on the transverse colon and partly adherent to a few coils of the small intestine, and it contained nodules of the size of a pea.

In the abdominal cavity there was about 3½ oz. of perfectly clear light yellow fluid. Spleen and kidneys normal. Liver very large (8 in. from side to side, 6 in. from before backwards, 2¾ in. from above downwards), extreme fatty-degeneration, with a few tubercles of the size of millet or hemp-seed. The whole of the left costal pleura thickly studded with tubercles, the pulmonary affected to a less extent. Both lungs hyperæmic, containing hæmorrhagic infarctions, but free from tubercles. Pericardial cavity entirely obliterated by adhesions, so that the heart was entirely surrounded by dense fibrous tissue. Muscular substance of the anterior surface of the right ventricle almost entirely converted into fibrous tissue; elsewhere it was normal. Severe tubercular meningitis on the base as well as the convexity of the brain. Brain œdematous, its ventricles widely dilated and filled with serum; choroid plexus tubercular.

Here, also, we have an extreme case of chronic tubercular peritonitis, in which there was no pain whatever, and where the presence of the disease was indicated merely by symptoms of ascites, increasing emaciation and cachexia. Of especial interest was the fact that the umbilicus, which had become steadily thinner owing to the enormous tension of the abdominal wall, permitted the escape of the serum accumulated in the abdominal cavity through fine fissures—an occurrence which I had never observed before.

We found from the puncture of the abdomen (which was performed twice without any bad effect) that, in ascites due to chronic tubercular peritonitis, one need not be afraid of operative treatment any more than of puncture in any other form of abdominal dropsy. We must first, however, convince ourselves by very careful percussion, that the fluid moves freely in the abdomen, lest we should puncture an adhesion, or even a coil of intestine with the trocar. In the case of this boy, I was especially struck by the advantage of using gentle percussion, which gave a dull note at the place I chose for my puncture, although any stronger stroke on the pleximeter gave an almost tympanitic sound there. I need scarcely add that in these cases puncture is merely to be used as a palliative to relieve the dyspnœa. Further, from laparotomy—which has recently been recommended and tried by surgeons in order to affect the evacuation of any abscess which may be present—I do not expect any great results; although, certainly, a few puzzling cases of recovery have been reported. Nor can we expect any better

results from medicinal treatment. Wet compresses persevered in during many weeks, salt-water baths, and painting of the abdomen with tincture of iodine or iodoform-collodion—have been no more successful than the internal use of codliver oil, iodide of iron and iodide of potash; the latter I gave in the last case for months continuously. Although a few cases in my private practice recovered under the use of this remedy, still I believe that these were cases of simple chronic peritonitis, and not of the tubercular form, as I have already pointed out (p. 98). At any rate, although such cases are rare, we learn from them that we must not content ourselves in cases of tubercular peritonitis with making a diagnosis and doing nothing more, but should always make an attempt at treatment. Further, painting with iodine must never be used over too large an area at once. I usually divide the abdominal wall into four quadrants by two lines intersecting at the umbilicus, and I have one of these painted daily with iodine or iodoform-collodion. This treatment may be persevered in for weeks without doing any harm; and I may mention especially that I have never in any any case found albuminuria—which, according to some French writers, is a frequent result of painting with tincture of iodine in children—although I have often examined for it.

I have already mentioned that chronic tubercular peritonitis is often accompanied by obstinate diarrhœa, which must be regarded as the result of tubercular ulceration of the intestine. These ulcers are either isolated, or else they lie in clusters, forming a zone around the mucous membrane, and we often meet with them also in children whose peritoneum is almost normal, and in whom the main lesion is pulmonary phthisis. We often see from the serous surface numerous desquamating patches surrounding the whole lumen of the intestine and thickly studded over with tubercles; in many of these there is great narrowing of the bowel, which corresponds to deep annular ulcers on the mucous surface. I have seen such annular ulcers even at the lower end of the rectum (as much as 2 in. from the anus). Where these occur, the lumen may be so much contracted that the bowel-scissors can only be introduced with difficulty. We also pretty often find adhesion of the intestinal coils to one another, ulcerated communications between them and perforations of a few of the ulcers with consequent acute peritonitis

or loculated peritoneal abscesses. Moreover, the whole series of symptoms, and the anatomical appearances, correspond so entirely with those in adults that I may confine myself here to mentioning a few details which I have found more particularly in children.

When tubercular ulcers of the intestine are few and isolated, diarrhœa is just as likely to be absent as in cases where there are a few catarrhal ulcers; and under these circumstanes a definite diagnosis is not possible. In a boy of 6 years, who was much wasted (post-mortem on 28th February, 1881), we found a large number of tubercular intestinal ulcers along with general tuberculosis. Perforation had taken place through one of these ulcers, setting up rapidly fatal purulent peritonitis, although during the 10 days that the child was in the ward no diarrhœa had been observed. In other cases the diarrhœa was only trifling, the chief symptoms being emaciation, pallor and increasing weakness. Since we now know that chronic diarrhœa without any distinct signs of tuberculosis in other organs but accompanied by increasing emaciation, exhaustion and remittent fever may also be caused by chronic intestinal catarrh with follicular ulceration (p. 47)—it follows that tubercular ulceration of the intestine can only be diagnosed with approximate certainty when more or less profuse diarrhœa is combined with definite signs of tuberculosis of other parts, whether of the abdominal or thoracic organs.[1] Tuberculosis of the intestine is only exceptionally combined with other—*e.g.*, dysenteric—ulceration.

Max H., 3 years old, admitted on April 1st, 1878. Had suffered for 3 months from constantly recurrent diarrhœa. Abdomen somewhat distended and tender on pressure, normal on percussion. Motions (3 or 4 daily) always very loose, yellowish-brown, containing mucus, and often preceded and accompanied by colic. Thoracic organs normal. No fever, but increasing emaciation and debility. On the 6th, œdema of the left leg, which disappeared after lasting 2 days; then on 16th, œdema of the face, which increased, and after a few days was accompanied by a return of œdema in the left leg. Diarrhœa, in spite of medicines given (calumba, cascarilla, &c.), persisted unchanged, sometimes accompanied by prolapsus ani. Increasing collapse. Death on 8th June.

*P.-M.*—A large number of small, caseous, very bronchitic deposits in both lungs. Caseous degeneration of the bronchial,

[1] Examination of the fæces for tubercle-bacilli must often give negative results.

tracheal, and mesenteric glands; fatty liver; thrush in the pharynx and œsophagus. In the situation of the second lowest Peyer's patch there was an irregular ulcer the size of a sixpence, in the margins of which a few enlarged follicles were visible, with caseous centres. On the corresponding serous membrane a few submiliary transparent grey nodules. Below the ileo-cæcal valve there was a very marked swelling of the follicles of the large intestine and of the whole intestinal wall. A little below the valve some ulcers were found, their number steadily increasing as one passed down, until at last they became confluent, and in the descending colon and rectum only a few patches of hyperæmia of the mucous membrane were left in the midst of them.

In this case the main disease was chronic dysentery occurring in a tubercular subject. It is note-worthy from a clinical point of view that the large amount of ulceration of the intestinal mucous membrane caused almost no fever at all, the temperature only on rare occasions rising a little above the normal; this is to be explained by that tendency of young children to collapse, which I have already referred to in several connections. In other cases, indeed, well-marked hectic fever occurred; and in such I have found the temperature for weeks 3—5½° lower in the morning than in the evening (*e.g.*, m. 97·7°—96·4°; ev. 103° F.). Of interest in this connection is the œdema of the left leg and of the face also, which occurred more than once in the case just given, and was not to be explained either by a kidney-affection or by mere debility of the heart-muscle. This local œdema could only have been caused by a thrombus in the course of the left femoral vein; which ceased to produce engorgement when a collateral circulation had become established, but in the further course of the disease again produced the same effect. Venous congestion resulting from simple cardiac debility would necessarily have occasioned œdema of both feet. Unfortunately, the femoral veins were not examined at the post-mortem. We know, however, that so-called marasmic thrombosis of these veins is by no means rare in phthisical adults and children, and that the process may—as in the following case—extend far up into the inferior vena cava; and when this takes place the prominence of the symptoms which it gives rise to obscures those of the original disease.

Emil M., 7 years old, admitted February 12th, 1878. Had been delicate ever since having scarlet fever in the preceding August

suffered almost continuously from diarrhœa, and had become much emaciated. Since the beginning of February, œdema of both legs, of the scrotum and penis; marked dilatation of all the subcutaneous abdominal veins. Abdomen normal. Urine scanty, no albumen. Râles in both lungs, those under the left scapula being sharp in character. Diarrhœa, 5 or 6 motions daily. Temp. ev. 102·2°; m. normal. The marasmus increased steadily; the œdema spread over the abdomen and loins; the veins—down even to the feet—continued to dilate, On 22nd, gangrene of scrotum and of dorsum of right foot. During the last few days the liver was felt prominently below the ribs. Death on 2nd March.

*P.-M.*—Complete thrombosis of the inferior vena cava, reaching to close below the entrance of the hepatic vein; it was continued downwards into the iliac and femoral veins, and into the cutaneous veins of both thighs and legs. Liver large and fatty. In the small intestine, tubercular ulcers the size of a shilling, few in number in the upper part, but in the ileum crowded together, and in some places coalescing so as to cover areas of the mucous membrane as broad as one's hand. Mesentery thickened and tubercular. Many similar ulcerations in the colon and rectum also. In the right kidney a caseous nodule the size of a hazel-nut and some miliary tubercles. A large cavity with caseous contents in the lower lobe of the left lung. Bronchial glands enlarged and caseous.

In the treatment of tubercular intestinal phthisis we have no remedies save those which I recommended to you for chronic catarrh and follicular ulceration of the intestine (p. 48), and you can hardly look for much success from their use.

## XVII. *Diseases of the Liver.*

In judging of enlargement of the liver it is important to remember that the lower margin of the organ reaches further down in children—especially during the first years of life—than it does in adults. If you do not take this fact into account you lay yourself open to errors in diagnosis with regard to the size of the organ. The cause of this lower level of the edge of the liver is explained in an interesting way by the recent researches of Sahli.[1] He shows that the mere condition of the liver itself in the child, especially its relatively large size, which is usually blamed for the fact of the organ reaching lower

[1] Sahli, *Die Topographische Percussion im Kindesalter*: Bern, 1892, S. 122.

down—does not really account for it; but that (as Henke has demonstrated) the lie of the ribs has much more to do with it. For as the ribs lie more horizontally at the sides in children than in adults, they allow a greater portion of the liver to remain uncovered, and its lower margin, therefore, under conditions otherwise identical, is situated further from the edge of the ribs. It consequently happens that even where the liver is not really much enlarged (as, for example, where there is a degree of fatty degeneration in cases of general tuberculosis), it may during life seem very considerably enlarged, and when the abdominal walls are thin its margin may even project so as to be visible. I have seen this condition in some cases of tubercular peritonitis where there was a considerable amount of ascites, occurring to such a degree that I was misled into assuming the presence of hypertrophic cirrhosis, although at the post-mortem there was no trace of it.

The liver is attacked by disease far seldomer in children than in adults. Interstitial inflammation ending in cirrhosis, which is so common among the latter, only occurs exceptionally in childhood[1]—perhaps because its commonest cause, the abuse of spirituous liquors, is hardly ever operative. Still a few cases have been published of hypertrophic or atrophic cirrhosis in children, apparently caused by the abuse of alcohol.[2] I have myself only once (post-mortem) met with a case of granular atrophied liver in a child, resembling the cirrhosis of adults. The patient was a boy of 5 years, slightly jaundiced, with an extreme degree of ascites, who had only been 9 days in the ward, and who was found after death to have had perihepatitis portalis fibrosa with partial atrophy of the liver. I have, however, often observed in children interstitial hepatitis with increase of size and a granular surface of the organ—the so-called hypertrophic cirrhosis. In these cases there was generally jaundice, palpable enlargement of the spleen and epistaxis, but only in rare cases much ascites.[3] Much more common are the cases in which clinical symptoms are either quite or nearly quite absent during

[1] Unterberger, *Jahrb. f. Kinderheilk.*, ix., 1876, S. 390.—Fox, *Ibid.*, xiii., 1879, S. 404.—Neureutter, *Oesterr. Jahrb. f. Pädiatr.*, viii., 1877, S. 14.—Birch-Hirschfeld, *Gerhardt's Handb. d. Kinderkrankh.*—Laure et Honorat (*Revue mens. Mars et Avril*, 1887).—Palmer Howard, *Arch. f. Kinderkrankh.*, ix., S. 380.

[2] Demme, 22. *Jahresber. d. Jenner'schen Kinderspitals*: Bern, 1885.

[3] Henoch, *Charité-Annalen*, 13. Jahrg., 1888.

life, and it is not till after death that we discover hypertrophy of the interstitial connective tissue along with fatty degeneration of the liver-cells.[1] In these cases we find either an increase of the connective-tissue, surrounding the acini in all directions in the form of whitish bands and perhaps already producing a granular condition of the surface and of the section, accompanied by a more or less green discolouration of the parenchyma; or, the affection may still be in a very early stage of its development, and only reveal itself, under the microscope, by the new-formation of an enormous number of young cells in the interstitial connective-tissue. The former occur especially as the result of infectious diseases (measles, scarlet fever), and may apparently be the cause of the jaundice which occasionally sets in during the course of these diseases.

Most frequently we see interstitial hepatitis in very young children, even during the first months of life, occurring as a manifestation of syphilis; and I have already referred to this in speaking of hereditary syphilis (vol. i., p. 106). In these cases also the liver was granular and almost always increased in size, but I do not deny that, if life is prolonged for a considerable time, the atrophic form may in the end develope from the hypertrophic. Besides syphilis, tuberculosis must also be regarded as a cause of interstitial hepatitis, either in chronic tubercular peritonitis from the inflammation spreading into the liver along the portal vein and the connective-tissue sheaths, or as a result of the irritation which a large number of miliary tubercles in the liver exert on the interstitial connective-tissue (p. 107). I have frequently met with cases of this tubercular form (which had been previously observed by Brieger[2] and others), but generally the hepatic symptoms were so slightly marked and so obscured by the chronic peritonitis that the change in the liver was not observed until at the post-mortem.[3]

In a few cases, however, the cause of the interstitial hepatitis remains unknown; and the opinion expressed by Bartélemy[4] —that these cases are due to syphilis tarda—I regard as quite unproved. Although I have not succeeded with an anti-syphilitic

[1] To this class belong most of the 15 cases observed by Neureutter, of which only 3 were diagnosed during life.

[2] Virchow's *Archiv*, Bd. 75, S. 92.

[3] Pitt, *Jahrb. f. Kinderheilk.*, xxvi. S. 402.

[4] *Arch. gener.*, Juin, 1884.

line of treatment, I have several times observed in children of 6—12 suffering from this condition, disappearance of the jaundice, and even diminution of the hepatic enlargement, under the steady use of Carlsbad salts. We may therefore, in children as well as in adults, have a form of chronic hepatitis which has nothing whatever to do with syphilis, and which—as long as there is no hypertrophy of interstitial connective-tissue—is capable of being successfully treated by alkaline mineral waters.

Abscesses[1] and malignant tumours of the liver are rare in children. I may add the following case to one of medullary sarcoma in the 8th month of life observed by West. The striking feature about it is the rapid increase of the hepatic enlargement.

> Child of 2½ years, of healthy family, brought to the polyclinic in the beginning of Feb. 1878. Had always been healthy till Christmas 1877, but since then his abdomen had increased in size without evident cause. On examination the liver was found much enlarged. During the next few weeks, rapid increase in size. On the surface of the left lobe, in the epigastrium, a flat, soft, almost fluctuating projection was distinctly felt, which, however, did not appear tender. Veins on the abdomen and on the lower part of the thorax distended. Increasing emaciation and weakness; slight jaundice. Death on 23rd March.
>
> *P.-M.*—Liver thrice the normal size, jaundiced, presenting on the surface, as well as in the interior, a very large number of soft, yellowish-white sarcomatous tumours, from the size of a hazel-nut to that of a walnut or larger, some of them projecting on the surface, especially a pretty large one situated in the left lobe. Gall-bladder distended like a cyst, and filled with turbid bloody fluid. The cystic duct compressed by one of the tumours. All the other abdominal organs normal, but jaundiced. We were not allowed to open the other cavities.[2]

One has often an opportunity of observing echinococcus-

[1] Abscesses in the liver caused by round-worms finding their way into the organ, have been observed in a very few cases in children. Scheuthauer, who published one such case (*Jahrb. f. Kinderheilk.*, xiii., S. 63) regarded them not as true collections of pus, but as areas destroyed by caseation. Some of them contained no worms but only their ova; and from this fact he concluded that the round-worms had found their way back again out of these deposits towards the ductus choledochus. Abscesses of the liver have also been observed after injuries and pylephlebitis (as the result of perityphlitis and extension along the inferior mesenteric vein), and owing to suppuration of the mesenteric glands after typhoid (Bernhard, *Jahrb. f. Kinderheilk.*, xxv., S. 303).

[2] *Cf.* Affleck, *Centralzeit. f. Kinderheilk.*, ii., S. 46.—Bohn, *Jahrb. f. Kinderheilk.*, xxiii., S. 143.

cysts in children, corresponding in every respect with those in adults. I can only recall one case which seems to me worth recording on account of the apparently good result of tapping.

> Boy of 11 years, admitted July 15, 1878; presented nothing abnormal but a prominence of the right hypochondrium. The liver was felt 2 finger-breadths below the margin of the ribs, and on it, between the umbilicus and the ensiform cartilage a tense elastic globular swelling, about the size of an apple, could be made out. On palpation and percussion of this, no thrill could be elicited. On the 19th we emptied the tense tumour with a fine trocar, obtaining about 2½ oz. of clear serous fluid, and at once applied pressure by means of a bandage. The fluid was free from albumen, and although it contained neither echinococcus hooklets nor succinic acid, it could manifestly only have come from a hydatid cyst, probably a sterile one. The further progress of the case was so satisfactory that the patient was discharged on the 27th (*i.e.* 9 days after the tapping). There was at that time no longer any trace of the elastic swelling to be found, and liver-margin also could scarcely be felt. Whether the cure was really permanent I cannot say; but judging from what one knows of similar cases it seems quite possible.[1]

Amyloid degeneration occurs in children far more commonly than the affections already named. The smooth enlargement of the organ, scarcely if at all tender to touch, occasionally reaches such a considerable degree that it occupies the whole upper part of the abdomen, extending downwards on the right side as far as the iliac spine. The diagnosis of this condition depends, apart from the large size of the liver, on the participation of the spleen and kidneys in the amyloid disease. This can be made out in the case of the former by palpation of the enlarged organ in the left hypochondrium, and in that of the latter by discovery of albuminuria; although, of course, we must not ignore the fact that in amyloid degeneration of the kidneys there may be no albumen in the urine (Litten). But even in the absence of these complications the conditions of dyscrasia accompanying the hepatic enlargement are sufficient to confirm the diagnosis. In children long-standing suppuration of bone is especially important in this connection. I have frequently observed in cases of disease of the spine or hip-joint, or other carious affections of the bones or joints, the liver and spleen become palpably enlarged, and albuminuria set in, which the post-mortem

[1] *Cf.* Edge, *Lancet*, ii., 18, 1881.

examination showed to be the result of amyloid degeneration. On the other hand I cannot agree with those who ascribe a similar influence to rickets. In spite of the enormous number of rickety children treated in our hospital and polyclinic every year, I cannot recall a single uncomplicated case of rickets in which the diagnosis of amyloid disease could be established either clinically or post-mortem. When this disease did occur there were always other important morbid conditions present also, undermining the strength—such as caries or tuberculosis. Syphilis, however, may give rise to amyloid processes in children, not indeed the congenital form in its early stages—the influence of which in exciting interstitial inflammation or gummatous hepatitis we have already (vol. i., p. 106) discussed—but syphilis of long-standing, whether hereditary or acquired.

Agnes Z., 11 years old, admitted on January 12th, 1875. Said to have suffered formerly from "glands." Her mother, at the time of her confinement, had had an "eruption" on her body, but it had disappeared without any special treatment. A year ago, swelling of the nose and offensive discharge from it (ozæna). Several fragments of bone removed; falling in of the bridge of the nose. For three months, pains in the left upper arm and in both tibiæ, marked wasting and general backwardness of physical development, with great mental precocity. The front of both tibiæ, especially at their upper part, covered with a diffuse hard deposit. Lower epiphysis of the left humerus much swollen, movement of arm difficult and painful, muscles less developed than in the right arm. Prominence of frontal bone at the glabella. Many of the teeth carious, the crowns of the incisors not markedly notched; the first left incisor considerably larger than the second. Lymphatic glands in the neck moderately enlarged; left tonsil fissured; uvula entirely gone. Liver-dulness began above at the lower margin of the fourth rib, and extended below the edge of the ribs for more than 1½ in. in the mammillary line, for 1½ in. in the parasternal line, and 1½ in. below the ensiform cartilage. The lower borders of liver and spleen were distinctly felt, the latter extended below the margin of the ribs, and its dulness reached to the eighth rib. Urine contained a moderate amount of albumen, but no casts were found.

Treatment:—Iodide of potash (grs. viii., thrice daily). After 152 grs. had been used, the pains in the bones quite ceased, the swellings on them were diminished and the arm could be more easily moved. The treatment was continued for 3 months, and when the child was re-admitted during the following year a course of inunction was used for 3 weeks without appreciable benefit. The nose liver, spleen, and kidneys remained in their former con-

dition, although the pains in the bones and the swellings on them had quite disappeared. Some months after the treatment, however, the pains recommenced; and we saw the girl (who was now 15 years of age) from time to time as she came to the out-patient department and asked for iodide of potash, this being the only remedy which relieved the nocturnal pains in the arm and tibiæ.

Bertha R., 12 years old, admitted December 2nd, 1875. Said to have suffered as a child from "glands" and suppurating "swellings" on the right knee and thigh, the scars of which were still visible. Had never had dysentery nor protracted diarrhœa. For several years the patient had been unable to retain her fæces owing to the very frequent onset of violent tenesmus and pain in the anus, followed immediately by a loose motion, sometimes containing blood. She was said to have been very subject to tonsillitis, and during the last 14 days she had again been suffering from difficulty in swallowing and severe pain in the throat, especially on the left side. The girl was very pale and delicate-looking, and she had a marked opacity of the left cornea, a thickened nose with coryza, a firmly adherent greyish-yellow fur on the dorsum of the tongue and also here and there on the mucous membrane of the cheeks, on both tonsils and on the uvula, which was much fissured. Just outside the anus there was a hæmorrhoid of the size of a bean. There was some bronchial catarrh, but otherwise the lungs were normal. Liver-dulness commenced at the lower border of the fourth rib and reached down to the level of the umbilicus, where the lower edge could be distinctly felt. Surface of the liver very hard and smooth, the enlargement causing visible distension of the abdomen. The spleen could not be felt, and was not found to be enlarged on percussion. Urine bright yellow, clear, albuminous, but without tube-casts, although these were looked for repeatedly later on without success. The motions varied much in character as the case proceeded; although sometimes they were normally formed, they were often passed with tenesmus (and so suddenly that the patient had not time to reach the chamber-vessel) in the shape of a small quantity of clay-coloured pulpy mass, streaked with blood. There was frequently also quite ineffectual tenesmus with sharp pains in the anus. When a local examination (with the finger and speculum) was made on the 8th, we found the mucous membrane of the rectum uneven and rough, much swollen, and red. Above the internal sphincter there was an annular stricture which was more easily felt than seen. This series of symptoms, to which were added nocturnal pains of the limbs and slight enlargement of many of the inguinal and cervical glands, favoured a diagnosis of syphilis; and when treatment was commenced with iodide of potash, and painting of the affected parts of the mouth and throat with a 1 per cent. solution of nitrate of silver, it had a very favourable effect on the latter affection in the course of a week, as well as on the

coryza and pains in the limbs. On the other hand, the rectal trouble and the alternation of normal and abnormal motions persisted, and the patient was often kept awake all night by the recurrent tenesmus. After the 8th, a solution of alum (2½ per cent.) was daily injected into the rectum and the forcible straining thus set up caused, on the 11th, a prolapse of about ⅝ in. in length. The prolapsed portion of bowel appeared extremely fissured, scarred, and cicatrised, deprived of its natural mucous membrane. As the condition remained pretty much the same, on the 20th a course of inunction was ordered (mercurial ointment; at first, 10 grs., later, 20 grs., daily). But after more than an ounce of the ointment had been used, everything remained as before, and on the 7th March the patient was transferred to the Eye Department on account of keratitis of the right eye. When she was re-admitted into my ward (24th May) the cachexia and emaciation had advanced considerably; the liver had increased still more in size, both upwards (dulness now began at the third rib) and downwards, so that we could feel its sharp border in the axillary line beneath the spine of the ilium, and in the parasternal line about 2 finger-breadths above Poupart's ligament, and in the middle line at the umbilicus. Pressure on the liver was rather painful. No change in other respects. The motions were loose and occurred 5 or 6 times daily, often accompanied by tenesmus and severe colic, and they contained traces of pus and blood. The urine was scanty and still contained a quantity of albumen. Occasionally almost pure blood was passed per anum. At the same time there was fever (100·7°—102·5° F. in the evening), intense thirst, loss of appetite and nausea. No lasting improvement was effected either by repeated injection of alum, or by the medicines which were ordered for the frequent attacks of diarrhœa (subnitrate of bismuth, tannin with opium, &c.); at most, there was a temporary lessening of the diarrhœa, and along with it always an improvement in the general condition and the state of the strength. In this way the disease continued for some months longer. In the middle of October the increasing loss of strength showed that the patient could not last much longer; and death took place on 16th November.

*P.-M.*—Extreme emaciation. Lungs normal. Heart small and flabby, its muscular substance pale, greyish-red. Pharynx normal, but on the upper part of the posterior wall of the larynx where the pharynx passes into the œsophagus, there was situated a pretty hard corded swelling the size of a hazel-nut, and moveable on the mucous membrane (gumma). Liver increased to thrice its usual size. General amyloid degeneration. Spleen comparatively small, presenting amyloid degeneration of the pulp, both on chemical and microscopic examination. The kidneys, which were pretty large, were similarly affected; as was also the mucous membrane of the stomach and intestine. At the junction of the ileum and jejunum there was an ulcer, the size of a sixpence,

with irregular infiltrated margins and a clean floor. No tubercle anywhere. The rest of the intestinal mucous membrane was much reddened and swollen. Peyer's patches prominent; just before the cæcum there was a smaller ulcer similar to that already mentioned. The mesentery and all the coils of the small intestine presented much fibrous thickening, the latter fixed in many places by very thin and long, tough pseudo-ligaments. Liver adherent to the diaphragm at many places. Below the splenic flexure of the colon the mucous membrane begins to be thickened irregularly and reddened. Below this again there were superficial ulcerations along with deeper ones, smaller than a threepenny bit, having a clean floor. These extended as far as the rectum, where only isolated patches of the mucous membrane remained intact.

Although the history was not ascertained in either of these cases, the gummatous tumours between the larynx and œsophagus were sufficient to prove that the whole complicated series of symptoms took their rise from syphilis. Liver, spleen, and kidneys showed evidence of amyloid degeneration. In the second case also the entire intestinal mucous membrane was similarly affected, and was covered with numerous ulcerations, so that, especially in the rectum, it was almost quite destroyed. Owing to fibrous contraction of the latter, the symptoms of intestinal phthisis became complicated with those of ulcerated stricture of the rectum. Unfortunately, both cases also illustrate the powerlessness of specific treatment at this stage of the disease. Iodide of potash and mercurial inunction only succeeded in removing, or relieving some of the symptoms; the amyloid process and the ulceration of the intestine remained unchanged —a fact which no experienced practitioner will regard as weakening the supposition of the case being syphilitic in origin. Seiler[1] was more fortunate in his treatment; but, at the same time, in both of his cases the pathological condition of the liver (whether amyloid or interstitial hepatitis) was doubtful.

Fatty degeneration is the commonest of all the diseases of the liver occurring in childhood; but although it is so frequently found post-mortem, it is not nearly so often possible to recognise it clinically. Thus we find it more or less well-marked in very many cases of serious infectious diseases, especially after diphtheria and scarlet fever, and likewise in children who are tubercular, phthisical, or exhausted by chronic

[1] Seiler, "Ascites im kindlichen Alter," *Berl. Klin. Wochenschr.*, 1881, No. 23.

diarrhœa. The liver is enlarged, greyish-brown, light- or greyish-yellow in colour, doughy, and pits on pressure; its cells are found on microscopic examination to be filled with a large number of oil-globules of different sizes. The organ is often, however, not much enlarged, although (for the reasons given on p. 113) it appears larger during life than at the post-mortem. Cases do occur, although they are much less common, in which there is considerable enlargement of the liver; and it then fills a more or less large part of the right hypochondrium and upper part of the abdomen.

Whether fatty degeneration of the liver may also occur in childhood from unsuitable feeding, as in adults, I shall not attempt to decide. At any rate, the conditions which produce it—excessive consumption of fat and alcoholic liquors, without sufficient exercise—very rarely come into operation in childhood; and I have notes of only one case which could be referred to this cause.

Richard M., 2½ years, admitted January 10th, 1875. Said to have had measles (?) long ago, and to have been suffering from diarrhœa for months. Whooping-cough also present on admission. Young as the child was, he had long been in the habit of drinking large quantities of Bavarian beer; and when in the ward was constantly asking for "beer-soup." On examination we found a fine desquamation of the epidermis on the trunk and slight œdema of the feet and eyelids, so that one could not but suspect the presence of nephritis following scarlet fever or measles. The urine, however, was perfectly normal, without any trace of albumen. Some fluid in the abdomen, liver enlarged and reaching down to the umbilicus, extending to the left about 3 in. beyond the linea alba. Spleen could not be made out. 4—6 very loose brown liquid motions daily; occasional vomiting; no fever. Lungs and heart normal. During the next few days evident collapse, disappearance of the œdema, dilatation of the subcutaneous abdominal veins, and visible projection of the edge of the liver in the umbilical region. Low temperature (96·8°—96·4° F.), wasting, great weakness of the pulse, apathy, drowsiness; purulent fragments on the conjunctiva and cornea. Death on 17th January.

*P.-M.*—Heart-muscle pale, greyish-red, fattily degenerated. Right auricle distended with fibrinous clots. Liver considerably enlarged, greyish-yellow throughout. Marks left by pressure with the finger take a long time to disappear. Under the microscope we found extensive fatty degeneration of the liver-cells. Mesenteric glands somewhat enlarged, pale. Mucous membrane of the

intestine very pale throughout, villi very distinct in oblique light (amyloid reaction not certain). Epithelial-cells in the cortical substance of the kidney very fatty.

The form of jaundice which is caused by catarrh of the duodenum and bile-ducts occurs in children almost as often as in adults, sometimes even in the form of an epidemic. Most of the patients are more than 3 years old; still, I have had some cases of jaundice affecting much younger children, *e.g.*, one of only 8 weeks and another of 5 months. The jaundice was in every case accompanied by loss of appetite, often with a clean tongue; during the first few days even nausea and vomiting; decolourised grey or clay-coloured offensive motions, which were sometimes very frequent and loose, but more generally scanty; bilious urine; languor, irritability and inclination to sleep. There was hardly ever any fever, or at most there might be a moderate degree at the commencement of the jaundice. The observation which I formerly published,[1] that in jaundice in children I had never met with the well-known slowing of the pulse-rate to 50 or less, has since then been confirmed. The pulse-rate always varies between 100 and 120; and I must therefore assume that the greater irritability of the child's nervous system, and especially the fright caused by the medical examination, is sufficient to compensate the retarding influence of the bile-acids on the heart's action. An observation of Traube's[2] is in favour of this: he observed in adults that the pulse when slow, either from jaundice or large doses of digitalis, at once increased considerably in rapidity if the patients sat up or otherwise moved themselves. When children with jaundice are very quiet it may, therefore, be possible to observe a retardation of the pulse; but I have never myself succeeded in doing so. It is rare to make out by palpation a considerable enlargement of the liver below the costal margin due to engorgement with bile. We are often able to ascertain its presence by percussion. All cases terminate favourably after lasting 8—14 days, and I have only notes of one, which was of especial interest owing to the frequent occurrence of extreme rises of temperature.

Gustav K., 8 years, admitted on December 13th, 1875, on account

[1] *Beitr. zur Kinderheilk., N. F.*, S. 342.

[2] Traube, *Die Symptome der Krankheiten des Respirations-und Circulations-Apparats:* Berlin, 1867, S. 29.

of necrosis of the ascending ramus of the right ischium, due to an injury, which was still keeping up a sinus in the perineum ⅛ in. long, leading up to the bone. On January 18th, 1876, the sinus was dilated by laminaria, the necrosed bone scraped and an antiseptic dressing applied. About 10 days later (on 29th), jaundice with high fever, 104·7° F.; pulse 122; no pyæmic rigor; general feeling of health. Liver somewhat projecting; temp. ev. 103·6° F. During the next few days the jaundice increased to a bronze-tinge; urine bilious, no albumen, no leucin and tyrosin; motions colourless, offensive. This condition remained almost unchanged till 21st March—*i.e.* fully 7 weeks—during which time the wounds, which continued to have a healthy appearance, gradually closed. Fever accompanied the jaundice on 29th January, but only lasted 2 days, and was succeeded by an afebrile period lasting from the 31st January to 2nd February. On this day the fever again set in (m. 101·1°, ev. 102·2° F.) and lasted till the 21st, at first with high evening temperature (*e.g.* 104·2 F. on 4th February), then gradually diminishing with the morning temperature almost normal (99·7°—100·8° F.), but still with considerable evening rises (101·3°—102·6° F.). On the evening of the 21st it rose to 105·8° F., then fell again, and on 21st March the fever had entirely disappeared, the jaundice and the enlargement of the liver passing off at the same time, and the urine and fæces resuming their normal character. The boy was discharged cured, with the sinus closed, on 18th June, after he had recovered from an attack of scarlet fever in the ward. The jaundice was treated by purgatives, hydrochloric acid, Wildungen and Vichy water. Quinine (7½ grs. at a time) had been given for the fever, without any result.

The suspicion that first presented itself, that the high fever and jaundice might be due to a pyæmic process arising from the bone disease, was invalidated by the complete absence of rigors, and especially by the further favourable progress of the case. The character of the fæces also was strongly in favour of the jaundice being hepatogenous and due to retention of bile, although its exact etiology was obscure. It might have been due to obstruction of the bile-ducts by concretions, which occasionally excite exacerbations of fever; but, apart from the great rarity of such a condition in childhood, the entire absence of pain was against this supposition. We were, therefore, reduced to assuming that the case was one of severe obstinate catarrh of the ducts extending far into their ramifications. The favourable termination under a lukewarm solution of soda after the case had lasted almost two months seems to support this diagnosis. Still the persistent fever, which sometimes reached from 104° F.

even to 105·8° F., is under the circumstances a noteworthy symptom.

The treatment of catarrhal jaundice which I have found most successful, is as follows :—in the first two or three days of the disease, purgatives (calomel 1—1½ grs. every 2 hours, mist. sennæ co., infusum rhei (Form. 39)), later on, hydrochloric acid (Form. 3), and in cases where there is diarrhœa this should be given from the beginning. Strictest rest and regulation of diet, even although there is no fever whatever; avoidance of all animal food, except beef-tea, with nothing but thickened soups, biscuits, rice thoroughly cooked, other farinaceous foods and preserves. The patient should also drink half-a-bottle of Wildungen water, in order to hasten the elimination of the bile-pigment excreted in the urinary canaliculi. In some very obstinate but afebrile cases where Carlsbad and Vichy water, as well as the other remedies just recommended, had been given without effect, I have seen surprisingly good results from copious injections (1½—3 pints) of cold water into the intestine by means of an irrigator.[1]

The fatal form of jaundice accompanied by cerebral symptoms which is caused by acute atrophy of the liver, is occasionally met with in childhood. I have myself met with 3 cases, of which only one, however, was examined post-mortem. But neither these cases nor those which have been published by others presented any features, either clinical or pathological, peculiar to childhood.

## XVIII. *Diseases of the Spleen.*

The commonest disease of the spleen in childhood is tuberculosis. It affects not only the serous covering and the pulp in the form of more or less numerous miliary and submiliary nodules, but it may also appear as pretty considerable greyish-yellow masses (larger than a pea) distinctly marked off from the dark red parenchyma. Since these, however, never produce any definite symptoms (judging from my own experience) we cannot diagnose them, but can only infer their presence from the fact of our finding other tubercular affections of other organs.

As a rule, the diseases of the spleen can be recognised with

[1] Krull, *Berl. klin. Wochenschr.*, 1877, S. 159.—Kraus, *Arch. f. Kinderheilk.*, viii., S. 1.—Löwenthal, *Berl. klin. Wochenschr.*, 886, 9.

certainty only when the organ forms a palpable tumour projecting more or less beneath the left costal margin. I say "palpable," for I do not trust absolutely to percussion alone, least of all in the case of children who struggle violently while they are being examined—in this way setting up muscular contractions which are apt to give rise to mistaken inferences. Consequently, I regard with distrust all reports of cases in which the position of the lower end of the spleen is given daily from the results of percussion alone. Just call to mind how great may be the effect exerted on the position of the organ by alterations of the level of the diaphragm or by flatulent distension of the intestine. "Palpable" enlargements of the spleen occur, as in adults, most frequently in certain infectious diseases —typhoid and relapsing fever—and after repeated attacks of intermittent fever; less commonly in acute miliary tuberculosis and cerebro-spinal meningitis.[1] On the other hand I have never been able to make out a palpable splenic tumour in scarlet fever, measles, erysipelas, or even (as others say they have done) in catarrhal sore-throat, unless it had been there before.

The principal chronic disease of the spleen causing a palpable enlargement of it is amyloid degeneration due to caries of bone or to syphilis. Yet cases do occur in which the organ is normal or even diminished in size. All that I have already said (p. 117) about amyloid disease of the liver applies to that of the spleen. As the enlargement of the spleen due to engorgement of the portal vein (*e.g.*, in cirrhosis of the liver) differs in no respect from the same disease in adults, I shall proceed at once to those forms of enlargement which are due to simple hypertrophy. These are by no means rare, especially during the first few years of life. You can usually recognise the presence of this disease by the peculiar yellowish-white tint of the patient's skin, especially on the face; it may best be compared to that of white wax. This characteristic complexion has frequently been the first thing to prompt me to examine the spleen, and I have hardly ever found that my suspicions were at fault. Only in three cases of splenic tumour have I found the complexion almost normal, and I was extremely surprised to discover that the organ was enlarged. In one child the colour of the skin resembled that

[1] See a case of this kind in vol. i., p. 370.

in Addison's disease. The spleen almost always projects below the costal margin as a hard smooth tumour; and not uncommonly it fills the left half of the abdominal cavity almost entirely, so that its sharp, notched anterior border reaches to the umbilicus or beyond it, and when the abdominal wall is flaccid it can be grasped by the hand or is even visible. Occasionally, the enlarged organ is movable to some extent, especially when its size is only moderate or it is in process of diminution. Tenderness on pressure is either quite absent or is very slight. We may find it difficult to recognise slight degrees of enlargement, owing to the abdominal muscles being very tense (especially if the child is crying). When such is the case we must wait for an interval of quiet, when the descent of the diaphragm during inspiration will make the spleen easily felt. All over the enlarged organ the percussion-note is dull; but there is generally no great change at its upper end. This is due to the great weight of the tumour, which draws it downwards and, by constant dragging on the splenic ligament, may occasion considerable displacement of the organ. For example, in a child of 1½ years whom I had under observation for more than a year I found the enlarged organ, which at first was felt in the left hypochondrium, finally lying in the left iliac fossa and pretty freely movable.

To these chief symptoms—the splenic tumour and the characteristic complexion—there are often (but not invariably) added œdema of the feet and eyelids and little blood-extravasations in the skin, generally visible in the form of a few petechiæ in various situations. Hæmorrhages from the mucous membranes[1], even fatal bleeding from small vaccination wounds, have been observed.[2] In some of my cases exhausting hæmorrhage occurred from the nose although there was no petechiæ on the skin. On examining the blood, I have only very rarely found well-marked leucocythæmia; as a rule I have not found the relative proportions of red and white blood-corpuscles varying much from the normal. In judging of this we must of course bear in mind the diminution in the number of coloured blood-corpuscles which takes place in extreme anæmia and the noticeable increase of white corpuscles which may occur

[1] Rilliet and Barthez, ii., 34.
[2] Pott, *Klin. Wochenschr.*, 1879, S. 655.

even in healthy children. It is exceedingly rare, as I have said, to find a marked increase (1 : 30 and in one case 1 : 12), that is to say a real leucocythæmia. I regard the name "pseudoleucæmia" as unsuitable, however, because I have scarcely ever been able to make out enlargement of the lymphatic glands in any of these cases.

The etiology remained obscure in almost all my cases. Only rarely could I discover that there had been a preceding attack of intermittent fever lasting for several weeks or months. In one case the mother stated that she had repeatedly suffered from intermittent fever during her pregnancy before the birth of the patient.[1] In some cases frequent dyspeptic disturbances had proceeded, but generally these also were absent, and the mothers were led to notice the disease merely by the growing pallor of the skin and the increased size of the abdomen, the child seeming in other respects perfectly healthy. Consequently, in most of these children the spleen is already pretty large by the time they are brought to the physician. The appetite and bowels are often quite unaffected and, generally, wasting and flabbiness do not appear for a considerable time. As to the relation often declared to exist between this affection and rickets, in the very great majority of the rickety children whom I have seen, I have been unable to make out any splenic enlargement—at least none that could be felt; and *that* alone seems to me of importance. In many of the cases, indeed, we could feel the spleen distinctly—at least during inspiration. No one who bears in mind how extremely common rickets is in Berlin, especially among the class who attend the hospital and polyclinic, will be surprised at the fact that a certain number of the cases of great splenic enlargement observed by me did, as a matter of fact, occur in rickety children. We may similarly, I think, explain its connection with syphilis (vol. i., p. 108). In one case, that of a child of 2½, the mother was suffering at the time from enlargement of the liver and spleen, but I was not able to make out any connection with malarial infection. It is remarkable that a later child of the same mother had a large spleen; this case recalls those published by Senator, Biermer and others,[2] of splenic leucocythæmia in twins, or at least members of the same family.

[1] Playfair has published a similar case (Schmidt's *Jahrb. f.* 1858, ii., S. 333)
[2] *Berl. klin. Wochenschr.*, 1882, S. 233.

While the cause is thus obscure, our prognosis must at least be uncertain; but it can only be absolutely bad in cases in which distinct leucocythæmia is discovered on examining the blood. We know from experience that most children who suffer from chronic splenic enlargement die, with progressing anæmia, wasting and, finally, dropsy of the cavities; that is, if death does not result earlier from some chance complication, *e.g.*, from broncho-pneumonia. In such cases we find at the post-mortem simple hypertrophy of the spleen, *i.e.*, nothing but a great increase of its cellular elements and also perhaps of its connective tissue. The organ is extremely tough and heavy (5 oz. and more), its capsule is occasionally thickened and adherent to the neighbouring parts, and on section it is flesh-red, brownish-grey or dark-red, with more or less distinctly marked Malpighian bodies. I have frequently found the spleen $4\frac{3}{4}$ in. long, $2\frac{1}{2}$—$2\frac{3}{4}$ in. broad and $1\frac{1}{4}$—$1\frac{1}{2}$ in. thick. In many cases we find numerous whitish streaks in its substance which are formed by accumulation of a large number of lymph-cells. A moderate degree of hypertrophy of the liver and lymphatic glands is found in some cases.

Still, some children have recovered completely from very great splenic enlargement, even after their case has been judged incurable. In cases of this disease we cannot expect a natural cure, and suitable treatment must be carried on with great perseverance for many months. I have met with several cases, but not one of them was combined with true leucocythæmia.

Marie E., $1\frac{3}{4}$ years old, brought to Romberg's polyclinic on January 14th, 1847, with extreme atrophy, waxy complexion, enormous splenic enlargement, and œdema of the face, hands, and feet. Treatment with iron along with saline and chalybeate baths. On 30th July—*i.e.*, 6 months after—some diminution in the size of the spleen was made out. On 2nd November, it was diminished by about a half. At the end of a year, on January 12th, 1848, the spleen only projected three finger-breadths beyond the costal margin. On 9th May it could no longer be felt; complete and permanent recovery.[1]

In this case, according to the statement of the mother (who, however, did not speak very positively), the child had had feverish attacks accompanied by sweating in the summer of 1846. In the following case any attacks of this kind were positively denied.

[1] Romberg u. Henoch, *Klinische Wahrnehmungen und Beobachtungen*, S. 160.

Adolf N., 1¼ years old, rickety, brought on May 8th, 1865. For the last 4 months, increasing enlargement of the spleen, which filled up the space between the costal margins, the spine of the ilium, and the umbilicus. No leucocythæmia; waxy complexion. Treatment with quinine and iron. Considerable diminution by 10th June, and complete disappearance of the tumour by the end of July; every appearance of perfect health.

George M., 1½ years, brought to the polyclinic on May 10th, 1878; wasted, waxy pale. Splenic enlargement as in the last case. Treatment with quinine and iron for five months. In November the spleen could only be felt like a small band under the ribs, and in the end of December this also had disappeared.

Similarly in a child of 10 months (brought in September, 1881) I found the very large spleen had diminished in size by about a half in the course of two months, the complexion had considerably improved, and all the functions were in perfect order. Again, in a child of 1 year (who came under treatment in October, 1881), with great enlargement of the spleen, this had diminished by January, 1882, to such an extent that the organ now projected only about 1½ finger-breadths beneath the ribs. We now for the first time discovered a second tumour lying near the umbilicus on the left side. It was freely movable, rounded, and not tender, and when the patient lay on the back, fell backwards and to the left side. It was separated from the splenic enlargement by a broad area, which was normal on percussion, and was very easily pushed far backwards and upwards. It was undoubtedly to be regarded as a floating kidney. Hitherto, this is the only case of floating kidney that we have met with in a child, and I shall not attempt to decide whether the splenic tumour had, by its mechanical dragging, brought about this displacement and mobility of the left kidney.

From these cases we may at any rate learn not to lose heart, and that it is well to go on perseveringly for many months and even years with a mixture of quinine and iron. Suitable nourishment, also, is absolutely necessary, by the breast in the case of infants, and in older children by good milk, beef-tea and wine; warm salt baths (1—4 lbs. of salt to each bath) are useful as auxiliary measures. But although this treatment was successful in the cases I have already given, there were others in which it was quite unsuccessful, or at most only improved the general health while leaving the condition of the spleen unaffected. Still, the other remedies which are recommended (bromide of potash, iodide of iron, and arsenic) also failed entirely in these cases. I can recommend the treatment with quinine and iron as

that which I have myself found to give good results in the greatest number of cases. I have no personal experience of the use of the induced current, which has been tried by Botkin and others in adults. At any rate, this treatment can do no harm; but on the other hand the use of injections of a 2 p. c. solution of carbolic acid and Fowler's solution (1 : 10 of water) into the substance of the organ which have been recommended by Mosler is extremely questionable, especially in very young children.

## XIX. *Abdominal Tumours.*

Apart from enlargement of the liver and spleen we may exceptionally, as seen in the case given on p. 96, have fibrous thickenings of the intestinal walls due to chronic peritonitis, giving rise to tumours in the abdomen. Oftener, although not at all frequently, we meet with tumours caused by new growths, especially by sarcomata which may originate in various parts of the abdominal cavity. The part from which they seldomest grow is the peritoneum; and consequently the following case is of especial interest.

Paul J., 11 years old, admitted on May 25th, 1880. In his second year had inflammation of the lungs and pleurisy; when 5 years old, measles and chicken-pox; health had been otherwise good, but the child had always been pale and thin. In September, 1879, "nervous gastric fever," lasting 6 weeks. Soon after, difficulty of breathing and palpitation, and in December vomiting and pain in the belly. In March, 1880, a visible tumour appeared in the right flank; in May the whole abdomen was distended. On admission, extreme emaciation, cachectic complexion, abdomen much swollen (diameter at the umbilicus 29½ in.) covered with dilated veins, tender on pressure. Fluctuation; several rounded tumours above the symphysis. Œdema of the scrotum and feet. Urine normal, as were also the thoracic organs. High level of the diaphragm; resp. 44. No fever. Purgatives and irrigations produced no change in the condition. On 28th, a slight discharge of blood from the anus. On 29th the abdomen was punctured, and nearly 9 oz. of turbid blood-stained fluid was evacuated; this fluid continued to trickle out, and by the following day about 17 oz. more had been discharged. On the 30th, continuous trickling of dark blood from the anus. On the 31st, collapse and death.

*P.-M.*—In the abdomen, about 12½ oz. of turbid whitish fluid, which in the pelvis was thick and milky, and under the microscope presented an innumerable number of partially fatty leuco-

cytes. The large omentum studded over with flat tumours of various sizes lying in clusters, so that it was almost entirely converted into a mass of tumours of a medullary consistence, and a milk-white colour. The coils of the small intestine felt hard, especially at their junction with the mesentery, and were diffusely thickened and studded with milky nodules, which in the case of one coil of about a foot long, formed a ring round the bowel about $1\frac{3}{4}$ in. broad, and about 1 in. thick. At this part of the bowel there was a place about the size of a half-crown, where a soft reddened mass of tumours projected through the mucous membrane. In this position also the mesentery was swollen to the size of a man's fist, by coalescence of a number of nodules. There were quite similar tumours on the smaller curvature of the stomach, at the juncture of the mesentery with the bowel, in the liver (especially in the porta hepatis), in the kidneys, on the serous membrane of the bladder and rectum, on the diaphragm, and in the anterior mediastinum. Nothing abnormal in the blood and bone-marrow.[1]

This case, as we found on microscopic examination, was one of multiple lymphosarcoma, the clinical symptoms of which were mainly connected with the peritoneum and bowel; in the latter, taking the form of hæmorrhages from places where the tumour had perforated, and in the former by the palpable tumour and the ascites. The ascitic fluid, which had a milky chylous appearance, due to the presence of innumerable lymph-cells, undoubtedly originated from the lympho-sarcomatous degeneration of the whole peritoneum. The absence of any glandular enlargement rendered the diagnosis more difficult, and the case was at first regarded as one of chronic peritonitis.

The sarcomatous growths more frequently originate in the connective-tissue and glands, which are situated in the pelvis or behind the peritoneum in front of the vertebral column. These may grow to an immense size, so as to be not a bit smaller than those found in adults.[2]

In a boy of 5, who, except for whooping-cough, had always been healthy, increase in the size of the abdomen and fretfulness formed the first striking symptoms; later on were added œdema of the face, lower limbs and genitals, pain in the abdomen, diarrhœa, and emaciation. In the hypogastric region we felt a hard irregular tumour, tender on pressure, which finally reached as far as the umbilicus, and its lateral processes extended into

[1] *Charité-Annalen*, viii., S. 557.
[2] *Beitr. zur Kinderheilk., N. F.*, S. 337.

both flanks. On October 29, 1862 (about 3 months after the enlargement had been first noticed), death occurred from exhaustion.

*P.-M.*—Out of the depths of the true pelvis, into which it was regularly wedged, there grew a hard greyish-white tumour, which was hyperæmic in places, and presented many lobes and fissures, which were slightly adherent to the right ilium, to the omentum, and to some of the intestinal coils. It had displaced the bowel and omentum upwards, and had filled up the whole abdominal cavity below the navel. No ascites, only a few tablespoonfuls of yellowish serum in the pelvic cavity. The epigastric glands, as well as those of the meso-colon and some of the mesentric glands, were similarly degenerated and partially softened in the centre. The upper end of the right kidney presented the same degeneration, and a few nodules, the size of a hazel-nut, were embedded in the cortex of the left. All the other organs normal. The tumour, the centre of which contained a cavity as large as a child's fist, filled with brown offensive matter, turned out to be a cystic medullary sarcoma (consisting only of small nucleated cells, and a few fibres of connective tissue), and seemed to have originated in the retro-peritoneal lymphatic glands.

The kidneys and the perirenal connective-tissue, however, are the most usual points of origin of the sarcomatous growths, which (especially during the first years of life) may give rise to enormous tumours in the abdomen. The form of tumour met with is generally that of medullary, cystic, and myxo-sarcoma; and along with these may be placed many of the cases which have been described as "carcinoma of the kidney." The striped muscular fibres which have been found in these sarcomata by Cohnheim[1] prove that some of them at least are of congenital origin; and their comparative frequency in very young children (under 2 years) agrees with this. As the new growth is almost always unilateral, and only affects both kidneys in extremely rare cases, the tumours may—according as they are situated on the right or left side—be mistaken for enlargement of the liver or spleen, especially if they have already arrived at a considerable size, and have come forward to the anterior abdominal wall (displacing the intestine to the other

[1] Eberth, *Virchow's Arch.*, Bd. 55, S. 518.—Cohnheim, *ibid.*, Bd. 65, S. 64.—Landsberger, *Klin. Wochenschr.*, 1877, S. 498.—Kocher u. Langhans, *Jahrb. f. Kinderheilk.*, xiii., 1879, S. 152.—Brosin, *Virchow's Arch.*, Bd. 96, Heft 3.—Neumann, "Ueber d. primäre Nierensarcom," *Deutsches Arch. f. klin. Med.*, 1882, Heft 3, u. 4.—Jacobi, *Compte rendu des travaux de la section de pédiatrie:* Copenhague, 1885.

on palpation; and within about 6 weeks it filled up the greater part of the abdominal cavity. This very fact aroused a suspicion that the swelling might possibly be due to an enormous collection of pus, but this was at once negatived by the result of the exploratory puncture. A fourth case (a girl of 8) was quite similar. In her, the tumour filled almost the whole abdominal cavity, and on post-mortem examination it appeared at first sight to have sprung from the right kidney, about ⅓ of which projected out of the sarcomatous mass. On closer investigation, however, we found that the kidney itself was merely compressed and partly atrophied, although closely surrounded by the tumour which had probably originated in the retro-peritoneal glands.[1]

[1] Arnstein, "Ueber ein Fall von primärem retroperitonealem Sarcom," *Diss.* Berlin, 1882.

## SECTION VII.

### DISEASES OF THE URINARY ORGANS.

### I.—*Nephritis.*

We often find at the post-mortem examinations of children that on section of the kidneys (which may be normal or only slightly enlarged), the cortical substance is more or less broader than usual, and of a greyish tinge. This condition, which is due to swelling and granular opacity of the epithelium of the cortex, and which may finally pass on to fatty degeneration, is the so-called "cloudy swelling." It is found especially in small atrophic children, and next to them, in those who have died of certain exhausting diseases accompanied by great loss of fluid, such as cholera, chronic intestinal catarrh, intestinal phthisis, dysentery, general tuberculosis, &c. What apparently occurs is an interference with the nutrition of the epithelial cells passing on to fatty degeneration of them, and this cannot be diagnosed clinically. It may also be caused by high temperature, as is proved by its frequently being found in children who had died of serious acute diseases, such as pneumonia, typhoid, scarlet and recurrent fever; and under these circumstances other cells also (*e.g.*, those of the liver, and the muscular fibres of the heart) are often similarly affected. But such minute tissue-changes, apart from a slight amount of albuminuria which occurs occasionally, are beyond the reach of diagnosis.

All the more important, therefore, is the diffuse nephritis which in children occurs for the most part acutely, and also, though far less commonly, in a chronic form—the so-called contracted kidney—the clinical and pathological features of which correspond entirely with those observed in adults. I shall therefore confine myself to a description of the acute form, which is often found as a sequela of infectious diseases, especially of scarlet fever.

The anatomical changes vary according to the stage of the disease. The kidneys are at first of normal size, they are hyperæmic and present red points on section, but they gradually become very large, almost cylindrical, dark-red, and of softer consistence. The capsule is readily stripped off, and on the surface we see arborescent injections and blood extravasations of various sizes. After the disease has lasted 4—6 weeks the surface becomes paler, and on section we find a marked contrast between the very broad, often somewhat bulging, yellowish-grey cortical layer, and the dark-red, hyperæmic medullary substance, in which only the very ends of the papillæ are pale. Less commonly, part of the cortical substance is still much congested, and there are hæmorrhages of various sizes which somewhat modify its appearance. In many cases the kidneys are enormously swollen, and they contain so many blood extravasations that they are quite soft and pulpy. At first, microscopic examination reveals nothing but cloudy swelling and slight fatty degeneration of the cortical epithelium, but patches of interstitial round-cell proliferation begin to appear round the vessels, and round the capsules of the glomeruli, and in the interior of the latter we find coagulated albumen and desquamated epithelium. According to the most recent researches, the characteristic feature of the scarlatinal form consists in the changes in the vascular loops, which are thickened with a colourless finely granular material until they become quite impermeable (glomerulo-nephritis). The glomeruli are completely empty of blood, and they project from the surface of the section as grey granules (see Friedländer[1]).

In most cases nephritis occurs as a sequela of scarlet fever, generally setting in about the 12th or 14th day—often not until the beginning of the 3rd week—after the appearance of the rash.[2] We do not know the reason why this sequela is so common. The idea, which so many maintain even yet, that it is owing to "a chill" or to "suppressed perspiration of the skin," I

[1] *Fortschritte der Med.*, i., 1883, S. 89.—Rosenstein, *Die Pathologie u. Therapie der Nierenkrankheiten*, 2 Aufl., 1886, S. 145.—Litten, *Charité-Annalen*, vii., 1882 S. 167.

[2] On careful examination we often find, even during the eruptive stage of scarlet fever, a little albumen and a few hyaline casts in the urine, or perhaps the latter only. But I shall return to this in considering scarlet fever. I have only in exceptional cases seen nephritis set in during the first week of the disease; Litten also (*loc. cit.*, p. 151) gives a few examples of this.

certainly cannot accept, for nearly all of my cases occurred in spite of the most careful nursing, and only in a few cases had the patients left their beds some days before. It is much more likely that the unknown *materies morbi* of scarlet fever exerts this specific irritation on the kidneys. But we are not justified in assuming off-hand that the said *materies morbi* is of "bacterial" nature.

The slightest form of the disease is manifested merely by a rapidly passing albuminuria. If the urine is examined daily about the time mentioned and during the whole of the 3rd week, even although there is no special symptom pointing to implication of the kidneys, we often discover unexpectedly a varying amount of albumen. This may pass off altogether during the afternoon of the same day, or else next morning; but it sometimes returns again for a time without in any way interfering with the general health. We may therefore question whether the condition is really one of nephritis (however slight) or merely one of albuminuria depending on some other influences which favour the transudation of blood-serum. At any rate the disease often enough developes from just such slight beginnings, and on the other hand, as we shall see presently, nephritis may be found post-mortem even in cases where there has been no albumen in the urine during life. I therefore advise you always to regard cases of rapidly passing albuminuria as serious, and to keep the children in bed, order a milk diet, and favour the secretion by giving diuretic mineral waters (Bilin or Wildungen water).

This is still more necessary when the albuminuria is not merely temporary but persists. It may last for many weeks without producing any other symptom except, perhaps, an increasing pallor of the complexion. During this time the secretion of urine is sometimes scanty and sometimes pretty free; it often contains a large quantity of urates, but almost always albumen, a few blood corpuscles and a very few hyaline casts, leucocytes and desquamated epithelial cells which are sometimes only found after careful and repeated examination. I have, for example, in one such case seen the albuminuria lasting from February 5th to March 10th, that is, more than a month, during which time the child seemed perfectly well with the exception of an attack of dyspeptic diarrhœa and there certainly was not a trace of œdema. Rest in bed for 4 weeks, acetate of potash,

warm baths, and finally iron, produced in this, as in many other similar cases, complete recovery. I have seen children who continued to feel perfectly well for 8—14 days, although the quantity of albumen was so great that almost half of the urine in the test-tube coagulated on boiling. Indeed, even when the urine was scanty and contained a large quantity of blood, I have known the appetite and temper to remain excellent for weeks. Hence we may lay it down as a rule for the practitioner—that in every case of scarlet fever the urine must be tested daily for albumen from the end of the second week.

Far oftener, however, we find certain symptoms which lead us to examine the urine. The children feel uneasy, become fretful and pale, lose their appetite and complain of headache. The urine becomes noticeably scanty and turbid, and often yields a yellowish-red deposit which dissolves on being boiled, thereby indicating that it is composed of urates. This condition of the urine frequently precedes the albuminuria by a few days. Occasionally there is complete anuria at the very beginning, lasting for 24 hours; or during that time only a few table-spoonfuls of turbid urine may be passed. Along with this deficient excretion of urine, or even before it appears, the parents' attention may be attracted by the occurrence of local œdema. Still this is by no means a constant feature; and you must always bear in mind that there may be no œdema whatever during the whole course of the disease. But in most cases œdema is observed sooner or later, not always at the very beginning, and its degree and extent may vary very much. In many patients the eyelids only, and perhaps also the dorsum of the feet and the ankles become slightly œdematous and the condition varies in severity from day to day. But in many cases other portions of the skin also become affected, especially the scrotum and the penis—which assumes a tortuous form; or the greater part of the skin is implicated by the general anasarca. When this is the case the eyelids are swollen, and can only be opened with difficulty, and the greatly enlarged thighs may become covered by an erythematous blush (intertrigo) at the place where they come in contact with one another and with the enormously distended scrotum. In children in this condition I have sometimes seen the epidermis so stretched as to give way in many places on the lower limbs, and out of the fissures thus

formed—especially on the flexor surfaces—serum oozed in drops until the whole epidermis became macerated and peeled off, leaving extensive excoriations. Under these circumstances, which are always to be regarded as very unfavourable, the skin (especially on the face) and the visible mucous membranes assume an anæmic waxy tint. One half of the face or body is often more swollen than the other, which is explained by the child's preferring to lie on that side. When the tension is great, the skin also becomes tender, and any pressure on it causes the patient to complain of pain.

Whether the œdema is extremely slight and limited or very extensive (or is even quite absent) has no effect on the condition of the urine, by which we judge of the state of the kidneys. The quantity of this is almost always scanty, there being often only about 3½ oz., or just a few tablespoonfuls in the 24 hours; but on other days more is passed, though it very rarely reaches a normal amount. The quantity varies from 2 oz., or even 1 oz., to 17½ oz. I have never observed pain on micturition, but I have often seen an unusually frequent desire to pass water, although only very small quantities came. The urine always has an acid reaction and its specific gravity varies between 1,006—1,024, the average being 1,010—1,015. It is generally turbid, reddish-yellow, resembling raw-meat juice; but the colour varies very often in the same case, being sometimes lighter and sometimes darker, changing often into a cherry-red or greyish-red, brown or blackish-brown, corresponding to the sediment deposited at the bottom of the urine-glass. The darker discolourations are due to the presence of a larger quantity of blood (nephritis hæmorrhagica). The microscope then shows a much larger number of red blood-corpuscles; but in the darkest blackish-brown urine these are quite bleached, like little pale rings. In addition the nephritic urine always contains a more or less considerable number of white blood-corpuscles (lymph-cells), desquamated renal epithelium and hyaline casts of various lengths studded over with white or red blood-corpuscles or epithelial cells. I need scarcely add that all these formed elements can only be seen distinctly in the sediment, and it is therefore a good plan to filter the urine thoroughly, and then to examine the residuum on the filter. We also very frequently find at the same time crystals of uric acid, and when the disease

has lasted some time, fatty epithelium, free fat globules and granular *débris*, which adhere to the casts or their fragments and indicate the presence of advancing degeneration of the renal epithelium. The amount of albumen also varies considerably, as already mentioned, just as does the colour and the amount of formed elements. On many days it is small in amount, on others there is enough to cause coagulation of almost the whole amount of urine in the test-tube on boiling. I have occasionally found the evening urine turbid, brownish-red, and containing a large quantity of albumen and blood, while that of the morning was light yellow and almost quite clear. In a girl of 9 years the urine was always free from albumen on the morning after a purgative had acted thoroughly, but again contained a distinct amount of albumen in the afternoon.

In a number of cases the whole illness consists almost exclusively of the symptoms already mentioned, *i.e.*, the œdema and the alteration of the urine, or even the latter alone. The general health is scarcely interfered with at all, and under proper nursing and treatment the symptoms gradually diminish and entirely disappear after lasting 2—3 weeks. At the same time we must always be prepared for relapses, in which the urine suddenly becomes bloody again or albuminous, and the œdema which had disappeared again sets in. These relapses generally last only a few days, and have no further evil results beyond lengthening the disease for a week or more and making the children more anæmic during their convalescence. Still, I advise you, however mild the course of the disease may be, to be on the look out, and in no case to give an absolutely favourable prognosis, because serious symptoms, especially uræmia (which we shall discuss presently), may arise quite unexpectedly and in the midst of apparently perfect health. I have also learned from experience that all cases of nephritis commencing with extensive and rapidly increasing anasarca are to be regarded with suspicion, especially if the secretion of urine is at the same time very scanty. Even in cases where only a few tablespoonfuls of urine are passed, or even where there is complete anuria, lasting for days, the general appearance of good health may deceive the inexperienced as to the seriousness of the condition. Many cases of this kind have been published, and I have myself seen several, among which the following seems specially noteworthy.

Carl T., 9 years old. Sudden anuria 2 weeks after the eruption of scarlet fever. No urine at all passed spontaneously, a few drops obtained with catheter, but only on one occasion as much as a dessert-spoonful, and this entirely coagulated on boiling. The anuria lasted 7 entire days without there being a trace of œdema, the pulse being 80—96. The tendency to sleep, which was noticed during the first few days, soon disappeared under the use of purgatives; but neither purgatives nor blood-letting, nor any other means used were able to restore the secretion of urine. The child seemed to feel almost perfectly well till, on the 7th day, uræmic convulsions set in, and death took place.

In these cases, however, we have not only to bear in mind the possibility of uræmia, but we must also be prepared in every case, however slight, for dropsy (which when it is present generally takes the form of anasarca) also developing in the cavities of the body. We then most frequently find ascites, with more or less enlargement of the abdomen and the characteristic signs on percussion; accumulations of serum in the pleural cavity or in the pericardium occur less commonly, and only in the last stage of cases which are ending fatally. When ascites alone is present, the general health, as I have often observed, continues pretty good; or, at most, there may be some difficulty in breathing owing to the thoracic space being diminished.

August R., 3½ years old (October, 1874). Œdema of the face and feet. Urine scanty, very turbid, albuminous, and somewhat hæmorrhagic. Moderate amount of ascites, and great flatulent distension with high level of the diaphragm; dyspnœa, resp. 60—70 in the minute. No fever; respiratory and circulatory organs entirely normal. Complete recovery after 3 weeks of treatment with purgatives and acetate of potash.

If hydrothorax is added to the ascites, the prognosis at once becomes much graver. When such is the case, steadily increasing dyspnœa sets in, sometimes assuming the form of asthmatic attacks, and compelling the child to sit up in bed or on a chair, leaning forward day and night. I have hardly ever seen œdema and hydrothorax occurring alone without ascites as in the following case, which is well fitted to encourage us in apparently desperate circumstances.

Marie Sch., 10 years old, was admitted into my ward in May, 1877, with scarlatinal nephritis. Urine very scanty, containing

very little albumen, sometimes none at all, and no blood. Great œdema of the face, feet, back and loins, but not a trace of ascites. The child was pale, but otherwise seemed pretty well. From the middle of the 2nd week there was rapid dyspnœic breathing (50—60 in the minute). On examination we found dulness and weak breathing of both sides behind, extending up to the angle of the scapula. In the course of 3 weeks this had extended as far as the middle of the scapula. At the same time several violent asthmatic attacks occurred in the course of the day, causing cyanotic discolouration of the face, coldness of the extremities and of the point of the nose, and lasting many hours. No fever. Complete recovery within 4 weeks under continuous treatment, first with purgatives, then with infusion of digitalis and acetate of potash, besides repeated application of dry-cupping and mustard plasters.

The most rapidly fatal complication is the sudden development of œdema of the lungs, less commonly that of the pharynx, ary-epiglottidean ligament and its neighbourhood (œdema glottidis). Orthopnœa and cyanosis are the characteristic symptoms accompanying this mode of termination, and along with them we have in the first case widely-spread crepitations, and in the second obstructive sounds accompanying inspiration and expiration. These may occur not only in cases where there is very extensive dropsy of the skin and cavities, but also in those in which there is no dropsy whatever, or only very slight œdema.[1]

Another of the commonest symptoms of scarlatinal nephritis is more or less frequent vomiting of food, tough mucus or watery fluid. This vomiting has not in my experience always the unfavourable "uræmic" significance which many ascribe to it; for it often appears, either at the very beginning or later on in the disease, without the case assuming an unfavourable character, and without there being any of the symptoms which cause "uræmic" vomiting to appear so formidable—especially headache and drowsiness. In most cases there is constipation, less commonly more or less profuse diarrhœa, and occasionally also some colic. Whether these loose motions

[1] According to Legendre (*Recherches cliniq.*, &c., p. 326), this œdema of the lungs is mainly situated in the interlobular and interalveolar connective tissue and by it the alveoli are compressed, and the portion of lung affected is consolidated. When the alveoli are blown up through the bronchi the serum trickles out at the root of the lung. When this condition is present we hear on auscultation not fine crepitations but harsh, almost bronchial breathing.

—which have sometimes an extremely fetid smell—form merely a chance complication, or are due to the excretion of urinary elements by the intestinal mucous membrane (Treitz), remains an open question. In view of this possibility I always refrain from stopping the diarrhœa quickly by astringents. In a boy of 8 years with ascites and slight pleural effusion, although there was no diarrhœa, there was almost constant tenesmus, which (after castor oil had been given without effect) was cured by small subcutaneous injections of morphia and doses of extract of opium ($\frac{1}{3}$ gr. thrice daily). The fact that diphtheritic inflammation of the intestinal mucous membrane is occasionally found as a result of nephritis remaining more or less latent during life (p. 52), warns us to give a cautious prognosis in these cases.

As to the condition of the temperature in scarlatinal nephritis, there is great variety of opinion among writers. It is a mistake to suppose that the disease always runs its course without fever when there is no complication. I certainly acknowledge that in a large number of cases, some of which are very serious, there may be no fever whatever, and the temperature may even remain somewhat subnormal (98·6°, 98·2° F.) whether the urine is hæmorrhagic or not. And, further, in some cases there may be more or less high temperature depending on other sequelæ of scarlet fever, which may be present at the same time, especially otitis, gangrenous pharyngitis, phlegmonous inflammation of the cervical connective tissue, or synovitis. I have, nevertheless, had some cases which prove that nephritis alone, without any complication, is sufficient to occasion a feverish condition of varying degree and duration. While there was occasionally only an initial rise of temperature to 100·4°—102·2° F., which after a few days permanently disappeared, in other cases I have seen an evening temperature of 101·3°—102·2° F., persisting for 2 or 3 weeks, during which time the morning temperature was almost normal. I have also seen quite unexpected ephemeral rises of temperature to 102·2° or even 104° F. and more, occurring in cases during the course of which there was (generally) no fever at all, and these were occasionally accompanied by vomiting and increase of the albumen or blood in the urine.

On the other hand, inflammatory complications due to

nephritis not uncommonly occur in various other organs, and these of themselves set up fever or increase it if already present. The complications, which may set in in any case—either in those commencing with rapidly increasing dropsy or in those which are comparatively slight—mostly affect the respiratory organs. Pneumonia, bronchitis, and pleurisy (even bilateral) occur in many of the cases, and often bring about a fatal termination, while slighter forms of bronchial catarrh very frequently accompany nephritis, and do not in any way exert an unfavourable influence on its course. In a boy of 4 years with extensive hepatisation of the lung, the urine which had hitherto been yellow and turbid became of a typical hæmorrhagic colour under the influence of the pulmonary condensation. In another child, who had synovitis followed by nephritis after scarlet fever, pneumonia of the right lower lobe developed, and was followed by purulent pleural effusion, filling the whole right half of the thorax, which was successfully treated by a radical operation after lasting more than 5 weeks. Pericarditis and endocarditis may also arise in the course of the kidney affection; the latter especially may be so latent that its existence would never be discovered without examining the heart (vol. i., p. 483).

I must here point out to you that during scarlatinal nephritis the pulse often becomes slower, of unusually high tension and even irregular, without our being able to find out any definite reason for it. In one girl of 12, the pulse fell to 48 in the minute, and also became very irregular, although nothing was wrong with the heart, and the general health was not affected. After some days the pulse rose to 60, soon after to 96, and became regular; at the end of a week the nephritis had entirely gone. I have frequently met with similar cases of retardation of the pulse to 64 and less, with or without irregularity, but only in one was it accompanied by abnormality of the heart-sounds.

Boy of 10 years, admitted into the hospital on November 18th, 1874, with scarlet fever. Nephritis 14 days afterwards. Pulse almost always between 120 and 124; but sank suddenly to 88, and became irregular, intermitting 10—15 times in the minute, and at the same time a loud systolic murmur was heard at the apex, which did not entirely obscure the first sound. By the following day the murmur had entirely disappeared, pulse again 96—100, perfectly regular. Complete recovery.

In this puzzling case there certainly cannot have been any valvular disease; nor could any anæmia or uræmia be found as a cause of the cardiac symptoms. I mention the latter especially, because in a few cases which presented more or less marked signs of uræmia, I have seen similar cardiac disturbance, sometimes even giving rise to an appearance of collapse; and yet on examination I was unable to discover any material abnormality of the heart. Thus, in a girl of 8 with hæmorrhagic nephritis, the pulse—which had hitherto been quite normal—became markedly slowed (72—68) and irregular, while headache, nausea, vomiting and drowsiness set in; and when these disappeared the pulse also returned to its normal condition. In another child extreme cardiac debility appeared after a uræmic attack which had lasted the whole night. The pulse was small, rapid and irregular, as was also the cardiac impulse; extremities cold, breathing rapid and shallow (60—70 in the minute), while on local examination we found nothing but reduplication of the first sound ("galloping" rhythm) which lasted a long time during convalescence. Quite similar symptoms with almost imperceptible pulse, cyanosis and extreme prostration were found in a girl of 7 years who on the previous day had suffered from a uræmic attack lasting several hours. Immediately after the attack I noticed the extreme smallness of the pulse, and I was strongly reminded of the state of collapse following diphtheria. Indistinct, hazy vision, vomiting, great pallor, sinking-in of the features, dyspnœa, slight cyanosis, small thready pulse (100—116) with a temperature between 99° and 96·4° F.—occurred in one boy and were successfully treated by repeated injections of camphor. This cardiac debility is especially to be dreaded in children with nephritis, on account of their tendency to serous effusion; for the latter may all the more readily lead to œdema of the lungs by occasioning engorgement in the pulmonary circulation—indeed it was of this that the child just mentioned died. In a boy of 9 years, there was persistent retardation (68—52) and irregularity of the pulse, along with repeated vomiting, for almost a fortnight before the uræmic convulsions set in; and, when they began, the pulse at once rose to 120 and more.

For many years, in a number of cases of scarlatinal nephritis from my wards examined post-mortem, the left ventricle was always found moderately hypertrophied and dilated. Since

then, C. Friedländer[1] has found that this pathological condition occurs in every case of scarlatinal nephritis. Similar observations have been published by Silbermann[2] and Riegel,[3] and we must therefore assume that hypertrophy or at least acute dilatation of the heart is just as likely to occur during acute nephritis as from the chronic form of the disease. This is probably due mainly to the heightened pressure in the aortic system which is caused by the obstruction of the glomerular loops; and the great diminution of fluid excretion is also to be taken into account. The greater the degree of the glomerular nephritis and the more scanty the urinary secretion, the more certainly may we expect the rapid development of eccentric hypertrophy, but when the disease is slight in degree it may be quite absent. In several of our cases, where we examined most carefully for it at the post-mortem, it was certainly absent. At any rate, we may gather from the large number of cases of scarlatinal nephritis which I have seen end favourably, and in which I have found the heart perfectly normal years afterwards, that gradual recovery from a slight degree of hypertrophy or dilatation is possible; and this is also the opinion of the writers above-mentioned. This acute enlargement of the heart can be discovered clinically only in very rare and extreme cases; and I must here specially warn you against overestimating the results of percussion, which are apt to be misleading. On the other hand, it has been demonstrated by Riegel that in acute nephritis there is generally a considerable slowing of the pulse from the very beginning, along with the increase of vascular tension; and, according to my observations, it may also be combined with irregularity of the pulse (p. 146). At the same time, an increase in the size of the heart is certainly not usual, and also, even when it does occur there may be no change in the pulse. In a boy of 8 years whose left ventricle was found, post-mortem, to be much hypertrophied and also fattily degenerated, I had observed during life neither slowing nor irregularity of the pulse. Moreover, neither the retardation nor the increased tension were invariably present in my cases; and, especially, in the case mentioned on p. 146, in which the symptoms connected

[1] *Arch. f. Physiol.*, 1881.—*Fortschritte d. Med.*, i., 1883, 3.
[2] *Jahrb. f. Kinderheilk.*, xvii., S. 178.
[3] *Berl. klin. Wochenschr.*, 1882, No. 23.—*Zeitschr. f. klin. Med.*, vii., H. 3.

with the pulse and heart lasted at most 24 hours, a merely mechanical explanation (*i.e.* by the blocking of the glomeruli) was hardly sufficient to account for them.[1]

The following cases show that the peritoneum may also become the seat of an inflammatory complication :—

In the first of these (boy of 9 years) there suddenly occurred during the nephritis high fever, dyspnœa, enlargement (with tension and great tenderness) of the abdomen, nausea, vomiting, and constipation. By local blood-letting, warm fomentations and mercurials we managed to remove the serious symptoms in the course of a few days.—In the second case (boy of 8 years) a joint-affection was observed during nephritis, followed by an extraordinary succession of inflammatory affections of various serous membranes. First, acute hydrocele, the scrotum being swollen to the size of a fist, transparent, very tense and tender. Next, after a violent uræmic attack, acute peritonitis followed in a few days by left pleurisy with considerable effusion. At the *P.-M.*, we found a considerable amount of pale yellow serous fluid in the abdominal cavity and the serous membrane of the small intestine was of a rosy-red colour, due to extremely fine injection. —The third case was that of a boy of 6 years, admitted on 24th May, 1876, with a severe attack of scarlet fever. On 13th June, during the nephritis which followed, the abdomen became painful and distended, and the temperature (which had been kept moderately high by otitis) rose suddenly to 105·6° F. A very few days afterwards there was collapse and coldness of the extremities (temp. 101·1°—99° F.). Pulse scarcely perceptible. In the abdomen a collection of fluid could be made out, and this steadily increased in quantity while the pain diminished. Death on 18th, in a state of collapse. *P.-M.*—Peritonitis purulenta universalis.—Boy of 6 years, November, 1883. Scarlatinal nephritis. Death from pneumonia. *P.-M.*—Pneumonia of right lung, empyema. Hypertrophy of the left side of the heart, in the abdomen a large quantity of serous fluid mixed with pus and flakes of fibrin.—Child of 1½ years. Nephritis, anasarca, ascites, fever, vomiting. Death in a state of collapse after 1½ days. *P.-M.*—Heart normal, a large quantity of milky purulent fluid in the abdomen, liver and spleen covered with a thick layer of pus. Intestines matted together in several places.[2]

Among the serious symptoms which one must always be prepared for in nephritis, those of uræmia are the most important. Although, as a rule, they are preceded by a marked diminution in the secretion of urine or even by complete

[1] *Cf.* Riegel, *Berl. klin. Wochenschr.*, 1882, No. 24.
[2] *Beitr. zur Kinderheilk., N. F.*, S. 353, u. 381.

anuria, still, I have seen cases in which the quantity of urine was not diminished to any great extent, or in which the secretion, after having been very much diminished had become re-established—and nevertheless uræmia set in.

The same thing may occur although there is no other sign of nephritis being present. In a child of 4 years, who took scarlet fever on December 28th, 1880, the urine although scanty contained no albumen when examined on January 9th, 1881. And yet on the morning of the 10th there suddenly occurred severe convulsions of the right side of the face and body, with coma; pulse hard, 144. The urine was drawn off with a catheter, and it now contained a large quantity of albumen. On 12th, frequent convulsions of left side of body. During the afternoon, return of consciousness and speech. Pulse 120, strong. Slight delirium and hallucinations. On following day diminution of the albumen, which disappeared by the 14th. Treatment with pilocarpine and baths of 99·5° F.

Also in a boy of 5 years, the uræmic symptoms set in suddenly 3 weeks after the eruption of scarlet fever, on the very day on which albumen was found in the urine for the first time. Complete recovery after 3 days; urine also free from albumen.

Epileptiform convulsions occasionally set in without any warning. In other cases they are preceded by vomiting, amblyopia, drowsiness, slowing and irregularity of the pulse. They succeed one another rapidly for hours, and in the intervals between them there is either complete coma or at least a condition of drowsiness. In one boy of 12 years the first convulsive attacks were followed by coma, which lasted without interruption for 9 days and was followed by a fresh series of convulsions. I have also occasionally met with violent excitement, screaming, and delirium (taking either a cheerful or a passionate form) during the interval. The temperature generally rises considerably during the attacks (even to 104° F. and over), and soon after the convulsions have ceased, it falls very much (sometimes as low as 97° F.), with coldness of extremities and extreme smallness and rapidity of the pulse. In these cases, death may ensue very rapidly. A boy of 5 years who became pulseless soon after the first convulsive attack (which only lasted 3 minutes) died in the second, which occurred soon afterwards. The convulsions vary in severity and extent; they are sometimes limited to single groups of muscles or to one half of the body, and are not very severe; sometimes, however, they are general and very violent. They are accompanied, as a rule, by complete loss of

consciousness. Reflex immobility of the pupils is almost always present; I have also, during the attack, seen the pupils alternately expand and contract. Derangements of the sense-functions often remain after the attack has passed off, *e.g.*, deafness, but especially amblyopia and amaurosis. But these are usually not permanent, and disappear in a few hours or days. Much more rarely the amblyopia precedes the uræmic condition, as in one of the following cases.

Paul R., 9 years old, admitted on June 6th, 1878, with scarlatinal nephritis. Had an irregular and slow pulse several days (falling to 55), the temperature remaining normal. On the morning of the 18th, suddenly vomiting and epileptiform convulsions, which recurred 7 times within 5 hours. The first attack only affected the right half of the face and the right arm. Immediately after it there was complete amaurosis, which soon passed off, so that the patient recognised the people round him; but after the second the paroxysm again set in, now affecting the muscles of the whole body. After the third attack the patient was again able to perceive the sunlight, and vision had become quite normal by the afternoon. About 5 o'clock, in spite of the administration of chloroform, there were repeated epileptiform attacks and delirium; and death took place in a state of collapse. At the *P.-M.* we found œdema of the brain, parenchymatous nephritis, moderate hypertrophy, and dilatation of the heart (especially of the left ventricle) along with a large amount of dropsy of the connective tissue, and of all the cavities.

Conrad R., 8 years old, scarlatinal nephritis with much dropsy. During the night of December 13th, 1864, repeated spontaneous vomiting. At mid-day on 30th, great languor, headache and very indistinct vision, so that the patient could scarcely recognise the people round about him. The pulse 96—100, small. Violent convulsions and coma set in during local blood-letting. After 2 hours, consciousness returned under the use of cold compresses. On the following day the intellect was quite restored. Vision perfectly normal; pulse fuller, 68 and irregular. Later, death from pleurisy and pneumonia, without return of the uræmic symptoms.

Ernst K., 12 years old. Nephritis in the 3rd week after scarlet fever. In the beginning of the 4th (January 4, 1876), uræmia preceded by vomiting. Twelve epileptic attacks within 12 hours, sometimes confined to the face, sometimes unilateral, and sometimes general. Treatment with leeches, an ice-cap, and purgatives. On the 5th, the intellect quite unaffected, but almost complete amaurosis; could perceive light, but nothing else. On 6th, urine copious and no longer albuminous. Vision restored in the course of the day.

The cause of the interference with vision is as little known as that of the uræmia. Nevertheless it is still a matter of dispute whether the retention of the constituents of the urine, or œdema of the brain with anæmia of that organ due to the increased pressure exerted by the left ventricle, occasions the dangerous cerebral symptoms.[1] Although in the first of the above cases and in some others œdema of the brain was found after death, I do not attach any particular significance to this fact; for the same appearance has occasionally been found in cases where there had been no uræmic symptoms during life. On this account also the view of von Gräfe (that amaurosis is caused by a more or less transient œdema of certain parts of the brain) requires further confirmation. The reaction of the pupils to light was normal in the last of my cases; but whether this was also the case in the other two (fatal) I cannot say. These cases were not examined ophthalmoscopically.[2] Other derangements of the nervous system—aphasia and hemiplegia—are occasionally left after recovery from uræmia. In one boy of 3 years, who had had scarlatinal nephritis with uræmic attacks in February, 1881, complete paralysis of the left half of the body and of the left facial nerve appeared after convulsions which lasted for 24 hours, and had not quite disappeared by the end of April. I have often met with similar cases, but I never had any opportunity of making a post-mortem. Occasionally the remains of hæmorrhagic patches have been found in the brain, but in other cases nothing at all abnormal was discovered.[3] Ataxia, epileptiform attacks and weakness of intellect have also sometimes been found to occur as consequences;[4] still we have as yet no knowledge of the pathological conditions which give rise to these symptoms. In two cases there was even actual mental derangement.

In a boy of 6 years with scarlatinal nephritis there occurred uræmic convulsions lasting several hours, immediately succeeded by a condition of mental confusion and excitement—there was

[1] *Cf.* Rosenstein, *Die Pathol. u. Ther. der. Nierenkrankh.*, 3 Aufl.: Berlin, 1886, S. 241.

[2] Œdema of the optic papilla is said to have been found in some cases; in others—*e.g.*, in one observed by Selberg and von Gräfe—nothing abnormal was found (Hirschu). Virchow, *Jahresber. f.* 1867, ii., S. 170.

[3] Jaekel, "Beitr. zum Symptomencomplex d. Urämie," *Diss.*: Berlin, 1884.

[4] Hajek, *Arch. f. Kinderheilk.*, 10 u. 11, H. 1880.

stupor, cheerful delirium, causeless laughter, staring look (especially towards the left) and slight twitching of the corners of the mouth. Complete recovery after the second attack.

A girl of 7 years had a first convulsive attack of uræmia lasting 3—4 hours, and immediately afterwards was found to be confused and delirious, had hallucination of sight and hearing, and was subject to rapid changes of mood; but her most usual condition was one of dulness and torpor, with a staring look. This mental condition also remained after recovery from nephritis, so that the child had to be transferred to the ward for Mental Diseases on account of the increasing restlessness and jactatation. Recovery without any special treatment. The psychosis lasted for about 6—7 weeks.

At any rate it is certain that the uræmia of acute nephritis may be recovered from even in spite of the occurrence of amaurosis, paralysis, or mental symptoms; and I may add (as the result of my own experience as well as that of others) that uræmic symptoms allow of a more favourable prognosis when they occur in scarlatinal nephritis than they do in other forms of the same disease. I have frequently observed that as soon as the uræmia is got rid of, the nephritis generally passes off more rapidly than it otherwise does. But as a fact this only occurs in a certain proportion of the cases, while others end fatally. Or the nephritis may persist after the uræmic symptoms have disappeared.

From the description which I have given, you might be apt to infer that nothing could be easier than the diagnosis of scarlatinal nephritis; and as a matter of fact the history of recent scarlet fever, and the chemical and microscopical characters of the urine put the nature of the case beyond a doubt, even if dropsy is entirely absent. But here, as elsewhere in medicine, the saying holds good—that there are exceptions to every rule. For there are undoubted cases in which in spite of repeated examination of the urine (at least by the methods ordinarily used in practice) neither albumen nor microscopic indications of nephritis could be found. The discovery of the latter may, however, be rendered extremely difficult by the large quantity of urates which remain on the filter; still I do not doubt that tube-casts as well as albumen may at times be entirely absent and may occasionally only be found for the first time during the last days of life, especially

difficulty by the catheter—now contained a large quantity of albumen and numerous casts covered with granules. Death on 27th, from collapse and œdema of the lungs, after consciousness had completely returned.

*P.-M.*—Typical nephritis, fatty liver, œdema of the lungs, and broncho-pneumonia.

Paul Sp., 4 years old, admitted March 8, 1876. According to the mother's account he had had scarlet fever 2 months previously, followed by nephritis with slight œdema of the eyelids and, later, purpura simplex. Complexion waxy pale, skin flabby, no œdema anywhere, mucous membrane very anæmic. A number of small purpuric spots on the skin of the thorax. Violent cardiac action, pulse 136. No abnormality of lungs or heart on physical examination. A certain amount of diarrhœa; the urine scanty, clear, straw-coloured and without a trace of albumen. During the 3 following days the temp. was high (varying between 102·6° and 104·4° F.), resp. 36—40, pulse 128—136. Slight delirium, occasional vomiting; a few fresh purpuric spots on the face. Urine turbid from large quantity of urates, without a trace of albumen. No tube-casts or other formed elements could be distinctly made out. Death on 11th.

*P.-M.*—Bilateral hæmorrhagic nephritis. Kidneys much enlarged, flabby and almost as soft as pulp. Surface thickly scattered over with dark punctiform and lenticular hæmorrhages. On section the cortical layer was thick, and its peripheral zone almost uniformly red, owing to the presence of numerous small hæmorrhages. The columns of Bertin presented the same character, while the cortical tissue lying between these two zones was yellowish-grey and transparent. The straight tubules were filled with uric acid. Liver fatty. Chronic intestinal catarrh.

In the latter case there was no albuminuria during life, while in the former it did not appear until the onset of the uræmic symptoms; for the urine had been examined for 3 days (and also, as I afterwards learned, before admission) without any albumen being found. And yet the microscopic examination during the last few days of life, and also at the post-mortem, proved that the nephritis must have existed for some considerable time. Also in a boy of 4½ years admitted in July, 1886, the urine remained free from albumen until the onset of the uræmic symptoms; then extreme albuminuria set in, and disappeared after lasting 19 days. These cases remain as yet inexplicable, and may well make us chary of assuming the possibility of scarlatinal dropsy without affection of the kidneys. Not only isolated cases but whole epidemics of this

form of the disease have been described.[1] Legendre has already given it as his opinion that in cases of this kind there has previously been albumen in the urine, but that it has disappeared by the time of examination. I have often myself met with cases of œdema and even of ascites after scarlet fever, in which no albumen was found in the urine although it was examined repeatedly (in one case twice daily for a whole week). On the one hand, however, the microscopic examination in these cases was not carried out with sufficient perseverance; and on the other hand, they nearly all recovered, while in the single fatal case which occurred a post-mortem was refused. Moreover, œdema occasionally occurs after severe scarlet fever of many weeks' duration, which has nothing whatever to do with the state of the kidneys, but is only to be regarded as a result of weakness and anæmia; and it soon disappears under a course of tonic treatment. Finally, in cases of violent scarlatinal inflammation of the skin, slight œdema of the face and feet may be found immediately after the redness passes off, and is the local result of the cutaneous inflammation. Here, however, such cases must be excluded, as also those of œdema of the face which are occasioned in the course of scarlet fever by phlegmonous inflammation in the neighbourhood of the lower jaw, or by severe rhinitis.

The duration of scarlatinal nephritis nearly always extends to 2—3 weeks even in the most favourable cases. A shorter course is rarely met with, although cases do occur—such as that given on p. 150—in which the whole process lasts only a few days, with uræmic symptoms, and ends favourably. Far oftener, however, the disease lasts for a number of weeks. Thus, for example, in a girl of 12, I found œdema of the face, with albumen and tube-casts in the urine, and this condition only began to diminish at the end of the 10th week, and did not entirely disappear till several weeks later. A girl of 8 years, also, who had had scarlet fever in January, 1875, still suffered from a varying degree of albuminuria in the end of May, and on 2nd June fragments of granular casts were still found in the urine. There is always a possibility of this condition passing

[1] Quincke (*Berl. klin. Wochenschr.*, 1882, No. 27) describes three cases of "simple" scarlatinal dropsy affecting three members of one family; but they are not conclusive, because in two of them the urine contained traces of albumen.

into chronic nephritis; but I have met with only a few cases of this kind.[1] In two girls, of 7 and 9 years, albumen was found in the urine (but no tube-casts) during 1 and 2 years respectively after the attack—not indeed daily, but from time to time—the general health meanwhile remaining unaffected. In a child of 8 years who was in my ward for a considerable time, there was a well-marked condition of chronic nephritis with much œdema and characteristic appearance of the urine; this was attributed to scarlatinal nephritis which had occurred a year before and had relapsed a few months afterwards. The fact of scarlatinal nephritis having once occurred seems, as a rule, to render the kidneys particularly vulnerable; for I have frequently observed fresh relapses (from chills?) years after its first occurrence. This was the case, *e.g.*, in a girl of 10 who had suffered from scarlatinal nephritis 6 years before, and whose urine had been perfectly normal for the last twelvemonth. She then caught a chill from sea-bathing, and this was followed by a relapse, which persisted for 5 years, although—apart from extreme pallor—the child seemed perfectly well during all that time. Similarly in a boy of 12 who had had scarlatinal nephritis and uræmia 5 years before, after which his urine was said to have been always normal (?), nephritis and uræmia suddenly re-appeared, and the child died.

There is a difference of opinion with regard to the treatment; one might almost say that every practitioner has devised a method of his own. And this state of things shows that Nature has had more to do with the recovery of these cases than has the art of Medicine. The chief questions are: whether it is in our power to limit the spread of the disease so that recovery may take place without the functions of the kidney being interfered with in such a way as to endanger life, and whether we possess remedies fit to cope successfully with certain serious complications and sequelæ.

We possess no direct means, it seems to me, of fulfilling the former indication. Perfect rest and proper diet are, however, indispensable.

Whenever you find albumen in the urine, even although its presence is merely transient, you must put the child to bed and

[1] *Cf.* also a case published by Leyden (of contracted kidney after scarlet fever in *Deutsche med. Wochenschr.*, 1887, No. 27.

order a strict diet consisting mainly of milk and milk-food.[1] Such a diet appears to me to be also urgently called for when the disease is more advanced. I sometimes allow beef-tea, but no meat of any kind. I have, however, occasionally known animal food to be expressly ordered with the view of "making-up as soon as possible for the loss of blood" which occurs in hæmorrhagic nephritis. You must bear in mind that you have to do, not with simple hæmorrhage from the kidneys, but with a state of inflammation and that this is favoured by an animal diet. Wine is to be forbidden as a general rule and is only to be allowed in moderate quantities in order to support the strength should collapse be imminent. When there is no diarrhœa I always commence the treatment with a purgative (Form. 7) and repeat this for 2 or 3 days running. But in cases where there is diarrhœa, I should advise you to adopt an expectant line treatment at first, as I have more than once known recovery to take place spontaneously in such cases, *e.g.*, in a boy of 2 years, who was treated only with bismuth and astringent enemata and in whom the nephritis and the diarrhœa passed off simultaneously after some weeks. After the purgative I order acetate of potash (Form. 41) which, in very delicate and anæmic children, may be given in decoction of cinchona (Form. 42). At the same time I order 3—4 wineglassfuls of Wildungen or Bilin water to be taken daily, in order to facilitate the elimination, from the renal canaliculi, of the tube-casts and other matters accumulated in them. I have never seen any bad effects on the kidneys result from these remedies, so long as the potash salt was not given in too large doses. The same applies to digitalis, which I have used either alone or combined with acetate of potash (Form. 22), both in febrile and in afebrile cases.

I have comparatively seldom needed to resort to the application of dry- or even wet-cups (6—10) to the renal region; only, in fact, in some cases where the urine was extremely scanty or there was positive anuria and the temperature was very high. For such cases blood-letting (about a teacupful) used to be recommended (Heim, Romberg) as the best "diuretic;" and I can yet remember a few cases during the time that I was house-

[1] I cannot agree with Jaccoud (*Gaz. des hôp.*, Mai. 7, 1885) in thinking that an exclusive milk-diet, during the whole course of the scarlet fever attack has also a prophylactic value and is able to prevent the onset of the nephritis.

physician in Romberg's wards, in which this method of treatment seemed to have been wonderfully successful—especially in some cases complicated with inflammation of other internal organs. Perhaps I should have saved the lives of many children had I not been so infected by the hæmatophobia of our time that I have entirely abandoned the practice of blood-letting for the last 20 years. Most of the severe cases are unsuited for general blood-letting, on account of the very anæmic condition of the patients; and I should therefore advise you, in cases of the sort just mentioned, to limit yourselves to dry-cupping, and only to apply wet-cupping in very strong children. Frankly, however, I must confess I have never seen any case in which this method of treatment yielded results the success of which was beyond a doubt.

Very many practitioners are fond of recommending that the child be placed in a warm bath of at least 95°—99·5° F., and afterwards be wrapped in blankets. I have also used this treatment very often, and I acknowledge that it has a distinctly good effect, provided that it really succeeds in producing copious perspiration—as it is intended to do. But when there is very marked œdema, the diaphoresis usually does not occur or is at least very unsatisfactory; and even in cases where there is very little dropsy or none at all, a whole series of such baths has often no effect. Indeed, in many cases of hæmorrhagic nephritis, I have observed an increase of blood in the urine after each bath, so that I was obliged to stop the treatment and to confine myself to ordering warm lime-blossom tea. The baths, then, I regard as merely an experiment which we ought not to shrink from undertaking even in complicated cases, but one the results of which must be carefully watched. According to my experience, the complication with pneumonia forms no contra-indication against the use of sweat-baths; for I have seen several cases of this kind in which their continued use resulted in recovery.[1] The use of the wet pack seems to me less to be recommended, and I have gradually given up using it. In regard to the subcutaneous injection of hydrochlorate of pilocarpin which has been much praised recently—especially by Demme—I cannot for my own part, commend it. In order to obtain a free perspiration we have sometimes to give as much as gr. $\frac{1}{3}$—$\frac{1}{2}$ and

[1] See my paper on "Nephritis" in the *Charité-Annalen*, xii., S. 651.

then we nearly always find (sometimes even with gr. ½ only) repeated vomiting, occasionally, too, signs of threatening collapse, even although a spoonful of strong wine had been given before. I have often been obliged to desist from this proceeding, because it seemed to threaten a dangerous depression of the heart's action. But in a few cases in which we were able to continue the injections for a week or longer without risk and in which they caused always copious perspiration but, generally, only slight salivation, I certainly observed a rapid diminution of the dropsy and an increase in the quantity of the urine, although the amount of albumen in it remained almost unaltered. I do not think, therefore, that I have ever succeeded in shortening the course of the disease as a whole by the use of pilocarpine. I have at most effected an increase of the urinary secretion and more rapid disappearance of the dropsy—with which, indeed, we may be satisfied, as long as we do not produce any of the collateral evil effects of the drug. As a rule I consider the sweat-baths altogether preferable to pilocarpine.

The medicines I have hitherto recommended must be used continuously, for 14 days at least, and I should not advise you to have recourse to astringents until after that time in cases where recovery is not progressing. I generally begin by giving tannic acid, and I only choose ergot in preference to it when there is a large amount of blood in the urine. But certainly neither of these remedies seems to me to diminish the elimination of water from the kidneys, but rather to favour it (Form. 44 and 45). If after 7—10 days they are still producing no effect, I have to resort to liquor ferri perchloridi (Form. 45). This is especially suitable in the hæmorrhagic form of the disease, and may also (like any other preparation of iron) be recommended for the anæmia which remains after recovery. But none of these remedies promise us certain—and, still less, rapid—success. Even though they are administered with the greatest perseverance it often happens that weeks and months pass before complete recovery takes place.

Inflammatory complications must be treated according to their nature. When uræmic symptoms set in, I determine, the treatment not according to the particular form of the symptoms but according to the general condition of the patient; and this seems all the more advisable as we possess no specific remedy

for this condition, the real nature of which is unknown. Wet-cupping to the neck, 5 or 6 leeches behind the ears or to the temples (after-bleeding to be prevented), an ice-bag to the head, along with a smart purgative of compound senna mixture and syrupus rhamni (Form. 7) and, when this has acted, enemata of equal parts of vinegar and water—I have often found to be extremely efficacious. I must remark, however, that this happened only in the case of very robust children with a hard tense pulse, in some of whom the face was dark-red and the conjunctiva injected. But I can assure you that in such cases I have obtained surprising results from this treatment. If you care to administer chloroform when the convulsions are protracted and severe, as in other epileptiform attacks (vol. i., p. 163), there is nothing to be said against your doing so. If, however, the uræmic symptoms are accompanied by indications of collapse and weakness of the heart—*i.e.*, with a small, very soft and rapid, or very irregular pulse, cold extremities, pallor, cyanosis and sinking-in of the features—then every form of antiphlogistic treatment must be given up and we must have recourse to stimulants instead, especially large quantities of wine, subcutaneous injections of camphor (Form. 14) and repeated warm baths (95°—99·5° F.) followed by wrapping in blankets. Since pilocarpine was first recommended for uræmia by Preetorius[1] (whose results, however, were not very encouraging), I have repeatedly used it. In 3 cases where pilocarpine was used (gr. $\frac{1}{13}$—$\frac{1}{7}$ injected 4 times daily) recovery took place with copious diaphoresis, which in one of the cases did not set in until more than a grain had been injected; but the majority died in spite of the treatment, and in these it almost always caused vomiting. I have therefore no reason to recommend this remedy, which even in cases of my first class must be regarded as somewhat dangerous on account of its tendency to produce collapse.

As I have already remarked, nephritis, at least in so far as it gives rise to symptoms, nearly always occurs as a sequela of scarlet fever. Although at the post-mortem examination of scarlet fever patients who have died during the first or second week of the disease with malignant symptoms, one generally finds some cloudy swelling of the cortex of the kidney or even a marked

[1] *Jahrb. f. Kinderheilk.*, xv., 1880, S. 375.—Demme, *Ibid.*, xvi., S. 369.

degree of nephritis—still, the symptoms of the kidney-affection are masked by the general ones of this terrible disease. We can only make the diagnosis in such cases by examining the urine. For example, in two children of 6 and 9 years respectively, I found—on the 4th day of an attack of scarlet fever complicated by "diphtheritic" pharyngitis and typhoid symptoms—the urine turbid, very scanty, extremely albuminous, and containing many lymph-corpuscles; and, after death, we discovered advanced nephritis. In a girl of 11 years great œdema and rapidly increasing ascites with very albuminous urine appeared on the 5th day of an attack of scarlet fever, along with broncho-pneumonia; and they caused death about the beginning of the second week. In severe cases there is occasionally anuria lasting 12—24 hours during the very first days, and ending in the discharge of a very small quantity of bloody urine. In these cases the condition really consists not merely of an exacerbation of the cloudy swelling which sets in after the high temperature (p. 138), but of a severe irritation of the kidney, exerted by the scarlatinal virus from the very beginning. As for the cloudy swelling, it usually passes off, and we have really no ground for fearing its passing into a condition of nephritis; for in several cases of scarlet fever in which the temperature was very high during the eruption and albuminuria had existed for several days, I have seen convalescence progress without any interruption whatever.

In very severe cases where there are symptoms of cardiac debility from the very beginning the albuminuria may also be referred to an engorged state of the renal veins.

Paul P., 7 years old, had an attack of scarlet fever on January 24, 1873. On the 26th, the pulse (140) was very small. On the following day, irregular and scarcely perceptible. Hands and feet cold, the scarlatinal eruption and the mucous membrane of the mouth cyanotic. Urine scanty, dark, and albuminous. By the 28th the pulse had been rendered more perceptible and more regular by the use of stimulants (wine, musk), and the rash had become of a brighter red colour. On the 29th the pulse was stronger, 120, the rash had regained its normal redness, the urine was copious and contained no albumen The albuminuria, therefore, was to be regarded, like the cyanotic colour of the rash, and of the mucous membrane of the mouth, as the result of the venous engorgement of the kidneys; for all these symptoms disappeared simultaneously whenever the normal conditions of the circulation were restored.

Acute nephritis in childhood is so extremely often a sequela of scarlet fever that you must always bear in mind its possible connection with that disease, even where the patient's friends declare that there has been no scarlet fever. Often enough, slight cases of scarlet fever where the eruption is not well-marked and lasts but a short time, are quite overlooked, and it is only afterwards when nephritis has developed that the parents remember (on being questioned by the physician) that two or three weeks previously the child had been feverish for some days, had complained of its throat, and perhaps had "red spots" or "some measles rash." In these cases the traces of desquamation, especially on the feet and hands, often afford a further proof of the scarlatinal nature of the attack.

Scarlet fever, however, is certainly not the only cause of nephritis in childhood. Next to it, the most prominent part is played by diphtheria; for nephritic symptoms often appear during the course of this disease, and, less commonly, during convalescence from it. But I shall defer the description of this form until I speak of diphtheria. A less common cause of nephritis is measles. Although cloudy swelling (parenchymatous nephritis) which occurs in all severe infectious diseases is also often found at the post-mortem of patients who have died of measles (Reimer found it 12 times out of 51 cases), still it is a very rare thing to find clinical manifestations of nephritis either accompanying or following an attack of measles.[1] I have only been able to verify its presence in 3 cases which had been under my observation from the time the measles began. I have seen others—to which, however, I could not attach much importance, because I had to trust to statements of the relatives, who very often confound measles with scarlet fever. But it is proved by Malmsten's[2] observations that nephritis—even a hæmorrhagic form of it—may set in during the first few days of measles.

That varicella like other infectious diseases may be followed by nephritis was unknown until I put my cases on record.[3] I have had 4 cases under treatment in which œdema with nephritic urine set in 8—14 days after the eruption of chicken-pox, which

[1] Kassowitz, *Oesterr. Jahrb. f. Pädiatr.*, 1874, i. 80.

[2] Cases like the one published by Loeb (*Arch. f. Kinderheilk.*, ix., S. 53) ought to make us cautious in diagnosing albuminuria. In that case of measles, there was not albumen but propeptone in the urine.

[3] Henoch, "Nephritis nach Varicellen," *Berl. klin. Wochenschr.*, 1884, No. 2.

in most of the cases was copious and accompanied by fever. In 3 of them recovery took place within a few weeks under diaphoretic treatment (sweat-baths) along with diuretics (Bilin water or acetate of potash). Only one of the cases proved fatal, and at the post-mortem we found, in addition to recent nephritis, slight fatty degeneration of the liver, œdema of the lungs, and a moderate amount of hypertrophy and dilatation of the left ventricle. Soon after the publication of these facts I received from Dr. Claussen of Itzehoe reports of 3 cases which he had observed of nephritis after varicella, and these resembled my own in every respect. Since that time the number of published cases has increased to such an extent that the existence of nephritis varicellosa has been completely established.[1]

I have only rarely known nephritis to occur as a sequela of intermittent fever. In a girl of 6 years, who had been cured of quotidian ague by quinine after 3 attacks, the urine was found a week later to be small in quantity, of a dark-brown colour, and to contain a large quantity of albumen, hyaline casts and blood-corpuscles; but it returned to its normal character in the course of 8 days under the continued use of quinine. Two other cases, one of which has been described by C. Küster,[2] had a quite similar course.

Girl of 4 years. Measles in the end of November, 1879, followed by otitis of both ears with perforation of the tympana. In the middle of December, quotidian intermittent fever with very high temperature (105·8° F.) which, although somewhat relieved by quinine (4½—6 grs. in the forenoon), persisted till the end of December. On 27th December the urine was scanty, of a reddish colour, with a tinge of olive-green, and contained albumen, blood-corpuscles and tube-casts. After a few days of apparent improvement there was a return of the severe feverish attacks from 30th December to January 1, 1880; and when these set in, the urine again contained much blood. On the latter day, I saw the child for the first time. The attacks of fever were quickly relieved by large doses of quinine. The urine at once became clearer, and on 3rd January it was already free from albumen and blood. Complete recovery.

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[1] Hoffmann, *Ibid.*, No. 38.—Rasch, *Jahrb. f. Kinderheilk.*, xxii., 248.—Semtschenko, *ibid.*, 259, (nephritis on the 3rd day of varicella).—Rachel, *Arch. of Pediatr.*, April, 1884.—Vichmann, *Nord. med. arkiv.*, xvi., No. 20.—Högyes, *Jahrb. f. Kinderheilk.*, xxiii., 337.—Newski, *Wratsch*, 1884, No 46.—Janssen., *Berl. klin. Wochenschr.*, 1887, No. 48.

[2] *Berl. klin. Wochenschr.*, 1880, No. 26.

In one case I found similar symptoms occurring as a sequela of infectious parotitis:—

> Clara S., 6 years old, healthy. Became affected with parotitis in the course of recovery from an attack of whooping-cough. A week after this had passed off, œdema of the face set in, with bloody albuminous urine. When I first examined the patient (April 20, 1869) this condition was still present. Slight evening rise of temperature, although the child seemed quite well. Urine abundant, greenish-brown, with a small quantity of somewhat bloody deposit, and containing a pretty large quantity of albumen, blood-corpuscles and epithelial cells. No tube-casts found. Complete recovery after treatment for 8 days with purgatives, milk-diet, and rest in bed.

In one child who was treated in my ward I found, in the course of whooping-cough, œdema of the face and feet, along with albuminuria, which soon disappeared; I have likewise observed nephritis (in a Russian boy of 10 years old) which had lasted for 2 years, and was said to have begun immediately after an attack of whooping-cough. These cases, then, seem to make it doubtful whether the albuminuria in the above-mentioned case (Clara S.) was really connected with the whooping-cough or with the parotitis. The very considerable venous engorgement which takes place in all parts, including the kidney, during the violent paroxysms of coughing may certainly be regarded as a factor predisposing to dilatation of the blood vessels and occurrence of exudation. But as the whooping-cough was in process of rapid recovery when the kidney symptoms set in, the connection of these symptoms with the parotitis appears to be almost beyond doubt.[1]

I have only once seen nephritis following erysipelas—in a girl of 9 years who had suffered from erysipelas a fortnight before. There was slight œdema of the feet, and the urine became dark-brown, scanty and very albuminous, and contained hyaline casts. There was no fever. Complete recovery took place within 13 days under treatment by purgatives, sweat-baths and Wildungen water.

A good many cases, however, do occur (and in the year 1885 there was a specially large number in my ward) in which the most careful examination fails to disclose any cause for the

[1] Hæmorrhagic nephritis after mumps has also been observed by Croner (*Deutsch. med. Wochenschr.*, 1884, No. 9).

nephritis.[1] The relatives are of course always ready to tell you that the child has had a chill; and it cannot be denied that this may sometimes be the cause.

I have known a girl of 9 years take ill after having fallen into the water when she was heated, and also a boy of 8 after having been thoroughly drenched by a shower of rain which had surprised him on his way to school. The latter sat at his lessons in his wet clothes during the whole forenoon, and 4 days afterwards, dropsy, fever, and very albuminous and bloody urine were observed. A child of 2 years got an obstinate attack of nephritis owing to the blankets having got thrown off the bed while he was sleeping in a cold room (October, 1881) so that he was found in the morning quite cold. In 4 cases the nephritis was superadded to an attack of diffuse bronchial catarrh occasioned by a chill, or to broncho-pneumonia; and in a girl of 9 years it occurred in the course of an attack of purpura rheumatica (assuming a hæmorrhagic character) and its occurrence seemed to be due to the child having been for a long time exposed to rain and cold. But only in very few cases can the occurrence of a chill be verified with certainty; it generally remains a matter of hypothesis, and when that is the case it is better not to pretend to explain the causation of the attack. In such cases we must bear in mind the possibility of "artificial" nephritis, which may arise as the result of various forms of treatment. It is well-known that the internal use of strong diuretics such as turpentine and cantharides may give rise to albuminuria and nephritis, and the same has been asserted regarding large doses of chlorate of potash. Much less attention, however, has been paid to the fact that the external use of these drugs may have an analogous action. In an epileptic girl of 10 years who for 4 weeks had had a blister dressed daily with cantharides ointment, I found albumen and hyaline casts in the urine, and these abnormal constituents disappeared a few days after the dressing was left off. You must, further, pay special attention to those cases in which balsamic remedies or tar are used in the form of inunctions for chronic skin diseases. The more carefully we examine the urine in these cases, the oftener will we be able to find albumen

[1] On the primary form of nephritis described by Letzerich (*Zeitschr. f. klin. Med.*, Bd. 13, S. 1) and Mircoli as occasioned by the invasion of certain bacteria, my own experience does not enable me to give an opinion.

and formed elements in it after a time, generally not until after some weeks or even later. In several cases of chronic eczema which were treated in the wards with inunctions of tar (picis liquidæ 1 : vaselini 10), we had occasion to observe this.[1] On the other hand, the painting with tincture of iodine which has been said by French physicians[2] to act very rapidly in children in producing the same result (even when only applied to very limited portions of the skin) was nearly always found to be harmless in this respect. Only in one case, in which pretty extensive raw areas of the skin had been painted 4 times with tincture of iodine, did there occur a severe attack of nephritis with œdema after about a fortnight. The urine contained a large quantity of albumen and many tube-casts and epithelial cells, and there were serious uræmic symptoms. I must, however, mention that this patient had previously been treated with tar-ointment. I have also frequently seen the onset of nephritic symptoms 8—14 days after the cure of scabies by inunction with Peruvian balsam.[3]

Emma H., 5 years old, sent to my wards on May 10, 1873, straight from the skin-department. Œdema of the eyelids; urine scanty, albuminous, and containing casts, epithelial cells, blood-corpuscles and many lymph-cells. The child seemed well, but the temperature was rather high (101·5° F.). Treatment with purgatives and tannin. By the 22nd, urine normal, œdema disappeared.

Adolf U., 3 years old, admitted May 27, 1873, with œdema of the face and feet, which had existed for about a fortnight, the child having been treated a week earlier for scabies with balsam of Peru. Eczema still visible on the feet. High temperature (ev. 101·1°—103·6° F.), restlessness, loss of appetite, slight catarrh. Urine clear, light-yellow, and containing a considerable quantity of albumen and epithelial cells; very few tube-casts. Treatment with acetate of potash, and afterwards with tannin. After 13th June, no fever, and the œdema had disappeared, although the urine showed from time to time a varying amount of albumen, but was also occasionally quite free from it for days. Complete recovery after the 20th.

Girl of 6 years, admitted April 24, 1883, a week after treatment for scabies with balsam of Peru. Œdema of the face and

[1] Jacubasch, *Charité-Annalen*, vi.

[2] Badin "De l'albuminurie consécutive aux applications de la teinture d'jode chez l'enfant, *Thèse:* Paris, 1876.

[3] Litten, *loc. cit.*, S. 139.

feet; urine scanty, somewhat tinged with blood, containing much albumen and many tube-casts, and lymph- and blood-corpuscles. No fever. Purgatives. Sweat-baths. Discharged cured on 1st May.

In a boy of 7 years admitted on March 18, 1885, who had been treated in the skin department with Peruvian balsam during 4 days for scabies—we found slight œdema of the feet, with a large quantity of albumen and formed elements in the urine. Recovery under the use of sweat-baths. Urine again completely normal by the middle of April.—In a boy of 2 years, inunction of Peruvian balsam on three occasions was sufficient to cause, within 12 days, fever (102·9° F.), œdema of the face and feet, and moderate albuminuria. Recovery after a few days under sweat-baths.

After the external use of carbolic acid also, in the form of dressings, compresses, and syringings, albuminuria and nephritis may set in as well as blackish discolouration of the urine. I have hitherto met with only one case of the kind, but it has been sufficient to make me cautious in using this remedy.

Agnes Sch., 6 years old, admitted June 14, 1879, with chronic eczema of the whole left forearm; otherwise healthy. Fomentations of carbolic acid lotion (1:20) applied to the arm, and continued without intermission for 4 days. After the 19th, only vaseline was applied, and, later, a plaster-of-Paris bandage to prevent scratching. On July 7, extremely scanty secretion of urine,—scarcely a test-tube full passed in 24 hours, containing much albumen, a few red blood-corpuscles and hyaline tube-casts —some of which were granular. On the 10th, œdema of the feet and abdominal wall, no fever, child seemed well. During the next few days, œdema of the face. Treatment with purgatives, then with tannin and wet compresses, which were invariably followed by profuse perspiration. After a fortnight (on the 28th) the urine, which was now abundant, became normal, but complete recovery did not take place till the beginning of August.[1]

I have already (vol. i., p. 18) drawn your attention to the fact that in new-born children some albumen may be found in the urine, at least temporarily; and we are not yet in a position to say whether in these cases the condition is to be referred to the irritation of uric-acid infarcts in the uriniferous tubules. Now although this extremely small amount of albumen generally disappears after the first 10 days of life, still cases do sometimes

[1] *Cf.* the experiments of Lassar (in Virchow's *Arch.*, Bd., 77, 1879) of which the case just given affords clinical confirmation.

occur (even in extremely young infants) in which nephritis developes with serious results without our being able to discover any cause for it. I am not now referring to the "cloudy swelling" of the renal epithelium which is very often found at the post-mortem examinations of atrophic children who are very young, and which is to be regarded as due to a disturbance of nutrition of the epithelial-cells, but to cases in which the disease can be recognised clinically, such as the following:—

> Catharina K., 5 weeks old, admitted on March 24, 1874, on account of intertrigo. Moderate atrophy. On the 25th marked œdema of the face and extremities (temp. 97·5° F.). Action of bowels normal. Secretion of urine extremely scanty; by means of the catheter, as well as by applying a clean sponge over the urethra, we could only obtain a few drops, which were turbid and could not be used for examination. On 27th dyspnœa, cyanosis, dulness at the base on both sides of the thorax. Death on the 29th. At the *P.-M.* we found diffuse nephritis and effusion of serum into the pleura, pericardium and peritoneum.

Also in a few cases of chronic nephritis in children in the first and second years of life that I have seen, the cause remained unknown. The connection with syphilis, which has occasionally been suggested, I could never establish; and in one suspicious case in which antisyphilitic remedies were used, the treatment was entirely unsuccessful. Nevertheless, in view of a case of this kind which was successfully treated by Bradley,[1] I would advise you always to bear in mind that syphilis may have something to do with the malady. The main symptom of chronic nephritis even at this very early age is always œdema. In one child of 7 months it was found only on the back of the left hand and on the left foot, while the right side was entirely unaffected. The chronic form of the disease generally presents no essential difference, either during the first years of life, or in older children, from the same affection in adults; and I feel all the more justified in passing it over as children are not nearly so often affected by it as adults are. I have not the slightest doubt that many of the cases of chronic nephritis which are reported as having occurred in children exhausted by tuberculosis, scrofula, malarial cachexia or syphilis have been wrongly diagnosed, and that the children were really suffering from amyloid degeneration of the kidneys, which we have already considered (p. 117). The

[1] Hirsch-Virchow, *Jahresber. f.* 1871, ii., S. 176.

diagnosis of this condition is easy if there is distinct enlargement of the liver or spleen and marked cachexia caused by syphilis, bone-suppuration, tuberculosis, &c., combined with œdema of various parts of the body and albuminuria. If this combination of symptoms does not exist—and especially if there is no albuminuria, as is sometimes the case—we can only suspect the presence of amyloid disease; we cannot make an actual diagnosis.[1]

I shall take this opportunity of adding a few words to what I have already said regarding the symptoms of dropsy, which may occur in children without the urine containing either albumen or the microscopic signs of nephritis. We have already seen in the description of œdema neonatorum (vol. i., p. 55) that there are a number of different causes which may produce œdema. The œdema which occurs in older children may also arise from various causes. I have especially often seen children in the two first years of life become affected by œdema of the backs of the hands and feet, of the legs, the cheeks, and the eyelids—sometimes to such an extent that the back of the hand felt like a tightly-stuffed cushion. I have already mentioned that such cases may possibly be due to nephritis or to amyloid degeneration of the kidneys. In my cases, however, the urine very seldom presented these characters; as a rule, it was quite free from albumen, although it was always scanty and often contained a large quantity of urates. I would, therefore, be inclined to disallow entirely the hypothesis that there was nephritis, were it not that our experience regarding the absence of albumen in this disease (p. 154) makes me hesitate to do so. As a matter of fact, in one such case where there was extensive œdema of the skin and also ascites, although the urine had never been albuminous, we found both kidneys very hard and their cortical substance indurated, owing to the presence of a large amount of connective tissue. To this case we may add two others of tubular nephritis, along with dropsy, observed by Dickinson[2] in very young children in which the urine had never contained any albumen. We must not, then, content ourselves with a mere naked-eye examination of the kidneys, but we must examine them microscopically. On this account I cannot regard as conclusive those cases in which the latter was omitted, even although

[1] On tumours of the kidney, *cf.* p. 133.

[2] Hirsch-Virchow, *Jahresber. f.* 1871, ii., S. 175.

the kidneys appeared perfectly normal to the naked eye. I am all the more inclined to emphasise this fact, because in one of these children the liver also appeared enlarged by a deposit of fat and increase of the interstitial connective tissue.

In addition to these forms of œdema, the exact nature of which cannot yet be definitely settled, others also not uncommonly occur in children as in adults, which are caused by exhausting diseases quite apart from any kidney-affection—especially by phthisis, chronic diarrhœa, and dysentery, or even by serious diseases of the blood, *e.g.*, leucæmia and pseudoleucæmia. In a large proportion of these cases the weakness of the heart and the engorgement in the venous system due to it, constitute the proximate cause of the œdema, which may be limited to certain parts and caused by "marasmic" thrombosis in the larger veins, *e.g.*, of one lower extremity. Further, the development of œdema must also be favoured by the congestion caused in the venous system of the body by the numerous patches of atelectasis of the lung which—especially in little children who are exhausted—are so apt to result from the diminished inspiratory power when bronchial catarrh is present. To the same category belongs the dropsy which accompanies disease of the heart in children as often as in adults.

That inflammatory diseases of the skin (especially erysipelas) may leave œdema behind them I have already mentioned (vol. i., p. 46); and I have sometimes observed the same thing after urticaria and erythema multiforme. In such cases the preceding skin-affection may have entirely escaped notice, and it is only the subsequent swelling of the eyelids or other parts that arouses the parents' anxiety. But sometimes no cause can be found to which we can refer the œdema, and we have to fall back on the usual ready-made explanation—a chill, which can hardly ever be verified.

George Sch., 9 years old, admitted October 8, 1878, with œdema of the face, scrotum, and prepuce, which was said to have existed for 24 hours. During this time, and also when admitted, the patient seemed perfectly well. Cause quite unknown. Urine normal in every respect. After the application of a warm cushion stuffed with herbs to the scrotum (which was very much distended) the œdema of it had become considerably lessened by the 10th. The child was kept constantly in bed, and the œdema of the face diminished rapidly without any medical

treatment. Discharged on 28th. The urine had been repeatedly examined, and was always found normal.

Child of 4 years, admitted November 15, 1881, with œdema of the face and of both legs. Perfectly healthy in other respects. Urine clear, abundant, free from albumen. No cause ascertained; no trace of desquamation anywhere. Sweat-baths. Discharged cured on the 27th.

Sometimes such attacks of œdema appear periodically, as was the case in a girl of 4 who suffered three or four times in the course of about 3 months from œdematous swellings of the feet as well as of the hands and face. Each attack lasted about a week, and was accompanied by general uneasiness, and, on one occasion, by vomiting; and yet nothing abnormal was discovered in the urine or in any of the organs on repeated examination. As the cause was quite obscure, and the child's pallor suggested anæmia, we gave iron and quinine, and under this treatment recovery rapidly took place. The quinine was given in view of the possibility (!) of a malarial element; but as to the presence of such an element I cannot give an opinion.

Among the local causes of œdema, compression of individual veins may also be mentioned, as in the following case:—

Child of 1½ years, examined *P.-M.* June 7th, 1873. During life there had been a great amount of œdema and pale swelling in front of and behind the right ear, so that it was made to stand out from the head just as in carious disease of the temporal bone. At the *P.-M.* we found miliary tuberculosis of the serous membranes, spleen, liver, and lungs, caseation of the bronchial glands, and compression of the right external jugular vein by an enormous mass of glands. The œdema had disappeared soon after death.

## II. *Derangements of the Excretion of Urine.*

According to the principle which I have laid down for myself, I shall limit myself here to a description, drawn from personal experience, of those derangements which occur mainly or altogether in childhood. I may first mention congenital hydronephrosis, due to congenital obliteration of the ureters, which is almost always unilateral, and can only very rarely be diagnosed during life. It is extremely uncommon to find it bilateral. This was, however, the case in a child of 3 weeks, who was admitted into my ward with a large swelling in each hypochon-

drium, both of which were fluctuating and dull on percussion. There had been absolutely no urine for about 10 days, but some was said to have been passed during the days previous to that time. I punctured the swelling on the left side, and withdrew a quantity of sero-sanguinolent fluid, and at the post-mortem we found that the condition was one of bilateral hydronephrosis with cicatricial obliteration of both ureters at the point where they leave the pelvis of the kidney. This obliteration cannot have appeared until after birth, on one side at least, for otherwise no urine could possibly have been passed during the first days of life. Moreover, the fluid which was removed by puncture came not from the hydronephrosis itself but from a cyst filled with bloody serum into which the trocar had penetrated. This cyst surrounded the whole kidney, and was probably the result of a perirenal hæmatoma which had arisen during fœtal life.[1]

Let me call your attention more at length to the derangements caused by lithiasis.

The uric-acid infarcts of the straight tubules invariably present in new-born children are generally washed out within the first few weeks, so that they do no harm. Still, this process is sometimes very protracted, and I have found, even in a few children of 7 or 8 weeks, fragments of the infarcts still remaining, some of them adhering to the lumen of the tubuli or to the papillæ, or even lying in the pelvis of the kidney as little reddish fragments. The irritation caused by these little concretions may at a very early period occasion difficulty of micturition, which is at first disregarded and is always hard to account for at this age, since the examination of the urine is so extremely difficult. Children are often brought to you in the first months of life who cry violently every time they pass water or before doing so. In spite of strong and repeated endeavours they never pass more than a few drops at a time, and are so very restless that we cannot doubt that they are in pain; at the same time the general health, during the intervals, may remain undisturbed. If we look at the diapers we often find that the places wetted by the urine are darker than usual and their borders have a reddish tinge, and sometimes we also find a few yellowish fragments like grains of sand. In these cases also the urine may by its acidity not only cause scalding in its passage through the urethra, but

[1] *Vide Charité-Annalen*, viii., S. 568.

may also redden the orifice of the prepuce and the inner surface of the labia and the parts in their neighbourhood. We often meet with precisely similar symptoms in older children, in whom they cannot be due to delayed excretion of uric-acid infarcts, but rather to newly formed uric-acid concretions (gravel) which are generally to be regarded as due to perverted nutrition.

Dysuria in little children is often difficult to explain. It is only when small uric-acid concretions are found on the diapers —and, in older children, in the urine—that the diagnosis is established beyond a doubt. In these cases, as in adults, there usually occurs some catarrhal irritation of the pelvis of the kidney, which in turn favours the formation of uric acid gravel, and may lead to symptoms corresponding to those of calculus-pyelitis in later life.

> Frida R., 3 months old, brought to the polyclinic on January 16, 1874. Had taken ill a fortnight before with vomiting and repeated twitching of all the limbs. Sickly appearance. Distressed screaming before and during micturition, and only a few drops passed in spite of violent pressing. Urine pale yellow, very turbid, containing a large quantity of albumen and no tube-casts, but innumerable pus-corpuscles covering the whole field of the microscope. There were also a considerable number of minute, angular, brick-red concretions, the size of a pin's head, and under the microscope these were found to be composed of uric-acid crystals.

I have already (vol. i., p. 189) given you a similar case, namely, that of a child of 5 months who suffered from severe dysuria while passing little calculi and had reflex convulsive attacks and contracture of many groups of muscles. When this condition lasts for a long time the catarrh of the pelvis of the kidney may gradually extend down the ureters to the mucous membrane of the bladder; and as catarrh of the bladder is known to favour the formation of calculi, it may there give rise to lithiasis vesicalis. Stone in the bladder is just about as common in childhood (even during the first year) as in later life, and it is therefore necessary in every case of chronic dysuria, whether combined with catarrh of the bladder or not, to examine the urethra and bladder under chloroform. In these cases micturition is sometimes quite checked, and in spite of violent pressing (which not uncommonly produces prolapsus ani) only a

few drops are passed. I have known the urine to be retained for 36—48 hours, the bladder being enormously distended and to be felt above the symphysis, so that it had to be emptied bya catheter; while at other times there was a continual trickling from the urethra. In this case the neighbourhood of the genital organs was kept constantly wet, and the decomposing urine not only caused an unpleasant smell, but by its irritation set up inflammatory and œdematous conditions of the prepuce, penis, and scrotum. In older children you find the penis unusually long and large, probably owing to its being constantly manipulated. There is frequently also the tendency to prolapse of the rectum, which I have already mentioned (p. 75), and I consider this a symptom of lithiasis which is not to be undervalued in children. Occasionally there is complete retention of urine, along with erythema and œdematous swelling of the genital organs, owing to a stone having got into the urethra and become impacted there.

> Alex L., 2 years old, admitted November 28th, 1877. Pretty well nourished, but pale. Complete retention of urine for last two days, slight redness and great œdema of the penis, scrotum and perineum. Prepuce could not be retracted owing to phimosis. Abdomen distended, hard, and tender; the bladder much distended, reaching two finger-breadths above the symphysis. The phimosis had to be operated on before a catheter could be introduced, and at the operation a stone the size of a pea, of a sulphur-yellow colour and friable consistence was found at the orifice of the urethra, which it had entirely occluded, and was extracted by means of a probe. A catheter was then introduced into the bladder, and a quantity of turbid urine was drawn off. The œdema rapidly disappeared under the use of lead fomentations, but cholera infantum set in on the night of the 29th with rapid collapse. Death on 1st December. *P.-M.*—the bladder was almost completely filled by a sulphur-yellow stone of the size of a hen's egg, with a defect corresponding to the fragment which had been passed per urethram. In the calyces of the left kidney there were quite similar stones from the size of a pea to that of a bean. Right kidney normal.

Other forms of dysuria also occur in childhood which have nothing to do with the actual formation of concretions in the kidneys or bladder. Even the passage through the urethra of very acid concentrated urine, as occurs in high fever, may give rise to pain during micturition, which is indicated by screaming,

and, in older children, by definite complaints. Moreover, when the urine is loaded with urates, attacks may occur resembling those of renal colic in adults. In two children of 3—4 years, violent pains set in in the abdomen, sometimes even accompanied by chilliness and heat, and lasting several hours; these sometimes kept recurring during a series of days, and always ended with a secretion of turbid albuminous urine loaded with urates. In the intervals, which often lasted for months, the general health was unimpaired and the urine perfectly normal. As the attacks were accompanied by nausea and constipation, the case had at first been diagnosed as one of intestinal colic, until the character of the urine at last attracted attention and led to an examination. Actual discharge of gravel—which in adults as in children may occasion inflammatory processes in the pelvis of the kidney and even in its parenchyma also—had never been observed in this case. All the more remarkable was the occurrence of temporary albuminuria with the attacks of pain, and yet it could only be regarded as due to the irritation caused by the morbid urine. The prolonged use of the mineral waters of Vichy, Wildungen, and Bilin, or of a solution of bicarbonate of soda, has served me as well in these cases as it generally does in similar affections of adult life.

I need scarcely add that in any case of dysuria in a child you must never neglect to examine the external genitals. On doing so you will not uncommonly discover the presence of phimosis, which more or less hinders the passage of the urine, and causes some of it to be retained behind the prepuce, and it may there decompose, giving rise to an inflammatory condition of the prepuce, balanitis, and painful micturition. I have observed occasionally in boys of 3—5 years a condition of well-marked gonorrhœa (with violent pain on micturition) which seemed to have been caused by the manipulations of other children. Congenital abnormalities of the urethral orifice also occur. Thus in a boy of 7 months I have seen only a slight furrow in the situation of the meatus, while the urine issued in three thin streams from three punctiform openings in its immediate neighbourhood. In such cases an operation is the only possible form of treatment. The same holds good of the adhesion of the labia minora to one another, which is occasionally met with in little girls. This, like the similar adhesion of the

two layers of the prepuce in boys, is almost invariably present in very early life, and it sometimes persists to the end of the first year and even longer. The adhesions can generally be separated by the handle of a scalpel, and only rarely require an incision. In some cases these adhesions have appeared to me to be the cause of dysuria, which at once disappeared on the labia being separated from one another. In other cases we found on examination some inflammatory redness of the entrance to the vagina and of the urethral orifice, and increase of the mucous secretion, and it was this condition that had caused the pain on micturition.

You will be called in far oftener to treat another condition which excites the parents' anxiety. I refer to incontinence of urine, especially nocturnal. Not only children in the first years of life, but also those who have already passed the second dentition and are approaching puberty, may suffer from this malady, of the cause of which we know so little that we are not always perfectly sure whether it is to be referred to a morbid condition or only to bad habit. "Wetting the bed," which is almost as common in girls as in boys, takes place first either during the earlier hours of sleep or only later on towards the morning. It may occur once or oftener in one night; sometimes at intervals of days or even of weeks, and this we generally observe more particularly during the course of acute diseases. The variety of opinion among physicians in regard to this complaint, but especially the number and variety of the remedies recommended, indicate that it is probably not always due to the same causes. First of all, I advise you never to neglect the examination of the urine; for cases are known of diabetes mellitus, and also of chronic nephritis, having first revealed their presence by nocturnal enuresis. But I have never as yet met with such a case myself, and therefore I cannot help regarding this as an extremely rare cause of incontinence. The proximate cause of the malady consists either in atony of the sphincter vesicæ or of a spasmodic contraction of the detrusor urinæ, which is able to overcome the contraction of the sphincter, which is less powerful during sleep. The first of these appears to me to be the less common and sometimes, though not invariably, it is combined with diurnal enuresis. When this is the case it always suggests a suspicion of lithiasis, and examination of

the bladder should never be neglected. In children who are otherwise healthy it seems, indeed, somewhat forced to assume the presence of atony limited to the sphincter vesicæ, and only in a few cases where there is well-marked debility—*e.g.* after an attack of typhoid or other serious disease, or in organic disease of the spinal cord—does this assumption appear to me to be justified. In the latter case especially, there sometimes occurs a continuous dribbling of urine, and it is only rarely that the patients are able to retain any considerable quantity at a time and to pass it in a stream. A boy of 13 afforded a characteristic example of this kind of enuresis, and had done so from early childhood. At the lower part of the lumbar vertebræ there was a flat doughy tumour about the size of a hen's egg, in the middle of which a defect in the spinous processes could be distinctly felt—probably the sac of a spina bifida filled with fat and granulation-tissue.[1] There was also involuntary defæcation whenever the motions were at all loose. It is only in such "atonic" cases that the much recommended treatment with tonic remedies (iron) and with ergotin and strychnia (internally or subcutaneously), is of any avail. These remedies, however, have not in my experience been at all successful. Also, electricity applied directly to the rectum, which has been so much recommended, and which is supposed to act reflexly on the sphincter of the bladder,[2] really acts (if at all) mainly by its psychical influence—to which I shall return shortly.

The condition is quite different in ordinary nocturnal enuresis; for in it the urine is always passed in a stream, and only during sleep, or when the child is half-awake. Here, in fact, there seems to be a reflex irritation of the detrusor, the action of which is all the stronger because the influence of the will on the sphincter is in abeyance during sleep. The first thing the physician has to do, therefore, is to try to discover the seat of origin of this reflex impulse. We sometimes succeed in finding either congenital phimosis, complete adhesion of the prepuce to the glans, stricture of the urethra, irritation due to thread-worms, fissure of the rectum, or even masturbation or vulvitis—to be the cause of the trouble; and when these are cured, the enuresis

[1] Some similar cases have been published by Blake (*Amer. Journ. of Obstetr.*, 1878, p. 146). In one of these the child seemed perfectly well with the exception of diminished functional activity of the bladder and rectum.

[2] Ultzmann, *Centralzeit. f. Kinderkr.*, i., No. 22.

also disappears. In the same way, renal and vesical calculi, or even the loading of the urine with lithates or phosphates may give rise to an irritation of this kind, and we must then try to cure the case by treating these abnormalities. Unfortunately, you will not in the majority of cases succeed in finding any of the abnormal conditions mentioned, nor even if they are found, will you manage to cure the enuresis by treating them. We then usually have to content ourselves by assuming a state of hyperæsthesia of the neck of the bladder or of the whole vesical mucous membrane, similar to what occurs in many cases of nocturnal emissions in adults—with which nocturnal enuresis has this further feature in common, that it is most apt to occur when the patient is lying on his back, and is often excited by dreams which appear to act directly on the detrusor. In such cases some pin their faith to sedative remedies, especially belladonna (extr. bellad. gr. $\frac{1}{12}$—$\frac{1}{4}$), but my own experience does not incline me to agree with them. Others recommend the frequent introduction of a bougie into the urethra or the cauterisation of the neck of the bladder, in order to diminish its excessive sensitiveness. I am far from denying that those means may possibly be successful, and I believe that they are always worth trying in very obstinate cases; but one must not forget that the psychical effect of these manipulations, and the pain which they cause to the little patients, is not to be overlooked, and especially that we can never be quite sure that it is not merely a bad habit. That this may be the case was proved by a few cases of incontinence of fæces which were cured with surprising rapidity.

In October 1879, a boy of 8 years was brought to the polyclinic who, for the last 2 years, had not been able to retain his fæces, but especially since an attack of typhoid a year before. He dirtied his clothes several times a day, but never during the night time. Micturition normal. General health perfectly good. On examination of the rectum we found no abnormality, and the sphincter ani contracted firmly. After nux vomica had been used without any effect, ergotin (gr. 1½) was injected subcutaneously near the anus, and the very first injection was completely successful. Three days afterwards defæcation was still normal. I told the mother that if the incontinence returned, she must bring him back at once—and I never saw him again.

Five other cases had an entirely similar course and were cured in the same way, although only after the second injection, in

which, however, we tried the experiment of using only distilled water. A few smart strokes on the nates immediately after the injection considerably enhanced the effect of the treatment. No one, surely, will deny that the only action in these cases was due to the psychical effect of the injection, *i.e.*, the fear of the needle and of the application which followed, and I therefore believe that many other cases of rapid recovery from nocturnal enuresis following the use of painful methods of treatment or faradisation are to be judged in the same way. I have at any rate been able, by this method, to cure within a few days a case of diurnal and nocturnal enuresis which had lasted for several years.[1] In any case, the amount of fluid taken before going to bed should be limited, and lying on the back must be avoided if possible; for the latter purpose it has been recommended that we should fasten a brush to the patient's back so that the bristles touch the skin. In many cases spontaneous recovery takes place after various remedies have been used in vain—often not until puberty is pretty well advanced.

## III. Diseases of the External Genital Organs.

I have already (p. 177) alluded to a fact, which in spite of the writings of Bokai and Schweigger-Seidel[2] seems to be still unknown to many practitioners. I refer to the adhesion of the inner surface of the prepuce to the glans by means of more or less dense tissue which, even where there is not a trace of phimosis, only permits of partial retraction of the prepuce. When we try to retract it, we soon encounter resistance, and we then discover that the inner surface of the prepuce is adherent to the surface of the glans; and the younger the children are, the more intimately and extensively are they united. This adhesion, which is to be regarded as the normal condition in the first period of life, may be found in children of 4 or 5 years, and even later; but as the age increases the adhesion becomes less extensive and less firm. The connecting material

[1] I may mention as belonging to the same category the case of a boy of 12, who almost every night for years had inserted his finger into the rectum and then passed his fæces into the bed. On July 18, 1881, he was brought into the hospital. Chloral was given with no effect, but he was at once completely cured on being threatened with the application of electricity and the actual cautery.

[2] Virchow's *Arch. Bd.*, 27, H. 2.—*Jahrb. f. Kinderheilk.*, v., Heft 1.

is formed of ordinary polyhedral epidermic cells, and probably arises in the following way. The cells of the rete Malpighi which come to the surface do not become cornified in the ordinary way, but remain filled with protoplasm, thus giving rise to adhesion between the two surfaces. By these adhesions (which, although they do not involve the whole surface of the glans, yet form a ring right round it) retention of smegma may be caused inside the prepuce, giving rise to balanitis, and this may cause a bladder-like swelling of the prepuce affecting the whole end of the penis. In cases of this kind, which we have sometimes seen at the polyclinic, rapid recovery ensues when the adhesions are broken down with a probe, and lead-fomentations are applied. The adhesion of the labia minora occasionally occurring in little girls, which I have already referred to as a cause of dysuria (p. 177), appears to originate in the same way as the adhesion of the prepuce, but only when the labia minora are in contact along their entire length.[1]

We pretty often (10 times in 100 newborn children) observe cryptorchidism, *i.e.*, the absence of one, more rarely of both testicles from the scrotum. In the normal condition the testicle passes down the inguinal canal in the 9th month of fœtal life; sometimes, however, this does not take place until after birth, so that we do not find the process completed until the 9th or even the 30th day of life. Unusual narrowness of the inguinal ring or inflammatory processes causing adhesion of the testicle may prevent its descent, so that it remains fixed in the abdominal cavity, or just outside the inguinal ring; and it sometimes remains there during the whole life-time without there being any interference with the functions of genital organs. The diagnosis of non-descent of the testicle is easy. You find the scrotum small and empty on one or both sides, while the testicle is generally of the normal size; sometimes, however, it is swollen, by serous effusion in the inguinal canal, to the size of a pigeon's egg or larger, and is either just outside the inguinal ring or else to be felt more or less distinctly within it, and if not firmly impacted it is movable to some extent. As a rule, the right testicle is oftener absent from the scrotum than the left. When the descent of the testicle is unusually long delayed —*e.g.*, till the end of the first year—there is always some risk

[1] Bokai, *Jahrb. f. Kinderheilk.*, 1872, S. 163.

inguinal hernia occurring. It is but rarely that the undended testicle becomes incarcerated in the inguinal canal, ag rise to very severe pain, and finally to inflammation, and en requires absolute quiet and antiphlogistic treatment (an ag, leeches). For the cryptorchidism itself nothing can be e. Further, we must not forget that in the normal state en the scrotum is firmly contracted, the testicles may be far drawn up by the cremaster that they can be felt just oute the inguinal ring, and it is only when the scrotum relaxes at we can make sure that it is not a case of non-descent of e testicle.

Diseases of the testicle, apart from hydrocele (which is ommon), are of rare occurrence in childhood. I would remind ou here of the enlargement due to hereditary syphilis which has been already mentioned, and which according to my experience seems to be commoner than the cases of medullary sarcoma, and enchondroma, which are occasionally observed, and which correspond entirely with those of the same disease in adults. Tuberculosis and caseous degeneration of the testicle is also undoubtedly rare, clinically at least, considering how extremely common the process is in other parts. I have myself met with several cases of this kind in children between the ages of 1½—7 years. The swelling generally affects the epididymis on one or both sides, which seems hard and uneven, and the testicles themselves are but rarely implicated. When they are, nodular tumours result, from the size of a walnut to that of an apple, and they may be further enlarged by the addition of a hydrocele; from time to time they become inflamed and burst, discharging caseous pus. Tuberculosis of other organs is almost always to be found, or caries of different bones, and in one case I observed chronic peritonitis which burst at the umbilicus. Local treatment (incision and scraping out) can only be used when suppuration has occurred. When this is not the case we must confine ourselves to the use of cod-liver oil, iodide of iron, and salt baths.

In little girls during the first days of life, we occasionally observe a small or even a considerable amount of hæmorrhage from the vagina, apparently connected with the desquamation of epithelium occurring about this time, and it generally has no bad effects. Premature menstruation occurring in the

11—12 year is not very rare, but its commencement before this period—*e.g.*, even in the 3rd or 7th year—[1] is certainly exceptional. Moreover, one must guard against putting down every hæmorrhage from the genital organs in little girls as a sign of premature menstruation. For we find on examination that this hæmorrhage arises not uncommonly from papillomata of the vulva and vagina, or from a polypoid swelling of the urethra. In these cases a dark-red swelling, which readily bleeds, protrudes from the urethral orifice, and may become so large as to push apart the rima vulvæ. The frequent desire to micturate which is often present is apt to be overlooked, so that the hæmorrhage is the first thing that attracts the mother's attention. In the cases of two girls, of 7 and 10 years respectively, who were observed in my ward, we obtained recovery in the course of a few weeks by cauterising the swollen and prolapsed urethral mucous membrane.

The commonest affection of the genital organs in little girls is vulvitis. These children are brought to you some days or weeks after a muco-purulent discharge from the genital organs has been noticed, and on examination you see a purulent fluid which issues from these parts and often dries up into thin crusts on the inner surface of the labia and thighs, and—like the fluor albus of adults—forms hard greenish-yellow spots on the linen. The mucous membrane of the vaginal orifice is reddened to a varying degree, and also perhaps slightly abraded, the labia majora and minora are often somewhat swollen and tender. This condition is often accompanied by dysuria, and many children are unwilling to walk because of the pain which is caused by the unavoidable rubbing together of the inflamed parts.

In such cases one is apt to suspect that there has been an attempted assault, and I know from my experience that this suspicion is sometimes only too well founded. I have notes of a whole series of cases of children of 4—11 years falling victims to brutality, indecency, or a certain superstition; in some of them there had been an actual attempt at rape, or there might only have been indecent manipulations. In some cases an older brother who has been sleeping with the child must be looked upon

[1] *Oesterr. Zeitschr. f. Päd.*, 1877, viii., S. 26.—*Deutsche Zeitschr. f. prakt. Med.*, 1878, S. 487.

as the culprit. Rupture of the hymen[1] is but rarely found, for the narrowness of the parts does not allow of a complete immissio penis. In spite of the facts, however, I advise you not to be too ready to assume that there has been an assault, for it pretty often happens that the mothers who declare that there has been such a thing, are either themselves deceived, or are trying to impose on the physician in order to get from him a certificate which they may use for ulterior ends. You ought never to accede to requests of this kind except in cases concerning which there can be no doubt; for, apart from the violent attempts at rape, we must not forget that there are other causes of vulvitis. Even want of cleanliness and the continual accumulation of sebacious matter between the labia may lead to a state of irritation, and frequent manipulations practised by the children themselves, or by their playfellows, or again, the irritation of thread-worms from the rectum (which causes frequent scratching of the anus and its neighbourhood)—may have even a more pronounced effect in giving rise to vulvitis. Where none of these causes exist, we must bear in mind the possibility of a local chill, but this can hardly ever be proved with certainty. I have frequently observed fluor albus following scarlet fever, probably rising from transmission of the inflammation of the skin of the labia to the adjacent mucous membrane. It is only rarely, according to my experience, that we meet with infection from gonorrhœa or fluor albus in the relatives. Still, the observations of many authors—especially the fact of several children who were living together being affected simultaneously, the ascertained possibility of transmission to the conjunctiva, and, finally, the fact that gonococci have several times been demonstrated in the secretion in my ward—all seem to indicate that infection may play some part in children also.[2] In one of these cases, that of a girl of 8 years, we also found swelling and pain of the left wrist without fever which lasted 7 days, and resembled in every particular the gonorrhœal synovitis of adults.

In addition to the above-mentioned symptoms of vulvitis we

[1] With respect to the form of the hymen in children which may give rise to mistakes, *cf.* Skrzeczka, *Vierteljahrsschr. f. gerichtl. Med.*, 1866.

[2] Pott, *Jahrb. f. Kinderheilk.*, xix., 1882, S. 71.—Hirschberg, *Berl. Klin. Wochenschr.*, 1884, No. 33.—Widmark, *Jahrb. f. Kinderheilk.*, xxiii., S. 210.—E. Fränkel, *Arch. f. Kinderheilk.*, vi., S. 372.—Cséri, *Pesth. med. chir. Presse*, 1885, No. 11.

not uncommonly find erosions and ulcerations of the labia minora and majora, which are calculated to strengthen any suspicion of syphilitic infection. In most cases, however, even where there had undoubtedly been an assault, the condition presents simply the appearance of an inflammatory affection due to external violence, and the only exceptions were certain very rare cases of regular chancres in girls of 11—13 years who had already practised prostitution. In other cases there was only superficial ulceration which was to be regarded as due to the maceration by purulent secretion and to friction. There was also sometimes an eruption of a number of herpes-vesicles, clustered together on the reddened labia majora. These vesicles entirely resembled those of zoster, but they were bilateral in their distribution and extended over the perineum as far as the anus.[1]

The treatment which we have been in the habit of using for vulvitis has consisted (except in the rare cases of syphilis) in making the patient lie absolutely still, constant application of lead-fomentation, and—when these did not soon act—vaginal injections of lead-lotion, or of tannin, or alum (5 per cent.), sulphate of zinc (2 per cent.), nitrate of silver (1 per cent.), or chloride of zinc (½ per cent.). It was often necessary to tie the children's hands in order to prevent them from touching the affected parts; but still it often took many weeks before the affection could be regarded as quite cured. Pott recommends, as being rapidly effectual, iodoform (5 parts to one part of gum arabic) to be blown through a narrow speculum into the vagina by means of an india-rubber insufflator, or that iodoform bougies should be allowed to melt in the vagina. We have often made trial in the ward of these bougies (iodoform or tannin, grs. 15—30, ol. theobrom. grs. 15, formed into thin rods); but only in a few cases did we see any speedy good result.[2]

In another class of cases there is not only an inflammatory affection of the mucous membrane, but at the very beginning there appears an extensive more or less hard and tense swelling of the labia majora. This usually continues to be limited to these parts, but in some cases it spreads to others and even

[1] Demme (*Jahresber. f.*, 1886, S. 31) has seen in two little girls tuberculosis of the genital mucous membrane with lenticular ulcers, the nature of which was established by the discovery of bacilli.

[2] Frühwald, *Wien. med. Wochenschr.*, 7, 1883.

to the mons Veneris. The mucous membrane of the vaginal orifice and the skin on the outside of the labia may remain quite unaffected, and the general health be quite undisturbed. Only once, in a girl of 3 years, have I seen urticaria come on simultaneously. I have never been able to make out for certain the cause of these swellings. Under the continuous use of fomentations of warm lead lotion the swellings either subsided within 8—14 days, or developed, with increasing redness and pain, into abscesses which required early incision.

From these simple inflammatory swellings we must distinguish a more serious kind, in which there is a marked tendency to gangrenous destruction of the affected parts. Gangrene of the vulva may arise from a simple very hard swelling of the labium, the mucous membrane and cutis both being intact. Oftener, however, it results from an ulceration which penetrates more or less deeply from the surface of the labium and is covered with diphtheritic membrane. The following case belongs to the first category.

> Marie K., 2 years old, admitted February 26, 1874. A healthy looking child. During the last 4 days, increasing hard swelling of the left labium majus, which was now dark-red, and presented on its inner surface a deep grey ulcer surrounded by a perfectly black border, which had only been noticed for 1½ days. There was high fever (ev. 104·5° F.); pulse 160—176. Otherwise everything normal. Thorough application of the actual cautery to the gangrenous area and its neighbourhood. On the following day, fever almost gone (m. 98·2°; ev. 100·9° F.; pulse 116). On 4th March the slough separated, leaving a clean surface. Complete recovery after a short time.

The rapid action of the actual cautery both on the fever and on the local process seems to indicate that the latter was due to some purely local, perhaps traumatic, cause which had rapidly produced an extensive hard infiltration of the whole labium and necrosis. The second form begins at once with a dark livid redness of the labium and of the neighbouring mucous membrane, the epidermis of which is raised up in vesicles or shed in fragments; and finally there is a formation of dirty-grey or bluish-red ulcers especially on the inner surface of both labia. The latter are œdematous or infiltrated, hard and swollen. The ulcers soon become covered with a yellowish-grey or blackish-green friable layer of gangrene, and rapidly increase in depth so

that not only are whole pieces of the labium eaten away, but the gangrene may even spread to the mucous membrane of the labia minora and to the vaginal orifice. We have here, therefore, conditions quite analogous to those of noma of the cheek, which, as I have mentioned (p. 12), may originate from the mucous membrane as well as from the substance of the cheek itself. The etiology of gangrene of the vulva is also identical with that of noma—extreme general cachexia of debilitated children, previous infectious diseases (especially measles, scarlet fever, and typhoid) and gangrenous processes in other parts of the body. For example, I observed the latter in a phthisical girl of 12 years, in whom gangrene of the lung was followed by gangrenous destruction of the labia.[1] In cases of this kind we must of course give an utterly bad prognosis from the first; but in less unfavourable circumstances we must not entirely abandon hope. We have been in the habit of fomenting and dressing the gangrenous areas with carbolic acid (2—3 per cent.), camphorated wine, chloride of zinc (2 : 1000) and—during the last few years most frequently—with iodoform. When needful, we must here also apply the actual cautery. When the labium is very hard and much swollen, deep incision may be recommended in order to relieve the tension and to prevent gangrene setting in. At any rate, in a girl of 3 years whose right labium presented an ulcer as big as a threepenny-piece, and was much swollen and extremely hard, this treatment seemed to succeed. After 3 incisions, yielding only blood and no pus, the hardness rapidly diminished and entirely disappeared in the course of a few days.

[1] *Charité-Annalen*, Jahrg. i., S. 618.

## SECTION VIII.

### INFECTIOUS DISEASES.

In spite of the very great progress of bacteriological research during recent years, hardly any results have been obtained affecting the subject of the infectious diseases of childhood, which we have now to consider. The fact that germs have undoubtedly been found to be the cause of recurrent fever, anthrax, and tuberculosis, induces us to regard measles, scarlet fever, and diphtheria as all being products of specific bacteria; and, considering the present stage of the science of bacteriology, there is certainly no objection to our doing so. Still we must not forget that the morbific fungi which are supposed to cause these diseases have not yet been discovered. Perhaps this is because we have not yet hit upon the method of staining which is required to demonstrate these particular micro-organisms. Meanwhile we certainly have no right to speak of a "diphtheria fungus," or "scarlet fever bacterium,"[1] &c., with that absolute certainty which can only be reached by very exact observation and experiment. But even when we have succeeded in demonstrating the pathogenetic fungus of any particular disease, further research into its chemical products is still of the greatest importance; for these (the so-called ptomaines) seem to play a more important part than the bacteria themselves. It is well known that in the diseases under consideration various forms of bacteria have been found in certain glands and in many other organs. These, however, are in no way specific, and are mainly of a "septic" nature. We must not overlook the fact that the germs of minute organisms which are always present in the atmosphere in enormous numbers will develop and multiply most rapidly on parts which are diseased, and are impregnated with the products of decomposition, and that from thence they may readily find their way by means of the lymph- or blood-

[1] Jamieson and Edington, *Brit. Med. Journ.*, June 11, 1887.—See also Escherich, *Centralbl. f. Bacteriologie u. s. w.*, i., No. 13.

stream into deeper parts even some distance away. The lesions of the pharyngeal and nasal mucous membrane in scarlet fever and diphtheria too often present the most favourable soil for their growth. I have myself seen cases of this disease in which we found, at the post-mortem, masses of those "minute organisms" within the lungs and on the cardiac valves; and they had evidently been carried thither by the blood-current. In such cases, then, we may at any rate assume that there are two distinct carriers of infection acting together, of which the first is specific but unknown, while the second is dependent on the entrance of certain micro-organisms well known to us under other circumstances as bearers of sepsis. The investigations recently published by A. Fränkel and Freudenberg[1] are worthy of special attention in this connection, even although they do not, strictly speaking, prove that the clinical symptoms are dependent on the bacteria which are present.

It has long been known that various acute infectious diseases may appear simultaneously in the same individual; and the fact has been rendered more intelligible by this "secondary" or "mixed" infection having been demonstrated microscopically. Most frequently we see acute exanthemata, especially measles and less commonly diphtheria, developing in the course of, or simultaneously with, an attack of whooping cough—which is also to be regarded as an infectious disease. A series of cases has been published in which two different acute exanthemata occurred together in the same individual, presenting symptoms which were hard to explain. Although all the observations of this kind would certainly not stand searching criticism—for many really seem to depend on a confusion with relapsing measles or scarlet fever, or with certain forms of erythema—still, there remains a number of thoroughly well established cases; and to those I am able to add certain others from my own experience.

Franz R., 9 years old, admitted on March 4, 1875, with varicella, which had set in 2 days previously. The whole body was covered with characteristic vesicles, some of which were umbilicated, and there were a few on the hard palate. Temp. 100·4°; ev. 103·1° F. On the following day there was a further eruption on

[1] "Ueber Secondärinfection nach Scharlach," *Centralbl. f. klin. Med.*, 1885, No. 45.—Cooke, *Fortschritte der Med.*, 1885, No. 20.

the lower limbs, and while some of the vesicles were becoming purulent, others were drying up. On the morning of the 7th, no fever, but complaints of pain on swallowing, and sore-throat. In the evening, an eruption of scarlet fever (Temp. 104°) which became diffuse on the following day, and then presented the interesting phenomenon of an intensely red skin—with chicken-pox vesicles—some of which were dried up and some still filled with pus—scattered over it. The further course was normal. Discharged on 13th April.

This patient was probably infected with scarlet fever in the hospital, for he had lain 36 hours in the ward for infectious diseases, which was partly occupied by scarlet fever patients. If we assume even a very short period of incubation for scarlet fever (1—2 days) the infection would still have taken place before the varicellar process had run its course and while the temperature was still high. In another child of one year, who was admitted with the eruption of chicken-pox just commencing, measles developed 5 days afterwards, so that both exanthemata were on view at the same time.

Otto W., 7 years, admitted October 31, 1876, with spinal caries and an abscess on the right side of the lumbar column. Abscess opened and dressed antiseptically. On 29th November, eruption of scarlet fever with severe sore-throat and high temperature. On December 5th, commencing desquamation, rash faded. On the following day, high temperature; dulness over the upper part of the right side of the chest before and behind, indeterminate breathing and crepitations; resp. 48; temp. 104·2° F. On the 7th, a fresh maculo-papular eruption on the face, arms and legs, with all the characters of measles; coryza with profuse purulent fetid secretion, extreme restlessness, drowsiness, croupy cough and obstructive breathing. Death during the night. *P.-M.*—Diphtheritic laryngitis and pharyngitis, croup of the trachea and large bronchi, croupous pneumonia of the right upper and middle lobe.

Alexander S., 4 years old, admitted February 16, 1877, with prolapsus ani. On the 27th, eruption of scarlet fever with high temperature and moderate pharyngitis. On 4th March, commencement of desquamation on the face, rash gone. On the 5th, the temperature rose again (104·4° F.), and there appeared a papular measles-rash spreading rapidly from above downwards, with much coughing and catarrhal sounds. During the next few days the temperature still high, dyspnœa, resp. 54, fine crepitation on both sides behind. Death on 15th. *P.-M.*—Capillary bronchitis, multiple broncho-pneumonia. Fatty degeneration of the liver.

That both of these children had regular measles and not

merely a relapse of scarlet fever, was proved not only by the character of the eruption, but still more conclusively by the accompanying affection of the respiratory organs. Now, since the period of incubation of measles lasts for at least 12 days, the measles infection must have taken place either simultaneously with, or even previous to, that of the scarlet fever.

Girl of 5 years, admitted with an eruption of measles, which had appeared on November 14, 1881. The fever continued after the eruption had passed off. The temperature rose gradually in the course of 6 days to 104° F. in the evening, although no local disease could be found on examination. On the 21st, the spleen was felt to be enlarged; on the 22nd, copious roseola on the thorax and abdomen. Apathy, diarrhœa, thickly-furred tongue. Typhoid fever which ran its course in the usual way.

When we take into account the known duration of the periods of incubation in the two diseases, it becomes evident that the child must have become affected with the two contagia almost simultaneously.

Much commoner than these cases are those in which two or more acute infectious diseases occur in the same individual not simultaneously, but successively, and after comparatively short intervals. My department in the Charité has afforded, unfortunately, particularly abundant material for such observations. This has been because the wards did not, previous to 1885, permit of the various infectious diseases being adequately separated from one another. Patients who had entered on the stage of convalescence from measles were often attacked by scarlet fever even within a few days, or *vice versâ;* children who had been tracheotomised for diphtheria and croup took scarlet fever, and so on. Sometimes even 3 or 4 such diseases followed one another in rapid succession.

Bertha W., 3 years old, admitted on November 29, 1876, with whooping cough. On 1st December, eruption of measles, with which the child must have become infected previous to entering the hospital; on the 4th, free from fever. On the 11th, return of fever. During the night scarlet fever followed by nephritis. Recovery. During the whole time the whooping cough remained unchanged.

Anna Th., 5 years old, admitted March 10, 1873, with whooping cough and diphtheria. Complete aphonia occasioning fears of croup; and the crowing sound of the whooping cough paroxysms changed into a harsh stridor. Albuminuria. Improve-

ment from the 14th; recovery on the 20th. From 10th April, fever; on the 12th, copious eruption of measles with severe bronchial catarrh and sore throat. Recovery.—During first few days of May, fresh rise of temperature. Typhoid fever which ended favourably in 3 weeks after a regular course.

Elise W., 8 years old, admitted on November 8, 1882, with scarlet fever. Fever prolonged by coryza and cervical adenitis. On 16th, eruption of varicella with increase of the fever (104·4° F.). On 18th, hoarseness, catarrh. Temp. ev. 104·5° F. On the 21st, temp. m. 104·4°; ev. 104·7°. On the following day, an eruption of measles which ended fatally.

Girl of 6 years, admitted on November 4, 1881, with whooping cough, on the 8th took scarlet fever with nephritis, and on the 14th measles; recovered.—Another girl, admitted on July 18, 1882, with measles, became more feverish on the 20th. On 21st, temp. 104·4°. Scarlet fever.—A third girl, admitted with typhoid on June 21, 1885, had an eruption of measles on 28th, and died of that disease on July 6th.

Cases like the last one, in which the infection with measles must, on account of the shortness of stay in hospital, be regarded as having taken place at home—occur, of course, in private practice also, although much more frequently, certainly, in children's hospitals. In the latter the spread of infection can only be prevented (or at least limited to a great extent) by building separate blocks which are so arranged as to allow the children affected with measles, scarlet fever, and diphtheria being accommodated in three different wards entirely cut off from one another, each being looked after by a separate staff of nurses. Happily the children's department in the Charité now at length affords this provision for such cases.

## I. *Scarlet Fever.*

Scarlet fever is one of the most dangerous and at the same time one of the most treacherous of the enemies of childhood. The complaint which I formerly made—that not nearly enough was being done by the authorities either for preventing or controlling the ravages of this enemy—has now lost a great part of its force, owing to the excellent police regulations now in force throughout the German empire. I do not, however, consider that these regulations are even yet all that could be desired. It is of the utmost importance that every child in whose family

even a single case of this disease occurs, should be strictly kept from attending school, in order to prevent the disease from hanging about the school—that hatching-ground of infectious complaints. Therefore, not only should the parents and the family doctor be required to give intimation of every case of scarlet fever; but, further, the neglect of this duty should be visited with legal penalties. Such stringent regulations would be disapproved of only by those who have no experience of the terrible ravages of this disease among children. What good does it do to shut the schools after the disease has spread till it has reached the proportions of a murderous epidemic? The fact that a number of cases have a more or less mild course, certainly cannot be regarded as proving that such rigorous isolation as I have recommended is unnecessary. For, apart from the varying rates of mortality in different epidemics (even in individual cases however slight they are to begin with), we can predict with less certainty in scarlet fever than in any other disease what symptoms are likely to develope, or what the end will be. From the following description founded on many hundreds of cases which have come under my own observation, you will perceive that I have by no means overstated the case.[1]

Scarlet fever generally affects children in perfect health. They go to bed in the evening quite well, and either waken in the morning with the initial symptoms of the disease, or show them on returning from school, or from a walk. Along with the general precursors of every febrile disease (irritability, loss of appetite, drowsiness, more or less violent headache) vomiting nearly always occurs once or oftener. This is (nearly always) soon followed by pain in the throat on swallowing, noticeable elevation of the temperature of the skin, and great thirst. The duration of those prodromata varies, but is always much shorter than in any other infectious disease; for the rash appears on an average within 24 hours, less commonly 36—48 hours after the onset of the first symptoms, but often sooner. We may take as example the following case which commenced while the child was under observation in the ward.

Child of 2 years. Quite well on previous evening. In the morning was fractious; loss of appetite, drowsiness, temp. 100·4° F.,

[1] Since the publication of the 2nd Ed., of these Lectures, 312 cases of scarlet fever were under observation in my wards during the years 1882 and 1883 alone.

pulse 144, all the organs apparently normal. At midday, vomiting. 4 P.M., red rash on chest and abdomen. Temp. 103·5° F., pulse 186. Pharyngitis, with punctiform hæmorrhages on the soft palate, diarrhœa, &c.

Sometimes the attack commences with a rigor—which I have observed even in a child of 2 years—or with sudden collapse resembling syncope, and then we discover that the temperature is very high. Much less commonly the disease is ushered in by one or more epileptiform convulsions; but I have very rarely seen this form of onset. I cannot endorse the opinion that the course of the disease will be more severe in direct proportion to the shortness and violence of the stage of invasion, and especially to the rate at which the temperature rises. In fact, this disease is one in which nothing can be foretold. Further, we must be prepared for cases of delayed appearance of the rash. In two fatal cases of mine, the rash appeared for the first time on the third evening after the onset of a persistent high temperature, which was accompanied by vomiting, mental dulness, twitching of the hands and of the angles of the mouth, sore-throat, and diarrhœa; in one of these patients, the eruption was first noticed in the two inguinal folds.

The scarlatinal eruption generally appears first on the throat and chest, a few hours later on the arms—especially round about the elbows—and less commonly on the face; and, in the course of a day, it spreads over the trunk and lower extremities. It does not, you see, progress in the same regular way from the face downwards as do those of measles and small-pox. The symptoms vary so much that it is quite impossible to give a description which will suit all cases. Generally speaking, a moderate form of the disease is what we most frequently find. In this the skin, when looked at from some distance, appears of a diffuse more or less intense red colour; and on closer inspection we find that this redness is caused by innumerable red points lying close together but separated from one another by very small areas of paler skin. The dark-red points seem to correspond to the hair-follicles; at least I have been able to observe in this disease, as in measles, that when the eruption has extended beyond the abdomen a darker colour and slight swelling is already noticeable at the roots of the hairs on the lower extremities. On the face only the cheeks and fore-

head are reddened, and that often only to a slight degree, whereas the nose and its immediate neighbourhood the upper lip and the chin are generally pale and somewhat yellowish. I have also often found the soles of the feet and palms of the hands quite free from eruption. As in all the exanthemata, the parts which are exposed to pressure—especially the back and nates—present the deepest and most diffuse redness. The rash is also marked over the skin of the chest and abdomen, while on the extremities it is less deep or may occur in patches separated by paler areas. When the hair-follicles are much swollen, the skin appears rough. On pressure with the finger the redness disappears for the moment, but at once returns. When one passes the finger-nail or any sharp body rapidly over the reddened skin, a white streak is at once produced, and remains distinctly visible for several minutes, so that one is able to write on the skin in characters which remain legible for some time. These "raies scarlatineuses" have been regarded as significant by French writers; but they occur also in urticaria and even in some healthy skins, so that they are in no way characteristic of scarlet fever. The varying intensity of the eruption seems to me more worthy of attention. It is generally more marked in the evening, but it may also on different days show alternations of intensity which do not always coincide with the variations in the temperature.

When the rash appears, the temperature rises and this persists without intermission as long as the eruption remains on the skin; *i.e.*, 4—6 days on an average. In nearly every case, even in those without any important complications, we find a continuous high temperature, which in the evenings reaches 103·6°—105·8° F., while in the mornings it is only 1·8° lower. Only very rarely have I observed the so-called inverse type during a few days, the morning temperature being 2° or 3° higher than in the evening.[1] The urine is often remarkably clear considering the high temperature; although it may also be of a dark colour owing to its containing a large quantity of urates. When the eruption is fading—that is, after about 4 to 6 days—the fever begins gradually to diminish, the evening and morning temperatures falling equally and reaching the normal level on the 6th or 7th day, simultaneously with the

[1] *Charité-Annalen*, iii., 1878, p. 513.

disappearance of the rash. On taking the temperature, however, we often find that it is still 100·4°—102·2° F. in the evening even for several days after the rash has entirely disappeared—and that in the absence of any complication; this represents the last remains of the infectious fever, and is similar to what we find regularly in typhoid. About this time there often occur severe perspirations, especially at night, and also eruptions of herpes labialis and nasalis. During the whole of this period the patient's general condition corresponds mainly with the state of the temperature. When the latter is only moderately high (not rising above 104° in the evenings) the children seem pretty well, except for their loss of appetite and great thirst. But when the temperature is higher, they are almost always very restless, and are apt to be drowsy and delirious. In a boy of 11, a regular fit of frenzy occurred on the evening of the 5th day (and yet the temperature was only 101·3°); he jumped out of bed several times, screaming fearfully, so that we had to tie his hands and feet, and administer chloral (grs. v.); he became quiet towards morning. The case ended favourably. The pulse-rate usually corresponds to the degree of the fever; but nevertheless in scarlet fever you must not be made very anxious—especially in the case of older children—even should you find a rate of 144 in the minute, so long as the quality of the pulse remains good—*i.e.*, so long as the tension of the artery is normal and the individual beats are distinctly marked.

Most children complain of pain in the throat from the first, especially on swallowing, and on examination we invariably find an intense bright or dark redness and swelling of the tonsils, soft palate and posterior wall of the pharynx which narrows the isthmus of the fauces more or less and interferes with swallowing (angina vel pharyngitis scarlatinosa). The uvula may also be regularly caught between the swollen tonsils or displaced backwards or forwards. In a few cases I have observed even in the first few days small blood extravasations on the reddened mucous membrane of the palate, and the sputum (which was brought up by hawking) then appears somewhat tinged with blood. Much oftener the reddened parts are covered here and there with muco-purulent matter which can be readily wiped off; or you may perhaps see on the tonsils those little purulent points of which I have already spoken (p. 15), and which I

would once more warn you against mistaking for "diphtheritic" membrane. The mucous membrane of the mouth is nearly always reddened all over, and it may also bleed when touched at all roughly. The tongue, which is generally covered by a yellowish-white coating during the first 2 days and is red at the margins, usually cleans on the third day, and then becomes dark-red all over, and resembles a strawberry all the more closely owing to the papillæ being more or less swollen and prominent (scarlet fever tongue). This condition of the tongue is not indeed quite invariably found, but it occurs so frequently that it may almost be regarded as characteristic of scarlet fever.[1] The affection of the mouth is sometimes accompanied by great increase of the salivation. When the rash fades the inflammation of the buccal and pharyngeal mucous membranes gradually disappears also, and only a few slightly enlarged lymphatic glands remain for some time under the angle of the jaw as the result of it. By the end of the first week the child has fully entered the stage of convalescence. About this time the desquamation of the epidermis begins, and the more intense the redness of the skin has been, the sooner does this process set in. On the face especially I have often observed it by the 4th or 5th day of the disease. The epidermis peels off, sometimes in branny scales, sometimes in larger flakes and fragments. On the body and thighs the desquamation is only noticeable as a dirty appearance of the skin, whereas from the fingers and the sides of the hands and feet large pieces of skin peel off. I have sometimes, however, seen the same thing on the nates, abdomen, and soles of the feet, and in some children the external auditory meatus becomes so blocked with desquamated epidermis that the hearing suffers and the accumulated masses have to be removed by syringing with warm water. In one case I observed large moist excoriations, which appeared on the 6th day after the desquamation, over the sacrum, knee, and elbow. I have never myself seen the falling out of the hair and nails, nor yet the decolouration of the new growth of hair which has occasionally been observed. As a rule the desquamation lasts for weeks, and during this time—as I have already mentioned (p. 139)—we often find temporary albuminuria.

[1] We must not, however, forget that a similar condition of the tongue is also pretty often met with in children who are not suffering from scarlet fever.

Only very rarely is the process of desquamation repeated, and in that case it lasts all the longer.

Unfortunately, the sketch of the disease which I have just given applies only to a certain proportion of the cases, which we may regard as normal; variations not only in regard to single symptoms, but in regard to the whole course, are so many and so frequently met with that it appears impossible to group them scientifically in definite classes. The deviations from the normal are much oftener of an unfavourable than of a favourable kind. We shall consider the latter first. In this, as in all other infectious diseases, the temperature may never rise beyond a moderate degree, or at least may show marked remissions in the morning hours, falling about 3·5° F. In such cases, also, the rash is generally pale, and the sore-throat not severe. In my paper on scarlet fever [1] I have given several cases of this kind, in which the temperature during the whole course of the disease did not rise in the morning above 100·4° or even 100°, and in the evening above 101·5° or 102·2° F., reaching 104° only on one or two occasions at most, and returning next morning to its former low level. From among the cases of this kind which I have observed since then I may select the following:—

Child of 3 years with scarlet fever beginning on 9th April.

| | M. | A. | P. |
|---|---|---|---|
| 9th April | | 101·3 | 144 |
| 10th „ | 100·4 | 102·4 | 152 |
| 11th „ | 100·4 | 100·9 | 132 |
| 12th „ | 100·4 | 100·4 | |

The fever and eruption had disappeared by the 13th.

In a girl of 13 the evening temperature never exceeded 101·3° (m. only 99·7°), and before the disappearance of the rash had fallen to 98·2 and 98·6.

In rare cases, after a smart initial fever, there is no rise of temperature whatever during the whole subsequent course.

Max P., admitted into the hospital on March 19, 1875, with an abscess under the right pectoralis major. Incision and antiseptic dressing. On the 21st and 22nd there was slight rise of temperature (100·6°—100·3°), while the child seemed perfectly well. On the evening of the 22nd the temperature rose suddenly to 104·4°; pulse 156. Deep, red rash on the arms, thighs, and face. On the

[1] *Loc. cit.*, S. 514.

following day diffuse scarlatinal rash, bad sore-throat. Temp. 96·6°—99·7°, pulse 131. The temperature rose on the evening of the 24th to 100°, but then rapidly fell again to 98·6°—96·8°, while the rash disappeared. On 2nd April, commencing desquamation which lasted about 12 days. No sequela.

In this child the infectious fever had come to an end when the eruption disappeared on the morning of the 23rd March, just as we often see in the course of a normal attack of measles. In a girl of 2 years, the exanthem appeared on 6th May with a temperature of 104·4°; in the evening the temperature was only 100·8°, next morning 99·5°, and the evening 100·9°—after which the fever did not return. In a girl of 4 years, the temperature rose, in spite of a diffuse rash, only to 100°; on the next day it was 100·4°, and then fell to the normal level. Probably such cases are commoner than we think, but are quite overlooked (especially among the poor) owing to the ephemeral character of the febrile symptoms and the slightness and short duration of the rash, and the fact that the child has had scarlet fever is only discovered afterwards from the subsequent nephritis, or from the traces of desquamation. We then often find that the parents have had no medical advice whatever, and have not even thought it necessary to keep the children in the house.

Least commonly of all it happens that the temperature after being very high (104° and over) for a few days, falls abruptly in the form of a crisis, and—in spite of the persistent prominence of the rash—the disease runs the rest of its course without any fever. Or the appearance of the eruption may be unaccompanied by fever, and the temperature only rise for the first time when the rash is fully developed.

Emma E., 2 years old, admitted into the hospital on April 25, 1878, with rickets. On the 29th, we noticed redness of the greater part of the skin, most marked on the scalp, back, and abdomen. Temp. 99·5°, pulse 96. Towards the evening the redness spread over the lower extremities, the temperature was 100·8°, pulse 160, and there was slight pharyngitis. During the next few days the temperature was remittent in type (as high as 103·1° in the evening). On the 4th May, general desquamation, followed by coryza and submaxillary adenitis.

The character of the exanthem, also, often presents variations which are perfectly compatible with an otherwise normal and favourable course of the disease. We have, for example, the

occurrence of the redness in large blotches instead of in the usual diffuse form, as has been already mentioned; or the appearance of miliary vesicles of a yellow or white colour rising abruptly from the reddened skin (scarlatina miliaris), and this may occur either locally (*e.g.*, round about the wrists), or may be visible almost all over the body. I have occasionally seen this miliary form occurring in all the members of a family, *e.g.*, in a mother and her 3 children (Dec. 1878). The miliary vesicles may become larger here and there like those of herpes, or may even assume the appearance of varicella or pemphigus; but in the latter form there are generally only single bullæ. Thus in a boy of 8 years, with a very copious eruption, I only saw a single bulla of the size of a shilling on the flexor surface of the right forearm. On the other hand, in a child of 10 months suffering from syphilitic condylomata, pretty numerous bullæ of various sizes appeared, during the very first days of scarlet fever, on the breast, back, and arms, causing gangrenous ulceration in some places (collapse and death on 5th day). In other cases dark-red nodules from the size of a lentil to that of a pea appear on the diffusely-reddened skin, and subside after a few days; or itching wheals like those in urticaria are seen here and there, and rapidly vanish again. All these forms are merely the products of aggravated dermatitis, and have in themselves no specially bad prognostic significance any more than have the small blood-extravasations (not bigger than a lentil) which are occasionally found as the result of the intense hyperæmia of the cutaneous capillaries. On the other hand it appears to me that a very irregular distribution of the rash (the so-called scarlatina variegata) is found mainly, if not exclusively, in cases which have an unfavourable course. In these cases some parts of the body are covered with a diffuse red rash, but on many others we find only patches of eruption separated from one another by areas either normal in colour or extremely pale. Should papular projections appear, as I have often observed, especially on the hands, forearms, and legs, an appearance may be produced which is apt to be mistaken for a confluent form of measles. These forms may also, in the further course of the disease, change and assume a more diffuse appearance. When the redness is very intense, we occasionally observe slight œdema of the feet, hands, and eyelids similar to

what we see in erysipelas, the skin seeming hard and tense, and becoming wrinkled when the rash fades. Only when the dermatitis is thus extreme in degree, do the little patients complain of itching of the skin; ordinarily, the rash gives rise to no discomfort.

Of greater significance for the prognosis than the variations in the appearance of the eruption is the persistence of the fever beyond the normal period—*i.e.*, after the disappearance of the rash. The redness of the skin certainly remains unusually long in some cases—*e.g.*, it may be visible for 8 or 9 days—and in these cases there is nothing striking in the persistence of the fever. Further, as I have already remarked (p. 197), an evening rise of temperature may linger as the last trace of the infectious fever for several days after the rash has disappeared. But wherever the fever, although only moderate in amount and with marked remissions, lasts after the rash has entirely disappeared—into the second week or longer—we have always reason to assume the presence of a complication or sequela, the nature of which cannot always be discovered at once. According to my experience, there are 3 morbid conditions which most commonly keep up the fever.

1. Persistence of the pharyngitis—which under these circumstances is always of the gangrenous and ulcerative character as I shall mention later on. This local process may last two or three weeks without greatly affecting the general health, and may continue to cause a markedly remittent temperature in the morning. Still, I have occasionally seen the pharyngitis persisting after the fever had entirely ceased.

2. The development of glandular and phlegmonous inflammation under the jaw—which is an extremely common complication even in mild cases of scarlet fever. During the first few days of the disease there is more or less swelling of the submaxillary lymphatic glands—most marked when the sore-throat has a gangrenous character. In many children these swellings entirely disappear later on, but in others they get very much larger during the second or third week and pass into a condition of diffuse hard infiltration of the connective tissue. This swelling may reach a considerable size owing to collateral œdema, and may extend upwards to the ear and downwards to the throat, the swellings on the two sides sometimes meeting

under the chin. Nearly all of the cases of phlegmonous inflammation end in suppuration; this generally takes place at the end of the second week or in the middle of the third, or even later. As long as this process lasts, the fever continues to be intermittent, with considerable evening exacerbations. When the pus is evacuated by incision the fever disappears rapidly (I have seen the temperature fall at once, after the incision, from 104° to 100°, and remain normal) or gradually; but it is often protracted by continued suppuration, or by the fact that the phlegmon on the other side requires a longer time to become ready for incision. In this way weeks may pass during which the children become seriously exhausted by the fever and suppuration.

These submaxillary abscesses—from which nearly all the children suffer in many epidemics—may also become the source of other dangers. If the incision is too long delayed, or the opening is not made sufficiently free, the pus may burrow downwards under the skin and in the connective tissue between the muscles of the throat, and may entirely undermine the whole side of the neck, reaching from the larynx down to the clavicle. Indeed, in a few cases we were obliged to make counter-openings much lower down, over the pectoralis major. At the same time a large portion of the undermined skin may become gangrenous, and at the bottom of the ulcer thus formed we see the cervical muscles exposed as if by dissection. In one case, as we found at the post-mortem, the pus burrowed as far as the apex of the right pleural cavity, which was surrounded by matter. In the following case perforation of the pharynx occurred.

Louise R., 1½ years old, admitted on February 6, 1877. Scarlet fever 14 days before; for the last 9 days phlegmonous inflammation of the left submaxillary region, the right affected during the last 4 days. Also nephritis and fever (ev. 102·9°). On incision of the swelling on the left side, a quantity of extremely offensive pus was discharged; similarly with the right side a few days later. This led us to infer that there was a communication between the abscess and the external air, in other words, that there was a perforation of the pharynx; and we found after death, which occurred on the 13th February, that this was actually the case.

In a girl of 3, perforation of the pharynx led to blood being swallowed and appearing in the fæces, and to liquids partially escaping from the wound.

Although recovery is by no means out of the question in such cases, still, even under the most favourable circumstances, suppuration lasting many weeks and accompanied by fever, must prove extremely exhausting to the little patients. Many die from marasmus or from complications, sometimes suddenly with exhausting bleeding from the abscess-wound owing to the jugular vein, more rarely the carotid artery, having been ulcerated into. Of this I have myself seen two examples. It is no less dangerous when the phlegmonous inflammation of the submaxillary connective tissue passes into a rapidly extending, diffuse, stone-hard infiltration of the whole region of the lower jaw as far as the thyroid cartilage, constituting the so-called angina Ludovici. I have seen two cases of this rigid infiltration (which deforms the face and renders the head almost immovable) on the fourth day of the disease. But it often does not appear until the second week of the disease, and is always accompanied by other threatening symptoms indicative of the malignant nature of the case—drowsiness, slight delirium, smallness and great rapidity of the pulse. There is extremely little tendency to suppuration in these cases, but there is very apt to be a gangrenous process resembling a carbuncle, and apparently due to the rigidity of the infiltration and to the obliteration of blood-vessels. In one of these cases a blackish slough was formed, in the middle of the second week, over the most prominent portion of the swelling on the right side. Two other children died from collapse before the skin had become gangrenous. On incision into the hard parts we found a hard laminated infiltration of the whole submaxillary region. Further, life may be endangered not only by this hard infiltration of the cervical connective-tissue, but also by the phlegmonous process previously mentioned, owing to its spreading inwards to the immediate neighbourhood of the larynx and setting up an inflammatory infiltration of the glottis, which ends fatally with symptoms of suffocation (vol. i., p. 369). I have occasionally seen well-marked œdema extending from the phlegmon into the subclavicular region, and even one arm becoming œdematous down to the hand, so that one could not help suspecting a thrombosis of the external or even internal jugular vein. In such cases I have seen thrombosis of these veins situated in the middle of the phlegmon, destruction of the thrombi, embolism, and death with

symptoms of septicæmia. You see, then, what serious complications may be produced by submaxillary phlegmon—which is one of the commonest complications of scarlet fever.

In rare cases the inflammation seems to spread from the pharynx to the muscles of the neck and throat. I have met with three cases in which these muscles were rigid and painful, causing difficulty of movement and wry-neck. Of these, two gradually recovered within a fortnight under the continuous use of hot fomentations and mercurial inunction; but the third ended in suppuration and had to be incised.

3. A third, and very common, cause of abnormal duration of the fever, is otitis. Although the scarlatinal inflammation of the skin may spread from the auricle into the external auditory meatus, giving rise to otitis externa with furuncular abscesses; still, this is much less common than otitis media, due to the inflammation spreading from the pharynx along the Eustachian tube. This form of otitis, which often affects both sides, is found in more than half of the cases in many epidemics, and often —especially in infants, who can only make known their pain by screaming—remains so latent that even an experienced physician is sometimes only led to examine the ear owing to there being no other apparent cause for the fever. Even older children do not always complain of difficulty of hearing or of pain in the ears; but pain is generally produced by pressure on the tragus or on the region behind the ear.

When a discharge of pus occurs from one or both ears, the pain ceases but the fever sometimes continues; and when we examine with the aural speculum we find perforation of the tympanum, but this need not cause us much anxiety. Fluid injected into the meatus often escapes by the mouth or nose. A large proportion of these perforations cicatrise under simple treatment within a few weeks, without leaving behind any noticeable defect of hearing. Much less commonly the inflammatory process is so acute that the child is quite deaf by the end of the second week, and the copious and offensive discharge becomes exceedingly disagreeable to everyone in the child's neighbourhood. In practice among the poor, such cases of otorrhœa are often very much neglected; but even with the best care the disease may spread from the tympanic cavity to the bone, and set up caries of the petrous bone. You will often meet with

children with the mastoid region swollen and presenting fistulous openings, with sequestra in the auditory meatus or behind it and with paralysis of the facial nerve (vol. i., p. 245), whose condition is to be referred to an attack of scarlet fever some years before. In a few cases I have even seen caries of the petrous bone (confirmed post-mortem) set in very rapidly, within 2—3 weeks and spread into the Fallopian canal—which was indicated by paralysis of the whole facial nerve.

> Child of 6 years, took ill with scarlet fever on June 25, 1884. Necrosis of the pharynx. Left otorrhœa on 8th July, with temperature always between 101·3° and 102·2°. On the 9th, the mastoid region was red, swollen, and painful. On the 10th, total paralysis of the left facial nerve; uvula inclined to the left. Collapse and death on the 15th. *P.-M.*—Left otitis media, caries of the petrous bone, implicating the Fallopian canal.

To this case I may add four others in which otitis and facial paralysis set in on the 13th, 17th, 18th, and in one case even on the 8th day after the eruption, and were still present at the time of the patient's discharge. As a result of this caries, and from the ulceration spreading into the petrosal sinus, hæmorrhages from the external ear have been known to occur and to end fatally. I have also several times seen an abscess form behind the auricle, between which and the otitis interna no certain connection could be traced, and which must assuredly be attributed to periostitis of the petrous bone.

> Max K., 11 years old, admitted with scarlet-fever on Dec. 3, 1873. By the 5th day the rash and sore-throat had gone, and the child felt well. Nevertheless the fever persisted for 3 weeks with temp. 101·3°—102·2°. On the 11th day of the disease there was pain and swelling behind the right ear, apparently due to periostitis of the mastoid process. Middle ear unaffected, hearing normal. An abscess formed in spite of the application of leeches. On the 28th day the pus was discharged by the external meatus. Rapid recovery.

Rupture into the external auditory meatus may occur from any abscess arising in the immediate neighbourhood, and that not only in scarlet fever but also in typhoid and in simple phlegmonous inflammations. In the cases which have come under my observation it has never caused any permanent injury, so long as one provided for good drainage; and this is best

ensured by making a counter-opening at the most dependent part of the abscess.

In addition to the causes named (pharyngitis, phlegmonous inflammation and otitis), which are often found existing together in the same case, the fever may also be kept up by various other complications, some of which are dangerous. We shall first consider affections of the serous membranes. We sometimes find at the post-mortem examination of cases of scarlet fever appearances of inflammation in the pericardium and pleura which we had never suspected during life, either because their course was latent or because they were masked by more prominent symptoms of a malignant nature—which we shall afterwards consider. But since inflammation of certain serous membranes does occur in cases of scarlet fever even after the disappearance of all malignant symptoms, it is the physician's duty to examine the respiratory and circulatory organs often and carefully whenever he finds the fever persisting, even should there be no subjective complaints which seem to call for examination. We are sometimes surprised to find the physical signs of endocarditis, the presence of which has only been indicated by a persistent high temperature. I have already given you a case of this kind which recovered (vol. i., p. 483), and the following is even more instructive, as the diagnosis was confirmed by post-mortem examination.[1]

Willy R., 10 years old, admitted on May 5, 1880, with diffuse scarlet fever eruption, moderately severe pharyngitis, high temperature (103·8°), delirium, drowsiness, frequent attacks of diarrhœa. Threatening collapse. After 5 days, unexpected improvement, although the temperature remained high. Hard infiltration of both submaxillary regions and pain in the left elbow-joint, and soon after in the knee- shoulder- and hip-joints, without swelling. Pulse never above 120. The phlegmon on the right side of the neck was incised, but without affecting the fever. On 20th May (the 17th day of the disease) the rate of the pulse increased to 144 and its tension diminished. Up to this date nothing abnormal was noticed about the heart. The remittent temperature, which still rose in the evening to nearly 104°, the persistent diarrhœa, the tenderness on pressure of the abdomen (which was somewhat distended), the apathy and weakness of the patient, and a certain amount of enlargement of the spleen, which could be made out on percussion, all served to arouse suspicion of typhoid following

[1] *Charité-Annalen*, vii., S. 649.

upon the scarlet fever. On the 24th May (the 20th day of the disease) the first sound of the heart was impure for the first time, but was not accompanied by a regular murmur. Notwithstanding this, however, I made the diagnosis of scarlatinal endocarditis (ice-bag to the cardiac region, salicylate of soda). During the next few days the cardiac impulse was visible from the 2nd to the 5th intercostal space, within the mammary line, and the dulness extended one finger-breadth beyond it. The "typhoid" symptoms gradually became more prominent, and on 28th May all the symptoms of typhoid fever were well-marked: high fever, delirium, drowsiness, blackened lips and teeth, diarrhœa, catarrh of the lower lobes in both lungs. On the 30th there appeared numerous little hæmorrhages (some like flea-bites, some as large as lentils) on the chest and abdomen, presently also on the face, eye-lids, and conjunctiva bulbi. The first sound of the heart was still impure, but there was never a distinct murmur. Collapse and death on 31st May.

*P.-M.*—Heart dilated, especially on the left side, with its walls slightly thickened. All three aortic valves destroyed, only some small warty prominences remaining, to which there adhered a quantity of clotted blood. Enormous quantities of bacteria in the valves and in the opaque tissue round them. Both tonsils converted into lax bags of pus. Hyperplasia of the cervical lymphatic glands. Spleen enlarged to thrice its normal size, bluish-red; the branches of the splenic artery almost all blocked by puriform emboli, which were in some places still solid. In the kidneys a few infarcts commencing to suppurate, cortical substance opaque. Slight enlargement and cloudy swelling of the liver.

In spite of the very extensive ulcerative destruction of the aortic valves, we did not find impurity of the first sound till the 20th day of the disease, and at no time a regular murmur. It is obvious that in such cases the endocarditis may be overlooked or else mistaken for typhoid fever. The typhoid symptoms and the high temperature seemed to be caused by the occurrence of embolisms (in this case we found these in the conjunctiva, spleen, liver and kidneys) and are therefore most strongly marked in cases—such as the above—in which there is bacterial ulceration. I have, however, seen this form[1] resulting from scarlet fever in children oftener than simple endocarditis; the latter is either recovered from in a few weeks or becomes the starting-point of chronic valvular disease. But we must be careful not to refer every systolic heart-murmur in scarlet fever forthwith to the presence of endocarditis, since such murmurs are often

[1] Litten, "Ueber septische Erkrankungen," *Zeitschr. f. klin. Med.*, Bd. ii., H. 2.

merely a symptom of the high fever and disappear along with it. I have often observed this, and in illustration I shall only mention the case of a child of 6 who was admitted on August 14th, 1883, with a temperature of 103·5° and a low systolic murmur, and whose heart-sounds became quite normal after a week.

Pericarditis occurs less commonly than inflammation of the endocardium; but pleurisy is commoner and is almost always accompanied by a purulent effusion. It is all the more necessary to bear these complications in mind, and consequently to examine very carefully, if the synovial membrane of the joints should be affected in the course of the fever—a pretty common complication which is more appropriately called scarlatinal synovitis than by the old name scarlatinal rheumatism. This affection, which sets in sometimes in the first but generally only in the second week of the disease, manifests its presence in the mildest form merely by pains in the joints without swelling and without any great interference with their movement. It sometimes only affects a few joints and sometimes several, especially those of the hands and feet; most rarely of all does it affect the hip- and sterno-clavicular joints.

In a girl of 7, these pains affected the joints of the right hand and only lasted one day (the 8th of the disease).—A girl of 12 was suddenly affected, on the 9th day, after the fever had ceased, by pains in the joints of both hands; on the following day, in the joints of the feet also. These lasted 2 days and were accompanied by fever (101·3°—102·9°).—In a boy of 10, there occurred on the 7th day acute pain in the joints of the hands, elbows, knees, and feet, without swelling or interference with movement (temperature 102·4°). Recovery on the use of quinine and warm baths (93°).—In a girl of 6 years, pains in both knee-joints began on 14th day of the disease without swelling. Evening temperature reached 104·7°. The fever gradually diminished and disappeared in about a week. Ice-bags applied constantly to the knee-joints.

In another series of cases we find swelling and difficulty in moving the joints, like that in acute rheumatism, in addition to the pain. This is almost always accompanied by a rise of temperature and usually, although not invariably, combined with other unfavourable symptoms—such as gangrenous inflammation of the mucous membrane of the mouth and pharynx, alarming collapse or inflammatory affections of other serous

membranes—the pleura, pericardium, endocardium and even the peritoneum. In these cases also, such inflammatory affections, especially of the pleura and cardiac membranes, may have such a latent course that their presence can only be excluded by local examination; and, when no such examination has been made, we may be very much surprised by finding after the acute stage is passed a pleuritic effusion or valvular affection, of the existence of which we had had no suspicion.[1]

Scarlatinal synovitis almost always ends favourably. It lasts from a few days to a week, after which the swelling of the joints, caused by effusion of fluid, subsides owing to its re-absorption. In rare cases the condition lasts longer, as, *e.g.*, in a case in which effusion into the knee-joint with floating patella persisted for weeks. I have also seen the hip- and elbow-joints and also those of the cervical vertebræ affected in a few cases. It is a much more serious matter in the cases where suppuration takes place inside the joint, but these are much rarer.[2] According to my experience this purulent synovitis in scarlet fever arises in two ways.

1. Least commonly it is due to ordinary synovitis passing on to suppuration, just as this also occasionally occurs in rheumatic polyarthritis. In these cases the suppuration is nearly always confined to one joint and it may become chronic. Thus many cases of suppurative inflammation of the hip or knee are to be attributed to an attack of scarlet fever occurring a considerable time before.

> Emil Sp., 10 years old, admitted on September 20, 1876. A year before, had scarlet fever, in the course of which there had been painful swelling of the right knee-joint and difficulty in walking. After several weeks, rupture had taken place, the pus being discharged from 2 openings—afterwards also little fragments of bone. On admission, suppurative inflammation of the knee-joint.

2. From embolism due to septicæmia. This form is distinguished by the gravity of the symptoms—the persistent high temperature, the advancing prostration, and the increasing inter-

[1] Depasse (*Revue mens.*, Sept. 1886, p. 403) in one case observed on the 23rd day of the disease acute hydrocele (without albuminuria) along with swelling of the joints of the hand.

[2] Rilliet and Barthez (*loc. cit.*, iii., p. 193) have never observed this termination—a remarkable fact considering their large material.—*Cf.* also Bokai, *Jahrb. f. Kinderheilk.*, xxiii., S. 305.

ference with the cerebral functions (ending at last in coma). It affects a whole series of joints and always ends fatally. The source of the septicæmia is generally found to be an extensive, usually bilateral, septic inflammation of the submaxillary connective-tissue (p. 202), or a gangrenous process in the pharynx. In one of these cases[1], the jugular vein, which was surrounded by the phlegmon in the region of the left lower jaw and throat, contained a thrombus for a considerable length. This process had resulted in pyemic fever with metastatic deposits in the pleura and kidneys, hæmorrhages in the skin, enlargement of the spleen and purulent synovitis in the joints of the hands, feet and elbows of both sides. In all cases of this kind I have found during life the affected joints swollen, very painful and immovable, the tissue over them being œdematous. In some instances this condition was only discovered 24 hours before death. We found, post-mortem, the cavities of the joints containing greenish-yellow creamy pus, the synovial membrane very opaque but otherwise unchanged. Both in the pus and in the synovial membrane we found the well-known septic micrococci. Löffler[2] injected these organisms into the circulation (making use of streptococci taken from the membrane of "scarlatinal diphtheria") and produced thereby multiple articular abscesses—thus rendering it extremely probable that these micro-organisms have a pathogenetic significance in connection with these conditions.

Occasionally in the course of scarlet fever periarticular abscesses occur which finally communicate with the cavity of the joint; but in these cases I have never been quite certain whether they have not been due from the very beginning to perforation by a previously existing suppuration inside the joint.

Max P., 5 years old, admitted on May 28, 1876, with nephritis 4 weeks after an attack of scarlet fever. Fluctuating abscesses on both feet near the ankles, which were incised (antiseptic dressings). During the next few days a large abscess formed over the left elbow-joint, and was opened on 6th June. On the 10th, a fresh abscess on the right foot, in which distinct crepitation could be made out. Similar crepitation appeared later (on 24th) in the left elbow-joint, and at the same time a fresh abscess appeared

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[1] *Charité-Annalen*, vii., 1882, S. 642.

[2] *Mittheil. aus dem Reichs-Gesundheitsamt*, ii.—Heubner, *Berl. klin. Wochenschr.*, 884, No. 44.—Schüller, Langenbeck's *Arch.*, xxxi. H. 2.

over the right elbow. The child was much weakened by the constant suppuration, the pain in the affected joints, and the hectic fever. On 27th July he was discharged at his parents' request after having made considerable progress under tonic treatment. Condition of the joints pretty much as before.

Franz M., 5 years old. Scarlet fever three weeks before. In the 2nd week, swelling of the upper part of the left humerus and rapid formation of an abscess. On admission, 3 fistulous openings in the upper third of the arm, into one of which a probe could be passed $3\frac{3}{4}$ inches into the shoulder-joint—the movements of which were limited to a certain extent. An abscess situated deeply in the right thigh and another on the right side of the neck. Nephritis, ending fatally.

In a child of 1 year, I saw abscesses arise round the right elbow and wrist 3 weeks after a scarlatinal eruption; they were incised, but, in spite of this, rupture took place into the elbow-joint. Death from pneumonia of the left lung.

I have no personal experience in regard to implication of the cerebral membranes by the scarlatinal process. The marked cerebral symptoms which are found in severe cases of this disease —and which we shall have to consider presently—do not, judging from my own observations, depend on meningitis. The most that we find is considerable hyperæmia or œdema of the pia mater and of the brain-substance, similar to what takes place under all sorts of other conditions. The symptoms are generally due to vascular engorgement resulting from the lowering of the heart's energy, which may also lead to thrombosis of some of the sinuses; but they are never due to regular inflammatory products. As to the observations published by other writers, especially those of Reimer[1] —I do not deny that meningitis may occur as the result of scarlet fever, but certainly its clinical symptoms would be hard to differentiate from those of "malignity."

On the other hand the mucous membrane of the bronchi and the parenchyma of the lung are more frequently affected in scarlet fever than is usually supposed. Not only catarrh, but also more or less extensive attacks of broncho-pneumonia, occur during the first and second weeks of the disease; but they are often overlooked owing to their being masked by a number of typhoid symptoms which engross the physician's attention. We have found bronchitis and broncho-pneu-

[1] *Jahrb. f. Kinderheilk.*, 1876, x.

monia in almost every severe case which we examined post-mortem; also frequently during life.[1] Croupous pneumonia I have not met with so often.

Hans K., 5 years old, admitted August 20, 1875, with eczema. Took ill with scarlet fever on 29th. During the whole of the first week, high fever (ev. 104·9°—106·9°) and gangrenous sore-throat. On 7th September, severe cough and dyspnœa; dulness and bronchial breathing over the left base behind, as high up as the spine of the scapula. On the 11th, sudden collapse, temp. 100°; pulse 166, thready; general cyanosis, cold extremities. Death in the evening. *P.-M.*—Hepatisation of the whole left lower lobe, and double fibrinous pleurisy.

Although complication with broncho-pneumonia or pleuro-pneumonia is always a serious matter, it is not necessarily fatal; for I have seen several cases recover. The prognosis depends on the concomitant symptoms and mainly on the condition of the heart. The strength of the heart, which is so very important in primary pneumonia, is still more so in the presence of a disease like scarlet fever, which tends to paralyse the heart's action. I may mention here the most important characteristic of this many-sided disease, both as regards the pathology and prognosis—what is called its "malignity." This "malignity" may express itself in a variety of forms, but on a comprehensive view of it we find two features to predominate—the great tendency of the disease to gangrenous inflammation, and the specific action of the virus on the heart.

1. Gangrenous Inflammation.—I prefer the term "gangrenous" to "diphtheritic" which has been hitherto in use, because to my mind nothing has done more to preclude a proper view of the nature of these processes than has that terminology. After Bretonneau[2] had described the specific infectious disease under the name of diphtheria in a clear and almost exhaustive manner, confusion was introduced by the pathological anatomists replacing this clinical conception by an anatomical one, and designating as "diphtheritic" all processes characterised by the deposit of a fibrinous exudation on the mucous membranes or even on the outer skin followed by gangrene. Thus it happened that physicians who were ready to follow Virchow's teaching on this matter referred to a compli-

[1] See the cases which I published in *Charité-Annalen*, Bd. iii., S. 539.

[2] *Traité de la diphthérie:* Paris, 1826.

cation with "diphtheria" conditions of the nature above-mentioned when they occurred in the most diverse diseases; and this misapprehension has become popularised. This was specially so in the case of scarlet fever, in which these processes are extremely common, especially in the pharynx. It is still, therefore, usual to speak of "scarlet fever with diphtheria" without meaning to imply that the specific disease which we call diphtheria is really present. The "gangrenous inflammation" (as I prefer to call it) is found in widely different diseases, most frequently in real diphtheria and in scarlet fever; also in small-pox, dysentery, pyæmia, and cholera—less commonly in typhoid and measles. The similarity of the pathological conditions does not, however, prove that the morbid processes are identical; just as the same kind of pustules may be caused equally well by variola, vaccinia, or the inunction of tartar emetic, and just as the anatomical basis of croup (the false membrane) may be occasioned equally well by strong ammonia, by the influence of heat or cold, or by true diphtheria—just so also may the exudation described by anatomists as "diphtheritic" be brought about by various causes such as those I have just described. Therefore we are not justified in talking of this condition uniformly as "diphtheria," and it is preferable to appropriate the term to the specific disease to which it has been applied since the time of Bretonneau. This view, which I have taught for many years now in my cliniques and have also published, is steadily gaining supporters (Demme, Heubner, E. Wagner, Lüttich[1]), and one strong argument in its favour is that this form of scarlet fever does not prevent the patient from being attacked by diphtheria soon after. Thus I have seen, among others, a boy of 2 years die of croup who had recovered from scarlet fever with severe gangrenous pharyngitis and become affected with diphtheria 4 weeks later. Cases of this kind—to which I shall return by-and-by—have been by no means uncommon in my hospital experience.

In scarlet fever this gangrenous inflammation attacks first of all the mucous membrane of the pharynx, which is already the seat of an inflammatory process. It is generally between the 3rd and 4th days of the disease (and sometimes even earlier) that we discover for the first time yellowish or greyish-white

[1] *Ueber Scharlachangina:* Leipzig, 1887.

patches of varying size on the tonsils (which are reddened and swollen), and especially on the sides opposed to one another. Sometimes only one tonsil is affected at first. Often similar streaks appear on the soft palate and uvula, or extend downwards from the tonsils towards the root of the tongue. The difficulty in swallowing is usually no greater than in ordinary scarlatinal sore-throat, varying mainly according to the degree of the inflammatory tension of the pharynx and its neighbourhood. This mildest form of gangrene of the pharynx need not occasion any immediate anxiety, for I have seen it in a large number of scarlet fever cases which presented no other variations of any importance from the normal course. After 5—6 days—often much later (in the 2nd or 3rd week)—the last remnants of the patches separate, and until this takes place they keep up a remittent fever (p. 202), but they may occasion no rise of temperature; they also leave behind shallow ulcers which readily bleed and which cicatrise after a short time. But the affection often developes to a more extreme degree, and then it is nearly always combined with other serious conditions and complications which may greatly endanger life. Not only the tonsils and the soft palate, but also the posterior wall of the pharynx and the hard palate become covered with the dreaded characteristic patches, tough adhesive mucus lies all over the mucous membrane and, when the mouth is open, stretches in thick threads between the tongue and the palate. There is a strong smell from the mouth and the swelling in the submaxillary region is more extensive and harder than usual. The process almost always spreads to the nasal cavity, and produces there that form of coryza which even the older physicians looked upon as very dangerous, and which differs essentially from the simple form of the same affection which occurs in mild cases of scarlet fever. The nostrils are excoriated, and an offensive serous discharge—often bloody—trickles over the upper lip, giving rise to irritation; in many cases the nose and its immediate neighbourhood as far as the eyelids becomes swollen and œdematous. Sometimes the conjunctiva also is affected, probably owing to the lachrymal ducts being implicated, either merely in the form of catarrhal inflammation with copious secretion and consequent gumming together of the lids, or, less commonly, from the occurrence of a deep gangrenous slough with great swelling of

the eyelids. In the very worst cases—which are fortunately rare—we may even have gangrenous perforation of the cornea with prolapse of the iris and complete destruction of the eye; I have several times seen this take place even as late as the 3rd week after the commencement of the fever.[1] Moreover, we are hardly ever able to convince ourselves, by inspection, of the extent of the gangrenous process in the nasal cavity, because it is generally situated far up and towards the back; examination with the speculum is often difficult and always unreliable. But I have often seen fragments of false membrane being discharged from the nose during several days, along with the ordinary secretion of coryza, and after this there could be no doubt as to the nature of the case. In a girl of 3 years who was suffering from scarlatinal nephritis these fragments of membrane continued to be discharged for weeks, the discharge occurring particularly when the nose was being syringed. They sometimes were so numerous as to cover the whole bottom of the glass, and some of them were in the form of regular casts of the posterior nares. The case was treated by nasal injections of a solution (1 per cent.) of sulphate of zinc, and recovered in the 6th week. Out of 6 cases of this kind only 2 recovered. In these cases also, repeated and even exhausting hæmorrhages took place from the ulcerated surfaces which had been left behind, both in the nasal cavity and in the pharynx after a gangrenous slough had separated. When the infiltration of the tissue of the tonsil and the corresponding gangrene penetrate very deeply, large pieces of the tonsil slough and separate, after hanging some time in the pharynx as blackish-brown offensive lumps. In several cases the soft palate became completely perforated on both sides by irregular holes which were generally situated above the tonsils. Necrosis of a portion of the hard palate (its rough surface being felt through the ulcer with a probe) is of rare occurrence.

All these symptoms, as we shall afterwards see, may also occur in real diphtheria in exactly the same way. In both diseases the condition consists of a fibrinous infiltration with a copious proliferation of nuclei and production of cells in the deeper layers of the mucous membrane, and, owing to this, the

[1] Suppuration of the eyeball may also occur as a result of the pyæmic processes mentioned on p. 210.

vessels are compressed and portions of the tissue become gangrenous; the bacteria found in the two diseases are identical.[1] All this, however, does not—as I have already said—prove that the two morbid processes are identical. Against such a view there is the further fact that forms of paralysis which might be designated "diphtheritic" do not occur after scarlatinal necrosis of the pharynx. I have never seen a case of paralysis of the accommodation of the eye, nor yet of the characteristic paralysis of the soft palate or of the muscles of the neck or limbs; for although fluids occasionally return through the nose in cases of scarlatinal pharyngitis, this is merely due to the inflammatory rigidity and immobility of the soft palate, and has nothing to do with the paralysis of the palate which occurs as a sequela of real diphtheria.

Another important difference consists in the fact that scarlatinal gangrene of the throat as distinguished from real diphtheria, has only a slight tendency to spread from the pharynx into the upper air-passages. Although one does well, in every case of primary pharyngeal diphtheria, to bear the danger of croup in mind, there is hardly any occasion to fear this in the case of scarlet fever. Hoarseness, which may even pass into aphonia, is certainly common enough, but this suspicious symptom gradually disappears in many cases, and seems to be due in general to catarrh having spread to the vocal cords; this catarrh may even, at a later period, spread further downwards and end in broncho-pneumonia. At the same time we must not be too sure. When Bretonneau says of scarlatinal pharyngitis, "elle n' a aucune tendance à se propager dans les canaux aérifères," he goes a great deal too far. I have elsewhere[2] published 8 cases of scarlet fever in which the pharyngeal affection undoubtedly did spread into the larynx, and in 7 of them this was confirmed post-mortem. In none of the latter, however, did the croup extend beyond the margins of the vocal cords. In the one case which did not come to a post-mortem, fragments of false membrane were discharged from the tracheotomy tube, indicating croup of the trachea or bronchi. The occurrence of croupy symptoms in scarlet fever

[1] The differences which Heubner has called attention to do not appear to me of sufficient importance to constitute an essential anatomical difference.

[2] *Charité-Annalen*, 1876, S. 529.

certainly renders the prognosis unfavourable, and according to my experience tracheotomy almost always fails. Moreover, the bronchial croup may be due in such cases merely to infectious particles having been inspired from the pharynx, and without the process necessarily extending continuously, through the upper air-passages. This idea seems to be favoured by the case of a boy of 3 in whom the gangrenous ulceration of the pharynx spread over the ary-epiglottidean ligaments and as far as the true vocal cords, but there came to an abrupt end, leaving the trachea unaffected. It was only in the bronchi, which were filled with muco-purulent secretion, that we found further fragments of membrane, and in some of the smaller ones even loosened casts.

I have also frequently met with similar cases of laryngeal croup at a later stage of scarlet fever. I may mention the following.

Emma H., 5 years old, admitted February 20, 1877, with fluor albus. On 2nd March, high temperature and simple pharyngitis. By the 5th, desquamation had begun on the face. During the next few days, the fever continued (100·4°—102·2°), and there was hoarseness, which on the 8th passed into aphonia and was accompanied by noisy inspiration. Dyspnœa set in on the 10th, and the temperature rose to 104·7°; ev. 105·3°. Emetics given, but had no effect. On the 12th, double broncho-pneumonia was discovered at the back. The temperature remained high, and finally collapse and death took place on 17th March, the 15th day of the disease. During the whole course of the case no membrane was ever visible on the pharynx; there was nothing but redness, swelling and increased mucous secretion. *P.-M.*—Diphtheritic pharyngitis, croupous laryngitis; double broncho-pneumonia. Left pleurisy with sero-fibrinous effusion. Enlargement of the spleen and mesenteric glands.

Helene Schw., 1½ years old, admitted February 12, 1877, with rickets. Scarlet fever eruption on the 14th, with sore-throat and small points of pus on the tonsils (temp. 103·8°—105·1°). During the next few days bronchial catarrh, moderate fever. On the 24th hoarseness, suspicious character of cough, rising temperature (102·9°). During the next 11 days, dyspnœa appeared and double broncho-pneumonia was discovered. Voice almost aphonic, slight obstructive noise with breathing. During the last 3 days, increasing collapse with an evening temperature of 104·4°. Death on the 7th. In this case also no membrane had ever been found on examination of the pharynx. *P.-M.*—Diphtheria of the pharynx and œsophagus. Croup of the larynx, double broncho-pneumonia. Caseous degeneration of the bronchial glands. Tubercle in the spleen and liver.

In neither of these cases was the gangrenous pharyngitis recognised during life, in spite of repeated examination—just as we not uncommonly find to be the case in primary diphtheria. The fact is to be explained partly by the concealed position of the patch, and partly by the impossibility of examining sufficiently all parts of the pharyngeal cavity in such children.

Martha H., 7 years old, admitted on March 20th, 1877, with scarlet fever, which had already lasted 5 days. Mental powers clouded, delirium; noisy breathing, submaxillary swelling on both sides. Rash now only partially visible. Pharynx reddened, swollen, covered with thick greyish-yellow patches and much mucus. Temp. 103·1°—104·4°. Pulse 144—168. On the 22nd, severe coryza and fœtor oris. Blackish-brown gangrenous spots on the uvula and arch of the palate. Grey membrane on the back of the pharynx; commencing collapse. On 23rd, coma and complete aphonia. Collapse and death. *P.-M.*—Pharyngitis et laryngitis diphtheritica ulcerosa. Diphtheritic gangrene of the œsophagus and of the pyloric region of the stomach. Multiple broncho-pneumonic patches. Slight parenchymatous nephritis.

Girl of 6 years, admitted April 2nd, 1884, with malignant scarlet fever. Temperature constantly 104° and over. Albuminuria, profuse diarrhœa. Drowsiness and delirium, extensive gangrene of the pharynx, otorrhœa and phlegmonous inflammation of the neck. In the end of the second week, symptoms of croup. On the 4th, tracheotomy, after which a piece of membrane was removed from the trachea. Collapse and death on the 19th. *P.-M.*—At the anterior end of the left vocal cord a deep somewhat triangular ulcer of the size of a pea, penetrating down to the cartilage. The larynx still very red and swollen. Trachea almost normal. Nephritis and parenchymatous myocarditis.

In these cases also, with the exception of the last, the gangrenous process was confined to the larynx, the trachea and bronchi being unaffected. Although, then, tracheal or bronchial croup does occur as a result of scarlet fever, the process does not extend beyond the larynx nearly so commonly as is the case in primary diphtheria. Add to this that—as we shall soon see—scarlet fever occasionally occurs along with true diphtheria, and when this is the case the occurrence of tracheal and bronchial croup is to be attributed to the latter and not to the scarlet fever.

From two of the above cases you will see that the gangrenous process in scarlet fever may affect the œsophagus, and even the mucous membrane of the stomach. I have only observed

the latter twice, but I have often seen fragments, or casts of fibrinous membrane, and specially longitudinal ulceration of the mucous membrane of the œsophagus, reaching almost to the cardiac end of the stomach. There is, however, no definite symptom during life which indicates the presence of these conditions, and even the implication of the larynx usually causes comparatively mild symptoms, which are not nearly so severe as those of real croup (hoarseness, aphonia, and noisy breathing). Indeed, in a few cases, the malignant symptoms of scarlet fever predominated so markedly that we entirely overlooked those of the laryngeal condition, and we were surprised to find at the post-mortem that the larynx was affected. Only once, in a girl of 7 years, was there marked tenderness on pressure over the larynx, as well as great hoarseness. These conditions seemed to depend on perichondritis, and gradually disappeared.

Dyspnœa, when it occurs in the course of gangrenous pharyngitis, by no means always depends on an affection of the larynx, even although it is accompanied by symptoms of obstruction. But it may also be caused by very great enlargement of the tonsils and neighbouring parts narrowing the isthmus of the fauces. A severe attack of coryza occurring simultaneously may, by contracting the nasal cavity, further aggravate the symptoms to a considerable extent, and these will attain their most extreme degree when there likewise exists a sero-purulent infiltration of the ary-epiglottidean ligaments (œdema glottidis). In all cases of this kind, however, it is absolutely impossible to attain to certainty as to the condition of the larynx; for, owing to the patient's state of mental stupor, the difficulty of opening the mouth, the extreme swelling of the tonsils, and the amount of gangrenous débris which covers everything—it is obviously impossible to use the laryngoscope with any success. In several cases of this kind in which the symptoms of laryngeal obstruction were very severe, we found at the post-mortem (in addition to the gangrenous pharyngitis and coryza) large abscesses of the tonsils and, in a few instances, peri- and retro-pharyngeal phlegmonous infiltrations situated laterally, or on the middle line—while the larynx was quite unaffected, apart from œdematous infiltration of the ary-epiglottidean ligaments. There can be no doubt whatever that in such a condition as this, tracheotomy is indicated. Still, I have

only once known it to give a successful result—in a boy of 8 years, in my private practice. All my other cases ended fatally owing to the accompanying malignant condition, in spite of tracheotomy or even of repeated incisions into the tonsillar abscesses.

The gangrenous pharyngitis is often accompanied by an analogous affection of the buccal mucous membrane (stomatitis scarlatinosa), in which the angles of the mouth, the lips, generally also the tongue, less commonly the hard palate, are infiltrated in isolated areas, or, more extensively, with greyish-yellow or greyish-white patches. This form of stomatitis may commence on the fifth day of the disease; but I have more frequently seen it begin in the second week or even later. The secretion of the saliva is increased, and the pain is often so great that the children cannot put out their tongues; they are unable to eat and the existing debility is thus further aggravated. Greyish-yellow patches are often seen extending from the bleeding fissures at the angles of the mouth and lips far over the mucous membrane of the mouth and tongue; and after they have become gangrenous and have separated, they leave behind them more or less deep ulcerations which produce notches, especially on the edge of the tongue. Even in cases where the ulcers were still superficial I have several times seen such severe hæmorrhage take place that the patients' lives were seriously endangered. Blood trickled in large quantity from the tongue and lips, especially when the child attempted to eat, and sometimes spontaneously; and whenever we tried to remove the thick clots from the lips, the bleeding started afresh. We were obliged to use constantly a solution of perchloride of iron, either applied on lint or painted on the tongue, in order to arrest the bleeding. But in many cases the affection is so slight that it presents the same symptoms and course as ordinary aphthous stomatitis (p. 1), while in other cases the dark-red mucous membrane of the tongue and gums is covered with a white croup-like membrane, which can be wiped off pretty easily, and leaves behind it shallow bleeding erosions. In this form of stomatitis I have often got excellent results from the use of a mouth-wash of chlorate of potash (2½ per cent.), but especially from the application several times a day of a solution of sulphate of zinc (grs. viii. to ℥i.) In one case only such marked cicatricial

contraction of the oral aperture resulted that it was reduced to a round opening the size of a hazel-nut—and had to be dilated by an operation. In one fatal case, the teeth were loosened by ulcerative stomatitis of the gum, and the lower jaw was in many places carious and stripped of its periosteum.

Fibrinous deposits may also be found covering the labia majora, the mucous membrane of the vulva, and also on any excoriation that may exist on the skin, such as eczema on the face, behind the ear, &c. In a child of 3 years with gangrenous sore-throat and rhinitis, I found the labia majora and minora swollen, bluish-red and covered with discoloured exudation during the second week of scarlet-fever; and a patch of eczema behind the ear became similarly affected soon afterwards. The constant application of a mixture of lead-lotion and carbolic lotion (2 per cent.) produced marked improvement in six days, but the child died afterwards of nephritis.

I have already said (p. 214) that the pharyngitis which occurs during the first days of scarlet-fever is usually of a simple inflammatory nature, and that the gangrenous character does not generally set in till the third or fourth day. But there are exceptions to this rule; for suspicious patches may occur in the pharynx at the very beginning and even before the appearance of the rash. In such cases the disease begins with a moderate amount of fever, sometimes indeed with a very high temperature (103·1°—104°) and "diphtheritic" sore-throat; and the scarlet-fever eruption does not appear on the skin till the second or third day, and in one case it was even five days later. Since the publication[1] of my former cases, I have had frequent opportunity of observing this unusual form of commencement.

Frida Th., 3 years old, admitted December 23, 1877. Had complained of pain in the throat for some days and had become feverish on the day before admission. A moderately large greyish-white patch on both tonsils, which were reddened and swollen. Enlargement of the submaxillary glands. Temp. 103·1°. Improvement during the next 2 days, the membrane separating completely. Temp. 101·1°. On January 1, 1878, violent fever began afresh (104·9°) with deep-redness of the pharynx. On the 3rd, a scarlatinal rash appeared on the chest. Death on the 6th, with symptoms of collapse.

Friedrich M., 7 years old, admitted December 28, 1878, with

[1] *Loc. cit.*, S. 525.

an extensive diphtheritic membrane on both tonsils, and on the arch of the palate, with swelling under the right angle of the jaw. Temp. 100·9°. During the next 2 days, the membrane disappeared entirely and the temperature fell (97·9°—99·3°). It rose again on the 31st (104·5°) accompanied by headache and sore-throat. The eruption appeared next day. Death from collapse on 6th January.

In both of these cases, you perceive, there was an interval of two days between the onset of the diphtheritic pharyngitis and the appearance of the prodromal rise of temperature of the scarlet fever—an interval during which the throat-affection and the fever were reduced to a minimum or quite disappeared. We have, therefore, to deal with the question as to whether the first affection was really connected with the second, or was not rather a true diphtheria which was rapidly followed by scarlet fever acquired in the hospital? For, as we shall soon see, the period of incubation of the latter disease is usually extremely short. This view is favoured by the fact that a boy belonging to the same family as the first child had died shortly before from diphtheria; and, further, that although I have had no case of this kind in private practice, several have occurred in the hospital—where infection with various contagia can hardly be prevented. I may also mention as an example of this the case of a boy of 6 years who was admitted on April 30, 1884, with diphtheria, took ill on the night of May 2nd with scarlet fever (which immediately assumed a malignant septic character) and died on the 6th. I have frequently seen patients—and especially children—on whom tracheotomy had been performed, take scarlet fever a few days afterwards. In such mixed cases we even find sequelæ, of which some have to be attributed to the diphtheria and some to the scarlet fever; *e.g.*, submaxillary abscesses and joint-affections, followed later by paralysis of the palate.[1] Now, if we exclude these mixed forms which arise from a combination of true diphtheria and scarlet fever, we get a confirmation of the fact that the pharyngitis occurring at the commencement of scarlet fever, although often very severe, is almost invariably simple, and only assumes a gangrenous character when the fever is at its height.

The malignant character of scarlet fever, however, consists

[1] See a case of this kind which I published in the *Berl. klin. Wochenschr.*, 1882, S. 599.

not only in the tendency to gangrenous processes which I have described, but depends even more on the specific action of the virus on the nervous centres and, through them, on the heart. Before discussing more fully this disastrous characteristic, let me draw your attention to certain symptoms which during the earlier days of the disease may give rise to greater anxiety than the circumstances really justify. At the very beginning, when the temperature is very high (104° and over) and only remits a little in the morning, and the rash is dark-red and diffuse—the patients often fall into a drowsy condition, from which, however, they can generally be easily aroused. Many of them are also more or less violently delirious and toss themselves about restlessly. Others are apathetic, do not answer when spoken to, and even seem hardly to recognise the people about them. This condition is not accompanied by any serious complications—the urine is free from albumen or only contains traces of it (as in any other extremely febrile diseases), the sore-throat is not particularly severe, and the pulse is of good quality and not very rapid. The presence of cerebral symptoms is the only point which arouses anxiety and makes us fear that the case may prove to be malignant. But when the temperature falls, on the 4th—6th day, the cerebral symptoms also disappear, the restlessness gives place to quiet sleep, consciousness soon returns, and the disease now continues its course in the ordinary way; but the incidents of that course, as already said, can never be predicted. Even in the case of a boy of 11 years, who became maniacal on the 5th day after a night of violent delirium, jumped out of bed repeatedly, ran to the window and screamed frantically, so that we had to tie his hands and feet—the administration of chloral (3 grains thrice daily) was sufficient to allay the symptoms, and recovery soon followed.

In this condition the apparently dangerous cerebral symptoms are merely caused by the continued high temperature; for in children analogous symptoms not unfrequently occur during the early days of other diseases which are characterised by continued high fever—*e.g.*, in croupous pneumonia. And I have in fact found antiphlogistic measures of great service in such cases, especially warm baths of 88°—90° F., in which the patient is to be kept about ten minutes. In many cases I have

even had the baths given twice a-day. An ice-bag was also kept constantly applied to the head, and—when there was severe pharyngitis—another was kept applied to the throat. I have also sometimes got good results from quinine (grs. viiss.—xv.), salicylate of soda (grs. xxxi.), and antipyrin (grs. iv.—viiss.), given in the afternoon, the temperature being reduced 2°—4° within 6—12 hours. On the other hand I have seen all these antipyretic remedies fail when the high temperature, delirium, and drowsiness indicated the approach of really malignant symptoms. In these cases, the temperature remained at the same level or even continued to rise, and I have therefore regarded the failure of antipyretic measures as justifying a bad prognosis, and indicating that the case was in reality one of the malignant form of scarlet fever due to the virulence of the disease. Of the essential nature of this virulence, however, we know practically nothing. In the same way, we are entirely ignorant of the reason why scarlet fever should be extremely mild in one series of cases, while in another series the course is malignant in a majority of the cases; and the "character of the epidemic," which people are so fond of talking about, really throws no light on the matter. In this connection, I have been much struck by observing (as many other physicians must have done) that if scarlet fever breaks out in a family and one of the children dies of a malignant form of it, it very often happens that one or two other children are carried off by the same symptoms, and in this way a whole family may be cut off. It seems probable that in such cases we really have the so-called mixed infection which has been proved to exist by the researches of A. Fränkel and Freudenberg already alluded to (p. 190).

Even in many simple cases of scarlet fever the bounding character (pulsus celer) and the extraordinary rapidity of the pulse (150 and more) give evidence of the action of the virus above mentioned, which I regard as consisting in a paralysis of the centre for the vagus. Although other infectious diseases—*e.g.* typhoid and especially diphtheria—sometimes present similar symptoms of collapse in very severe cases, still these are most frequent in scarlet fever and constitute the chief danger, which we must always be prepared for during the course of this treacherous disease. These cannot be due to the molecular changes in the cardiac muscle which are often discovered post-mortem; for

the symptoms of paralysis of the heart may set in at a period when we cannot believe that there is any extensive molecular degeneration. Every physician occasionally meets with cases in which vomiting, diarrhœa, convulsions, and extremely rapid and small pulse set in in the midst of perfect health, and collapse follows so rapidly that even within 8—12 hours the pulse disappears, the face and extremities become cold, and death occurs with comatose symptoms, and less commonly with more or less violent convulsions—without any rash ever having appeared. The diagnosis remains obscure till, in the course of a few days, one or more of the other children take scarlet fever, whereby all doubt is at once dispelled. But it is more usual even in very rapid cases for violent fever to set in and some rash to appear—although the latter may certainly be confined to certain parts and unequally distributed—and for death not to take place for 24—48 hours.

Child of 3 years, admitted June 21, 1879. Quite well on the preceding evening. Vomited twice during the night; in the morning, scarlet fever eruption on parts of the trunk; pharyngitis, coma, conjunctivitis. Temp. 104·9°; pulse 180, very small; resp. 72. In the afternoon, collapse, pulse scarcely perceptible. Death at 11 P.M. Duration 24 hours.

Another child of 2 years died just as quickly, but had convulsive movements towards the end. The temperature was constantly 104·4°—104·9°, there were small petechiæ on the chest and arms. After the disease had lasted 18 hours, collapse set in and the child died 6 hours later.—In a boy only 6 months old, whose temperature on the first day was 102·6°, ev. 104·4°, on the second day 105·4° and 106·5°—deep coma, collapse and death took place on the evening of the second day.—In a child of 2 years whose temperature was continually between 104° and 107·2° and whose pulse-rate was 180—192, death took place on the fourth evening.

This paralysing effect on the brain and heart is often noticed during the first days of the disease, most frequently in children under 3 years. In such cases we find that the pulse is extremely rapid (170 and more) from the beginning, and very compressible; the coldness of the hands, feet and nose is in striking contrast to the high temperature of the body (104°—105·8°), and the rash assumes a cyanotic livid tinge in consequence of the venous engorgement caused by the cardiac debility. The little patients are extremely collapsed, they are delirious, try to get out of bed, grind their teeth and may also have trismus and rigidity of the

limbs. Coma soon sets in and they die in this state within a few days, the pulse steadily diminishing in volume and increasing in frequency; but sometimes death is preceded by repeated epileptiform attacks. These "foudroyant" cases are invariably fatal. The most vaunted stimulants are absolutely powerless in the presence of the paralysing action of the poison.

The prognosis is somewhat more favourable when the symptoms of cardiac debility do not occur at the very beginning, but only after the complete development of the rash—within the first or second week of the disease. As these symptoms generally progress less rapidly in such cases, however, they are all the more likely to be overlooked in their earlier stages by an inexperienced practitioner, for a number of other morbid symptoms come to the front and distract his attention. The children are more or less drowsy, seem completely apathetic and toss themselves about restlessly. The eyes are closed or half-shut, the conjunctiva is generally congested and there is photophobia. The rash develops in various degrees, often occurring as scarlatina variegata (p. 201), or it may resemble measles or have a coppery-red tinge or the skin may be studded with little blood extravasations. The face is puffy, especially round the nose; and the fact that the nasal cavity has become affected by the gangrenous inflammation (p. 216) is indicated by the coryza, the snuffling and snoring, the bleeding and the repeated discharge of fragments of diphtheritic membrane from the nose. The tongue, lips and gums are dry and covered with brownish sordes as in typhoid fever; the pharynx, when its examination is possible, is almost always found to be "diphtheritic" and emits a very offensive odour. There may be, simultaneously, otitis and phlegmonous inflammation of the submaxillary regions; the urine may show traces of commencing nephritis, and there may even be a complication with inflammation of the respiratory organs or of the serous membranes, especially purulent synovitis, which I have already described to you (p. 210). This group of "malignant" symptoms, which is always pretty much the same in its essential features and is very characteristic, may last 8—10 days or more. In some cases there is comparative freedom from cerebral symptoms and the child answers the questions which are put to it, but with stammering and nasal intonation. This absence of cerebral symptoms, however, must not lead you to indulge in

rash expectations. During all this time and until death, the temperature remains 103·1°—104° and over. In a few cases, indeed, I found the temperature shortly before death 104·4°—108·9° although the pulse was scarcely perceptible and the extremities were already cold. In other cases, again, which appeared quite analogous, the temperature varied much and fell considerably during the last days, even as low as 97·7°. I may further mention more or less profuse diarrhœa as a symptom which is tolerably rare in simple scarlet fever, but is much commoner in malignant cases. It occasionally sets in at the very commencement of the disease, so suddenly and severely, that it induces a state of collapse resembling that of cholera. I have also in many cases observed more or less deep jaundice, but it was not always of very serious significance, as it was often caused either by catarrh of the duodenum or bile-ducts or by interstitial hepatitis (p. 115); but in severe cases its appearance may be a symptom of septicæmia. However grave and ominous the general aspect of these malignant cases may be, we must not give up hope so long as there are no signs of progressive collapse. Here is another case in which we have a striking proof of the prognostic value of the pulse. As long as the pulse does not exceed a certain rate (120 or 140 according to the age), and still retains almost its normal tension and fulness, we need not lose heart, however bad the other symptoms may be. Should the pulse become very small and compressible, dicrotic, and irregular in rhythm and force, but especially if it become extremely rapid (180 or even 200 and 240 as I once found in a boy of 4 years), and likewise should the skin of the extremities become cold, should any rash that remains become cyanotic and the coma more profound, finally should we observe grinding of the teeth and tremor of the hands and tongue—the prognosis is utterly hopeless. We must be prepared to have symptoms of collapse setting in in all malignant cases, even in those which are only moderately severe. They frequently come on quite suddenly and unexpectedly, belying the favourable prognosis given, perhaps, just the day before.

At the post-mortem of even the most malignant cases of scarlet fever we find nothing which can be looked upon as characteristic of this disease. In addition to the various complications which we can make out during life, and to which I

need not recur, we find those forms of albuminoid and fatty degeneration of the muscular fibres of the heart, of the liver-cells and the renal epithelium which are common to all severe infectious diseases. We also often find moderate swelling of many of the lymphatic glands, of Peyer's patches, of the solitary glands of the intestine and the mesenteric glands; and this condition is often found even where there has been no diarrhœa during life. In such cases there are no noticeable changes in the mucous membrane of the intestine.[1] In the mouth and pharynx, and also in the œsophagus, we often find thrush developing in addition to the gangrenous process. Not uncommonly also we find small multiple blood-extravasations in various organs, especially in the lungs. I have never noticed any characteristic change in the blood at any of the post-mortems which I have had an opportunity of making, and they have unfortunately been very numerous; and only in a small number of cases have I found any considerable enlargement of the spleen. I have already (p. 189) mentioned the occurrence of septic cocci in various organs, and their probable significance.

From the description of the disease, its numerous varieties and complications you will have seen that I was justified in speaking of scarlet fever as an affection the course and issue of which cannot be predicted, and one in which our prognosis must always, even in apparently most favourable cases, be given in very guarded terms. But as various sequelæ may set in after the acute process has quite passed off, and may constitute a fresh source of danger to the health and life of the patient after all risk seemed passed, I advise you to tell the parents at the very beginning of the disease that you can give no guarantee of recovery until the end of the fourth week. Among these sequelæ the most important is nephritis, which I have already (p. 137) spoken of in detail. Next to it, we have to fear the results of otitis media, its spreading to the bones, the implication of the sinuses and meninges or paralysis of the facial nerve and permanent deafness. In several cases I have observed during convalescence—generally in the third week—the occurrence of pneumonia; and in two of the cases it ended fatally.

[1] I have never as yet observed in children acute yellow atrophy of the liver or the dysenteric changes of the mucous membrane which Litten has described (*loc. cit.*, S. 120 and 128), although I have seen intestinal catarrh with swelling of the solitary glands and Peyer's patches.

I have also repeatedly, as already mentioned, seen children in the clinical wards who had just recovered from scarlet fever, take real diphtheria owing to direct infection and die from collapse or croup. On the other hand I have but rarely seen gangrene of the skin or mucous membranes. The gangrene in some cases took the form of bed-sores over the sacrum or on other parts of the body exposed to pressure. In one, there was necrosis of the cartilage of the nose; in another, gangrene of an inguinal bubo in the 3rd week of the disease, which ended fatally. I have also often seen a gangrenous phlegmon in the connective-tissue of the throat, but never noma of the mouth or female genitals, which other writers have observed. A number of cases have been published in which, during the first weeks of the disease, the patients have lost the point of their tongue from gangrene, or portions of the alveolar process. Very common sequelæ, also, are abscesses on the neck, back, hands and eye-lids and in the immediate neighbourhood of the joints, which by their repeated appearance and the copious suppuration from them, finally lead to marasmus. Occasionally also they burst into neighbouring joints (p. 211). Papular, eczematous and pustular eruptions especially on the face and ears, occur not uncommonly during the first weeks or months after an attack of scarlet fever. In one case I saw a bulbous eruption on the extremities on the 13th day. Only once have I observed an attack of acute pemphigus setting in with repeated extreme rises of temperature, and I am therefore uncertain whether it was not merely a chance complication. In a few cases fluor albus develops immediately after scarlet fever and is probably caused by spreading of the dermatitis to the mucous membrane of the genital organs (p. 185).

The nervous system, according to my experience, is less often affected than any other. I have only observed a completely ataxic gait in two children on their first getting out of bed after the attack. In the first of these it lasted a few days, and in the second some weeks. I have only twice seen chorea during the acute stage of scarlet fever. In both cases it was accompanied by pain in the joints (synovitis); I have never seen it as a sequela. Considering the very large number of choreic children whom I have had the opportunity of observing, this fact seems remarkable—for other authors (Gubler and Bouchut) say they have often seen chorea after scarlet fever. I may mention,

finally, the manifestation of a hæmorrhagic diathesis in the form of purpura, which I have frequently met as a sequela, but always hitherto with a favourable termination. Other authors, however, have described cases which proved rapidly fatal. I have notes of 8 cases of purpura after scarlet fever, all of which began on the 3rd or 4th week after the eruption; but in none of them had the scarlet fever varied in any important respect from the normal course. In 4 of the children, nothing was observed beyond blood-extravasations into the skin of various parts of the body, *e.g.*, in a girl of 3 years, there was a very large patch of purpura on each cheek, almost symmetrical in character, and others on the extensor surfaces of both fore-arms. In the other cases there also occurred, simultaneously, hæmorrhages from the mucous membranes, especially from the nose. In one of these children large blackish-red extravasations formed very rapidly on the right upper arm and on the right gluteal region, and over its surface there appeared several bullæ filled with bloody serum. Complete recovery took place within a fortnight, in spite of a violent epileptiform fit; and 4 other cases which were complicated with nephritis, also ended favourably.

Marie U., 6 years old, admitted November 23, 1874. Said to have been ill for 3 weeks. During the last 3 days, she had bled almost constantly from the nose, mouth, and external ears; she had almost no appetite, and was extremely wasted and anæmic. Pulse scarcely perceptible, extremities and face cold—general tremor. The skin of the extremities presented a few ecchymoses of various sizes, some of which coalesced, and there were a great number of them on the chest and abdomen. The right upper eyelid swollen from extravasation of blood. Slight œdema of the legs. Thoracic and abdominal organs normal. Urine contained a moderate amount of albumen, epithelial cells, and tube-casts. Motions black and bloody. Temperature 100·4°—101·1°. We were told that the child had had scarlet fever, and this was confirmed by the distinct remains of desquamation. Treatment.—Plugging of the right nostril to stop the hæmorrhage, injection of ergotin (grs. iss.). After the 25th, ergotin grs. iss. every 2 hours. On the very next day the fever was gone, the appetite improved, and a few loose but not bloody motions were passed. The ecchymoses gradually disappeared. No fresh hæmorrhages. Nephritis continued till 1st December, at which date the urine was normal and the œdema had passed off.

How the hæmorrhagic diathesis comes to arise after scarlet

fever, we do not know. Perhaps it is owing to the occurrence of molecular changes in the walls of the small vessels producing a greater liability to rupture. It is worthy of note that the fact of an attack of purpura having occurred shortly before does not give rise to any special tendency to this diathesis; for in a boy of 10 who was treated for purpura rheumatica in the hospital in the beginning of May 1875, and took scarlet fever on 16th May, no recurrence of the purpura took place after the fever.

Although not so commonly as in typhoid, still relapses do occur in scarlet fever. After the patient has been quite free from fever for several days or even weeks, and desquamation has begun in the normal way, the rash suddenly sets in afresh, either over the whole body or only on some parts of it, the temperature again runs up—and the disease goes through its course a second time. In such relapses the symptoms may be more severe than they were in the first attack. The reddening of the skin during the process of desquamation is very interesting, and has a quite peculiar appearance. Practitioners have given more and more attention to these relapses since the publication of the observations of Trojanowski, Thomas, and Körner[1]; and I have seen at least a dozen cases. I shall only mention the following.

Flora M., 12 years old, took simple scarlet fever 12 days previously. For the last 5 days had been cheerful and quite free from fever. Suddenly high fever set in again with slight delirium; there was also cough and rapid breathing. On December 27, 1876 (that is, 12 days after the first eruption) I found the resp. 52, stertor, and harsh breathing on both sides posteriorly and over the left side in front with numerous fine crepitations. Tongue dry. Marked desquamation over the whole body, along with a diffuse dark-red rash, which had not been noticed on the previous day. Pulse 144. During the next few days severe pharyngitis and conjunctivitis set in, and on the 30th while the bronchitic symptoms gradually improved and the fever diminished (101·3°) we found swelling of the submaxillary lymphatic glands, prominence of the lingual papillæ and both of the tonsils inflamed and covered with yellowish-white membrane. Temp. ev. 103·1°. On the 31st the rash faded, and by the next day it was entirely gone. The sore-throat and bronchitis also diminished, and by 6th January the fever had entirely disappeared. On the other hand otorrhœa and deafness

[1] *Jahrb. f. Kinderheilk.*, 1873, S. 417; *ibid.*, 1876.

set in, and lasted several weeks, as did also the desquamation. The treatment consisted of wet compresses to the thorax, and tartar emetic; later, infusion of ipecacuanha and chlorate of potash (grs. xv. to ℥i.) as a gargle.

A boy of 5 took ill with scarlet fever on 28th August. On 22nd September—that is, 25 days later—there was a fresh rise of temperature (101·3°), vomiting, general pale-red rash and sore-throat. On the 27th the rash and fever disappeared. Renewed desquamation.

Marie S., took ill with scarlet fever on October 11, 1878. Normal course. On the 13th day, sudden return of fever (103·1°) and diffuse red rash on the body and thighs; this faded after 24 hours, and then entirely disappeared. Temperature 101·7°, due to otitis and a few gangrenous patches which still remained in the pharynx. Complete recovery.

A boy of 2½ years (April, 1880) had a relapse 4 weeks after the first attack, which had been followed by purulent pleurisy on the left side. Recovery, after being twice punctured and aspirated.

Boy of 3 years. Eruption on February 26, 1883. Admitted 1st March. Free from fever 4th—6th March. On the 7th, a relapse. Temperature 104·4°. On the 12th, skin bright red. Pneumonia of the right lower lobe. Death on the 16th.

Girl of 4 years, admitted with scarlet fever April 20, 1883. Free from fever on the 24th. Desquamation on 2nd May. On the 9th, fresh rise of temperature (104·9°). On the 10th, the rash recurred, with sore-throat. Until the 13th, the temperature always varied between 104°—105·8°. Baths. On the 15th, both rash and fever had disappeared.

Boy of 3 years, took ill with scarlet fever on May 18, 1883. On the 20th, the rash had faded, but the temperature was still between 102·2° and 104·2° owing to gangrene of the tonsils and stomatitis. A little albumen in the urine. On the 26th, fresh rash, drowsiness, submaxillary phlegmon, œdema of the face, small quick pulse. Death, in a state of collapse, on the 28th.

Boy of 2 years. Eruption of scarlet fever on April 23, 1883; faded by 27th. No fever. On 29th, desquamation; on 30th, relapse. Temp. 102·7°. On 6th May, double broncho-pneumonia. Temp. 104·9°. Death on the 9th.

In these cases we cannot speak of a fresh infection, any more than we can do so in the relapses of typhoid fever; and I can only account for them by supposing that the scarlatinal virus had not been completely eliminated by the first attack, and that therefore a relapse was inevitable. If stricter attention were given to the subject it would probably be found that relapses are much commoner than is generally supposed. But you must not

expect to find in every instance such a typical series of symptoms as in our first case. We are more likely to find single symptoms, fever or rash, appearing so transiently that they are apt to be overlooked, especially in practice among the poor. On the other hand, we must take care not to mistake simple erythema or urticaria (which I have often seen following scarlet fever) for a relapse; because with the latter there is invariably renewed desquamation. In any case, we must not regard the relapse as at all less important than the first attack. Several cases, including some in my own practice, seem to show that a relapse may end fatally from pneumonia or with malignant symptoms even when the primary attack has had a perfectly normal course.[1]

The desquamation seems to me to be also of very great importance in determining the presence of the so-called scarlatina sine exanthemate, *i.e.*, scarlet fever in which there is no rash. That such cases do occur is beyond a doubt, although they are not very common. You often find several members of a family take ill of ordinary scarlet fever with a normal well-marked rash, while other members of the household—especially parents and servants, but sometimes also children (who may or may not have had scarlet fever)—are only affected by more or less severe pharyngitis with fever, unusually rapid pulse (Trousseau) and marked derangement of the general health, but have no rash at all. It follows, of course, that in these cases no desquamation takes place; for desquamation is only to be looked for where its anatomical cause—dermatitis—has previously existed. I have several times observed (in adults) nephritis occurring as a sequela of this scarlatina sine exanthemate, and this fact must dispel any doubt as to the nature of the cases. We learn from the following case that this variety may even sometimes be accompanied by articular pains.

In October, 1878, I was consulted about 2 children, the elder of whom was suffering from scarlatina variegata with gangrenous pharyngitis. The rash was still distinctly visible on the arms; on the face desquamation had already begun on some places. The younger boy had a constant high temperature (ev. almost 104°). He had a severe but simple sore-throat, but not a trace of eruption, although it was carefully examined for every day. On the 8th day, while the temperature remained high, very severe pains

[1] *Charité-Annalen*, vii., S. 661.

commenced in the hip-, knee-, elbow-, and ankle-joints, preventing all movement but not accompanied by any swelling. The joints were wrapped in cotton wadding. Recovery after a few days; no desquamation.

All stages of childhood are characterised by susceptibility to scarlet fever, but it is less marked in children under 2 years. Out of 312 cases, only 37 belonged to this period of life; but, on the other hand, it was amongst these that the rate of mortality was highest. The great majority of the children were between 3 and 8 years. Generally speaking, the number of persons who escape having scarlet fever is much greater than the number of those who escape measles—which often comes on in adult or even in advanced life in individuals who have not had it in childhood. But a great many people remain free from scarlet fever all their lives, even those who have often to expose themselves to its infection. We know nothing definite in regard to the mode of infection. It is an established fact that the infection is most readily taken by living for some time along with scarlet fever patients and breathing the air surrounding them. Probably, although not certainly, the virus may also be transmitted through the medium of clothing and other things, perhaps also by articles of food. In England, milk is especially blamed for carrying the infection of scarlet fever as well as that of typhoid and diphtheria. If this view is correct, the most careful supervision will not suffice to prevent the disease from spreading; and I recall with especial regret a visit which I made to a baker's child who was suffering from malignant scarlet fever. The sick-room was just next to the shop and communicated with it by a door which was always being opened and shut, or else left open, so that the bread could not but be impregnated by the infectious atmosphere. If infection can be carried as supposed, just imagine the consequences in a case like this!

The fact that any one with an open wound is especially susceptible to the contagion of scarlet fever—which is borne out by the well-known liability of lying-in women to this disease—I have had repeated opportunity of confirming in my wards.[1] Children with recent operation-wounds from phimosis, tracheotomy, ophthalmic operations, &c., often contract scarlet fever; and this generally takes place 4—7 days after the operation. The

[1] *Charité-Annalen*, i., S. 599.

observations of Hillier,[1] and Riedinger,[2] are substantially to the same effect. This also further confirms the well-known fact that scarlet fever has an extremely short period of incubation compared with other infectious diseases (measles, small-pox). Although it is often very difficult, even impossible, to determine exactly the precise moment of infection, yet a large number of observations which I have made both in private practice and hospital work go to prove that the period of incubation is not longer than 4 days, and sometimes only lasts 36—48 hours; Trousseau, Murchison, and others say that they have observed an even shorter duration (24—8 hours). The case given on page 190, in which varicella and scarlet fever existed simultaneously, and also the following one, may serve as examples of this rapid development.

> Boy of 10 years took scarlet fever December 5th, 1866. It could be shown that he had taken the infection from a schoolmate who was sitting next to him. Although his two younger sisters were at once isolated, one of them took ill of scarlet fever on the 8th, *i.e.*, after 3—4 days.

At what period of its course scarlet fever is least infectious we do not know. Meanwhile we must regard it as being infectious during its whole duration, from the commencement of the disease until the end of desquamation; and must isolate the patients accordingly. The possibility of infection being transmitted even during the stage of incubation, has determined me to lay down very strict rules (p. 193) with regard to school attendance.[3]

A second attack of scarlet fever in the same individual is certainly a thing of very rare occurrence if one takes into account the relapses mentioned above (p. 232). Personally I have only seen one indubitable case—in the child of a medical friend, who was re-infected one year after an undoubted attack of scarlet fever, owing to his brother having the disease, and this second attack presented the most typical symptoms and ended in profuse desquamation. In this matter also we have to guard against mistaking feverish erythemata—as we may readily do—for repeated scarlet fever eruptions.

[1] *Diseases of Children*: London, 1868, p. 289.

[2] *Centralbl. f. Chirurgie* 1880, S. 57. On the other hand, the experience of Treub (*ibid.*, No. 18) seems not to be in favour of wounds having this effect.

[3] *Cf.* also Uffelmann, *Handb. der privaten* [illegible] *ne des Kindes*: Leipzig, 1881, S. 395.

We now come, finally, to treatment. In cases with a normal course and without complications no medicines are required. The patient should be isolated from the other children, or, better still, the latter should be sent from home when the thing can be managed. Pure air and a moderate temperature (61°—63·5° F.) of the sick room are to be urgently enforced. You would hardly believe how deeply the old notion, that the children should be kept as warm as possible, is implanted in the popular mind. We must have the windows frequently opened, at least in the anteroom, or have them left constantly open (during the day at any rate); the patient should be lightly covered, and the room should be darkened only in the rare cases where there is photophobia. Cooling drinks (water with fruit-juice), pieces of orange, milk, thickened soups, and a little pigeon- or chicken-broth should form the diet during the days of fever. When there is constipation we should administer enemata or give a mild laxative every second day; for example, a teaspoonful of magnesia or an effervescent aperient, a wineglass-ful of mineral water, &c.

Should the fever remain persistently at a considerable height, and should the apparently malignant symptoms which we have already mentioned (p. 224)—drowsiness, restlessness, and delirium—set in, in consequence of it, we apply an ice-bag to the head and give a dose of quinine (grs. vii.—xvss.), antipyrin (grs. iiiss.—viiss.), or antifebrin (grs. iss.—ivss.) between 4 and 6 p.m.; or we may give a luke-warm bath (not under 88° F.). I do not approve of baths of a lower temperature, because in a disease like scarlet fever which in itself has a tendency to produce collapse from cardiac debility the low temperature is more liable than in any other disease to cause unexpected sinking. On the other hand, I can strongly recommend the sponging of the whole body every 2—3 hours with cold water and vinegar; and the children like it when they are very feverish. If you want to prescribe something, the best thing to give is hydrochloric acid (Form. 3).

Antipyretic treatment does not succeed, however, unless the symptoms are only apparently malignant, and are caused by the high temperature. In all really malignant cases, antipyretics, as I have already said, have no effect. I have never seen any successful result from the use of large doses of quinine, internally or subcutaneously; and I regard salicylate of soda, as

well as antipyrin and antifebrin, as remedies which are dangerous in such cases, and may favour the occurrence of collapse. Nor yet have cold baths and wet compresses any material influence on the temperature, which either remains quite unchanged under their use or only falls slightly for a very short time, while the pulse becomes even smaller and the general collapse more imminent.[1] On several occasions I have known collapse to occur while the child was in the bath, and in one case it even ended fatally. In this form of the disease the extreme elevation of temperature is evidently kept up by such a high degree of infection that no antipyretic can have any effect upon it; and on this degree of infection, according to my opinion, the whole result of the treatment depends. Here as in any other case of poisoning, the issue mainly depends upon the quantity of the poison taken. In all severe cases of scarlet fever, what the physician has principally to contend with is the paralysing effect of the virus on the nervous system of the heart. If the continuous use of strong stimulants suffices to keep the heart's action going until the organism has overcome the other serious results of the infection, we may still hope for a favourable issue; unless, indeed, other severe complications arise (pneumonia, pericarditis, endocarditis, pleurisy, &c.). Should the degree of the infection, however, be so great that the heart is either paralysed within the first 12—48 hours (p. 226), or should coma, delirium, great rapidity and smallness of the pulse, coldness of the extremities and cyanotic discolouration of the skin occur and steadily increase, the resources of the stimulant line of treatment are just as inefficient as all the "disinfecting" and "germicidal" remedies—which I have never yet seen do any good at all. I have experimented, in a large number of serious cases, with quinine, carbolic and salicylic acids, benzoate and hyposulphite of soda; but the results were very discouraging. The hyposulphite of soda, moreover, caused repeated diarrhœa, and had to be given up. I have therefore abandoned all these remedies and

[1] We may compare the following chart, which I select from among a number of similar ones :—

| | | | | | |
|---|---|---|---|---|---|
| On the 5th day of the disease | | | 11 | A.M. | Temp. 104·7 |
| ,, | ,, | ,, | 11·30 | ,, | 105·8 Bath 83·8 |
| ,, | ,, | ,, | 12 | ,, | 106· |
| ,, 6th | ,, | ,, | 10 | ,, | 105·1 Bath 81·5 |
| ,, | ,, | ,, | 12 | | |
| ,, | ,, | ,, | 6 | | &c. |

now confine myself to the use of stimulants, which have at least a palliating effect in so far as they revive the failing heart's action.

Among stimulants I should give the first place to alcohol (wine, cognac), coffee in large doses, and camphor. The latter, according to my experience, is to be preferred in those cases to the much-praised musk—which, however, in large doses is always worth trying. I have seen exceedingly good results from the continuous use of these remedies in a number of serious cases, in which however the symptoms of collapse had not yet reached an extreme degree. For an account of these cases I must refer you to a former paper.[1] Since then, the number of my cases has considerably increased, and in a few of them recovery took place in spite of the enormous pulse-rate of 180 and over.

Wine (tokay, sherry, champagne) should be given every hour, 1—2 dessert-spoonfuls each time; strong coffee, about half a cupful several times a-day; camphor, grs. i—iii; musk, grs. ½—iii, according to age, every two hours. Where the swallowing is rendered difficult by great swelling of the pharynx, one may give twice daily a nutritive enema of peptone, or of a small cupful of beef-tea with the addition of the yolk of an egg and a spoonful of wine. You may also give, every three hours, a hypodermic injection of sulphuric ether (♏. xv) or of camphor, either in the form of camphorated oil or (better) as a solution of camphor (1 part) in rectified spirits and distilled water (5 parts of each). These injections sometimes occasion small yellowish infiltrations surrounded by a reddened margin, which afterwards become gangrenous and separate with suppuration. I attach very little value to the much-praised carbonate of ammonia, or to valerian; for I consider both these remedies too weak to be able of themselves to maintain the sinking cardiac energy. Warm baths (93°—95° F.) with cold effusions to the neck and back are of more use; but their action must be carefully watched, for the effusions seem sometimes to induce symptoms of collapse, and then strong stimulants are required in order to restore the temperature.

Should the malignant form of scarlet fever be unaccompanied by any serious symptoms of cardiac debility (as it may be during

[1] *Charité-Annalen*, Bd. iii., S. 561.

many days), I would recommend the steady use of decoction of cinchona (1 in 20 to 1 in 12) with liquor chlori, and if the pulse is falling one may give tincture of valerian (m. xlviii—lxxii). As to the disinfection of the mouth, pharynx and nose: you should order syringing every 2—3 hours with a solution of carbolic acid (2 per cent.) or permanganate of potash (2½ per cent.). I have also got good results from syringing the nose with a solution of sulphate of zinc (1 per cent.) and from painting it with a solution of chloride of zinc (5 per cent.).

The various complications connected with the ears, the respiratory organs, and the serous membranes, must be treated according to their nature. When there is synovitis, the painful or swollen joints should be wrapped in cotton-wadding. The attempt to disperse the phlegmonous swelling in the submaxillary region by painting it with tincture of iodine is nearly always unsuccessful, and this treatment is more likely to favour the occurrence of rupture owing to the irritation of the skin which it produces. Warm poultices, when there is fluctuation, free incision and antiseptic dressing, and when the suppuration is burrowing deeply, regular washing-out and drainage—constitute the main resources of treatment.

During convalescence whenever desquamation begins, you should order regular luke-warm baths. The inunction of the whole body with lard, which was formerly recommended and is still practised by many, I have for my part entirely given up, as I should not venture while using them to send a child out of doors before the 4th or 5th week.

## II. *Measles.*

Although measles is by no means always a mild disease,[1] it is nevertheless considerably less serious in its symptoms and complications than scarlet fever. Above all there is not the same impossibility of predicting the course as in scarlet fever; nor do we find serious symptoms appearing so suddenly and treacherously as we do in even apparently slight cases of the latter disease. An experienced practitioner is always far better prepared for any unfavourable symptom that may occur in the

[1] According to the mortality statistics of 1887, there was in Berlin a death-rate from measles of 0·74—almost as much as that from scarlet fever (0·85).

course of measles, and can give a prognosis with greater certainty than in scarlet fever, for you will recollect that in the latter a sure prognosis cannot be given within the first 4 weeks.

From certain observations made in localities which had remained unvisited by measles during a great number of years (especially those of Panum in the Faroe Islands) we know that the period of incubation—*i.e.*, the time between infection and the onset of the first symptoms—is about 9 days; and that from the infection to the commencement of the eruption is 13—14 days. Owing to this prolonged incubation as compared with that of scarlet fever (p. 236) it may happen that in families with many children, where one child is infected by the other, several months may elapse before they have all had the disease, for every member of the family is nearly always affected, whether the patients be isolated or not. And this fact seems to indicate the great volatility of the contagium, and its diffusion through all the rooms. Thus it comes that only comparatively few people get through their childhood without having measles, and even those who do escape it at this period of life, with a few exceptions, have it sooner or later in after life. On the other hand, a much greater number of individuals escape having scarlet fever altogether.

The period of incubation is nearly always free from any symptoms. I have but rarely been able to confirm the observations of Thomas, Rehn and others, according to which transient rises of temperature (101·8°—102·2°) may occur even at this time. In such cases it would certainly be hard to say whether the transient rise of temperature was really due to the measles or to some other, unknown, cause.

Child of 2 years, admitted with ozæna on March 5, 1873. Suddenly on the 12th, fever (100·8°), some cough, and diarrhœa. On the 14th, again free from fever. On the 28th, the initial rise of temperature first appeared. Eruption of measles on 1st April.

Child of 4 years, admitted with hip-joint disease. On 5th April, general feeling of malaise; temp. 100·2°; on the following evening 102·9°. After that, the child was free from fever and quite cheerful till the 15th—that is, for 10 days—at which date the initial fever began.

The commencement of the prodromal stage is indicated in most children by a general feeling of malaise, irritability, loss of appetite, and slight catarrhal symptoms. The eyelids

are a little reddened, and are somewhat swollen. The eyes dim and somewhat moist. It is often accompanied by frequent sneezing, attacks of epistaxis, and a short dry cough. Sometimes the patients also complain of pain on swallowing, and on examining the pharynx we find slight inflammation of the tonsils. These catarrhal symptoms—which are sufficient, during an epidemic of measles, to announce the impending onset of the disease—may indeed be so trifling that the children's health seems scarcely disturbed, and it is only the thermometer that reveals the approaching danger. We almost invariably find that the temperature is more or less raised; sometimes it is only 100°—100·4° in the evening, and in other cases, even in the morning (and especially on the first day), it is 100·4°—102·2°. It always varies greatly, however, *e.g.*, on the 2nd day it may again be quite normal, or nearly so, and may again rise on the 3rd day. Under these circumstances you must never neglect to examine the pharynx, even although the children do not complain of pain on swallowing. In most cases, after the second day, you observe a rash on the hard and soft palate, especially in children who are strong and plethoric. This rash is either diffuse, with dark spots scattered over it; or, less commonly, the mucous membrane as a whole is still pale and presents more or less numerous punctiform and stellate red patches. The former of these conditions almost always precedes the eruption of measles. The duration of the prodromal stage is, on the average, 3 days (much less commonly 4—6 days), although one is not always able to account for these variations from the normal type. Take for example the following temperature-charts.

Child of 1½ years.

| | M. | EV. | |
|---|---|---|---|
| 2nd Dec. | — | 103·3 | Conjunctivitis. |
| 3rd ,, | 100·6 | 102·9 | Cough. |
| 4th ,, | 102·9 | 103·1 | |
| 5th ,, | 100·9 | 103·6 | Eruption. |

Child of 3 years.

| | M. | EV. | |
|---|---|---|---|
| 28th March | — | 102·7 | |
| 29th ,, | 99·7 | 101·1 | Catarrh. |
| 30th ,, | 102·6 | 102·6 | Conjunctivitis. |
| 31st ,, | 101·5 | 103·6 | |
| 1st April | 103·6 | 103·6 | |
| 2nd ,, | 102·4 | 104· | Eruption. |

Child of 4 years.

| | M. | EV. | |
|---|---|---|---|
| 16th Aug. | — | 102·7 | Sore-throat. |
| 17th ,, | 100·9 | 100·8 | |
| 18th ,, | 99·9 | 100·9 | Felt well. |
| 19th ,, | 100·6 | 102·9 | Cough. |
| 20th ,, | 101·1 | 103·3 | Eruption. |

Child of 3½ years.

| | M. | EV. | |
|---|---|---|---|
| 15th Nov. | 99·7 | 100·6 | Catarrh. |
| 16th ,, | 102·7 | 103·3 | Worse. |
| 17th ,, | 101·8 | 104·7 | |
| 18th ,, | 101·3 | 103·3 | |
| 19th ,, | 104·5 | 104·4 | Pneumonia. |
| 20th ,, | 103·1 | 104·4 | Eruption. |

Only in the last of these four cases did the delayed eruption of measles coincide with an attack of pneumonia which developed during the prodromal stage. But cases of this kind are common enough, and we cannot but suppose that the occurrence of such a serious complication at such an early stage retards the appearance of the rash; the laity, however, are wont to talk in such cases of the rash "receding," or "striking inwards." In delicate or sick children the prodromal stage is apt to be somewhat prolonged. The skin over the surface of the body generally presents no morbid symptoms during the prodromal stage; sometimes, however, one does notice even at this stage minute pale-red papules and—in very rare cases—also a transient erythema.

A child of 2 years took ill suddenly on March 1, 1877, with fever and cough. Temp. 101·1°; ev. 104°. On the face and chest, a diffuse bright-red rash. On the 2nd, this had disappeared, but there was broncho-pneumonia (especially in the left lower lobe) and severe diarrhœa. During the night of the 3rd, eruption of measles.

The commencement of the eruption (except in very sickly children suffering from chronic disease) is always indicated by a considerable increase of the fever. The temperature rises rapidly to 103°—105°, and while the rash is appearing there is great restlessness and an almost incessant short cough. The eruption begins first on the face, generally on the temples, in front of the ear and on the chin, and appears in the form of small bright-red spots (papules) from the size of a pin-head to that of a lentil-seed, and very flat. This rash spreads very rapidly over the whole face, throat, chest, and further down, so that usually the whole body, right down to the feet, is covered in the course of 24 hours. The upper part of the body is then more thickly covered than the lower extremities, on which discrete points are still to be seen. The eruption is not, generally, fully developed until the following day. The papules, which are small at first and evidently arise round the roots of the hairs, increase in size and redness during this stage, owing to their being surrounded by a hyperæmic area (roseola); and when the eruption is completely developed they form roundish or crescentic patches with irregular borders, and vary from the size of a pea to that of a bean. These disappear momentarily on being pressed, and one

aid to diagnosis. The reappearance of the fever, after the temperature has been normal for several days, or its persistence after the rash is fully out, or even after it has faded, ought to lead you to make a thorough examination of the chest at once, even although neither the breathing nor the cough presents as yet any threatening features. Even should you find nothing but the dry or moist râles of bronchial catarrh, these should be sufficient to make you cautious in your prognosis, for even within 24—36 hours, dyspnœa, "grunting" expiration, noisy breathing and the other symptoms of broncho-pneumonia may become fully developed. We learn from experience that the inflammation of the lungs is most serious when it sets in during the stage of subsidence, and that this is by far the commonest immediate cause of death from measles. The younger the children are, the more severe does the attack of broncho-pneumonia generally prove. It is especially threatening in young infants, and in several cases I have seen the attack ushered in by a number of violent epileptiform convulsions, which sometimes went on for 24 hours. But even in older children—especially in those who have previously suffered from bronchial catarrh, or even from pulmonary tuberculosis—a very guarded prognosis must be given.

The fever may also be kept up beyond the normal time by complications connected with the mucous membrane of the alimentary tract. Occasionally I have seen an attack of tonsillar sore-throat, lasting into the 2nd week of measles or developing then for the first time, and in these cases the high temperature (up to 104°), although it was only transient, and the greyish-yellow purulent spots on the tonsils gave rise to a suspicion of diphtheria. On the tongue, sometimes also on other parts of the mucous membrane of the mouth, we find occasionally—more frequently in some epidemics than in others—a form of stomatitis quite similar to that described in connection with scarlet fever (p. 221), and this may prove very serious owing to the pain and consequent interference with eating which it causes.

Marie St., 1¼ years old, admitted May 7, 1876, with measles in the stage of subsidence, the rash being still visible as yellowish-grey pigmented spots. Slight bronchial catarrh. Temp. 103·1°; pulse 160; resp. 40. Mucous [illegible] of the lips, cheeks, and

tongue much swollen, red, apt to bleed on being touched, with yellowish-grey patches in streaks; profuse salivation. During the next few days these symptoms continued, with very high temperature (up to 104·5°), great restlessness, sleeplessness, diarrhœa. Improvement after the 10th. The fever became remittent, and under painting with sulphate of zinc (p. 221), the mucous membrane of the mouth gradually returned to its normal condition. On the 25th, all fever gone. Diarrhœa successfully treated by subnitrate of bismuth (grs. iii. every 2 hours). Recovery.

Diarrhœa forms a much commoner complication, and may set in during the very first days of the attack. In many epidemics it constitutes an almost constant symptom, and in the stage of subsidence it is often accompanied by very severe bronchial catarrh or broncho-pneumonia. This diarrhœa, owing to its frequent severity, is by no means a trivial matter. The motions are sometimes very profuse, occurring 12—20 times a day, and often accompanied by violent colic, and—owing to the tenesmus and presence of blood—are apt to assume a dysenteric character, which may end in fatal collapse. As a matter of fact, we find on post-mortem examination of such cases more or less severe acute catarrh of the colon, with swelling or even ulceration of the follicles, and also perhaps enlargement of the Peyer's patches and mesenteric glands. Now, although many cases do perfectly well notwithstanding a moderate amount of diarrhœa, still we must not forget the tendency to intestinal catarrh in measles, and we must be particularly careful about using purgatives. When, therefore, there is constipation, we should order either enemata or mild laxatives (pulv. glycy. co., ol. ricini) in small doses.

After measles—as after scarlet fever—we may have otitis media, rupture of the tympanum, and offensive otorrhœa. These may keep up the temperature for a considerable time, and persist for months and years after all the other symptoms have disappeared. If the temperature continues high after measles without any discoverable cause, we should always institute a thorough examination of the ears. Serious diseases of the ears, deafness, and caries of the petrous bone are often to be referred to a former otitis morbillosa which has been neglected, as so often happens with scarlatinal otitis (p. 205). The latter, however, is commoner, for its proximate cause—the pharyngitis—is always present in scarlet fever, and is rather uncommon in

measles. In a similar way we may explain the fact that the submaxillary glandular swellings and abscesses, which are found mainly in the 2nd week of measles, are far less common in that disease than in scarlet fever.

Among the infectious diseases, whooping cough is the one which is most frequently combined with measles (vol. i., p. 465), both in epidemics and in single cases. Usually, the whooping cough has already lasted for some weeks, and the addition of measles is then always a serious matter, because the tendency to broncho-pneumonia, common to both diseases, is materially increased by this combination. Although cases of this kind often end favourably, still, the prognosis is always doubtful, especially if broncho-pneumonia, due to the whooping cough, has already set in and the measles appears as an additional complication. In cases of this kind I have often found the rash either very scanty, entirely absent from some part of the body, or of a cyanotic tinge from the beginning. Meanwhile, the previously existing dypsnœa was very much increased, the pneumonic accompaniments extended over the whole back, and even over the front of the thorax, and the pulse became steadily smaller and more rapid. The children almost invariably became extremely cyanotic and died from collapse within 36—40 hours. This rapid fatal termination from extensive broncho-pneumonia and cardiac paralysis is to be dreaded in all children who have been suffering for any length of time from exhausting diseases —chronic pneumonia, diarrhœa, tuberculosis &c.—and acquire measles in addition. In such cases the measles is a "terminal" disease in the true sense of the word, and the condition of the temperature generally differs considerably from the normal course. In many cases of this kind which I have observed in the hospital there was no high fever accompanying the eruption, but the scanty spots made their appearance without there being any further elevation of the temperature, already somewhat high (100·4°—102·2°). Even in cases where the primary disease (*e.g.*, chronic intestinal catarrh with ulceration) was entirely unaccompanied by fever, the onset of this "terminal" attack of measles was, in the case of very debilitated children, only accompanied by a slight rise of temperature in the evening, while it remained almost normal in the morning.

Child of 9 months, very weak and debilitated—

| | M. | EV. | |
|---|---|---|---|
| 17 Dec. | ..... | ..... | 101·1 Catarrh. |
| 18 „ | ..... 99·5 | ..... | 97·8 Resp. 60. |
| 19 „ | ..... 96·3 | ..... | 103·5 Eruption of measles. |
| 20 „ | ..... 100·2 | ..... | 103·8 |
| 21 „ | ..... 98·4 | ..... | 102·6 Collapse and pneumonia. |
| 22 „ | ..... 100·9 | ..... | 101·8 Death. |

Under certain circumstances, diphtheria also occurs as a complication of measles—real diphtheria, not merely the gangrenous inflammation of the pharynx which we have become acquainted with as a common accompaniment of scarlet fever. I have scarcely observed a single case of this complication in private practice, but I have frequently met with it in the hospital, where complete isolation of the different infectious diseases from one another was not practicable until 1885. It is easy to understand how, after one or two of the children suffering from measles had become affected with diphtheria, the other measles patients in the same ward would be readily infected, so that one could not, perhaps, from the fact of a large number of cases occurring together, legitimately infer that the cases belonged to a special variety of the disease—a kind of "genus diphthericus." It has occasionally happened that children who were admitted with an attack of pharyngeal diphtheria were infected with measles in the hospital; but the converse occurred much more frequently. The diphtheria generally developes in the course of the second week of an attack. Sometimes it occurs at first on the conjunctiva, and it is but rarely confined to the pharynx. The majority of cases end fatally, owing to the disease spreading to the larynx and bronchi; tracheotomy only succeeds in extremely rare cases. In a few cases I have certainly seen diphtheria and the resulting croup set in at an earlier stage, *e.g.* on the 4th day. Or they even came on simultaneously with the appearance of the measles-rash, so that tracheotomy had to be performed on the very first day of the eruption. We found at the post-mortem that they were cases of real diphtheria and not merely of simple inflammatory croup. As, however, these children had already been some weeks in the hospital on account of rickets, caries, &c., we may assume that the diphtheritic infection had taken place soon after that of the measles; so that it thus happened that the two diseases set in simultaneously.

Cases have been published by several authors (Klüpfel,[1] Steiner,[2] Löschner,[3]) in which measles was complicated by acute pemphigus. I have observed one case of this kind myself.

Girl of 4 years. In October, 1881, an attack of measles with normal course during the first 2 days, but with persisting fever. On the 3rd day, an eruption of bullæ over the whole body, which varied from the size of a hazel-nut to that of a half-crown. On the 4th day, each cheek covered by a single bulla, and the dorsum of each hand in a similar condition. The bullæ contained yellowish serum. In some places they lay close together, and between them there was a dark hæmorrhagic measles-rash, which was partly confluent. The bullæ were situated partly on the site of the eruption, but partly on the unaffected areas of skin. The eyelids were much swollen, likewise the lips and cheeks, so that the mouth could not be opened for examination. Temp. m. 100°; ev. 101·3°. Pulse small; collapse threatening, as in cases of extensive burns —to which this case had a considerable general resemblance. Between the 6th and 7th day the fever rose (104·9°). Pneumonia of the right lower lobe. Death on the 8th day. Post-mortem not allowed.

I have elsewhere[4] stated the reasons which made me assume that in this case and the similar ones published by others there was complication with acute pemphigus. In a few of these cases, many of the bullæ appeared before the measles-rash, and they recurred after it had faded; in one case this went on for 13 days. Still, it is worthy of note that this eruption was observed several times in members of the same family. Three cases ended fatally from pneumonia. In cases of this complication, in fact, all the dangers are present to which children with extensive burns are exposed; and the condition is all the more serious because measles has in itself a tendency to produce pulmonary and intestinal affections. The occurrence of even a single bulla, or at least of very few, either along with the spots or after these had faded (which I have seen in several cases of measles), I am in the habit of regarding as of unfavourable significance. These were generally filled with bloody serum, gave rise to more or less deep, even gangrenous ulceration, were accompanied by other

[1] Hirsch-Virchow, *Jahresbericht*, 1875, ii., 517.
[2] *Jahrb. f. Kinderheilk.*, vii., 1874, S. 350.
[3] *Ibid.*, vii., S. 43.
[4] *Berl. klin. Wochenschr.*, 18

dangerous symptoms, and ended in collapse and death. I must not forget to mention that in rare cases measles is complicated with varicella, the vesicles of which may coalesce here and there so as to form pretty large bullæ; and these may mislead the inexperienced into diagnosing pemphigus.

The least frequent complications of measles are those connected with the nervous system. In little children during the first 2 years of life the fever preceding the eruption is sometimes ushered in by epileptiform convulsions. Older children often complain of headaches, especially over the forehead, arising partly from the fever and partly from the coryza which is almost always present. The drowsiness and closed eyes which we often observe while the rash is coming out, and during its acmè need cause no anxiety, for they disappear when the temperature falls. Serious nervous symptoms are rare, as Rilliet and Barthez have also pointed out. In one child who had passed through an attack of measles quite normally, violent maniacal attacks, with intervals of drowsiness, occurred without apparent cause at the end of the first week. These disappeared after a few days, and were to be ranked along with the transient attacks of mania which occasionally occur after other highly febrile diseases. The following case was of a more serious nature.

> Carl J., 3 years old, took an attack of measles in the beginning of November, 1876, the course being entirely normal. In the middle of the second week after the eruption, a state of drowsiness suddenly set in, out of which the child could scarcely be roused. Rigid contraction of the muscles of the neck (head-retraction), moderate fever, irregular pulse. Treatment with leeches to the head, an ice-bag, calomel, and thorough mercurial inunction into the neck. Rapid improvement. The head was then held straight. The mental powers were unaffected, the pulse regular; but the patient was still unable to walk. After a few days, a relapse without apparent cause. On the first of December, there was again rigid cervical contracture, left internal strabismus, but no rise of temperature or other cerebral symptoms. Iodide of potash given. On the 7th, the head freely movable, squint less marked; child apparently well. After other 8 days, complete and permanent recovery.

My apprehension that this case might turn out to be one of meningeal tuberculosis following measles, was fortunately not confirmed. We can therefore only suppose that it was a case of

moderately severe simple meningitis such as has also been occasionally observed by other writers as a sequela of measles.[1] That in this disease as in scarlet fever serious cerebral attacks, drowsiness, coma, delirium and tremors, may be produced by the malignant character of the attack (*i.e.*, by the pernicious influence of the virus on the brain and heart) is certain; but this "malignity" is but rarely observed in measles. I have, myself, hitherto met with only a few cases of this "typhoid" or "adynamic" form of measles, which, like scarlatina maligna, is accompanied by laryngitis, broncho-pneumonia, and hæmorrhages into the skin as well as into various other parts (mouth, nose, bowel, kidneys), and is always fatal. In two cases of this kind, the symptoms were such that one was tempted to assume a complication of the measles with typhoid fever; but, as a post-mortem was not allowed, this matter remained uncertain.

Relapses similar to those which I have described in scarlet fever also occur now and again in measles; but I have met with only a couple of cases myself.

Child of 2 years, admitted with condylomata May 24, 1880. Recovery after 13 inunctions of mercurial ointment (grs. x.). On 29th June, eruption of measles. Normal course, with moderate amount of catarrh and diarrhœa. On the 11th July, a fresh rise of temperature (103·3°) which continued till the 12th, and on the evening of the 13th, rose to 104°—when a second eruption of measles appeared. This new attack ran its course, accompanied by pretty severe broncho-pneumonia, especially of the right lower lobe. On the 20th, disappearance of the fever.

Boy of 4 years, admitted with measles in the end of January, 1885, and discharged on 12th February. Re-admitted on 17th with a fully developed measles-rash. Temp. 104·4°; photophobia, coryza, catarrh. Normal course.

Almost all the sequelæ of measles are simply complications which have become chronic. Thus we often find blepharitis, blennorrhœa of the conjunctiva, keratitis, and otitis—lasting for many weeks, and even months. In other cases, again, ulcerative processes of the laryngeal mucous membrane (which

[1] We must not forget that there may be a chance coincidence for which the measles is not to blame. Certainly some at least of the cases collected by Thomas (Ziemssen's *Handb. d. spec. Path. u. Ther.*, ii., S. 91) belong to this category; on the other hand, a case of diffuse myelitis published by Barlow (*Med.-Chir. Transact.*, vol. 70: London, 1887), which ended fatally on the 11th day of measles, was certainly directly due to that disease.

may even end in perforation of the cartilages and abscesses on the front of the neck), chronic broncho-pneumonia and diarrhœa are left behind as sequelæ. In the last-named cases, we may finally have intestinal ulceration, which (if situated favourably in the rectum) may be successfully treated by local measures. By far the commonest of these sequelæ is chronic broncho-pneumonia, and I have already described to you (vol. i., p. 390) its dangerous symptoms and its resemblance to pulmonary phthisis. As a matter of fact we see this sequela end fatally in a number of cases, after increasing emaciation and hectic have lasted for months. In these cases, on post-mortem examination we find either chronic broncho-pneumonia only, with dilatation of the bronchi and small abscesses in the lung, arising from destruction of the alveolar wall and the running together of pulmonary air-cells filled with pus; or, more frequently, caseous degeneration of the lungs and bronchial glands. The idea that measles has a special tendency to cause tuberculosis is due, I believe, to the fact that this disease (like whooping cough) prepares a particularly favourable soil for bacillary infection owing to its being frequently complicated by broncho-pneumonia, and causing hypertrophy of the bronchial and tracheal glands.

The sequelæ of measles also include various affections of the skin—abscesses, eczema, impetigo, and ecthyma, but especially gangrene, which is commoner in this disease than after scarlet fever. Cases of noma and gangrene of the pharynx and lungs are pretty often seen in sickly children among the very poor, some weeks after the eruption. I have only met with a single case of true noma following measles, and have more frequently seen gangrene of the skin (especially in the form of ecthyma cachecticum—to be described later on), of the subcutaneous tissue, of the cartilages of the ear and nose (which, when recovery took place, left little defects behind), and of the gum with dropping out of the teeth. All these cases ended fatally. In a child of 3, an abscess occurred on the forehead as the result of an attack of erysipelas of the face which set in on the 8th day of the disease. This penetrated to the bone and caused death owing to extensive gangrenous destruction of the skin laying bare the frontal bone.

In a girl of 3 years, who was still suffering from adenitis and

phlegmonous inflammation in the submaxillary region following an attack of diphtheria, measles set in on Feb. 14, 1878. The phlegmon ended in suppuration, was incised, and in the course of 10 days, became gangrenous under the influence of the measles. Temp. constantly from 104°—105·1°. Face extremely œdematous; air of the room poisoned by the gangrenous smell. Death from collapse and double broncho-pneumonia.

In 3 other children, circumscribed gangrenous patches of the skin formed in the 3rd week of measles. These arose from bullæ (rupia or ecthyma) and resulted in sharply defined, round ulcers from the size of a sixpence to that of a shilling, which seemed as if punched out, and were covered with blackish debris; these were situated on the occiput, in the region of the clavicles, on the hips and other parts of the body. In 2 of the cases, death ensued from collapse and broncho-pneumonia, and the 3rd recovered.

Child of 2 years, admitted March 1, 1877. On the 3rd, an eruption of measles. On the 9th, the left arm was swollen and tense, and there was a bulla on the olecranon, with sero-sanguinolent contents. During the next few days the infiltration spread to the clavicle, scapula, and nipple. An incision made on the 11th let out nothing but blood and a little yellow serum. On the 12th, a fresh eruption of bullæ with bloody contents on the trunk and left arm, which burst and left behind sloughy infiltrated ulcers. On the 19th, the whole skin, from the elbow to the shoulder, was undermined. Counter-opening at the back, from which there was a discharge of pus. Connective tissue gangrenous over its whole extent, and could be drawn out in sloughing fragments. Persistent fever and collapse. Death on the 23rd. *P.-M.*— Double broncho-pneumonia, parenchymatous nephritis. Fatty liver.

This pathological condition (cloudy swelling of the renal cortex and a moderate amount of fatty degeneration of the liver) is on the whole pretty common in measles, as in other infectious diseases, and does not give rise to any clinical symptoms. It is only comparatively rarely—as I have already (p. 164) mentioned —that we meet with cases of nephritis analogous to those following scarlet fever. I have learned from experience, however, that many cases of nephritis attributed to measles are really scarlatinal ones which have been wrongly diagnosed either by the parents or by the doctor. I have met with but few cases of nephritis following measles hitherto. I may give as an example the following, which I had under observation from their very commencement and for the authenticity of which I can therefore answer.

Carl B., 7 years old. In the end of June, 1875, an attack of measles with perfectly normal course. Three weeks after the eruption, œdema of the face, feet, and scrotum. Urine albuminous, scanty, containing epithelial cells and casts and, after a few days, hæmorrhagic. Treatment with warm baths, acetate of potash and Wildungen water. On the 28th July, œdema almost gone. Urine still containing blood. Ergotin given. Complete recovery after 10 days. Iron given on account of the anæmia which was left.

Fritz R., 3 years old, admitted April 24, 1885, with coxitis. Took ill on 13th May, with measles. Convalescence delayed by catarrh of the larynx and trachea, and interrupted by febrile disturbances. On the 30th—*i.e.*, in the middle of the second week—the urine contained a large quantity of albumen, epithelial casts and leucocytes. Slight œdema of the face. Sweat-baths. On 6th June, no change. On the 11th, only traces of albumen left, along with a few casts and epithelial cells. On the 26th, complete recovery.

In one case, which occurred in a child of 3 (likewise three weeks after an eruption of measles) and ended fatally from œdema of the lungs, we found at the post-mortem (which took place in the Charité on Dec. 24th, 1874), besides pulmonary œdema and multiple broncho-pneumonia, typical double nephritis. In another child, we found hæmorrhagic nephritis with enlargement of both kidneys, numerous punctiform hæmorrhages in the cortex, which was much enlarged and greyish-yellow, and fatty degeneration of the epithelium. It is also worthy of note that in cases of measles which commenced a few weeks after, or during, an attack of scarlatinal nephritis, the influence of the measles on the nephritis was entirely different. At any rate, an unfavourable influence was not invariably found.

I have only once observed purpura as a sequela of measles (p. 231)—in a girl of 8 who was brought to me on Nov. 9th, 1851. In this case, 3 weeks after the eruption (which had been accompanied by bloody diarrhœa) there suddenly occurred hæmorrhage from the mouth, nose, ears, and bowel, numerous petechiæ appeared on the skin, and the left palpebral conjunctiva became suffused with blood. The child at the same time appeared perfectly well. I know nothing of the further course of the case.

While scarlet fever is never wholly absent in large populous cities, cases appearing sporadically all the year round, and only

at times—especially in autumn and during the earlier winter months—prevails endemically; measles on the other hand sometimes disappears almost entirely and then suddenly breaks out again as an epidemic. This usually starts from a particular quarter of the town, spreads gradually to neighbouring parts, and thus lasts for months. That one individual may have measles twice (just as he may have scarlet fever twice) is certain; still I think that the number of such cases is greatly overestimated, especially by the laity. In spite of my scepticism, however, I must admit that they do sometimes occur, as, for example, in the following case :—

> Boy of 13 years, had measles in 1872, along with 4 brothers and sisters (under my treatment). In November, 1876, a second attack of measles with characteristic prodomata—high fever with crisis on the 3rd day, catarrh, photophobia, &c. Rash copious on the face and trunk, slight on the extremities. Infection at school during an epidemic of measles was ascertained.

However, such cases are exceptional. Most of those one hears of are really cases of other similar rashes being mistaken for measles. Such rashes are therefore spoken of as "false measles" (morbilli spurii). At the same time we must always bear in mind that this name does not by any means denote a definite morbid process, but that it includes a number of diverse affections which have as a common feature a measly rash composed of small maculæ, which readily become papular. I need only remind you of the common rashes described as roseola vernalis, autumnalis, æstiva and infantilis—rashes which do not always remain macular but may also present flat central elevations and are often taken for measles. The chief point for the diagnosis of the latter consists—apart from the cachectic and "terminal" forms with an abnormal course—principally in the characteristic temperature-curve being found along with catarrh of the respiratory and pharyngeal mucous membranes; a combination which is never found along with simple erythema or roseola.

I shall take this opportunity of saying a few words about the affection which has long been described as rötheln (rubeola), the existence of which is still a matter of lively controversy. Some physicians regard röthel[illegible] -n independent epidemic, or, more commonly, endem[illegible] ginning with scarcely

noticeable fever (generally only during the stage of invasion), sometimes also with slight catarrhal symptoms, but which is chiefly characterised by an eruption consisting of small red points. On the other side it is contended that all such cases are nothing more than very slight, almost afebrile forms of measles or even of scarlet fever. My own experience does not enable me to give a decisive judgment in this matter. Although I have occasionally seen two or three children in one family take ill with an affection corresponding to the "rötheln" described by other writers, and have frequently seen cases in which I felt doubtful about the diagnosis—still, I have never yet had an opportunity of observing this condition occurring to any great extent, either epidemically or endemically, as several (Steiner, Thomas, Nymann and Roth) have described it. Such being the case, I am not in a position to give my judgment in favour of rötheln being an independent disease.

The susceptibility to the contagion of measles[1] is present at all ages. It is greatest between 2 and 6 years, and least in new-born children and very young infants; in these, however, the danger of respiratory complications is greatest. The presence of other diseases, either acute or chronic, forms no protection from the infection of measles; some, like chicken-pox and whooping cough, seem to produce a predisposition to it. In what period the disease is most infectious is as difficult to ascertain as in scarlet fever; still there is no doubt that infection is possible during both the prodromal and eruptive stages. Most children become infected after having been once or twice in contact with a patient who has the disease. But some children can be with their brothers and sisters for 3 or 4 weeks before they take the infection from them. I, at least, have never seen a case of absolute immunity from the infection of measles as I have sometimes seen in the case of scarlet fever.[2] Similarly, I have never observed a single assured case of morbilli sine exanthemate. The notion that the danger in measles is slight, which is entertained by the laity and many physicians, is practically correct in regard to private practice (and especially where the circumstances

[1] We know as little of the nature of the contagion in this disease as we do in scarlet fever. The bacteria described by Babesin (*Arch. f. Kinderheilk.*, iii., S. 143) stand in need of further and independent confirmation.

[2] At the same time I certainly do not deny that cases of such immunity may exist. *Cf.* Biedert, *Jahrb. f. Kinderheilk.*, xxiv., S. 94.

are favourable). But in hospital it is a very different matter. In my wards during 1882 and 1883, out of 147 cases of measles there were 74 deaths; and in the epidemic of 1885-6, out of 90 cases 36 died, mostly from broncho-pneumonia, croup, diphtheria,[1] and tuberculosis.

I have but little to say on the subject of treatment, for the morbid process as such requires nothing but that the patient should be kept in bed in a room with a temperature of 65°—68° F. (warmer than for scarlet fever), be lightly covered, and—as long as the temperature remains high—be kept on a diet of milk, water-gruel, and cooling drinks. The room should only be darkened as far as is pleasant to the children, and never entirely. Unfortunately the good old (or rather bad old) custom of completely darkening the room is still too prevalent. I do not consider it is at all necessary to separate the patients from their brothers and sisters, although I am so strongly in favour of it in cases of scarlet fever. Such a precaution seems to me quite unnecessary when one considers how much less is the danger connected with measles and bears in mind that the children are almost bound to have it sooner or later. Only in the case of very young children (under 2) or those who are already ill, especially with tuberculosis, should we endeavour to protect from infection by complete isolation. In simple cases no medicine is required. When the cough is severe we may order ipecacuanha along with aqua laurocerasi (Form. 16), and, if required, a blister of the length of one's finger-joint to be applied over the larynx. Moderately severe diarrhœa, which is not uncommonly met with during the first week, may be disregarded; but when the motions begin to be pretty copious (4—6 times daily, or oftener) you should try to control them by giving ipecacuanha along with opium (Form. 29), or subnitrate of bismuth (Form. 30). But even in perfectly normal cases I advise you to keep the children for a whole week in bed, and to confine them to their room for some time afterwards (for 3 weeks in summer and 4 in winter).

For the treatment of complications I may refer you to what I have already said about that of croup, broncho-pneumonia, and diarrhœa. In the broncho-pneumonia of measles, especially, I have frequently obtained extremely good results quite unexpectedly, even when it was accompanied by lowering of the

[1] *Cf.* Pennel, *Revue mens.*, Juin, 1885, p. 279.

heart's energy, small pulse, cold extremities, drowsiness and slight delirium—from the bold use of warm baths (with cold douching) camphor and benzoin. In cases where the pneumonia has set in thus during the period of eruption the rash generally becomes rapidly cyanotic, but the baths bring back its rosy colour. The following case illustrates the rapid success of local treatment in a case of ulceration of the rectum which had remained after measles :—

> Marie S., 1½ years old, brought to my polyclinic January 30, 1877. Formerly healthy. An attack of measles at Christmas with bloody diarrhœa lasting 3 weeks. Since then, one tolerably natural pulpy motion once or twice daily. But several times there occurred during the intervals tenesmus, prolapsus ani, and the discharge of 1—1½ teaspoonfuls of pus mixed with blood; sometimes pure blood or muco-purulent matter. The child somewhat emaciated and pale, but otherwise seemed well. Nothing was found on examination of the rectum. Diagnosis: ulceration in the upper part of the rectum. Treatment: enema of nitrate of silver (1 in 600). Recovery after 5 injections; further injections of alum (1 teaspoonful to a cupful of water) to make matters sure.

When there is gangrene of the skin, we cover the affected parts with iodoform, or with lint soaked in camphorated wine, carbolic lotion (2 per cent.), or solution of chloride of zinc, and give wine freely and decoction of cinchona (Form. 23).

The opinion sometimes expressed, that measles is capable of exerting a curative influence on certain chronic diseases (especially of the skin) does not agree with my experience; at any rate I have seen chronic eczema and prurigo persisting unchanged after recovery from an attack of measles.

The attempt to transmit measles to children by inoculating them with blood, tears, and nasal mucus (Home, Katona, Mayr) in order to induce a milder course by a kind of vaccination as it were, have yielded no satisfactory results.

## III. *Chicken-Pox.*

Varicella is one of the few diseases which are confined almost exclusively to childhood. For my own part I have not met with a single well authenticated case in an adult.[1]

[1] J. Seitz (*Corresp. Bl. f. Schweizer Aerzte*, Jahrg., 18, 1888) describes a few cases observed in adults.

There cannot be any doubt as to the infectious nature of chicken-pox. Although inoculation-experiments with the contents of the vesicles have only succeeded in a small number of cases (my own attempts have led to as little as those of Thomas, Hippius[1], and others); still, we have the most positive proof on the matter in our daily practice—instance the successive infection of all the children in a family and the endemic prevalence of the disease in institutions (which I have often observed in my own wards). The stage of incubation is 13—14 days, like that of measles, and is succeeded by the rash, generally without prodromal symptoms. The patients only rarely complain of headache, vomiting, and fever. Conjunctivitis or sore-throat is also sometimes observed; but these I am rather inclined to regard as chance complications than as symptoms connected with the varicella. In one child of 10 months, whose body was covered with a copious rash, I observed severe epileptiform convulsions which set in during the eruptive stage, lasted 24 hours and were accompanied by high fever. In a few cases, also, I have seen diffuse erythema preceding the eruption of varicella by several hours and persisting during the first day.

The eruption appears simultaneously on various parts of the body without any definite order, in the form of round red spots about the size of a lentil seed, in the centre of which a vesicle about the size of a pin's head forms immediately. But only in exceptional cases have I been able to observe this first period, for the vesicle grows so rapidly that after an hour one finds the vesicles all over the body, varying from the size of a lentil to that of a pea (sometimes being as large as a sixpenny-piece, and resembling those of pemphigus) filled with clear serum, and surrounded by a very narrow red border. In a few cases I have found the rash composed (as Thomas also observed) of round red spots, nearly all of which had a miliary vesicle in their centre. The number of the vesicles, which appear in rapid succession, varies greatly. Sometimes they are isolated, and sometimes they are clustered closely together—especially on parts which are exposed to irritation through pressure or stretching of the skin (*e.g.*, on the back or over the tuber ischii, in which position I have seen a number of vesicles crowded together over

[1] *Centralzeit. f. Kinderkrankh.*, ii., 1879, S. [illegible]

an area of the size of one's palm, like a patch of zoster). In this way it happened that in a child who always lay on his left side, that side was more severely affected than the right; and in a boy who had an acute abscess the size of a child's head in the left groin, the extremely tense skin over the abscess was covered with a thick eruption of varicella, while over the rest of the body it was very scanty. The most beautiful eruption I have ever seen was that of a negro child, whose brownish-black skin looked as if it was scattered over with pearls. Very often a few vesicles also form on the mucous membrane of the mouth, on the hard palate on the inner surface of the lips, and on the tongue; but these very soon come to look like whitish or greyish-yellow round erosions, owing to the rapid desquamation of the epithelium. Now and then I have met with a few isolated turbid vesicles, surrounded by a congested area on the ocular conjunctiva and on the genital mucous membrane in little girls. In one case there was a regular wreath of vesicles on the inner surface of the labia majora.

Many people still hold the opinion that varicella is not a febrile disease, but this does not agree with my experience. The notion has taken its rise in private practice, where the affection is regarded as too trivial to require the use of the thermometer. When the temperature is taken it is almost always found to be elevated during the eruption, that is, during the first day, and sometimes even on the second; but in the majority of cases it is only moderate in degree. As a rule, I have found the temperature on the first day 100°—101·8° (ev.), while by the second day, the fever had often disappeared or was only present in the evening (101.3°, or higher). But rarely is the temperature very high or of long duration.

Child of 2½ years. Eruption of varicella on May 11, 1875, with high temperature (104·2°; pulse 168). Back covered with diffuse erythema. Very copious eruption of vesicles, especially on the chest and thighs. On the 12th, erythema still visible. Temp. 100°; ev. 102·9°; pulse 144. On the 13th, redness gone, temp. normal. Vesicles beginning to dry up.

In another child, the temperature on the evening before the eruption was 101·5°; on the evening of the second day, still 102°. In a third case, which like the first was accompanied by erythema, the initial temperature was 104·9°, and on the 2nd evening it was still 100·9°. In a boy who had been successfully tracheotomised

on account of croup following measles, an attack of varicella which broke out during his convalescence after an initiatory rise of temperature of 101·5° (evening before the eruption) had a febrile course which continued fully 4 days; the temperature on the 2nd and 3rd days being 104·4° and 104·9, and on the 4th evening rising to 105·8°. This boy had a general predisposition to high temperature, so that, during a trifling gastric attack, he had for 3 days an evening temp. of 104° and over.

An unusually high temperature is generally, so far as my observation goes, associated with a very extensive and close rash, the individual spots of which are not uncommonly united by an erythematous blush, and some of them become purulent on the 2nd or 3rd day. More frequently the form of the somewhat opaque vesicles remains unaltered, and they generally cease to appear by the end of the second day. After the 3rd day, the vesicles begin to dry up, owing to the evaporation of their contents; they collapse and are changed into thin brown or blackish crusts (of a size corresponding to that of the original vesicle), which quickly lose their red border and fall off after 8—14 days, leaving behind them red spots but no cicatrices. The formation of cicatrices only occurs when the violent itching has caused the children to scratch the eruption and the scabs during the process of drying up. When they do this some of the vesicles pass into small shallow ulcerations, ending finally in cicatrisation, and sometimes also ecthymatous pustules and erythematous rings are produced. The eruption is not always completed by the end of the second day, however; fresh crops of vesicles often appear quite irregularly, so that, *e.g.*, the back and lower parts of the body may be first affected, and the face not until the 3rd day. Between those which have already dried up, there appear other fresh watery vesicles, so that one can see the different stages of the eruption side by side on one part of the body. I have met with this so often that I cannot agree with Thomas, who asserts[1] that such fresh outbreaks do not occur. In one child of 13 months, I have even seen this fresh eruption take place on the evening of the 3rd day along with a considerable rise of temperature.

I must now return to what I said before about the contents of a certain number of the vesicles becoming purulent—an occurrence which is often observed. Those are just the cases

[1] *Arch. der Dermat.*, 1869, Heft 3.

that keep alive the controversy, which has not yet received its quietus, concerning the connection between varicella and the forms of variola. In my opinion, every unprejudiced observer must certainly take the side of the dualists, *i.e.*, of those who do not recognise the existence of any such relationship, but regard varicella as a quite independent infectious disease having nothing whatever to do with variola. I have already, elsewhere,[1] given my opinion to this effect, and stated definite reasons. I would in the first place refer to the anatomical differences between the two eruptions—that of varicella at once becoming vesicular, while that of variola begins with red papules, on the summits of which vesicles appear later on. There is also the fact that the varicellar vesicles are more simple, and when pricked collapse, letting out all their contents at once; while the small-pox vesicles are multilocular, so that only the compartment which happens to be pricked discharges its contents. All this, however, is inconclusive, for among the normal chicken-pox vesicles we frequently find a varying number of more complex ones which present a central depression and end by becoming opaque and purulent—that is to say, they behave quite like those of variola or varioloid. Likewise, cases are not very rare in which the vesicles seem abortive, *i.e.*, appear in some places at any rate as small red papules, which here and there are capped by very minute vesicles. The main thing always is the fact that chicken-pox affords absolutely no protection against small-pox, but that on the contrary the latter may set in within a few weeks after an attack of the former and *vice versâ*. Varicella, further, may set in immediately after successful vaccination, and, again, children may be vaccinated successfully shortly after or even during the presence of chicken-pox. It is also important to remember that in small epidemics of varicella, such as I have often seen in my wards, not even a single case presents the characters of variola or varioloid. And, finally the cases of varicella which have a superficial resemblance to varioloid, invariably induce only chicken-pox in other individuals. Two children in one family who had been well vaccinated suffered simultaneously from varicella, the elder had a copious eruption, partly umbilicated and purulent, and accompanied by high fever—while the younger brother presented

[1] *Berl. klin. Wochenschr.*, 1874, No. 18.

nothing but a few watery blebs, and appeared otherwise perfectly well. In another family, a child of 3 years who had been successfully vaccinated, took a very severe attack of chicken-pox, and the vesicles were so copious, and many of them so distinctly umbilicated and purulent, that I should have been led into error had I not myself observed the first eruption of transparent vesicles. After 14 days, the elder brother took quite a mild attack of unmistakable varicella. I have never known the disease to spread to the adult members of the family, as small-pox would be pretty sure to do, nor has a nurse ever become affected by variola or varioloid during any of the epidemics of varicella observed in our wards. Add to this that inoculation with fluid out of the chicken-pox vesicles, when it has been successful, has always produced varicella and never variola.[1] Against such facts as these, which are exemplified daily, all the sophistical arguments with which the supporters of the unity theory try to maintain their view,[2] are of no avail, and surely it will not be long ere everyone is brought to recognise that the two diseases are essentially distinct.

Chicken-pox may affect very young children even in the first months of life, and in them is not more dangerous than at a later period. Until quite recently I have regarded the disease as perfectly harmless; and I have only been led to change this opinion by having met with varicellar nephritis (p. 164), to which I need not now recur. I am not acquainted with any other complication or sequela.[3] Serious consequences may of course arise from the combination of varicella with other infectious diseases (scarlet fever, measles, diphtheria) which I have sometimes known to occur. Varicella is, I believe, the disease by aid of which we can best demonstrate the simultaneous occurrence of two acute exanthemata, because its characteristic vesicular eruption differs so strikingly from the diffuse or maculo-papular eruptions of scarlet fever and measles. I have twice met with varicella in children affected with ichthyosis, once in a patient with extensive psoriasis, and once complicated by tertian intermittent fever.

[1] Steiner, *Wien. med. Wochenschr.*, No. 16, 1875.—D'Heilly et Thoinon, *Revue mens.*, Dec. 1875.

[2] Kassowitz, *Jahrb. f. Kinderheilk.*, 1873, Heft 2.—*Ibid.*, Heft 4, S. 420.

[3] Semtschenko (*Jahrb. f. Kinderheilk.*, xxv., S. 171) says that in two cases he has observed purulent pleurisy and purulent synovitis, respectively, following varicella

We need scarcely speak of treatment; but even in the slightest cases I confine the children to bed for some days, and to the room for a week.

## IV.—*Diphtheria.*

It is unfortunately an indisputable fact that this disease—which was well-known to the old Arabian physicians, was almost lost sight of at a later period, and was again brought to light by Bretonneau[1]—is steadily becoming commoner and more malignant. The fact is being recognised and deplored in all countries. According to Kalischer,[2] a yearly average, since 1875, of more than 40,000 children have died in Prussia alone of diphtheria, about 20,000 of scarlet fever, 8,000—12,000 of measles, and 10,000—16,000 of whooping cough. In the large towns, this progress of the disease is readily traced to the growing number and increasing severity of the cases occurring in the hospitals.[3] I need only mention that in Berlin, according to municipal statistics, out of 65,521 deaths in the years 1882 and 1883, 5,066 were due to diphtheria alone; while out of 31,483 deaths in the year 1885, 1,816 only were due to this disease.

I base the following description on 853 cases of diphtheria observed and noted in my wards, exclusive of many others occurring in my private practice of which I have only short notes. I have excluded all cases of doubtful sore-throat as well as those of so-called "scarlatinal diphtheria" which—for the reasons already given (p. 214)—I regard as entirely distinct from real diphtheria. Among these 853 children there were almost as many girls as boys. All ages were represented: the 1st year by 108; the age between the 1st and 6th years by 542; and the 7th and 8th years by 107 cases. The remaining 96 were distributed over the period between the 8th and 14th years. The disease was thus found to be most frequent between the 1st and 6th years, and on this point all writers are agreed.

Although at this age infection acquired at school is out of the question, yet centres of infection do exist. Kindergartens, the playing together in lobbies and rooms, and on the stairs

[1] *Des inflammations spéciales du tissu muqueux et en particulier de la diphthérite*: Paris, 1826.

[2] *Verhandl. der deutschen Gesellschaft f. öffentliche Gesundheitspflege zu Berlin*, 1883.

[3] Henoch, *Charité-Annalen*, x., 1885, S. 498.

(especially in very poor neighbourhoods) afford such centres in plenty. But still it has been proved experimentally (Peter, Trousseau and others) that diphtheria is not so exceedingly infectious as it is customary to assume. For it was found that the contact of diphtheritic products with the mucous membrane was not in these experiments followed by any infection.[1] In my wards during a period of 13½ years not a single nurse, and only one house-physician was affected by diphtheria, although there were almost always in the wards cases of the worst form of the disease. Here, as in other infectious diseases, we are therefore almost obliged to assume the existence of a predisposition—a favourable soil for the development of the infective material.[2] Although adults are less susceptible to this disease than children, still the necessity for caution is impressed on us by the well authenticated cases of doctors being infected by their patients, and mothers by their children.

As to the influence of different seasons of the year, I have not been able to come to any definite conclusion. My cases are divided pretty equally among all the months of the year. Diphtheria, like scarlet fever, never disappears entirely from Berlin; the disease has unfortunately become endemic here. But from time to time, at quite indefinite intervals, it spreads so much as to constitute an epidemic. We especially often meet with small endemic outbreaks limited to a single house, or to several houses or streets, and these are to be referred either to spreading by direct transmission, or to a common cause. The destructive endemic and epidemic outbreaks which not uncommonly occur in certain neighbouring villages arise in a similar way. We have no certain knowledge with regard to the nature of the infective material, which is assuredly capable of being transmitted from one individual to another, perhaps also from animals (fowls) to men. It is probable that the contagion in this disease as in some others is of a vegetable (bacterial) nature. The researches hitherto made have not as yet furnished any indubitable results, and what has been described as the "diphtheria fungus" is merely one of the putrefactive bacteria which are present in all septic conditions. I need not trouble you with a discussion of alleged causes which

[1] Monti, *Ueber Croup u. Diphtheritis*, 2 Aufl., 1884, S. 145, *et seq.*

[2] My experience does not go to confirm the opinion of Unruh (*Festschr. zur Jubelfeier d. Kinderheilanstalt in Dresden*, 1884) that tuberculosis of the joints and bones gives rise to a special predisposition.

are hardly susceptible of proof, but which are always being called in to account for all sorts of infectious diseases—sewer-gas, tainted water or infected milk. I may mention, however, that the wife of a proprietor in West Prussia told me that her children had taken diphtheria at long intervals, and some of them had died; and that these attacks only ceased to occur when a dung-hill under the nursery window was removed. We are also quite uncertain how long the period of incubation lasts. From certain observations made in my wards, I think I may conclude that the average duration is 7 days; but we can never be quite sure that the infection has not taken place before admission.

In a large number of cases the diagnosis is easy from the very first, because the symptoms at once suggest some pharyngeal trouble. Older children almost invariably complain of their throats or of pain on swallowing, and this attracts the parents' attention. When the physician is called in he finds the entire pharyngeal mucous membrane reddened to a varying degree, the tonsils swollen and covered with white or greyish-white patches, especially on the inner surfaces which face one another. It is difficult or even impossible to remove these with a brush or spatula, and the attempt to do so is almost always followed by a slight hæmorrhage from the exposed mucous membrane. I have only in very exceptional cases found the tonsils free from deposit, and perhaps the soft palate or even the posterior wall of the pharynx, and least commonly of all the mucous membrane of the hard palate more or less covered with membrane. At the same time we must be careful not to mistake for real membrane the secretion which flows from the nose over the back wall of the pharynx; for it can always be wiped off, or, in older children, removed by gargling. As a rule the children are feverish, but the temperature is, generally speaking, not so high as that which ushers in an ordinary attack of follicular sore-throat (p. 14). It usually varies between 100·4° and 102·2°, with evening exacerbations. Cases do also sometimes occur in which at first, at any rate, there is absolutely no rise of temperature, the children are as a rule unusually depressed and ill-humoured; they lose their appetite, have a grey fur on their tongues, and complain of headache. Generally even during the first days we find some swelling of one or two of the lymphatic glands under the angle of the jaw; but this may be absent in

diphtheria just as it may be present in catarrhal sore-throat. Its absence in diphtheria, however, is by no means so rare as I formerly supposed, and I have observed it even in very serious cases; for example, in two children in the same family, one of whom died with croup and the other with collapse.

As I have already remarked (p. 16) it is not always easy, in forming our diagnosis, to distinguish between commencing diphtheria and catarrhal sore-throat. Sometimes, indeed, it is simply impossible during the first 24—48 hours, so that it is as well to reserve one's final judgment, and in any case to isolate the patient from the other children. The yellowish colour and rounded form of the little circumscribed plugs of pus which are scattered over the red and swollen tonsils is indeed characteristic of this form of catarrhal sore-throat. Further, the fact of the disease beginning on one side and spreading to the other afterwards is also in favour of the case being non-diphtheritic. Still, cases do occur in which diphtheria begins in just the same way —on one side and in little patches and not spreading to the other tonsil until the next day. It is even more difficult still to distinguish between these two conditions if, in a case of catarrhal sore-throat, we find on the tonsils instead of the above-mentioned purulent plugs, greyish-white longitudinal streaks which really have a misleading resemblance to diphtheritic membrane, but differ from it by the fact that they lie loosely on the mucous membrane like a croup membrane, and are made up of a large quantity of epithelial cells held together by an amorphous (fibrinous) material. I cannot agree with those who always ascribe a specific diphtheritic character even to this kind of membrane, for I believe that it is merely the product of croupous inflammation of the mucous membrane, because I have not uncommonly observed it to occur simultaneously or alternately with the ordinary yellowish plugs in children and adults particularly predisposed to catarrhal angina; and also because I have occasionally found it accompanied by the formation of an abscess in the tonsils (p. 16). The incision-wound in these cases never assumes a diphtheritic character even although it has been made through the middle of the croupous membrane. These things at any rate increase the difficulty of the situation, and we must neither be in a hurry to diagnose diphtheria, nor yet, on the other hand, to give [illegible]aring verdict. When we remove

the patches and subject them to microscopic examination we find nothing which is, as yet, recognised as a sure criterion for the diagnosis; for in both conditions there are epithelial cells, amorphous matter, and micrococci.

The state of things is more serious, however, if we find patches of the dreaded white membrane not only on the tonsils but also on the margin of the soft palate, the uvula, the angle between these two, and the palatal arches. Even when such a condition is present there may be little or no difficulty in swallowing, the fever may be only moderate in degree and the child may feel almost quite well in other respects. I have often seen children with very extensive pharyngeal membrane come to the polyclinic on foot. They either complained of nothing at all or only of their "belly"; or they were brought because they had no desire for play, or because their parents had been made anxious by another child's having taken ill in the same way or having died, and had therefore examined the throat and so discovered the disease. I cannot too strongly impress upon you the tolerance which many children show for diphtheria in its early stages, because, owing to this, the disease is often entirely overlooked. I should urgently recommend you to make a careful examination of the pharynx of every child who is feverish or even only changed somewhat in temper, although there should be no local symptom whatever. My students have often enough witnessed instances of far-advanced diphtheria being discovered in this way, when neither the doctor nor the parents had had any idea of its being present. The disease, in such cases, remains latent either until threatening symptoms suddenly set in or until the occurrence of certain sequelæ — especially paralysis — reveals the fact of previous neglect. Therefore, while in pharyngeal diphtheria even when it is extensive both the local and general symptoms may be trifling (during the first few days at least), the implication of the nasal mucous membrane which is often observed, forms a characteristic symptom (coryza or rhinitis diphtheritica) which at once arouses the anxiety of every experienced physician, and which I have never yet seen accompanying a simple case of sore-throat. The children snore heavily during sleep and even breathe noisily through the nose while wide awake, and a large quantity of thin purulent secretion either flows spontaneously

from the nostrils or can be pressed out. This discharge is noticed particularly during crying and other expiratory actions; and the nostrils and upper lip finally become reddened and irritated by it. Affection of the nasal cavity in diphtheria has the same unfavourable significance as the gangrenous process has in scarlet fever (p. 215). I certainly do not wish to assert that the presence of diphtheritic coryza always makes a fatal issue inevitable, for I have frequently found a moderate degree of it present in quite slight cases. Still, I generally regard the implication of the nasal mucous membrane as a bad omen, especially when it is severe. The sero-purulent discharge from the nose is often mixed with blood and severe epistaxis also occurs, being due to the separation of the diphtheritic crusts, which are washed out in varying quantity when the nose is syringed. These hæmorrhages further increase the debility, and ought always to arouse anxiety. I regard the coryza as an extremely important symptom, especially in little children who are too young to speak: it has often been the first thing to suggest to me diphtheria and to lead me to examine the pharynx. Indeed, in one girl of 6 years, it was only the repeated hæmorrhage and the swelling of the nose which induced me to look at the throat, whereupon I discovered the diphtheria. The snoring due to the swelling of the nasal mucous membrane may be so loud, especially during sleep, that it resembles the sound of the obstructed breathing in croup; but as soon as we open the child's mouth the noise is lessened and we see our mistake.

Diphtheritic coryza does not always spread from the pharynx. It may also appear as the very first symptom of the disease. When this is the case, the diphtheria very seldom remains limited to the nasal mucous membrane, but generally spreads through the posterior nares into the pharynx. We often find that the children have been suffering from severe coryza for 8—10 days, which has scarcely attracted any attention until anxiety was aroused by the further extension of the disease or even by symptoms of croup. Unfortunately we cannot, at first, distinguish with certainty this form of diphtheria from ordinary severe coryza unless we can distinctly make out (as we often can) patches of white membrane reaching down to near the nostrils. Rhinoscopy, especially in young children, is attended with almost insuperable difficulty. We must therefore pay particular attention

to any rise of temperature which may be present (I have seen such a case of coryza begin with a temperature of 104°) œdema of the nose, loud snoring, sero-sanguinolent discharge from the nostrils, general apathy and pale collapsed appearance—a series of symptoms which is in favour of the coryza being diphtheritic. We can never, of course, be quite sure until the disease spreads downwards into the pharynx or fragments of false membrane are discharged from the nose, which I have repeatedly seen in this disease as well as in scarlet fever. I have already given you an example of this kind (p. 216), and I have seen the same thing happen in a child of 3 years who was also suffering from diphtheritic otitis and conjunctivitis, and who had a few furuncular abscesses on the neck and chest, covered with diphtheritic membrane. There was also the case of a girl of 13 with moderately severe pharyngeal and nasal diphtheria, out of whose nose a large fragment of membrane was extracted on the 14th day of the disease. Sometimes, though much seldomer than in the nose, the disease begins on the mucous membrane of the lips in the form of greyish-white membrane resembling the confluent patches of aphthous stomatitis (p. 3), and in such cases I have known pharyngeal diphtheria only appearing 36—48 hours later. In one case of this kind the diphtheria spread from the lips to the dorsum of the tongue and proved fatal on the very next day with symptoms of croup. As I have hitherto only observed this form of commencement in hospital practice, I am inclined to assume that certain erosions or fissures of the lips already present had become infected with the diphtheritic poison. I also found the floor of the mouth and the frænum linguæ affected in a couple of cases, one of which ended in recovery. In several children who were referred to my ward from the Eye Department, the diphtheria had commenced on the conjunctiva, and in some others it had begun as a diphtheritic membrane over a patch of long standing eczema on the face or ear; and we then saw the extension to the membrane of the lips and the pharynx taking place under our eyes. Most rarely of all, I have seen the disease begin on the genital organs of little girls, and, proceeding from thence, infect the whole organism.

Clara D., 3½ years old, admitted July 29, 1878; well nourished, scrofulous, with phlyctenular conjunctivitis of both eyes, very pale. Both labia majora had been much swollen and

about the room, or play as they sit in bed. After an average duration of 8—12 days, when the membrane has become loosened and partially separated, the shallow ulcerations left behind (which readily bleed when touched), generally become skinned over and soon there is nothing left but a dark-red spot at the affected place. You must, however, always be prepared for a fresh formation of membrane, either on the same spot or close beside it, after the first has separated; and when this happens, the fever (which had quite gone) may return, and the whole process be prolonged for 2—3 weeks. I have often enough seen cases of such recurrent attacks taking place after an interval of 2—3 days. Even in this mild form the urine which at first sight appears normal and is at most only rather scanty, may contain a small quantity of albumen without the prognosis being therefore really any worse. Only in a very few cases have I found herpes labialis—once in a girl of 11 in whom the vesicles passed into excoriations of the nostrils covered with diphtheritic deposit without the favourable course of the case being at all interfered with.

2. The moderately severe form. Here, in addition to the tonsils you find the soft palate, uvula, and perhaps also the posterior pharyngeal wall covered with greyish-white patches, which have the appearance characteristic of true diphtheria—that is to say, seem to infiltrate the tissues of the mucous membrane somewhat deeply. An experienced physician may be able to draw conclusions as to the prognosis from the mere external form of the local condition, even in cases where the rest of the symptoms and especially the general feeling of the patient are altogether favourable. In all these cases there is a danger of serious symptoms, if not of death. Not uncommonly, as I have already said, the muco-purulent secretion which flows from the back of the nose over the posterior wall of the pharynx is mistaken for a diphtheritic membrane. It, however, can be easily removed by gargling or brushed off, whereas in this situation particularly, the regular membrane is generally very adherent. The nasal cavity is more severely affected, the discharge is more copious, greyish-yellow in colour, and bloody; and it, as well as the patient's breath, is often offensive. There is considerable swelling of the pharynx and of the nasal mucous membrane, the snoring is louder, and the isthmus faucium more contracted; the difficulty of swallowing may be ve[illegible]ut it may also be very

slight, or there may even be none at all. In a few cases I have seen the uvula swollen to the size of a finger, and covered completely with whitish-grey membrane as with the finger of a glove. Although, in this form, the fever may still be only slight (100·4°—103·1°) still the general feeling of health is usually much more affected, the desire for play is lost, the apathy is greater, and sometimes we even notice that the children become drowsy as soon as they are left to themselves. The appetite is generally lost, the tongue is covered with a greyish-yellow fur, rarely it is bright-red with prominent papillæ at the tip. There is often vomiting during the first days of the disease. The urine is scanty and frequently, though by no means always, contains albumen, epithelial cells, and pale tube-casts. Even in these cases this symptom, at least in my experience, has no very great significance as regards the prognosis; for I have often seen children with a moderate amount of albuminuria get well, while others died whose urine was free from albumen throughout. A change of voice is also a common occurrence which causes anxiety. The children become more or less hoarse, even aphonic, and may also have a rough harsh cough, forcibly reminding one of croup. Under these circumstances it is very natural to fear that this symptom is due to the diphtheritic process spreading down into the larynx. We ought, therefore, always to be prepared for the onset of violent croupy symptoms, and for tracheotomy if necessary. Nevertheless, in a considerable number of cases, I have seen these apparently serious symptoms gradually diminish and disappear after causing great anxiety for many days—in one case for a week and a half; and in these cases we are probably right in assuming that there is merely catarrh of the mucous membrane, which has spread from the pharynx to the vocal cords. I learned, however, from a post-mortem which I made on July 16, 1878, that even serious changes in the larynx cannot be excluded in such cases. In a child who had had the above-mentioned laryngeal symptoms for 4—5 days following pharyngeal diphtheria but without any dyspnœa, and had afterwards recovered his normal voice and almost stopped coughing—I found after death, which took place suddenly from collapse, that the mucous membrane of the larynx and trachea was covered here and there with a thin croupy membrane. This shows that we are not justified in always putting down every

Separation of all the sloughs. Recurrence on both tonsils on the 19th; attack lasted till October 8. Interval till the 18th, on which day the pharynx became affected with diphtheria anew, with swelling of the submaxillary lymphatic glands. On the 26th, the membrane was most marked, and there were croupy symptoms (harsh cough and hoarseness). Recovery then followed, and the child was discharged on November 9, after having been ill for 2 months.

The swelling of the submaxillary glands generally disappears rapidly on the ulcers healing. Phlegmonous inflammation and the formation of abscesses requiring incision are much rarer occurrences in this disease than in scarlet fever. Destructive burrowing of pus, which is not very uncommon in scarlet fever, or extensive hard infiltration with a tendency to gangrene (angina Ludovici), I have only very rarely observed in diphtheria.

During the stage of recovery we sometimes find gangrenous fragments which are still partially attached to the mucous membrane, and move up and down in the pharynx with every inspiration and expiration; or the children expectorate large portions of membrane, which sometimes represent a complete cast of the uvula. Quite large pieces of the infiltrated and gangrenous tonsils may separate in this way, and in that case we are apt to have more or less copious hæmorrhage from the pharynx and nose, and this may considerably increase the debility already existing. In a few cases, I have also seen the whole uvula, or at least a part of it, destroyed by ulceration, and deep cicatricial notching left behind on the soft palate. I have further observed, in a number of children, complete perforations of the soft palate from the size of a pea to that of a bean either on one or both sides. In rare cases, I have seen the process extend into the middle ear through the Eustachian tube, leaving behind obstinate otorrhœa and deafness.

Thrombosis of the larger veins may occur during convalescence owing to the great debility and the lowered energy of the heart's action.

Alice M., 11 years old. Diphtheria 5 weeks previously, which left behind extreme debility with scarcely perceptible pulse. In the beginning of February, 1881, paralysis of the soft palate, galloping rhythm of the heart. Urine somewhat albuminous. Pulse 140, extremely small. On the 7th, œdema of the whole left lower limb, reaching [illegible]he inguinal fold with

great tenderness to touch and on movement. After 10 days, disappearance of all morbid symptoms under tonic treatment.

3. The severe form. The danger of diphtheria arises mainly from two circumstances: firstly, a paralysing influence on the nervous system of the heart, similar to that in scarlet fever; secondly, its tendency to spread from the pharynx into the air-passages (diphtheritic croup). These two sources of danger have to be taken into account in every case of diphtheria, whether the disease appears in its mildest form or is moderately severe. It is not at all uncommon for the pulse to become suddenly very quick and small (less commonly slow and irregular) and fatal collapse to set in rapidly within the first few days or later on in the disease; and yet the case may not have appeared at all serious up to that time. It is also common for croupy symptoms to develope all at once at a stage when the pharyngeal affection is passing away, and when everything seemed to promise a speedy recovery.

Occasionally diphtheria assumes its severest form at the very beginning, and this is usually indicated by an initial fever of great severity (104° ) and an unusually rapid pulse (140—160). This is accompanied by great apathy, drowsiness, complete anorexia, and often also vomiting. I regard the invincible anorexia as one of the most serious symptoms. The children refuse every kind of nourishment, and, as feeding by means of an œsophageal tube is often impossible owing to the pharyngeal affection, they have to be nourished by enemata (of peptone, beef-tea, or milk with yolk of egg, or wine); unfortunately, the result is generally unsuccessful. Along with the pharyngeal affection, which is not always very severe and may even be extraordinarily slight, there is always severe rhinitis with a copious fœtid secretion, œdematous swelling of the nose and often of the eyelids, and loud snoring—the children breathing with their mouths wide open. The voice is nasal and often difficult to understand. Many can hardly be got to speak at all, can only be wakened with difficulty, and then relapse into apathy and drowsiness. The breath is extremely offensive, and the salivation, which is occasionally present, has always appeared to me a particularly unfavourable symptom. The submaxillary lymphatic glands are much swollen, but I have only rarely seen an extensive board-like infiltration of the whole submaxillary

connective tissue, reminding one of angina Ludovici. In three cases, there was also a diffuse doughy swelling of one parotid region, reaching as far as the eyelids, while on the other side there was submaxillary adenitis. The hæmorrhages already mentioned, from the nose and from ulcers on the pharynx, generally come on with particular violence in the severe form of the disease, and hasten the fatal issue by the increasing exhaustion which they cause. We have often been obliged to plug the nose on account of this bleeding, or to inject solution of perchloride of iron into the nose or pharynx, and yet have failed to avert the imminent collapse. Petechiæ, also, and larger purpuric spots frequently appear on various parts, even on the ocular conjunctiva; the urine is scanty, and rarely free from albumen and nephritic elements. I have never observed the swelling of the joints, which some authors have mentioned; for in one case—that of a child of $3\frac{1}{2}$ years, in whom the joints of the right knee and foot were swollen for some days—I was uncertain as to whether the disease was real diphtheria or only scarlet fever. The eyes are frequently affected; the cornea finally becomes ruptured, and the whole anterior portion of the eyeball sloughs, leaving the lens uncovered.

In all descriptions of diphtheria, a great deal is said about eruptions which, it is alleged, appear in the course of the disease, especially when the case is severe; and they are described either as forms of diffuse erythema or else as more or less extensive roseola. Although I have always looked for these rashes carefully, I have only been sure of their presence in a comparatively small number of patients; and those were not always very severe cases, for sometimes they were of the moderately severe form and ended favourably. In a child of 2 years who died of collapse, there was a confluent red rash which appeared for the first time on the face and nates on the day of death, while papules the size of a pin's head were visible on the abdomen and back. In all the other cases there was merely roseola or erythema, very rarely papules or erythema urticatum, which lasted several days without there being any increase of the fever. I attach particular importance, however, to this very point, because I am convinced of the fact that the majority of the rashes which have been described as diphtheritic are neither more nor less than scarlet fever, and that their onset is always accom-

panied by a characteristic rise of temperature.[1] I have thrice seen varicella break out during the course of diphtheria, accompanied by a rise of temperature (104°); and this is a further example of two infectious diseases occurring together (p. 190). All the three cases recovered.

But among all the symptoms of the severe form, by far the most formidable is the tendency (already mentioned) to paralysis of the heart, which is indicated by steadily advancing collapse. The pulse gets quicker and quicker (160 and over) and becomes weaker, and is also often irregular in force and rhythm. The hands, feet, and cheeks are cold, the skin and visible mucous membranes somewhat cyanotic and sometimes jaundiced. The temperature does not always fall, for, as I have often observed, it may remain high (105°—105·4°) to the very last; or it may only reach its highest point on the last day. Still, cases of this kind certainly do also occur which run their course to the very end without any rise of temperature, especially when the larynx and lungs remain unaffected. Some children in this condition have severe vomiting, others are delirious, and most of them lie quite relaxed and in a comatose condition, with pale yellowish face, staring or half-open eyes, and can only be wakened with difficulty if at all. Still, I have met with such patients who, although their extremities were cold, and their pulse thready, were still quite conscious and could sit up and speak to their parents in a toneless voice. The pulse disappears entirely under the finger, and the apex-beat also gets weaker and weaker, and often irregular, and the second sound less distinct. The number of the respirations sometimes falls to 20 in the minute, unless there happen to be a complication with broncho-pneumonia raising the number more or less above the normal. When the child is in this state it is hardly possible to get a good view of the throat, but the presence of actual gangrene of the pharynx is often revealed by the unusually offensive and even gangrenous smell of the breath. When we do succeed in making an examination, we see one or both tonsils, part of the soft palate, and even the back wall of the pharynx transformed into a blackish, ragged, bleeding surface, with a smell like that of noma (the septic form). Often the case is terminated by an attack of broncho-pneu-

[1] The observations of Cadet de Gassicourt agree with mine. Out of 932 cases of diphtheria only 37 were accompanied by erythema.

monia, which, however, can scarcely be made out during life; for the cough may either be entirely absent, or may be so trifling as to be overlooked, and the rapid shallow breathing may be the only thing pointing to disease of the respiratory organs. An exact physical examination of the chest can scarcely be made, owing to the great general debility; besides, it is of no importance whatever for practical purposes. In two cases (in one of which tracheotomy was performed) there was, at the last, cervical rigidity, with bending forwards of the body, of which the post-mortem furnished no explanation.

As far as my experience goes, this form of diphtheria is almost invariably fatal, often very rapidly, within a few days or within a week at most; and it is only in cases where this form of the disease has developed out of the second (moderately severe) form, that death is deferred for 2—3 weeks. I am here speaking, however, only of those cases in which the fatal collapse sets in while the disease is at its height, and not of those (to be described by-and-by) in which paralysis of the heart proves fatal quite unexpectedly, after the local condition has been completely recovered from. I have only met with two cases of recovery from the severe form. One was in a child who had a temperature of 104°, a small shabby pulse of 144, noisy delirium and collapse, but only a slight local affection of the pharynx. The other case was that of a girl who had very severe pharyngeal diphtheria with great apathy, entire loss of appetite, numerous petechiæ, aphonia, croupy cough, and an extremely small and very rapid pulse. Both of these recovered very slowly under strongly stimulant treatment, to my great surprise. We must not therefore despair utterly even in these cases.

The extension of the diphtheria from the pharynx to the epiglottis, larynx and trachea in the form of croup is not quite so unfavourable. I have seen recovery taking place even after the disease had undoubtedly spread into the medium-sized bronchi. Even in the mildest cases you are never secure against the disease spreading in this way. Experience even proves that there is a stronger tendency to croup in the mild and moderately severe than in the very severe form just described, in which the general constitutional symptoms prevail. Still, I have frequently met with cases of the very worst septic form of diphtheria which ended with croup. The exact period at which

the diphtheria spreads to the larynx and trachea cannot be exactly determined; the laryngeal symptoms do not appear till, on an average, 4—6 days after, but sometimes not for a week or 10 days. Sometimes the local affection in the pharynx has been entirely recovered from, so that one has thought that all danger was passed; and then the sudden onset of croupy symptoms is all the more painfully surprising. In one boy of 8, I saw diphtheria set in 14 days after the beginning of diphtheria, of which nothing was left but two shallow ulcers, and tracheotomy was successfully performed. Cadet de Gassicourt also found tracheotomy necessary in three cases of prolonged diphtheria, on the 18th, 23rd, and even on the 43rd days. In a few cases the croupy symptoms mentioned on p. 279, continued for 1—2 weeks with varying severity, indeed with complete intermissions; and we thought that recovery had fairly begun, until presently croup set in and tracheotomy had to be performed. On the other hand, some cases have been reported in which the croup was said to have begun on the second day of the attack, or even earlier, as the first symptom of diphtheria. Still, I cannot help thinking that in all of these the diphtheritic affection of the pharynx or nose has probably been overlooked. I have often seen croup in children who, according to their parents' account, had been quite well only 24—36 hours before; and yet on examination we found that there was a diphtheritic affection of the nose and pharynx. But even when we find the pharynx apparently normal, this is no proof that it is really quite unaffected; for that can only be determined by a post-mortem. The physician is often betrayed into error by the fact of the diphtheria being situated out of sight. The examination of the pharynx is often so very difficult that one can only make out redness and swelling, with copious mucous secretion, and should there be any laryngeal obstruction one assumes it is due to simple inflammatory croup. And yet, at the post-mortem, a diphtheritic process may be discovered, limited to parts which (in most children) are beyond the reach of inspection during life—particularly the fossa pyriformis at both sides of the root of the tongue, and the epiglottis or even the posterior surface of the soft palate, the anterior being unaffected.

The pharyngeal diphtheria which precedes croup is most frequently overlooked in those cases in which it occurs at the

termination of some severe disease, especially in children with advanced tuberculosis, chronic pneumonia, severe typhoid, tubercular meningitis, intestinal phthisis, &c. I do not deny that I have myself frequently been taken at unawares by the sudden onset of croup under such circumstances, and was only led by this to examine the pharynx carefully. Similarly, we often find at the post-mortem of such cases diphtheria of the pharynx of which during life there had been no indication whatever, or at the most only offensive breath or coryza. To guard against such surprises, one would need to have the throat of every child in the hospital examined at least once a day—and that would be a good deal easier to order than to carry out. Fortunately, this "terminal" diphtheria is in any case such a hopeless condition, that really there is nothing very disastrous in our having overlooked it.

Least common of all are the cases in which the diphtheria of the pharynx only becomes visible after the croup has set in; and even then I am much more inclined to assume that the disease has existed for some time in a concealed position, and has only gradually come within sight, than that the case was one of diphtheria ascendens in the true sense of the word. Take for example the following case.

> Carl O., 4 years old, admitted into hospital with severe croup on January 26, 1878. Pharynx reddened, contained much mucus, but no membrane visible. Tracheotomy performed with success. On the 31st, fever with evening exacerbations to 103·1°—104·9°. Pharyngitis increasing; on February 4, a grey membrane on both tonsils seen for the first time. After this the temperature fell, and the child recovered.

I need say but little about the symptoms of diphtheritic croup, since they are just the same as those of primary pseudo-membranous laryngitis (vol. i., p. 365). Hoarseness, noisy breathing, and harsh cough interrupted by "sawing" inspiration are here also the earliest symptoms. I have but rarely found the voice still unaffected when the croupy inspiration was present. The state of the temperature varies very much in different cases. In many, it rises on the croup setting in, while in others it remains almost normal (100·2°—100·9°) even when the disease is fully developed. Indeed, in a few cases I have found the temperature 98·4°—99·3° just before tracheotomy,

although the accompanying symptoms did not indicate the presence of collapse. The pulse is always very rapid, varying between 140 and 180, while its fulness and tension rapidly diminish and it not uncommonly becomes irregular in rhythm. In a boy of 7, the pulse which had formerly been irregular, became intermittent shortly after tracheotomy had been performed, and a few days afterwards diminished in frequency (56—80) and assumed the character of the pulsus alternans (bigeminus) as described by Traube. After two beats which followed pretty quickly on one another, there was always a pause, then two beats and another pause, and so on. At the same time, the second beat was invariably lower and weaker than the first. This phenomenon was not affected by the respiration, and it only lasted three days and then ceased, the second beat disappearing gradually, and fatal collapse setting in. The extensive fatty degeneration of the muscular fibres of the heart which was found at the post-mortem cannot be looked upon as the cause of this phenomenon, as that condition is met with often enough in cases of diphtheria with or without croup, without any pulsus bigeminus having been observed during life. For example, in a boy of 10 years who died of diphtheritic collapse and in whom we found extensive fatty degeneration of the heart after death, I observed the pulse become very irregular towards the end, and fall from 132 to 72, but there was not the least trace of pulsus bigeminus or alternans. Moreover, I observed the bigeminal character in another case, in which there was no croup, but which was accompanied by a state of utter exhaustion which roused the utmost anxiety; the case ended fatally.

The expulsion of large fragments of membrane or tubular casts by coughing or retching is of no more favourable significance for the prognosis in diphtheritic than in simple croup (vol. i., p. 375). I have seen a child expectorate a complete cast of the trachea on the third day; and a boy of 12 brought up a small glassful of casts corresponding in their size to the trachea and main bronchi. In other cases I have seen tubular clots expelled from the medium-sized bronchi. Almost all these cases ended fatally, although in some of them there was a short period of improvement which aroused our hopes. For bronchitis and broncho-pneumonia arise even oftener in diphtheritic than in simple croup. The presence of these affections,

as I have already said (vol. i., p. 375), is mainly indicated by a considerable increase in the rate of the respiration and by increase of the fever, while the physical signs—apart from impairment of the percussion-note, which is not always found—are generally concealed or at least very much obscured by the loud laryngeal sounds. The expulsion of fine or dendritically branched coagula proves conclusively that the croupous bronchitis is spreading to the further ramifications of the air-passages.[1] The occurrence of these casts and especially the putrid bronchitis which I have seen in a few cases and gangrene of the lung occurring in small patches—are all, I think, to be attributed to the inhalation of fragments of diphtheritic or gangrenous tissue from the pharynx, which may act not only by exciting inflammation but also by directly infecting the small bronchi and the surrounding lung-tissue. It was not, however, possible to diagnose this putrid bronchitis or even circumscribed pulmonary gangrene in the cases I have met with; for there was no expectoration, and the offensive smell of the breath could quite well be explained by the pharyngeal diphtheria alone.

The prognosis is always considerably worse when the diphtheria is spreading into the air-passages. While out of 463 cases of pharyngeal diphtheria, 237 recovered and 226 died of collapse, broncho-pneumonia, and other complications—out of 384 in which there was croup, we lost 336. Only 48 recovered, and all these, excepting 3, had been tracheotomised—an extremely large mortality, to the causes of which I shall presently return. Thus, in spite of all the dangers which lie in the mere fact of being infected by diphtheria, we find that most of the deaths are due to the onset of croup. Its occurrence adds another fresh danger to those already present, for the tendency to fatal collapse lasts even after the disease has spread to the air-passages. And thus it often happens that in a case in which the immediate danger of choking has been warded off by an operation, and the child has been able to breathe perfectly well

[1] Some authors (Gerhardt and Riegel) base the diagnosis of croupous bronchitis on a peculiar kind of dyspnœa (absence of inspiratory indrawing and of movement of the larynx with respiration, weak breathing in the upper parts of the lung). I do not know whether these symptoms are always present, but I have found them in one case at least, that of a girl of 3 years, who suffered from great dyspnœa and brought up cylindrical coagula (some of which were dichotomously branched and from 1¼ to 1½ inches long) in such quantity that in the course of 36 hours she filled several wine-glasses (temp. 102·2°—104°; resp. 44).

without a tube for some time, it dies quite unexpectedly from collapse.

At the post-mortem, in cases which have died when the disease was at its height, we always find much more serious and extensive changes than the difficult and unsatisfactory examination during life has led us to expect. There is a ragged membrane of a dirty grey colour with a brownish or greenish tinge covering to a varying extent the posterior pharyngeal wall, the soft and, less commonly, also the hard palate, the tonsils, the posterior part of the tongue, and the mucous membrane of the nose; and this can sometimes, on careful dissection, be followed into the nasal ducts and Eustachian tubes. Generally the exudation is infiltrated into the superficial layers of the mucous membrane in such a manner that it cannot be stripped off, but has to be scraped away with a scalpel, leaving a raw surface just like that which is left when the patch separates spontaneously. This infiltration is also often found in the mucous membrane of the epiglottis, and, if the case has been one of croup, in the upper portion of the larynx extending as far as the vocal cords; while lower down in the larynx, and also in the trachea and bronchi, the exudation lies so loosely on the surface of the mucous membrane that one can pick it off with forceps or wash it away with a stream of water, leaving the subjacent mucous membrane more or less reddened but otherwise intact. This character of the exudation, which depends perhaps on the different kinds of epithelium (squamous or cylindrical) is not, however, always found; for we have often been able to strip off the deposit from the pharynx and tonsils pretty readily in the form of a more or less thick membrane, and it was only found to adhere rather more firmly to the enlarged follicles. On the other hand I have several times observed an exudation on the mucous membrane of the trachea and even of the bronchi, which was "diphtheritic" in the anatomical sense of the word, *i.e.*, infiltrated and yellow, and which could only be removed by scraping with a knife, leaving a raw surface behind. From this fact you perceive not only that both the forms of exudation, the infiltrated and that which lies free on the surface of the mucous membrane, may be met with close together, but that the nature of the epithelium by no means always determines the form of the exudation.

After the false membrane has been removed, the mucous membrane of the pharynx and of the upper part of the respiratory passages, appears reddened to a varying degree, cyanotic and œdematous. The tonsils are often much swollen from being infiltrated with exudation, and occasionally they have small recent or caseous deposits of pus. Less frequently, I have seen abscesses in the retro-pharyngeal connective tissue. Actual gangrene of the pharynx with discoloured greenish-brown sloughs and gangrenous fœtor is also of rare occurrence. It is much commoner to meet with more or less extensive ulcers, of the tonsil especially. These may be either superficial or deep, and are covered with discoloured *débris*, which arises from the separation of portions of affected mucous membrane which have sloughed. We may also find considerable destruction of the tonsils, soft palate and uvula, and also—as I have already (p. 282) mentioned—perforation of the palate on one or both sides. In many cases the diphtheritic process extends to the commencement of the œsophagus, the mucous membrane of which is found to be infiltrated, hæmorrhagic, or covered with very sinuous ulcerations. Rarely we find similar alterations in the mucous membrane of the stomach, especially in the pyloric region, in the form of a bloody infiltration covered by a discoloured false membrane. In one case, the tough whitish deposit which lay loosely on the surface of the reddened and swollen mucous membrane of the stomach extended more than 1½ inches into the duodenum. The submaxillary lymphatic glands are almost invariably enlarged; less commonly there is serosanguinolent infiltration, or even phlegmonous inflammation of the surrounding connective tissue. Least commonly of all, we find gangrenous destruction of it. Only in two cases have I found actual purulent parotitis, with suppuration of the surrounding connective tissue. In these cases in which the process has spread into the air-passages,[1] we find the changes which I have already described in speaking of the croup—infiltration and ulceration of the epiglottis and of the ary-epiglottidean ligaments, œdema of these, degeneration of the laryngeal muscles, rarely necrosis of the cartilage, membranous deposit and super-

[1] In "septic" cases, aphonia may be the only symptom during life of the laryngeal affection, and yet at the post-mortem we may find false membrane in the larynx, and even in the trachea.

ficial ulceration of the vocal cords, membranous exudation in the larynx, trachea and bronchi in the form of fragments and casts, and a varying amount of redness, swelling and hæmorrhagic spots in the mucous membrane of the parts, their lumen being filled with purulent matter. The false membrane varies very much, both in extent and thickness, in many cases these are only fragments of thin deposit on the trachea, while in others thick greyish-white cylinders are formed, which coat the bronchi and reach far down into their ramifications, affording complete casts of their interior. But these casts often occur only here and there in the bronchi, and are separated from one another by unaffected spaces filled with mucus. In several cases, we found that only one lung was affected by this croupous bronchitis, while in the other there was merely a catarrhal condition. I have already mentioned that there may be a regular diphtheritic infiltration of the mucous membrane of the trachea and bronchi. There is almost always more or less broncho-pneumonia, and it is often accompanied by numerous patches of atelectasis of the lung-tissue, as well as by adhesive pleurisy, œdema of the lungs and emphysema of the margins and of the upper lobes. Only in a few cases have I found putrid bronchitis and little gangrenous patches in the lung-tissue. The tracheal and bronchial glands are nearly always swollen, and sometimes hæmorrhagic and infiltrated. The muscular tissue of the heart is often flabby, reddish-grey, and opaque, and under the microscope it appears fattily degenerated. These changes are seen specially in the left ventricle and its musculi papillares, while the endocardium never presents any alteration worth speaking of save, perhaps, small ecchymoses. In particular the endocarditis which was stated by Bouchut and Labadie-Lagrave to be almost invariably present, was never found in any of our cases. Small ecchymoses nearly always occurred, and in the most various situations—in the pericardium and endocardium—the muscular tissue of the heart, the pleura, omentum, mesentery, mediastinum, and skin. The liver was usually flabby, reddish-yellow, fattily degenerated, the spleen often (but not always) hypertro phied. The kidneys were nearly always in a condition of parenchymatous nephritis, the mesenteric glands, the Peyer's patches and even the solitary glands of the intestine were sometimes enlarged to a moderate degree. In several cases we found, along

with diphtheria, an abundant deposit of thrush in the pharynx and œsophagus, and in one case, also thrush of the gastric mucous membrane.

All the attempts hitherto made to supplement these naked-eye appearances by the aid of the microscope, and to obtain an insight into the real nature of the disease, have as yet led to no certain results. There is no unanimity with regard to the mode of commencement or the composition of the diphtheritic deposits and infiltrations. Besides the ordinary conception of it—as a fibrinous exudation with profuse proliferation of nuclei in the superficial layers of the mucous membrane—there is another, according to which a peculiar degeneration of the epithelial cells is held to be the chief cause. Then there is also the parasitic theory, which finds its collections of bacteria not only in the pharynx but far beyond this, in the renal canaliculi and in many other parts of the body—although it is not able to prove that these bacteria are really the specific elements of infection and not merely the carriers of a general septic process. Moreover, the fact of bacteria having formerly been found in the renal canaliculi, and even in the urine of diphtheritic patients, has been rendered more than doubtful by the negative results of the most recent researches (Fischel,[1] Weigert, Fürbringer[2]).

As to the significance of diphtheritic nephritis (p. 164) we must, therefore, content ourselves in the meantime with knowing that it is due to the influence on the kidneys of an unknown poison. An important difference between this form of nephritis and that in scarlet fever consists in the fact that it almost always sets in at the height of the diphtheria, about the fourth day or so, its presence being manifested by more or less copious albuminuria, generally by diminution of the quantity of the urine and by the discovery of nephritic elements in it. Further, the bloody discolouration of the urine which is so common in scarlatinal nephritis is here only exceptionally met with. Albuminuria is very common in diphtheritic patients, occurring in about half the cases. We have often enough found an opalescent opacity and slight precipitation of albumen in perfectly

[1] *Zeitschr. f. klin. Med.*, vii., Heft 5.

[2] Virchow's *Archiv*, Bd. 91.—With regard to the anatomical relations *cf.* also Brault, *Journ. de l'anat. et de la physiol.*, 6. 1880, and Fischel, *loc. cit.*

mild cases. Only where the coagulated albumen amounts to one-third or more in the test-tube, and where there are numerous nephritic elements, are we justified, I think, in giving a more unfavourable prognosis, for I have hardly ever observed this save in serious cases. However, this rule also is not without exceptions. These cases, even after the diphtheria has passed off, generally last for weeks and delay the convalescence, which is slow under any circumstances. These cases also present a striking contrast to the scarlatinal form of the disease; for while in the latter, at least half of the cases show a varying amount of dropsy, this is very rare in the diphtheritic form according to the experience of all writers. I have myself only observed œdema of the face, feet, and scrotum in 8 cases, and have scarcely ever met with uræmic symptoms. Others say that they have seen them in rare cases, and I myself can adduce the case of one child of 3 years, who took ill of diphtheria on April 13, 1884, had albuminuria, vomiting and collapse on the 19th, œdema of the face on the 25th, and died, with violent convulsions, on the 26th. At the post-mortem, we found severe parenchymatous nephritis and œdema of the brain.

I have frequently seen the albuminuria disappear along with the diphtheria, and the urine remain free from albumen for a week and longer; then suddenly the condition would return, looking as if it was really a sequela, as in the case of scarlet fever. In order to be able to assume that this was actually the case (which it very rarely is) we must be able to prove that the urine has remained constantly free from albumen during the whole course of the diphtheria. And I was able to do this in the only case of this kind which I have met with.[1]

> Marie St., 4 years old, suffering from hip-joint disease, on January 14, 1874, took a slight attack of diphtheria beginning with some rise of temperature (ev. 101·1°). This had disappeared on the 22nd, but a deep ulcer was still visible on the right side of the soft palate. The general health was perfectly good. No rash was ever observed, and the urine, which had been examined several times, was always found free from albumen. On the

[1] The existence of primary diphtheritic nephritis—*i.e.*, of nephritis commencing without there having previously been any diphtheritic symptoms in other parts—has sometimes been assumed, but never proven.

31st, *i.e.*, 17 days after the onset of the disease—the child was ill-humoured, pale, complaining of vague pains, and vomited frequently. Temp. 101·7°. On the following day, œdema of the face, urine scanty and very albuminous. These symptoms lasted for more than a week with a moderate amount of fever (temp., m. normal; ev. 100·4°—102·2°), and were accompanied by catarrh of both lower lobes. The urine during this time always contained an abundance of urates, and the amount of albumen varied—on some days there was not a trace of it. Tube-casts were found only rarely, epithelial cells often. After 11th February, the œdema and albuminuria disappeared. Treatment, acetate of potash.

Unfortunately, even after the diphtheria is cured and the consequent kidney-affection has disappeared, we cannot be quite certain about recovery. Risk of sudden death from failure of the heart's action hangs over the convalescent like the sword of Damocles, and when death does thus occur, the shock to the relatives is all the greater because of the confident hopes which they had begun to entertain.

Clara R., 9 years old, suffered in December, 1873, from a moderately severe attack of diphtheria of the pharynx, and after about 10 days was completely restored to health. The child had recovered her appetite, looked quite well, sat playing in bed, and all danger seemed to be over. On the 12th day, after the slough had entirely separated, I happened to call in and found her sitting in bed and playing. She looked well, but to my surprise her pulse was 144 and strikingly small. The apprehensions which I at once expressed to the parents were only too well borne out, for although we had recourse to every kind of restorative, the rate of the pulse steadily increased, the strength rapidly sank, the extremities became cold, the pulse imperceptible, the respiration intermittent, and death occurred on the following day.

Unfortunately, I have met with many similar cases, and I therefore regard it as the duty of the physician, even in apparently mild cases of diphtheria, not to give an absolutely favourable prognosis until 4—6 weeks after the diphtheria has been recovered from. Indeed, I had one case in which sudden death from syncope took place at the end of the seventh week. The same holds good with regard to those cases in which tracheotomy has had to be performed. In them also, everything may go on as well as possible for some time, and then—when the tracheotomy wound is already in process of recovery—fatal

collapse may suddenly set in. The symptoms of collapse are not always the same. Sometimes, repeated vomiting is the first thing noticed; less commonly, severe cardialgia; the pulse becomes slow, weak and irregular, much more commonly it is very rapid and small, while the extremities become cold, the skin somewhat cyanotic, and the heart-sounds (especially the second) get weaker and weaker and may perhaps have a "galloping" rhythm. There is not always dyspnœa, but the respiration is extremely rapid (50—70); the children moan and murmur and sink into an apathetic and finally comatose condition, which I have seen lasting from a few hours to three days. These cases invariably end fatally, sometimes even within a few minutes, although in a few cases we succeeded, by the use of strong restoratives, in keeping the heart going for several days.

Otto T., 6 years old, admitted with diphtheria on September 15, 1877. Chronic course. The pharynx was free for the first time on the 8th October, and the fever had gone. Slight albuminuria, which after the 13th was only occasionally found. The child seemed quite well in its general condition. On the 19th, *i.e.*, at least 4 weeks after the commencement of the diphtheria, sudden symptoms of collapse. Pulse 152, very small, cold extremities, great apathy and weakness. After this, a large tablespoonful of sherry or tokay was given every hour, and this was continued for 3 days until the child was slightly inebriated (redness of cheeks, cheerful delirium, and continual chattering); meanwhile the pulse improved and the hands became warm. In spite of all, however, the collapse got the upper hand at last, the former symptoms returned, Cheyne-Stokes' breathing developed towards the end, and death took place.

There cannot be any doubt that these cases are due to a paralysis of the heart, although after death we often find more or less extensive granular and fatty degeneration of the heart-muscle or the appearances of myocarditis in a few cases,[1] still, it is questionable whether these changes are really to be looked upon as the cause of the fatal collapse, or whether a diphtheritic paralysis of the cardiac nerves, especially of the vagus, is not rather to blame for this termination. This, at any rate, is certain, that in several cases of this kind we found far the larger part of the muscular tissue of the heart perfectly

[1] Leyden, *Deutsche med. Wochenschr.*, 1882, 7.—Unruh, *Jahrb. f. Kinderheilk.*, xx., S. 1.

unaffected and its striation normal. Nor did we find the large blood-clots in the cavities of the heart or in the trunk of the pulmonary artery, which many have described as the cause of death. The important connection between the nervous system and the production of this cardiac paralysis, seems to me to be indicated by the fact that these cardiac symptoms not uncommonly set in in the course of diphtheritic paralysis even in ordinary cases of paralysis of the palate only, and cause death from syncope almost at once. In April, 1879, I saw a boy of 8 years become completely ataxic about 14 days after recovering from diphtheria, and he died a few hours afterwards with severe dyspnœa, stertorous breathing and disappearance of the pulse. Less commonly, this termination only takes place after the threatening symptoms have lasted for some days.

Otto M., 9 years, examined by me for the first time on October 3, 1876. Five weeks previously, he had had a moderately severe attack of diphtheria, which had been followed by paralysis of the palate. This was now almost recovered from, and the boy had been out of hospital for a week. Ataxia and paresis of both lower extremities had set in a few days before I saw him. He was quite unable to stand or walk; while sitting or lying, the legs could be moved pretty well, but clumsily. The arms were also weaker than in the normal condition. Further, the pulse was extremely rapid (150 and more) and very irregular, and this made me fear that paralysis of the vagus was about to set in. Heart otherwise normal on examination. Also paresis of the left abducens, so that the eye could not be moved outwards. The motility gradually improved under the use of decoction of cinchona and daily injections of strychnia (gr. $\frac{1}{12}$), while the pulse remained unchanged—indeed, the irregularity in rhythm and force of the single beats rather increased. At the same time it continued to get smaller and more rapid, and collapse set in with repeated vomiting on the 7th day after the onset of the first serious symptoms. The boy remained conscious to the end, but death came on very rapidly. Post-mortem not allowed.

A case which Dubrisay[1] has published proves that recovery may take place in such cases even after the threatening symptoms have lasted much longer than they did in the above; and I have myself seen a few cases—which I shall refer to when I come to the treatment—in which the most threatening symptoms recurred, for a week and longer, and yet the patient recovered.

[1] *Centralzeit. f. Kinderheilk.*, i., S. 72.

The longer the case lasts, the more probable, I think, is it that recovery will finally take place.

Diphtheritic paralysis is such a common sequela of diphtheria that we must be on the look-out for it in every case. I myself have never seen paralysis following any form but pharyngeal diphtheria; but other writers say they have observed it after diphtheria of the skin, *e.g.*, of the finger. In spite of all researches there is far from being unanimity of opinion with regard to the nature of this paralysis. The remarkable fact about it is, that the *materies morbi* does not seem to exert its action on the nervous system until long after the patient has, apparently, got over the original disease. Déjerine[1] has found formation of fatty granules and disappearance of the axial cylinders in the anterior roots of the spinal nerves, and also in many of the peripheral nerves. Likewise atrophy of the ganglion-cells in the anterior horns and increase of the interstitial connective tissue—that is to say "parenchymatous neuritis and myelitis." P. Meyer[2] has also seen distinct signs of parenchymatous neuritis (disintegration of the medulla, proliferation of nuclei in the white substance of Schwann, formation of granular cells, the occurrence of nodules due to œdema and swelling of the connective-tissue) in almost all the peripheral nerves in cases of very extensive diphtheritic paralysis. The same changes were found in the roots of the spinal nerves and in many spinal ganglia, while in the spinal cord itself many of the ganglion-cells had lost their processes either altogether or partially. These results of microscopic examinations, as well as some observations which were made in 1862 by Charcot and Vulpian and in 1867 by Buhl, show the necessity of further investigation of the peripheral nervous system in diphtheritic paralysis. Also, in one case of diphtheritic paralysis of the heart with sudden death, Gombault[3] found the vagus, the medulla oblongata and the muscular tissue of the heart quite unaffected; and yet (just as in two other analogous cases) the anterior roots of the spinal nerves were affected, at least partially, in a similar way to that which was described by Déjerine. There can hardly be any doubt that the paralysis of the palate is of the nature of peripheral paralysis.

[1] *Jahrb. f. Kinderheilk.*, 1878, xiii., S. 132.—*Arch. de physiol. normale et pathol.*, 1878.
[2] Virchow's *Arch.*, Bd. 85, Heft 2.
[3] Cadet de Gassicourt, *loc. cit.* iii., p. 349.

Diphtheritic paralysis occurs most commonly after the milder attacks of the disease, and generally shows itself first in the form of paralysis of the palate, two or three weeks after the diphtheria has been recovered from. Less frequently it occurs earlier. Thus, in one of my cases it appeared on the 5th day of the disease, and death from paralysis of the heart occurred a few days later. In many cases the affection of the palate is the only paralytic symptom; the children's speech becomes nasal and more or less unintelligible, and whenever they drink, some of the fluid returns at once through the nose. When we examine the pharynx we notice that both during inspiration and phonation the soft palate is almost, if not quite, immovable and hangs loose, so that during drinking the pharynx is not completely shut off from the nasal cavity, and fluids therefore find their way through the posterior nares into the nose. In many cases only one half of the palate is paralysed, so that on phonating it is drawn to one side. Sometimes I have found it insensitive, *e.g.*, a touch with a brush was not felt and did not excite a reflex contraction. This paralysis of the palate may, as I have frequently observed (and especially among the lower classes), be the first sign which reveals the fact that there has been an attack of pharyngeal diphtheria which has been quite overlooked by the parents and has recovered without any treatment. That such an oversight should occur is, in my opinion, more probable than the suggested explanation that in such cases the diphtheritic paralysis may be primary, *i.e.*, that it may be the first manifestation of the diphtheritic infection—just as some are occasionally inclined to assume a primary diphtheritic nephritis without there having been any previous pharyngeal affection.[1] Very often there are also disturbances of vision, especially inability to read distinctly or to see things distinctly at the same distance as usual, a flickering haze and mist before the eyes, diplopia — symptoms which are produced by the derangement of accommodation due to paralysis of the ciliary muscle. The movements of the pupil are often sluggish, but they may be quite normal. The vision for near objects is especially apt to be impaired; thus *e.g.*, one of my little patients did not know, when writing, whether he was keeping on the same line. Reading for any length [illegible] and especially when the

[1] Guidi, *Revue* [illegible]

print is small, is often impossible. Most of these children are in an anæmic condition, due to the disease from which they are recovering, and there may still be albumen in the urine. As gradual recovery very often takes place in the course of a few weeks without the aid of art, we must not overestimate the value of the different methods of treatment which are recommended.

In another set of cases, the paralysis spreads further, but in these also, the affection of the palate and of accommodation are nearly always the first symptoms to occur; but sometimes, though less commonly, these are either entirely absent or they have already recovered before the paralysis affects other parts of the body. I have pretty often met with paresis of the muscles of the neck, so that the head fell forwards and could only be held erect with difficulty, unless help was given; and I have seen this even in cases where there was no other paralysis excepting that of the palate and ocular nerves. The next thing to appear is ataxia and powerlessness of the lower extremities, so that the patient can only stand or walk with great difficulty if at all; consequently, the patients throw their limbs about as if they had locomotor ataxia and are apt to fall, especially when they are turning round. There may also, as Trousseau observed, be a swaying to and fro of the body when the eyes are shut. The paresis and ataxia but rarely amount to complete paralysis, and when such is the case the upper extremities also may be affected. I have myself only very rarely seen complete paralysis of the extremities or of individual cranial nerves (facial, oculo-motor, abducens), which one sometimes finds mentioned.[1] I have twice seen paralytic aphonia, which was cured by the faradic current; more frequently I have met with symptoms of paralysis of the respiratory muscles. When this is present, the breathing becomes very shallow, laboured and rapid (50—60), if there is a cough, it is ineffective and unable to dislodge the masses of mucus which accumulate in the bronchi. As any moderately severe attack may under such circumstances lead to death from suffocation, the affection of the respiratory muscles must be looked upon as a very formidable variety of diphtheritic paralysis, only less dangerous than the cardiac paralysis already

[1] Uhthoff (*Jahrb. f. Kinderheilk.*, xxii., S. 327) describes a case of almost complete bilateral ophthalmoplegia.

described. The dysphagia caused by paralysis of the palate is seldom so great as to give rise to an amount of exhaustion sufficient to endanger life seriously; for the patient can generally swallow solid food. Paralysis of the sphincters I have not seen except in the last stages of the disease, and I have never been able to ascertain the presence of any considerable disturbances of sensibility—either anæsthesia, analgesia, feeling of coldness or even hyperæsthesia. But as it is particularly difficult to ascertain the presence of these conditions in the case of children (and, indeed, in the case of little children it is generally quite impossible) I shall not dispute the accuracy of the observations of this kind which have been made in adults. Still, I must point out that the electrical conditions and the nutrition of the paralysed muscles in my cases remained undisturbed, even after the paralysis had lasted a long time; but others have observed diminution of the electrical excitability or the reaction of degeneration. In all cases, however, even those in which there was only paralysis of the soft palate, the tendon-reflexes (especially the patellar) were almost invariably absent and generally they did not return for some months.[1] All the more remarkable, therefore, was the case of a boy of 12 years who had paralysis of the palate and of accommodation at the beginning of the 5th week of the disease, but yet presented a normal, and on the right side a rather increased, patellar reflex.

It is still open to doubt whether diphtheritic paralysis may occur in the form of hemiplegia. Writers are not at one on this matter. I have not myself seen any case as yet that I could with certainty regard as diphtheritic hemiplegia. In one girl of 8 years, I observed paralysis of the entire left side of the body, which set in suddenly with gradually increasing symptoms of collapse, but at the post-mortem we found that it was caused by embolism of the right Sylvian artery, which had originated in a marasmic thrombus in the left side of the heart, and I am therefore probably right in thinking that another similar case which recovered (and to which I shall shortly return) was to be explained in the same way.

[1] According to Bernhardt (Virchow's *Arch.*, Bd. 99) the patellar reflex pretty often disappears after the diphtheria, even although there are no paralytic symptoms, and this phenomenon sometimes does not appear till 6—8 weeks after the disease has passed off, and it may last for months.

Treatment. I can only repeat to-day the opinion I expressed in 1874.[1] "According to my experience all the remedies hitherto recommended (and, with the exception of preparations of sulphur, I think I have tried nearly all of them) are of no use whatever in severe cases of the disease; and it is only in such that they are wanted, for the slighter cases recover without treatment." I believe all physicians of experience will agree with me in this. The fact that we find such marvellous things (not altogether untrue) reported of such an immense number of remedies, and that methods are sounded abroad under the use of which, it is alleged, scarcely any patients ever die —is simply due to this, that these vaunted remedies have been followed by success in slight cases, or at any rate in moderately severe ones, or perhaps in the catarrhal and croupy (p. 15) sore-throats, which are so often mistaken for diphtheria. In really severe cases they are absolutely worthless. You will therefore excuse me if I do not here recapitulate the numerous remedies which one finds mentioned in medical literature, and which I have never seen used with success. Although my duty as Director of the Children's Clinique has made it incumbent on me to have every new remedy tried, I must confess that the innumerable mischances have caused me to perform this duty with reluctance and distrust. Old and half-forgotten methods are always being re-discovered and extolled by young practitioners, and, especially, the endeavour to destroy the supposed bacteria is always producing new "antiseptic" remedies; but when the latter are more rigorously tested they are found to have at most only a favourable local action—and in many cases not even that. And how can we expect it to be otherwise in a disease in which the general infection of the whole organism is the most important factor, and the local pharyngeal affection has become of secondary importance, at least by the time at which the patient generally comes under treatment. The consequence is that after a few months we hear of these wonderful remedies no more. I certainly should not find fault with any practitioner for not losing all hope of finding a specific for this fearful disease—only, it is to be desired that men should not recommend such things until they have submitted their observations to the most searching criticism; and still more, that the remedy recom-

[1] *Charité-Annalen*, Bd. i., S. 589.

mended should not be found to exert a deleterious secondary influence. We had very recently an example of what I mean, in the silly and utterly irresponsible way in which people took to praising pilocarpine, and I have myself repeatedly seen instances of its deleterious action in this disease, in which there is, in any case, such a tendency to collapse.

Of all the infectious diseases, malarial fever is the only one for which we know of a specific. For scarlet fever, measles, small-pox, typhoid, cholera, plague, &c., we have long ago recognised the fact that we have no remedy, and entertain no great expectations of ever finding one. And the hope of being able to do so in the case of diphtheria must be moderated somewhat. Hitherto every hope has ended in disappointment.[1] We must, above all, guard against deceiving ourselves. If, therefore, I recommend to you the method of treatment which I have myself finally, and after various experiments, decided to adhere to, this is only because I regard it as at least rational, and because I have seen more than half of my cases—and some of them very serious ones—recover when thus treated. Nevertheless I say expressly "post hoc," not "propter hoc." Locally, I order gargling, and in very young children I have the pharyngeal and nasal cavities regularly syringed with a solution of chlorate of potash (2 per cent.), acetate of alumina (5 per cent.), carbolic acid (1—2 per cent.) equal parts of lime-water and distilled water, and, when there is a very offensive smell, permanganate of potash (about ¼ per cent.). The most convenient way of applying these remedies is to use them in the form of spray, for even in refractory children it is easy to force the thin nozzle of the spray-producer between the patient's teeth, and thus one can act equally upon all parts of the pharynx. The oftener this is repeated the better; in general, it should be done at intervals of 1—2 hours. We certainly cannot suppose that these injections have any specific action, destructive of the supposed "germs." The aim is simply to wash out the decomposed matters as thoroughly as possible, to remove the fœtor, and to gradually loosen and dissolve any exudation that remains adherent. Painting the affected parts with any sort of fluid, I do not regard as efficacious, because it usually throws the children into a state of great excitement; besides, when they are refractory it can only

[1] *C.* Lunin, *Petersb. med. W[illegible]r.*, 1885, No. 6.

be done by force, and in that case the mucous membrane is apt to be injured. Such injuries are to be avoided at any price, because any abrasions of the mucous membrane are almost certain to become infected with diphtheria. I cannot therefore understand what could have induced Letzerich to recommend that we should scratch off the false membrane with the finger nail; for this is just the very way to throw open a door whereby the "bacteria" may find entrance into the organism. For the same reason, also, I have long since given up all forms of cauterising the pharynx, although this was at one time held to be indispensable. And I am only surprised that methods such as these, which have long ago been tried and discarded, should be always cropping up afresh and finding new supporters. The local destruction of the bacteria, supposing the thing possible, can only be of use at the moment of infection, and not at a time when the false membranes have developed and the bacteria have long ago penetrated into the lymphatic and blood circulation.[1] When there is bright inflammatory redness and swelling of the pharynx, I order an ice-bag to be applied to the throat and let the patient swallow little bits of ice from time to time, in order to moderate the inflammation. For internal use, I recommend from the first decoction of cinchona (1:24—1:12) with liquor chlori, and also nourishing diet (milk, meat-soups, and wine), although the latter is often almost impossible to administer because many of the children entirely lose their appetite. When the repugnance to food is invincible, one must have recourse to nutritive enemata (meat-extract, peptone, beef-tea with egg and wine).

The treatment by which we endeavour to fulfil the indication of cleanliness, is antiphlogistic and tonic also, and has at least the virtue of being perfectly safe. I should strongly advise you against all forms of treatment which increase the tendency to collapse which is already present, especially against large doses

[1] Owing to the use of the galvano-cautery being so strongly recommended (Bloebaum, *Deutsche med. Zeit.*, 1885, No. 88; 1886, No. 39) I next tried Paquelin's thermocautery. The patient was put under chloroform with great difficulty, and the pharyngeal mucous membrane, which was characteristically infiltrated, was thoroughly cauterised by Dr. Bungeroth. The case ended favourably, but a submaxillary phlegmon formed which had to be incised. So far, I have not tried this method further, because I have seen a few entirely analogous cases recover just as quickly under quite simple tonic treatment.

of chlorate of potash (small doses, it is true, are harmless, but they are also quite useless) which may result in dangerous poisoning; similarly against large doses of quinine and salicylate of soda. In my experience hyposulphite of soda has proved utterly useless; likewise the much vaunted benzoate of soda, which we have used both internally and locally applied in the form of powder. I have succeeded no better with bromine, the inhalation of which failed to arrest the gradual spread of the process, or with chinoline, or, finally, with oil of turpentine. We administered the latter to the children mixed with wine (in teaspoonful doses).[1] I have also repeatedly tried carbolic acid without any success, both internally and in the form of subcutaneous injections in the neighbourhood of the hyoid bone (gr. $\frac{1}{2}$—$\frac{2}{3}$ each time). In proof of the inefficacy of this treatment I may mention the case of a boy of 9 years with fracture of the upper arm, who had a carbolic dressing applied to his arm on July 19, 1873, after removal of a portion of the humerus. The urine became blackish; but in spite of this distinct sign of absorption, the boy took diphtheria on 18th August, followed by paralysis of the palate. The use of arsenic and of subcutaneous injections of perchloride of mercury (gr. $\frac{1}{4}$ daily) which I tried in several severe cases, were similarly unsuccessful.[2] The local effect of perchloride of mercury (1:5000) was neither better nor worse than that of the other remedies which I have tried for the disinfection of the pharynx. Moreover, I regard continued use of this drug as not free from danger, even although the inhalation of a spray of $\frac{1}{2}$—2 per cent. solution of it repeated at intervals of 1—3 hours is said to have produced no serious symptoms of poisoning.[3] Again, I have very recently tried, without success in severe cases, the application as spray of a solution of corrosive sublimate acidulated with hydrochloric acid. I have no experience of the local use of iodoform,[4] but I have tried painting the pharynx with papayotin (also with pepsin) in many cases, and I have only found it locally efficacious in cases in which the false membrane was loosely attached, more

[1] *Cf.* Siegel, *Arch. f. Kinderheilk.*, vi., 46.—Bungeroth, *Charité-Annalen*, xi., 1886, S. 587.

[2] Kaulich, *Prager med. Wochenschr.*, 1882, Nos. 19, 20.

[3] Stumpf, *Münchener med. Wochenschr.*, 1887, 12.

[4] Korach, *Deutsche med. Wochenschr.* —Frühwald (*Wien. med. Wochenschr.*, 7, 1883) obtained no succ its use.

like that of croup; while in all serious cases presenting the characteristic infiltration it was of no use whatever.[1] But we know that in the first class of cases the separation of the patches takes place very readily without any treatment, and therefore I see no good reason for tormenting the children by painting the throat.

In the presence of diphtheritic collapse we are helpless, whether it sets in at the beginning or when the disease is at its height. The most highly prized stimulants—camphor, musk, large doses of wine, even given up to the point of intoxication (p. 297), strychnia, and the application of the electric current to the vagus—all are powerless against the mighty adversary. At one time, while I was in the habit of giving iron from the beginning in all cases of diphtheria, especially in the form of liquor ferri perchloridi and tinctura ferri chlorati ætherea, this sad termination was quite as common as it is now, when I have long given up these useless remedies. Only in exceptional cases have I seen recovery take place, and it came as a surprise to me, but I cannot say whether these recoveries were to be ascribed to the curative power of nature or to the remedies used—of which strychnia is best worth trying (in the way which I shall recommend presently).

Similarly, there is no remedy which can prevent the diphtheria from spreading into the air-passages. Unfortunately, we have to content ourselves with expectant treatment; and it is only when croupy symptoms set in that we have occasion to change the previous treatment. The antiphlogistic measures which we use in primary croup (vol. i., p. 367), are here to be altogether avoided. Not only are they worthless, but they may also become a source of danger by weakening the patient. The same may be said of emetics, from the use of which I have never seen any permanent good result; and they likewise often give occasion for anxiety owing to the collapse which they produce. In two or three cases, I thought that I had brought about recovery by the use of energetic mercurial inunction (grs. x., every 2 hours), and was therefore induced to try the same treatment in a larger number of cases; but the result was so unsuccessful that I can only ascribe the few successful cases to a happy chance, and I have now quite abandoned the mercurial treatment. At the

[1] Kohts und Asch, *Zeitschr. f. klin. Med.*, Bd. v.

same time I can recall the case of a boy of 1½ years old who was treated in the hospital by mercurial inunction on account of syphilis, and was even slightly salivated, but who, nevertheless, became affected with pharyngeal diphtheria at the end of this treatment. I certainly do not deny that diphtheritic croup is occasionally recovered from without treatment, and also under the use of very various remedies; and I have already (p. 279) referred to this; but these recoveries are at any rate exceptional, especially if the croupy symptoms have reached an advanced degree. Out of 384 cases of croup which I have treated, only 3 ended favourably in this way, while 40 which were not fit cases for tracheotomy died without an operation, and tracheotomy had to be performed in the remaining 341. This operation is, I am convinced, the only measure from which we can expect help, and I therefore advise you to perform it in all cases, except in patients who are already in their death-agony or in those who present very severe general symptoms. We need not be deterred by the tender age of the children; for although the prospect of the operation being successful is much more favourable after the end of the third year than before it, still there have been plenty of cases of recovery in children in the second or even in the first year of life. In my own ward, we have frequently succeeded in saving children of 2 and even of 1½ years by tracheotomy. It is of great importance not to perform the operation too late, when cyanosis, coldness of the extremities, and extreme orthopnœa have come on. Although even under these circumstances we should not shrink from the operation, still it is always advisable to operate as soon as the local signs of laryngeal obstruction (constant stridor with the breathing, croupy cough, indrawing of the jugular fossa and lower ribs) have set in. We have then nothing to lose by it and everything to gain. Even if we discover the presence of pneumonia, this is not, to my mind, a contra-indication, for the operation is often successful in spite of this complication. It is only when the croupy symptoms are accompanied by those of extremely severe general infection—drowsiness, coma, septic fever, purpura, shabby pulse, fall of temperature, and very extensive hard swelling in the submaxillary region—that I regard the case as unsuited for tracheotomy.

We must never forget that the operation only deals with the

laryngeal obstruction, enabling the child to breathe freely again, but having no influence on the disease itself. The diphtheria may become arrested after the operation and recover, but it is just as likely to spread into the bronchi (causing fibrinous bronchitis and broncho-pneumonia) or to prove fatal by collapse, and we may thus explain the large mortality among the patients operated on. Out of 341 cases on which tracheotomy was performed in the hospital, scarcely 15 per cent. recovered. But in connection with this we must take into account the very unfavourable conditions which we have hitherto had to contend with, and which have often exposed the children to the risk of fresh infection (especially that of scarlet fever) after the operation, and made a proper amount of ventilation exceedingly difficult to obtain.[1] You must also remember the fact that these children were operated on at every age and at all stages of the disease, that the majority belonged to the poorest classes, and that many were also tubercular. In private practice the conditions are much more favourable.[2] I must here again remind you that our statistics refer only to diphtheritic croup, and that the tracheotomies performed on children with simple inflammatory croup show much more favourable results (vol. i., p. 378). We have often had an opportunity of observing the well known fact that the operation has at certain times strikingly good results, while at other times almost all the children operated on die.[3]

The chief cause of the mortality after tracheotomy, as we learned from post-mortem examination of our cases, is bronchitis (in some cases croupous) and broncho-pneumonia; and these cannot be regarded as necessarily due to the operation, for they occur often enough in cases of diphtheria where there is no croup. Very rapid and shallow breathing is therefore always a

[1] I hope that the recently erected isolation-block, which has been constructed according to all the rules of hygiene, will have a favourable influence on the results of our tracheotomy cases. The comparatively brilliant results obtained in other institutions are, I think, mainly to be attributed to these local conditions, and to the fact of the patients being more robust (*cf.* Jenny's thesis, "Zur Tracheotomie bei Diphtherie und Croup im Kindesalter": Leipzig, 1881—which gives a rate of recovery of 42 per cent.).

[2] This, also, has to be kept in mind in considering the unusually favourable results of Ranke's operations, for all of his cases were either private or polyclinic patients (*Jahrb. f. Kinderheilk.*, xxiv., S. 225).

[3] Rosenthal, "Die Tracheotomien auf der Kinderabtheilung," *Charité-Annalen*, x., 1885.

bad sign, even before the operation, because it indicates that the bronchi and lungs are becoming affected. For the same reason, the expectoration of tubular (and even branched) casts through the tube after tracheotomy is a bad sign. Cases, like some I have seen, in which recovery takes place after continuous casts of false membrane ending in two or even many branches, have been coughed up or extracted, are of very rare occurrence. The bronchitic and pneumonic complications scarcely allow of any alleviation taking place in the condition after the operation; for although the symptoms of laryngeal obstruction disappear, the rapidity of the respiration increases (to 60—70 in the minute), the temperature remains at 102·2°—104° and over, and death generally ensues in the course of 24—48 hours.

In most cases, however, the operation brings about such a strikingly favourable change that the inexperienced and especially the laity begin to entertain the most confident expectations of recovery. Unfortunately, these are often bitterly disappointed. The breathing, which has been quiet, becomes rapid and laboured, the temperature again rises, and on examination we find that the implication of the bronchi and lungs which had formerly been dreaded has now actually taken place. We have no remedy which is certain to avert this danger. We have often tried the effect of filling the room with a cloud of watery vapour from a spray-producer (which is recommended by many[1]) or have ordered inhalations of a spray of salt-solution or of lime-water to be administered regularly through the tracheotomy-tube, and yet a large number of our patients have died of croupous bronchitis and pneumonia. But life may also be endangered by other complications which set in after the operation. Firstly, there may be diphtheritic infection of the incision; and this sometimes gives rise to extensive gangrenous destruction of tissue on the front of the neck. Then there may be erysipelas serpens which I have several times seen spreading upwards as far as the chin, and downwards to the epigastrium with the formation of a large number of bullæ, and ending fatally with high fever and collapse. Dangerous and even fatal hæmorrhage may also occur from the wound when it has become diphtheritic, from the divided thyroid body, or from the trachea where it has been eroded by

[1] Jacubasch, *Berl. klin. Wochenschr.*, 1882, No. 22.

the tube, and even from the innominate artery if it has been ulcerated into.[1] Another occurrence which is not uncommon, and which may be a cause of much anxiety, is the return of fluids through the tracheotomy-tube or through the wound, if the tube has been removed; solid food is more easily swallowed but has yet some tendency to get into the air-passages and set up violent coughing. The cause of this symptom, which occasions serious difficulty in the nourishment of patients after operation, we do not properly know. It can hardly be due to the epiglottis being incapable of shutting off the larynx during deglutition, for a like difficulty does not usually occur in the case of people who have lost a large part of the epiglottis. It seems more likely that it is caused by the glottis remaining open owing to paralysis. We may lessen this difficulty by introducing one of Trendelenburg's "tampon" tracheotomy-tubes, but I have also in several cases saved the children's lives by giving nothing but solid food (eggs, pounded meat), by the use of nutritive enemata, or by feeding through an œsophageal-tube; for the interference with deglutition does not usually last longer than a few days or a week at most. You can easily understand that there is a considerable risk in these cases of the children acquiring pneumonia owing to food getting into the air-passages. Still, the following instance shows that even when such a serious complication does occur, the case may ultimately recover.

Anna K., 6 years old, admitted on January 28, 1874, with diphtheritic croup. Tracheotomy on 29th, during which a number of fragments of false membrane were removed. During the ensuing week (until the 7th day) all fluids returned through the tracheal wound, and the child steadily lost strength (enemata of milk, yolk of egg and beef-tea, and later on Leube's meat-enemata). On 7th February she swallowed a soft-boiled egg for the first time, and after the 18th she could take any fluids (for a week past she had had twice daily 8 drops of liquor ferri perchloridi in a teaspoonful of finely pounded beef). There was albuminuria from the very beginning with tube-casts in the urine, and this lasted until 19th February (3 weeks). The tracheal wound became diphtheritic and the bronchial catarrh which already existed became complicated on the 16th day after the

[1] Zimmerlin, *Jahrb. f. Kinderheilk.*, xix., 1882, S. 39.

operation by broncho-pneumonia of the left upper lobe (temp. 101·8°). After the 18th, the fever disappeared. On 2nd March, the voice had recovered its normal tone. Discharged cured.

In 5 cases epileptiform convulsions set in about 12—48 hours after the operation, and ended fatally. Whether these were to be regarded as the initial symptom of an attack of pneumonia or as indicating inanition or as a uræmic symptom, I shall not decide. At any rate, nothing was found within the cranial cavity that could account for their occurrence.

When the case does well, the tracheotomy-tube may generally be removed on the 6th day after the operation; but this must of course always be done tentatively at first and under medical supervision. The removal of the tube may be delayed by considerable swelling of the mucous membrane (especially that covering the arytenoid cartilages) which narrows the lumen of the larynx. Thus, for example, in the case of a boy who was operated on on January 7, 1877, we could not take out the tube until the 31st, because up till that date every attempt to dispense with it at once occasioned difficulty of breathing, and on laryngoscopic examination we found that there was still a considerable amount of swelling of the mucous membrane. Under these circumstances we must bear in mind that a tracheotomy-tube which is allowed to remain unusually long in the trachea may, however well made, cause irritation of the mucous membrane by its pressure, and even give rise to an ulcer. Such ulcers are generally situated $\frac{1}{2}$—1 inch below the incision, and may occasion pretty severe hæmorrhages, and finally polypoid growths. When such growths have formed, removal of the tube is out of the question, and the children have just to wear them for an indefinite time until the growths on the mucous membrane are cured and the danger of obstruction is thereby removed. Tubes which are too large, and especially those which have sharp edges, are of course the most hurtful, especially if they are not removed and cleaned regularly.[1]

[1] On the whole, the operation is but rarely followed by any bad result. Neukomm (*Ueber spätere Folgezustände nach der Tracheotomie:* Zürich, 1885) found in 79 children whom he examined 1—3 years after tracheotomy, that 58 were quite normal while only 8 presented any derangement which could be regarded as due to the operation. Jenny (*loc. cit.*, S. 32) has seen marked functional disturbance (dyspnœa, hoarseness) as a result of the operation in only 2 per cent. of the children who were discharged as cured.

I shall never forget the case of a boy of 4 years who had been operated on in the city 4 weeks previously, and whose tracheotomy-tube, according to the father's statement, had never been removed from the wound during all that time. When the tracheotomy-tube—which seemed far too big for the case—was with difficulty removed in the hospital (March 6, 1878), a large quantity of bloody pus was discharged from the trachea and surrounding connective tissue. Two hours afterwards, dyspnœa set in so that we had to introduce a narrower tube, and on the following day we were able to remove this permanently.

Finally, I may refer once more to the fact that the children in my wards who had been tracheotomised showed a special tendency to become affected with scarlet fever, like all other patients with open wounds (p. 235). The scarlet fever often appeared within 24 hours or two or three days of the operation; it sometimes ended in recovery but more frequently in death. One of these children, a boy of 6 years, who had been operated on on March 10, 1877, and had at the time brought up many fragments of false membrane, suffered within the next few weeks from broncho-pneumonia and nephritis as well as from scarlet fever, and during all this time continued (sometimes for days running) to bring up fragments of false membrane out of the wound. In another case fragments of membrane were still, during the fifth week after operation, being expectorated through the tube, which for this reason we were afraid to remove. Cases of this kind, in which the croupous process in the trachea and larger bronchi persists for weeks after the operation, are upon the whole rare, and experience proves that they may end favourably. Instances have been published of cases in which fragments and casts of false membrane have continued to be discharged from the bronchi for 61, and even for 151, days after the operation, and yet recovery took place.[1]

We now come to the treatment of diphtheritic paralysis. The slighter cases, especially those in which only the palate is affected, often recover spontaneously or under simple tonic treatment (good diet, iron, fresh air). When recovery is delayed, I would recommend subcutaneous injections of strychnia, which I have used in a number of cases since 1874, and which

[1] Cadet de Gassicourt, *Revue mens.*, Janv., 1883.—Sanné, *Traité de la diphthérie*, p. 55.

were tried in adults about the same time by Acker.[1] Although this drug has been formerly recommended (*e.g.*, by Trousseau in the form of syrup of strychnia), it seems to have been generally regarded as a dangerous remedy to give to children. The large number of cases which I have treated in this way, however, proves that there is no danger when it is given to the children cautiously and in moderate doses.

In two cases which I formerly published[2] the patients had altogether $\frac{1}{6}$ and $\frac{2}{7}$ of a grain of sulphate of strychnia, respectively, before recovery took place. The rapidity of the improvement in these cases, which was noticeable even after the first injection, proved that the strychnia was either the cause of the recovery, or at any rate favoured it to a considerable degree. My subsequent experience has confirmed my favourable opinion of the drug.

Otto H., 7 years old, brought to the polyclinic on June 21, 1875. Diphtheria three weeks previously. During the last 8 days, nasal speech, dysphagia, food—especially fluids—returned through the mouth and nose. Soft palate immobile, but not insensitive. Vision for near objects disturbed, clouds before the eyes. Head bent forwards, and only raised with difficulty. Uncertain gait; child easily tired and unable to stand steady with the eyes shut. Health otherwise good. Injection of sulphate of strychnia (gr. $\frac{1}{65}$ and $\frac{2}{65}$) into the neck every second day. Improvement distinctly noticeable even by the 28th. Everything normal on 14th July, after 15 injections.

Ida W., 8 years old, brought on August 16, 1875. She had had diphtheria on July 3, and had now been well of it for a week. For 14 days at least, nasal speech, return of fluids through the nose, derangement of accommodation, paresis of the lower extremities. Iron given. At the same time injections of strychnia (gr. $\frac{3}{65}$) daily. Complete recovery after 11 injections.

Clara Z., 4 years old, brought on June 16, 1875. Diphtheria 3 weeks before. For the last 10 days, nasal speech, fluids returning through the nose, soft palate immovable and anæsthetic; uvula very flabby. Paresis of the bones. Strychnia (gr. $\frac{1}{65}$) injected into the neck daily. On the 22nd, after only 4 injections, drinking was easier. Dose now increased to gr. $\frac{2}{65}$. Recovery on the 30th after 10 injections.

Anna W., 7 years old, brought April 3, 1876. Diphtheria 5 weeks before. For the last 3 weeks, paralysis of palate and weak-

[1] *Deutsches Arch. f. klin. Med.*, Bd. xiii., H. 4 & 5.
[2] *Berl. klin. Wochenschr.*, 1875, No. 17.

ness of vision. Iron and injections of strychnia. The speech was a little better on the 5th; on the 7th, slight movements of the soft palate; on the 10th, speech and drinking better. On the 22nd, everything normal. Iron as after-treatment.

Child W., $3\frac{1}{2}$ years old, examined October, 1876. Diphtheria 4 weeks before, followed by paralysis of the palate, great weakness and pallor. Three days before examination sudden paresis of the legs, so that the child could walk with difficulty, and only when supported. Slight albuminuria. Iron and daily injections of strychnia (gr. $\frac{1}{65}$). Complete recovery after 14 injections.

Adolf D., 4 years old, brought to the polyclinic October 8, 1877. Diphtheria 3 weeks previously, and for the last 14 days very marked paralysis of the palate (speech almost unintelligible) and paresis of the lower limbs with ataxic unsteadiness. Health good in other respects. After 5 injections of strychnia (gr. $\frac{2}{65}$), speech already much more distinct, drinking almost normal, soft palate could be moved a little, walking better but still greater unsteadiness in turning round. On the 30th, almost all the symptoms had disappeared.

Elise S., 4 years old, admitted into the ward on December 17, 1877. Six weeks previously, diphtheria and croup. Successfully tracheotomised. For the last 3 weeks, paralysis of the palate and now of the lower extremities also. The former was now almost recovered from. Legs quite flaccid, absolutely immovable; the arms also weak, so that the child could not change her position at all without help. Sensibility normal. Strychnia (gr. $\frac{2}{65}$) injected daily. On the 21st, that is after 5 days, the knee-joints could be flexed. On the 23rd, could walk a little with support. Complete recovery after other 14 days.

Gustav K., 8 years old, admitted with unilateral diphtheritic paralysis of the palate on October 3, 1882. There was also at the same time a frequent powerless cough, dyspnœa, diffuse bilateral bronchial catarrh. Dulness and sharp râles at the lower part of the left back. Temp. ev. 101·3°. Diagnosis.—Broncho-pneumonia, threatening paralysis of the respiratory muscles, paralysis of the palate. Treatment.—Daily injections of gr. $\frac{3}{65}$ of strychnia, and later of gr. $\frac{4}{65}$. Internally, camphor gr. 3 every 3 hours, abundance of wine and strengthening diet; nutritive enemata of wine, yolk of egg and beef-tea, given on account of difficulty of swallowing. During the next few days expectoration better, but condition otherwise unchanged. After 10th October, improvement and disappearance of fever. On 1st November the strychnia was discontinued. On 22nd November discharged cured.

Girl of 6 years. Diphtheritic paralysis of the palate, aphonia. Paralysis of the extensor communis digitorum of both hands,

with the fingers in a position of flexion, ataxia and trembling of the legs. Patellar reflex absent. Recovery after 14 days under the use of strychnia-injections.

Girl of 9½ years took ill with diphtheria November 21, 1885. On the 25th, tracheotomy was performed on account of croup, and a piece of false membrane an inch in length was removed. Copious albuminuria. On 1st December, paralysis of the palate. On the 2nd, the tracheotomy-tube was removed. On the 7th, great weakness; pulse 52, irregular, small; fainting fits, cadaverous pallor. Improvement after injections of camphor; pulse 104. After the 10th, respiration short, shallow and laboured, 52; repeated vomiting. Albuminuria and dysphagia persisting, the latter absolute, so that the child had to be fed several times a day through an œsophageal tube. On the 18th, resp. 68, dyspnœic. Slight tracheal râles; examination otherwise normal. Pulse 120 small. Paralysis of the muscles of the neck—aphonia, and ataxia of the lower extremities. Treatment.—Injections of camphor thrice daily, and of strychnia (gr. $\frac{2}{100}$ and later $\frac{3}{100}$) once daily. After the 22nd the respiration improved, and by the 31st it had returned to the normal rate. Albuminuria gone. After January 1, 1886, "galvanism of the phrenic nerve" was also employed, but the daily strychnia injections were continued. While all the symptoms were improving, there took place between the 2nd and 12th an attack of hemiparesis of the left side of the face and body, which I regarded as due to an embolism (p. 302). Complete recovery in the middle of February.

Thus you see that even in severe cases like the two last given, with threatening respiratory and cardiac paralysis, we must not despair, but must rather persevere in administering strychnia and stimulants (camphor, wine). In such cases I should also advise you to use electricity at the same time. This agent is deservedly esteemed, and you must not infer, from my recommendation of strychnia, that I wish to undervalue it in any way. When the dysphagia produced by paralysis is complete, the administration of milk, yolk of egg and beef-tea through an œsophageal-tube as in the last case, is preferable to the use of nutritive enemata. Bodily rest cannot be too strongly enjoined in cases of diphtheritic paralysis. Any muscular exertion may cause serious mishap owing to overstimulation of the exhausted cardiac muscle. We ought, therefore, to confine the patients to bed as long as possible, especially those in whom the paralysis has extended beyond the palate. For the same reason baths are only to be used with great caution. But if, in giving

them, one avoids as far as possible all active movements of the patient, the carbonic acid ferruginous baths (Pyrmont, Schwalbach, and Cudowa[1]) are especially useful.

## V.—*Typhoid Fever.*

The following account is founded on 302 cases, of which 256 were observed in my wards in the hospital and 46 in private practice. In addition to these I have made use of a number of others of which I have only short notes and no regular clinical reports.

From these numbers alone you will see that the opinion formerly held, that typhoid is rare in children, was quite mistaken. To Rilliet[2] and Taupin[3] belongs the credit of having by their works disposed of this fallacy. The majority of the cases described by the older writers under the names "febris meseraïca" or "febris gastrica remittens" were really typhoid of the mild form which is mainly characteristic of childhood. More minute investigation of the pathological appearances and, especially, the use of the thermometer have removed all doubt about the matter.

The pathological anatomy of typhoid fever in childhood certainly does not present in every particular the same typical features as in adults. Although the parenchymatous changes in the internal organs (the myocardium, liver, kidneys, &c.), and the enlargement of the mesenteric glands are the same in both—yet there are certain differences within the intestine itself. Rilliet drew attention to the milder character of the process, especially the fewness and smallness of the intestinal ulcers, which he attributed to the preponderance of the so-called "plaques molles," *i.e.*, swelling of the Peyer's patches due to proliferation of the lymphatic cells in the follicles. On the other hand, the hard plaques—in which the soft infiltration not only affects the whole tissue of the gland, but also the underlying mucous membrane—are said by him to be but rarely found. It is, however, the latter that are most apt to give rise to large ulcers owing to gangrene penetrating deeply, while the former ultimately end in

[1] Scholz, *Ueber schwere diptherit. Lähmungen:* Berlin, 1887.
[2] "De la fièvre typhoïde chez les enfants," *Thèse*, 1840.
[3] *Journal des connais. méd. chir.*, Nov., Déc., 1839; Jan., 1840.

resolution by fatty degeneration of the newly-formed cells. It is difficult for any single person to dogmatise with regard to this view, which is shared by other French writers besides Rilliet (Barrier, Bouchut); for typhoid in childhood is such a mild disease that opportunities for post-mortem examination do not often present themselves, and we cannot arrive at any very reliable results by merely grouping together other people's observations. When Gerhardt, for example, states that he found ulcers in 29 cases out of a total of 43 post-mortems, he yet omits to mention the very important facts as to the nature and size of these ulcers. Of my 302 cases, only 21 were examined post-mortem.

1. A girl of 4 years. Duration of illness 11 days. The Peyer's patches extended far into the upper part of the ileum, but were not very large. Those situated in the lower part were all much swollen, and not very vascular, but extremely soft in consistence. The solitary glands were less swollen, the mesenteric glands, especially those in the ileo-cæcal region very large—a few being as big and even bigger than hazel-nuts, much reddened and of medullary consistence.

2. Girl of 3 years. Duration about 3 weeks. Numerous typhoid ulcers in the ileum. The intervening mucous membrane resembling that in dysentery. Mesenteric glands markedly soft and swollen.

3. Boy of 7 years. Duration unknown, but short. Enormous development of the Peyer's patches and all the solitary follicles, which projected very much all over the mucous membrane of the ileum. No ulcers. Mesenteric glands considerably swollen.

4. Boy of 3 years. Recovery after an attack lasting 8—10 days. Death from diphtheritic croup 3 weeks after. Peyer's patches and the mesenteric glands enlarged, especially those in the ileo-cæcal region; no cicatrices of ulcers.

5. Girl of 10 years, duration 4 weeks. Relapse. A few ulcers in the ileum in process of healing. Solitary and mesenteric glands black and pigmented.

6. Boy of 10 years. Duration 13 days. Peyer's patches and solitary glands very much hypertrophied, the latter condition also found in the colon. Mesenteric glands also enlarged. No ulcers.

7. Girl of 4 years. Duration 4 weeks. Peyer's patches and solitary follicles in the ileum much enlarged. Just above the ileo-cæcal valve some pale-grey and yellowish gangrenous follicles as large as a millet-seed. Mesenteric glands soft in consistence, besprinkled with yellowish-white gangrenous patches. No ulcers.

8. Girl of 13 years. Duration 16 days. Four inches above the ileo-cæcal valve, 2 soft P[illegible]s with central ulcera-

tion and firmly adherent yellow sloughs. Further, just in front of the valve a number of diffuse, confluent, soft swellings with ulceration and sloughing. A single ulcer, almost clean, at the commencement of the colon. Mesenteric glands of medullary consistence and enlarged.

9—10. The 9th and 10th cases were those of children who had died of severe typhoid fever in the 3rd and 4th week. In both cases there was nothing but a soft enlargement and in some places a retiform character of the Peyer's patches. There was no ulceration.

11. Boy of 9 years. Duration about 28 days. Peyer's patches and solitary glands still moderately enlarged. No ulcers.

12. Girl of 4 years. Death in a relapse. Duration of the disease about 22 days. Retiform character of many of the patches; no ulcers.

13. Boy of 8 years. Death at beginning of 3rd week. Small superficial ulcers on some of the patches.

14. Girl of 2½ years. Death during a relapse. Entire duration about 7 weeks, that of the relapse 7 days. Peyer's patches soft, not ulcerated. Just above the ileo-cæcal valve 2 ulcers the size of lentil-seeds, with clean floor, extending down to the muscularis, with narrow laminated border.

15. Girl of 6 years. Duration at least 3 weeks. Patches swollen and soft with sloughing on some of them and some places denuded. A few follicles of the size of a lentil-seed and ulcerated. A few typhoid ulcers in the ascending colon.

16. Boy of 4 years. Duration 17 days. Patches much swollen, sloughy in some places and ulcerated, especially near the ileo-cæcal valve. Many sloughing follicles in the colon.

17. Boy of 5 years. Duration 21 days. Patches soft and swollen, partly clean, partly sloughy. On the valve, the mucous membrane almost entirely softened with extensive ulcerations. Ulcers also in the ascending colon, roundish, smooth, and with softened edges.

18. Boy of 4 years. Duration at least 3 weeks. Intestinal follicles enlarged. Peyer's patches very little swollen, a few ulcers on the ileo-cæcal valve.

19. Girl of 4 years. Duration 14 days. Patches and solitary glands soft and enlarged. No ulcers.

20. Boy of 10 years. Duration 14 days. Ulcers of the ileum and colon.

21. Boy of 8 years. Duration 3 weeks. Very large typhoid ulcers in the ileum.

Among these 21 cases, then, we find 11 with ulceration in the intestine, and this occurred in cases which had lasted from 14 days to 7 weeks. Only in cases 2, 17, and 21 had the

ulceration and the condition of the intervening mucous membrane the same characters as we often find in adults. In the remaining 10 cases we only found a soft swelling or a reticular condition of the glands without ulceration; and this was the case not only in those which had lasted merely a short time (13 days or less), but also in several (cases 7, 9, 10, 11, and 12) in which the fever had been protracted for 3—4 weeks. The comparative rarity of ulceration in infantile typhoid is therefore confirmed by my observations also. And in cases in which there were ulcers, these were generally less numerous, flatter and smaller (*e.g.*, only occurring in the centre of the patches) than is usually the case in adults. This fact is to be borne in mind in accounting for the rarity of perforation of the intestine and of hæmorrhages from it in infantile typhoid.

Then, peculiar soft swelling of the intestinal glands is found in typhoid fever even in children in the first two years of life, but in them it loses a great part of its significance, because at this age the Peyer's patches and the solitary glands become enlarged in a similar way in intestinal catarrh and in various infectious diseases. Moreover, they may show signs of inflammation and ulceration, even although no typhoid symptoms have been observed during life. On the other hand, such symptoms may have been present in the most characteristic form, and yet at the post-mortem the expected enlargement of the intestinal glands is not to be discovered, and we either find nothing at all worth mentioning, or merely the signs of inflammation in the mucous membrane of the small and large intestines (Rilliet and Barthez's "entérite typhoïde"). In my paper on infantile typhoid,[1] I gave a few such cases, which had occurred in the wards almost simultaneously in children of 6—7 months. In these there was diarrhœa, enlargement of the spleen, bronchial catarrh, otitis, drowsiness; and, above all, the characteristic temperature-curve. And yet, in the single case which was examined after death we found only one Peyer's patch enlarged and reticular in character, trifling enlargement of a few mesenteric glands, while the mucous membrane was healthy throughout and the spleen normal; there was broncho-pneumonia of the left lower lobe with serous effusion between the dura and pia mater. In such cases, which have also been observed by

[1] *Charité-Annalen*, ii., 1876; S. 542.

other writers (Barrier, Bouchut) and even in adults,[1] the clinical symptoms of typhoid were so well-marked that the mere absence of the ordinary pathological conditions must be considered of less importance. We must therefore assume that the latter may be either very slightly developed or may even be absent altogether, without our being therefore justified in denying that the case was one of typhoid.

Moreover, children under two years of age are much less frequently affected by this disease than older ones are. Of my 256 cases, only 8 were of this age while the majority (143) were between the 5th and 10th years, 50 were between the 3rd and 5th, and 55 between the 11th and 14th years.[2] The two sexes were affected in almost equal numbers. With regard to the seasons, there seems to be a special predisposition to the disease in autumn and at the beginning of winter. Out of 226 cases, 80 occurred during the months of October and November, 52 in July, August, and September, 16 in December, and 19 in March and April, while the remaining cases were distributed over the months of January, February, May, and June.

The contagiousness of the disease—if we are to assume its existence at all—is but slight in degree. I have never been in the habit of isolating patients with typhoid fever, but have always let them be along with the other children; and yet it has only happened two or three times that any of the children in the adjacent beds have taken the disease. And in all of these cases the typhoid patients were very young and passed their motions almost always in bed, so that perhaps the evaporation of the fluid fæces had here an infecting influence. The fact which we so often observe in private as well as in hospital practice, namely, of two or more children in the same family taking ill simultaneously, or one after the other (sometimes along with their parents) is better explained by supposing that the same injurious influence has been operative in all the cases, than by supposing that there has been direct transmission from one patient to the other. The worst cases I have seen have occurred in these family- or house-epidemics. Thus, in July 1881, I saw a whole family (mother and three children) carried off, the father alone escaping.

[1] Griesinger, *Infectionskrankh.*, S. 138.

[2] Montmollin (*Observ. sur la fièvre typhoïde de l'enfance*: Neuchatel, 1885) found among 295 cases only 15 under 2 years of age.

Under such circumstances we cannot help supposing that we have to do with the "mixed infection" already referred to on p. 190, or at least with a particularly malignant variety of infective material. That the infective material is of the nature of a bacillus has been rendered extremely probable by recent microscopic and experimental researches. We have no certain knowledge as to how the specific infection enters the organism. The cases of typhoid which we have observed in the children's department almost all came from outside. Only in very rare instances did the disease originate in a convalescent patient, or in a child under treatment for another disease; and when this did happen, it was often noticed that there had been no cases of typhoid in the wards for a long time. The drinking-water could not be to blame, for if that were the case many more of the children would have been affected. The virus must therefore have been brought from outside in some way or other (on one of the visiting days). I shall not venture to decide from my own experience whether the period of incubation lasts 3—4 weeks, as is generally stated. When the infection has once taken place, the onset of the disease may be favoured by various influences, among which I may mention emotional influences and severe chills.

A healthy boy of 12 was surprised by a shower on his way to school in June, 1875, got wet to the skin and had to sit in school in this condition for 4 hours. On the following day he complained of headache and an attack of typhoid fever commenced, for which he was confined to bed for 5 weeks.—An orphan boy of 11 went to church in perfect health, according to his own statement. While absorbed in reading the hymn book, he was suddenly startled by the sound of the organ, and shuddered violently. Giddiness and vomiting at once took place, and, by the same evening, fever set in and developed into typhoid. Certainly, in both of these cases, the typhoid would have occurred without the determining causes which were given—although, perhaps, a little later.

The comparatively mild character of typhoid in children compared with the same disease in adults, had been pointed out by most writers even before the "antipyretic" method of treatment had come into use. Out of 266 cases, some of which were very severe, I find that 40 (that is about 11¼ per cent.) ended fatally; and certainly no one can call this a very small rate of mortality. The rate, however, certainly differed much

in different epidemics, so that in some years it was extremely small and in others startlingly great. We must also deduct 10 cases of children who died from diseases which they acquired during their convalescence from typhoid fever, or which had existed previously to it. The mortality in private practice was far lower, and the above-mentioned pathological facts—the great rarity of perforation of the intestine and of copious hæmorrhages—are to be regarded as the main causes of the mildness of the disease. I have often seen cases recover completely after a very long and continuous fever with high temperature, copious diarrhœa, lung complications, and the formation of thrush in the mouth and pharynx. Nevertheless I must acknowledge that the experience of the last few years has very much shaken my faith as to the mildness of typhoid in children, which is so generally asserted.

We now come to the clinical symptoms of the disease; and I must first point out that the practitioner may find it difficult to make up his mind in slight cases (in children even more than in adults) whether the case is really one of typhoid or only one of "gastric fever." I am not prepared to deny the existence of the latter condition and its dependence on a form of gastro-intestinal catarrh which has nothing to do with typhoid infection; and that in spite of the fact that many writers are strongly opposed to this view. There will always be cases in regard to which the opinion of physicians differs on this point; for all the intrinsic symptoms of typhoid—fever, enlargement of the spleen, roseola, and diarrhœa—are not necessarily always present, and as a fact some of them may be absent. Even the most important criterion, the characteristic temperature-curve, which is the same in children as in later life, occasionally varies in a most perplexing manner. Thus a girl of 3 years[1] who was already in the ward under treatment for an old cardiac lesion, pleuritic effusion and catarrh of the large intestine, began to be feverish only 11 days before her death, but so irregularly that even in the first stage she was almost free from fever in the morning. Perhaps the fact of the fever coming on in a child who was already much weakened may have had to do with its abnormal course; but still you see

[1] *Loc. cit.*, S. 398.

from this that typhoid fever may, in exceptional cases, run its course without the characteristic temperature-curve.[1]

Only in a very few of my cases did the attack commence suddenly with a rigor followed by fever, and in one case also by copious profuse perspiration; and even when it did do so it was always doubtful whether there had not previously been some rise of temperature which had been overlooked, and the rigor was due to a sudden increase of it. For example, in a boy of 11 who had begun to get better, and whose morning temperature had become normal, a relapse suddenly set in with a violent rigor. In these cases the temperature almost always rose rapidly immediately after the rigor, reaching as high as 104°—105·8° on the first evenings. In some cases, however, after the sudden abrupt rise of the first day, a fall of the temperature was noticed on the second, while on the third day the temperature again rose as high as before, or even higher. We need not form an unfavourable prognosis from this sudden rise of temperature at the very beginning, for only one of these cases had a tolerably severe course, the fever lasting 29 days, and two ended fatally, while the other fifteen had a very mild and extremely short course of 8—18 days.

Much oftener than a sudden commencement we found gradual rising of the temperature in the so-called "ladder form." In such cases the temperature often did not reach 104° till the second half of the first week. In these there was no initial rigor; there was at most a slight shivering which set in while the fever was increasing, in the afternoon or evening. After this, just as in adults, the temperature assumes a continuous remittent type, being high in the evening and about 1·8° F. lower in the morning, and it remains for some time pretty much about the same level (acme), and then gradually falls into an intermittent state in which it is normal in the morning but still febrile in the evening. When the evening temperature returns to the normal level, the fever is at an end. To determine the duration of the fever as a whole, we must be able to ascertain, at least with approximate accuracy the com-

[1] *Cf.* Fräntzel (*Zeitschr. f. klin. Med.*, Bd. ii., Heft 2), who has observed many cases of typhoid fever end fatally which had a very low temperature or even no rise at all, but serious cerebral symptoms. These occurred especially in patients in a state of exhaustion due to fatigue or insufficient nourishment.

mencement of the disease, which I was only able to do in 167 of my cases. The entire duration of the fever (relapses were not counted) was as follows :—

| | | |
|---|---|---|
| In | 9 cases | 7— 9 days |
| „ | 10 „ | 10 „ |
| „ | 15 „ | 11 „ |
| „ | 4 „ | 12 „ |
| „ | 38 „ | 13—15 „ |
| „ | 14 „ | 16—17 „ |
| „ | 24 „ | 18—19 „ |
| „ | 35 „ | 20—23 „ |
| „ | 13 „ | 24—30 „ |
| „ | 1 „ | 35 „ |
| „ | 1 „ | 42 „ |
| „ | 2 „ | 48—49 „ |
| „ | 1 „ | 70 „ |

Thus in the greater number of my cases (111), the fever had quite disappeared between the 13th and 23rd day of the disease. Eighteen cases were prolonged beyond this period, two of these lasted till the end of the 7th, and one till the end of the 10th week. Thirty-eight cases ended between the 7th and 12th days. In these (the so-called "abortive" cases of typhoid) there would have been ground for doubt, on account of the short duration of the fever, as to whether the cases were to be regarded as typhoid or merely as "gastric fever"—had not the diagnosis generally been confirmed by the presence of palpable enlargement of the spleen, roseola, and diarrhœa. The degree of fever during the period of continuous elevation of temperature varies greatly in different cases. While in many patients the maximum temperature never exceeds 102·7°—103·6°, and in a very few cases does not even rise above 100·8° in the morning, in the majority the evening temperature varies between 104° and 105·1°, and sometimes reaches 105·8°—106·3°. I do not think it advisable to take the temperature oftener than thrice daily, because the variations thus ascertained are more apt to cause perplexity than to afford guidance. We found as the total result that the morning temperature was almost always one or two degrees lower than the evening; greater differences (apart from the use of antipyretics) are rare, and only occur on single days. Thus, for example, one boy of 8 years had for two days a temperature of 100·8° and 101·1° in the mornings, and

104·5° and 104·4° in the evening. There is often a rise of temperature beginning about midday, which generally reaches its highest point between 2 and 3 P.M. and then falls, and about 5 P.M. it begins afresh, so that in many cases there is both a midday and an evening exacerbation, the first of which is sometimes 1—2 degrees higher than the second. Cases in which the evening and morning temperatures are almost equally high are less common, but they are always extremely obstinate. I have frequently seen children whose temperature for a whole week at least was never below 104·1° or 104·9° in the morning, and in the evening was always from 104·4° to 105·8°; and these were the cases which most obstinately resisted all antipyretic remedies. Only in 9 cases have I seen an inverse type of the fever—*i.e.*, the temperature higher in the morning than in the evening. In five of the children, this type only lasted a day or two; in other five it lasted for 5, 7, 13, and 18 days respectively. This type was almost always found either at the very beginning or (even oftener) during the stage of improvement, and only once during the acme of the disease.

The duration of the acme generally varies between 8 and 20 days; most commonly it comes to an end on the 10th, 13th, or 18th day. Much less frequently its duration is shorter (5—7 days) and it may also be considerably longer (35—44 days). When the acme is at an end the temperature comes down gradually, falling to 101·8° and lower in the morning, and usually only reaching 103·1° in the evening. The duration of this stage of the fever (stadium decrementi), which can only be definitely ascertained in cases where we have an opportunity of observing the patients from the beginning, was 2—4 days in more than half of the cases. Sometimes it only lasted one day while in others it went on for 5—9 days. You must, however, always be prepared to find, even at this stage, sudden inexplicable evening rises of temperature to 104° and even higher; but these are merely transient and have no influence on the further course of the disease. I have sometimes observed a rise of this kind to 104° even on the last evening of this period, and yet on the very next day the thermometer was at 99·1°—the beginning of the intermittent stage which then took its regular course.

In this last stage the morning temperature is normal or

subnormal, while the patient is still feverish during the afternoon and evening. It generally varies in duration from two to five days; often I have seen it last only one day, and less frequently for a week or more. In many of my cases the intermittent stage was unusually prolonged, even for 2—3 weeks, and then it gave rise to great anxiety as to whether miliary tuberculosis might not be in process of development. These apprehensions, however, were always unfounded. The evening rise of temperature generally only reached 103·1°, and very rarely, and only for a short time, 104°; this state of things has in several cases seemed to me to be connected with constipation.

In a girl of 9 years who was in the intermittent stage after a relapse which had lasted 14 days, the evening temperature was still about 101·5°. On the 7th and 9th September, it rose again suddenly to 104° and fell at once on each occasion to 101·1° after some enormous nodular fæcal masses had been got rid of by means of an enema.

This unusual rise of temperature may also be caused by overloading of the stomach, by too early and too prolonged sitting up in bed, and by emotional causes; and we may thus explain the fact that occasional transient rises of temperature may occur even in the mornings, which are generally free from fever at this stage, and during any part of the perfectly afebrile convalescence, which need not necessarily cause any anxiety. We find this happening not at all uncommonly in our wards after the visiting days, on which the convalescent patients are secretly regaled with cakes, &c., by their relatives. In a small number of the cases, the intermittent stage, or even the stadium decrementi is quite absent or is only so slightly indicated that the continuous fever passes immediately, after the manner of a crisis, into an afebrile state. I have already in a former paper[1] published a series of charts illustrating this fall of temperature by crisis, and I may now add the following, which like almost all similar ones occurred in a case with a rapid course and high temperature.

Boy of 3 years. Duration of the acme 7 days. Temp. m. 103·8°—104°; ev. 104°—106·2°. Diarrhœa, drowsiness, delirium, &c. On the 7th day, temp. 103·3°; ev. 105·3°; pulse 160. On the following day temp. m. 98·1°; ev. 99·5°; pulse 88. After this no return of the fever.—In a boy of 4, whose temperature fell within

[1] *Charité-Annalen*, ii., S. 561.

24 hours from 102° to 95·4°, symptoms of collapse (vomiting, almost imperceptible pulse) set in, requiring the administration of the strongest stimulants. The pulse remained irregular in volume and rhythm for days.

Convalescence begins whenever the fever has entirely disappeared, and during this period, as in other highly febrile diseases, the temperature is often subnormal (96·8°—95·9°) in the morning, or even in the evening, until owing to nourishment having been taken and digestion going on regularly, the normal temperature becomes permanently re-established. Ephemeral rises of temperature, such as I have already mentioned, and even rigors may also occur from time to time during this stage without, however, doing any harm.

The pulse-rate of children with typhoid generally corresponds to the degree of the fever. Still, as in adults, this rule is not without exceptions, and you may have, for example, pulse-rates of 90, 108, and 120, with temperatures of 104·4° and 106·2°. Moreover, the pulse-rate varies considerably, and even in favourable cases reaches a rapidity (152—180) which in older patients would most certainly justify a fatal prognosis. Even during convalescence a high pulse-rate, due to the cardiac debility which the fever produces, not unfrequently persists for days. Less frequently there is an extremely low rate (*e.g.* 60), or irregularity, as is also observed after other serious diseases,[1] for example pneumonia. Dicrotism of the pulse, which is pretty common in adults, I have also seen in children, but only in a small number of cases. The quality of the pulse is more difficult to estimate owing to the artery being so much smaller than it is in older patients. In children under five, especially, the pulse is always small and easily compressible. It is only when, besides being very rapid, it can scarcely be felt on gentle palpation, and its beats run into one another, and the extremities become cold and cyanotic, that collapse from cardiac failure is to be apprehended.

I now come to the nervous symptoms, which used to be

[1] I have never observed slowing of the pulse along with irregularity during the course of typhoid fever. Revilliod (*Notes cliniques sur quelques maladies des enfants:* Paris, 1886, p. 35) gives a case of this kind in which, with a temperature of 104°, the pulse always kept between 42 and 68 and was somewhat irregular. Under such circumstances one is apt to mistake the case for one of tubercular meningitis.

regarded as the point of most interest in typhoid fever. As a fact, however, these symptoms are considerably less severe and less frequent in children (even when they have reached the age of 11 or 12 years) than in adults. In quite a number of cases there are either very slight nervous symptoms or none at all. Many children even sit up in bed now and then, smile, and have a pretty good appetite, although the temperature-curve and the palpable enlargement of the spleen put the existence of typhoid beyond a doubt. Indeed I have seen cases in which the only symptom of typhoid present was the characteristic temperature-curve, while not a trace of any of the others (diarrhœa, enlargement of the spleen, or roseola) was to be found; so that for 3 or even 4 weeks we were kept in a state of continuous anxiety by the thought that the case might possibly be one of another nature—miliary tuberculosis or an insidious form of endocarditis. Certain nervous symptoms, to be sure, do occur more frequently; but still only to a limited degree, which by no means corresponds to the persistent high temperature. Headache and apathy without mental derangement, slight drowsiness, restlessness, a moderate amount of delirium especially in the evening and at night, difficulty of hearing, hyperæsthesia of the skin, especially over the abdomen, sleeplessness, dreams and giddiness are the symptoms we most commonly meet with. Serious nervous symptoms are extremely rare. In young children we often have, in place of delirium, very violent and passionate screaming without apparent cause, which, especially at night, disturbs the rest of the family extremely. The same thing occurred in the case of a boy of 10 years, and was so bad that we had to give him chloroform repeatedly. Liebermeister's view—that the nervous symptoms are simply due to the influence of the high temperature—is, I am convinced, untenable. Because, as I have elsewhere[1] shown, the severity of these symptoms is not necessarily proportionate to the degree of the fever. There must, therefore, be other causes to be taken into account besides the fever, and foremost among these is the action which the typhoid poison exerts on the brain. The older the children are the greater is their liability to have serious nervous symptoms. After 10 years of age, I have pretty often seen active delirium, stupor, coma,

[1] *Loc. cit.*, S. 567.

tremor of the hands and tongue, and attempts to jump out of bed. In cases where the children have died in a state of deep coma I have several times noticed the conjunctiva bulbi become red, and the cornea covered with mucus—just as occurs in tubercular meningitis and in cholera—and finally get quite dry and opaque. In one little girl of 3 years, it was even perforated. In this case slight spasmodic contractions of the extremities occurred on the last day of life, and yet at the post-mortem we found no abnormality of the brain to account for them. In a girl of 10 years who took a relapse in the 5th week of the disease, there was, towards the end, contracture of both legs and of the right arm, also repeated grinding of the teeth, and yet at the post-mortem we could find nothing but a moderate amount of serum in the ventricles and œdema of the pia mater; and these are conditions that we may also find in cases of typhoid without the above-mentioned spastic symptoms. We found the same condition in a girl of 4, who had had distinct cervical rigidity during the last few days of life, especially when she was set upright. Cervical rigidity, grinding of the teeth, and starting when touched, were also found in a few other cases, which all ended fatally; and yet at the post-mortem nothing unusual was found in the brain. Finally, I have seen a boy of 9 years who had trismus constantly during the last week of typhoid fever; but no examination of the cranial cavity was made.[1] Only in a very small number of cases have I seen a short epileptiform attack at the beginning of the disease, such as occurs in the initial stage of other infectious diseases.

Among the forms of psychical derangement, apathy is the commonest, and it is often accompanied by slight delirium, especially during the night. In exceptional cases I have seen violent delirium or hallucinations: one boy, *e.g.*, was always thinking he heard his father's voice. I would especially point out that in many cases the psychical derangements only set in when the temperature was falling, or even after the fever had entirely disappeared, so that one could only regard them as forms of delirium ab inanitione due to cardiac debility and cerebral anæmia.

[1] *Cf.* Förster (*Jahrb. . Kinderheilk.*, 1863, vi., S. 114), who had a case complicated by trismus and opisthotonus which ended in recovery. Röth also (*Arch. f. Kinderheilk.*, ii., S. 375) gives a few cases in which spastic contractures occurred in the course of typhoid in children.

A boy of 4 years continued, for days after the fever had gone, to imagine that he had a black cat sitting beside him in bed.—A girl of 6 years, when her temperature was only 97° had difficulty of speech, frequent delirium, and wanted to leave the hospital. After the mental symptoms had quite disappeared she suffered for weeks from low spirits and nocturnal and diurnal enuresis.—In a girl of 6, extremely debilitated and anæmic, regular maniacal attacks took place immediately after the disappearance of the fever; she screamed wildly, struck out round about her, jumped out of bed. During the intervals, the child lay perfectly apathetic, staring in front of her, and only at times seemed fully conscious. Although the general impression in this case suggested that the mental state was due to inanition, nevertheless copious diet and abundance of wine failed to ward off fatal collapse.—Another kind of psychical change took place in a boy of 12 years, in the stadium decrementi (end of the 2nd week). Hurried, almost unintelligible speech, childish obstinacy and continuous and passionate screaming, lasted for days and greatly alarmed the parents. When, in the 5th week, there was a relapse of the typhoid, it was followed a few days afterwards by a recurrence of the same mental condition; and this time there were distinct symptoms of mania. The patient treated his parents and brothers and sisters with contempt, kept constantly chattering unintelligible stuff, screamed passionately, took absolutely no sleep, and vomited almost everything he ate or drank. Pulse very rapid and small, breathing quick, extremities and point of nose cold and cyanotic. Such being the condition, I tried chloral hydrate (35 grains in an enema) and within 15 minutes the hands and feet became warm, the cyanosis disappeared, the pulse and breathing became slower, and the pulse stronger. The chloral enema was repeated on 3 consecutive evenings and caused a rapid disappearance of the threatening symptoms, while morphia and warm baths had had no effect.

I ought, however, to mention that this boy had always been extremely ill brought-up and exceedingly irritable; for we find that the character of children, due to natural disposition and upbringing, evidently exercises some influence in their mental condition in typhoid fever.

I have rarely observed actual paralysis following this disease. One boy of 9 had paresis and ataxia of both lower limbs which had developed after typhoid and had existed for two years before I saw him. I know nothing about the further course of the case. A girl of 11 was seized during the stadium decrementi with bilateral ptosis, paralysis of the right abducens, and aphasia

which lasted seven days; and after these had passed off a childish and peevish disposition continued for weeks. I have met with complete aphasia in 13 cases, while in a few others only one particular word (*e.g.*, "ja!") could be pronounced and nothing else whatever.[1] The intelligence may at the same time remain quite unimpaired; one boy of ten could write his name, and when asked how old he was he held up his ten fingers but could not speak a single word. Further, the aphasia always sets in during the intermittent stage or at the beginning of convalescence, and never while the fever is at its height, and it generally lasts 8—14 days. I have never observed the occurrence of fits of eclampsia nor transient rises of temperature such as have been described as ushering in aphasia.[2] Nor have I ever seen paralysis of the laryngeal muscles (posterior crico-arytenoid) which necessitated tracheotomy in a case described by Rehn.[3]

All the cases of typhoid fever which were followed by aphasia were severe and of long duration or else very acute, and yet all of these children recovered, with the sole exception of a boy of 6 years who died of a relapse. In two cases, in a boy of 9 years and a girl of 14, I observed amblyopia lasting for several days while the temperature was falling. In the second of these, it turned out to be due to paralysis of the accommodation; but in both children it entirely disappeared. Whether all the nervous symptoms are caused by definite changes in the brain, is quite unknown. Still, one can readily imagine that under the influence of the disease degenerative changes may take place in the tissue-elements of the brain similar to those which have been demonstrated in the muscular fibres (especially those of the heart) and in the cells of the liver and kidneys; and that when these recover during convalescence, the symptoms depending on them would also disappear. Particularly interesting was the case of a boy of 7, who, after a relapse of 14 days' duration, got into a state of extreme debility with tremor, exceedingly small pulse and involuntary evacuations, and was then suddenly seized with hemiplegia of the right half of the body and also of the facial and sixth nerves. The paralysis rapidly improved under galvanic treatment, so that after 14 days the patient was discharged in a

[1] Bohn, *Jahrb. f. Kinderheilk.*, xxv., S. 69.
[2] Semtschenko, *Jahrb. f. Kinderheilk.*, xviii., S. 300.
[3] *Deutsches Arch. f. klin. Med.*, xviii., Heft 1.

state of almost complete recovery. I have often seen him since at the polyclinic and found him perfectly well. The appearance of the hemiplegia during a period when there is great cardiac debility seems to me to make embolism the most likely cause in this case (*cf.* pp. 302, 316, on hemiplegia after diphtheria).[1] On the other hand, the possibility of hæmorrhage into the brain is not to be dismissed off-hand, especially as Bouchut (*loc. cit.* p. 71), says he has observed hæmorrhage into the retina in children with typhoid.

Enlargement of the spleen and roseola are among the most important symptoms of typhoid fever in children as in adults. In regard to the spleen, the difficulty of making ou the fact of its enlargement can hardly be exaggerated; in particular the percussion of the spleen may give utterly fallacious indications owing to distension of the colon with gas, to morbid conditions of the left lung and pleura and to the restlessness of many children during examination. Only in 120 cases, have I been able to carry out the examination of the spleen with the necessary exactitude and perseverance, and in 55 of these I found the spleen palpable—either constantly $\frac{1}{2}$—$1\frac{1}{2}$ inches below the costal margin or only felt distinctly during deep inspiration, sometimes also evidently painful on pressure. The gravity of the case has in general no influence on the degree of enlargement. In the other cases we could only make out the splenic enlargement by means of percussion, generally as far as the lower margin of the 7th rib; there was also tenderness on percussion and on pressure under the costal margin. In many other cases, however, no splenic enlargement could be discovered either by palpation or by perfectly unprejudiced percussion. In considering when (that is, at what stage of the disease) this enlargement is found, I shall only make use of the 55 cases in which I could feel the spleen distinctly; for only in these was there no room for doubt. We found that the more acutely and rapidly the temperature-curve rose, the earlier did the splenic enlargement appear, sometimes even on the 3rd or 4th day. But in most cases it cannot be felt till the 6th or 9th

[1] De Montmollin (*Observations sur la fièvre typhoïde de l'enfance:* Neuchatel, 1885) records the case of a girl of 8 who, on the 24th day, had an embolism of the left brachial artery, symptoms of which persisted for a month.—Marasmic thrombosis of the larger veins, especially those of the lower extremities, have been observed during convalescence.

day or even later, and after the enlargement of the organ upwards and forwards has already been demonstrated by means of percussion. The enlarged organ can generally still be felt during the intermittent stage. In a girl of 11, it could be felt a whole week, and in a boy of 8, at least two weeks after the fever had disappeared. In several cases I was not able to make out the enlargement distinctly until during the course of a relapse, whereas in the first attack it could only be made out by percussion.

The diagnosis of the roseola also demands the greatest caution, especially when one has not had the case under observation from, at least, the end of the first week; for the rash is often overlooked, or has entirely faded before the child comes under treatment. Yet I can affirm most positively that in 19 cases which were carefully observed, from beginning to end, in my wards, no roseola was ever found. But in all the others it made its appearance just as it does in adults, *i.e.*, in the form of a very small number of pale-red spots, generally projecting a little. These vary from the size of a pin-head to that of a lentil-seed at most, and are situated especially on the abdomen and the lower part of the thorax, less commonly on the back and on the inner surface of the thigh. The number of the spots is generally very small, 5—10 altogether often even fewer; so that one may be left in doubt whether they are really of the nature of roseola or merely some other chance rash. Only in 10 cases, was there an unusually copious rash, resembling that of typhus; in a few instances it extended almost all over the body. As regards the time at which the roseola makes its appearance, the rule is pretty much the same as in regard to the enlargement of the spleen. In cases where the temperature rises very rapidly and to a high level, the roseola also often appears by the 3rd—5th day after the beginning of the fever (in one case it had spread almost all over the body by this time); but in the great majority of cases it does not make its appearance until the 7th—10th day; in a very few, not until the end of the second week, during the stadium decrementi. As a rule, after the first spots have entirely passed off, a few of them reappear in the course of the next few days, rarely later. Thus, *e.g.*, we may find them for the first time on the 12th—18th day of the fever; and the temperature, which has already [illegible] runs up again to 104° or

more. Although the individual spots generally last only 2—3 days, yet, owing to the reappearance of the spots, the eruption often remains visible to the end of the second week or even somewhat longer, and there is usually left behind for some days a slight yellowish pigmentation in the situation of the roseola. Should a relapse occur later, it is generally but not invariably accompanied by a fresh eruption of roseola.

In addition to this eruption we often find in children, just as in adults, the well-known sudamina-vesicles appearing as soon as copious perspiration sets in on the fall of the temperature, and followed by branny desquamation. Only in 4 children, have I observed the occurrence of erythemata, sometimes diffuse sometimes more limited, in a few cases in the form of erythema urticatum or annulare; and this was on the 3rd, 8th, 10th, and 18th day of the disease respectively. This rash almost always remained fully out for only 24 hours, then rapidly faded, leaving behind it, in one case, a bluish pigmentation, but never any desquamation of the epidermis. In two severe cases, one of which ended fatally, there was a formation of flaccid bullæ on the thorax and back which left behind superficial ulcers. Actual petechiæ are but rarely found, although false ones, due to flea-bites, are often enough met with in hospital cases and are apt, on hurried examination, to be mistaken for true ones. Moreover, an eruption of real petechiæ does not at once justify an unfavourable prognosis, even although they recur repeatedly.

Martha W., 14 years old, admitted September 19, 1874, in the second week of typhoid fever. A large number of bluish-red petechiæ, from the size of a pin-head to that of a lentil, scattered over the whole body. Here and there also larger ecchymoses, the largest of which was under the left trochanter, and in the middle of it one could feel a solid mass of exudation as big as a sixpence. On the 23rd, fresh ecchymoses the size of a half-crown over the sacrum. On the 26th, another on the right thigh with central hardness, while the stadium decrementi of the fever had already begun. All these blood-extravasations were completely reabsorbed within ten days. The case was, on the whole, a serious one: high temperature (reaching 104·7°) extremely small pulse, coldness of extremities, very weak heart-sounds, great restlessness with delirium, diffuse catarrh and broncho-pneumonia, and finally a relapse which set in after an afebrile interval of 11 days and lasted 12 days. Nevertheless the child finally recovered. The cause of the hæmorrhagic diathesis

remained unknown. The disappearance of the petechiæ on the subsidence of the first attack of fever was remarkable, seeing that no fresh extravasations appeared during the relapse.

According to my experience bedsores are less common in children than in adults. Altogether, I have seen 14 such cases at most, and these were seldom very severe. In the case of Martha W., a tolerably deep bedsore over the sacrum healed up even during the relapse while the temperature was still continuously high (ev., as much as 105·4°). In another child, a nævus situated right over the great trochanter, and in a third, the occiput, was the seat of the bedsore. Only three of the cases ended fatally. We also frequently found whitlows, abscesses, boils, and ecthyma-pustules, especially in the sacral region, over the iliac spines, and on the abdominal wall. In the latter situation there was, in two of the cases, such a deep gangrenous sloughing of the subcutaneous connective tissue that the abdominal muscles were laid bare. In one boy of 10, while the temperature was falling, a large abscess formed in the right thigh, and afterwards another in the left; these necessitated deep incisions, kept up the temperature for weeks, and were healed only by the end of the third month. Finally, in a fatal case in which the temperature had been 104·4° during the last few days, and there had been repeated rigors and large extravasations of blood with a few bullæ filled with bloody serum on the right lower limb, we found at the post-mortem a marasmic thrombosis of the right femoral vein reaching far into its branches.

The symptoms connected with the digestive organs are the same as those which occur in adults. There is nearly always loss of appetite, which lasts to the intermittent stage, and in obstinate and badly brought-up children it sometimes leads to outbreaks of passionate violence whenever an attempt is made to get them to take the necessary nourishment. Very rarely and only in quite mild cases do the children themselves express any desire for food. But when convalescence sets in, the appetite is nearly always enormously increased. In one case, when this hunger was not immediately satisfied, regular paroxysms of fury with screaming, followed.

The character of the tongue varies very much. It is very rare to find it clean and moist throughout, but also it is rare to see it as dry, fissured, and covered with blackish-brown crusts as

we so often find it in adults. In the early stages of the disease we generally notice a thick whitish or greyish-yellow fur on its surface, while its edges and tip are very red, or the anterior half is smooth and red while the posterior half is much furred. When the fever is at its height, the coating often desquamates entirely or partially, and the surface then becomes of a shining red appearance as if varnished, somewhat dry and brownish in the centre, often with rather prominent papillæ. On the whole, however, the appearance of the tongue changes from day to day. Generally speaking, its tendency to become dry, which usually proceeds *pari passu* with the severity of the nervous symptoms (especially of the drowsiness), is less marked than in adults; and the same may be said of the offensive smell from the mouth, of the dryness and coating of sordes on the lips and gums, which are often absent and rarely reach the extreme degree which is so common in older patients. On the other hand, we nearly always find loose fragments of epidermis on the lips, and these get gradually torn off owing to the habit, which the children almost invariably have, of picking at their lips. This symptom, which belongs to the same category as the equally common boring in the nostrils and rubbing of the eyelids, is observed in the first few days of typhoid but may last into the later stages of the disease. It is, however, of no characteristic significance, for it is also very often observed in the first stage of tubercular meningitis (vol. i., p. 319). In a girl of 5 years, a hard infiltration extending to the edge of the lower jaw formed a few days before death, and had apparently started from a fissure on the lower lip; there was also at the same time an extremely hard swelling of the left parotid gland.

Small aphthous patches and superficial ulcers on the edges of the tongue and the angles of the mouth are often found, causing the children to be constantly putting their fingers into their mouths. Sometimes typhoid fever is accompanied by tonsillar sore-throat. Indeed in the case of a girl of 10 years, such an attack of sore-throat occurred, both with the first attack of the fever and with the relapse which followed, although the mucous membrane of the mouth remained quite unaffected. I have observed thrush in 27 cases, either at the height of the fever, or when the temperature was already falling, but always only in cases where there was extreme general debility.

Generally, thrush was limited to the arch of the palate, but occasionally it affected the whole mouth and pharynx. Thrush of the pharynx is especially liable to be mistaken for diphtheria, and this mistake is to be avoided by examining for the filaments of the thrush-fungus with the microscope.

Vomiting occurred certainly in 42 of my cases, generally only at the beginning and during the first week of the disease. Less commonly it was also found later on in the disease, or recurred again and again until the end of the fever. It generally comes on after eating or drinking, less commonly it occurs spontaneously, or is quite absent for some days, and then recurs without any evident cause. In any case, vomiting is commoner in children than in adults, and in regard to this the experience of other observers agrees with my own.[1] I cannot agree with those (*e.g.*, Löschner) who ascribe a specially bad prognostic significance to repeated vomiting, although I certainly do grant that it occurs principally in severe cases. It goes without saying, that vomiting at the commencement of the illness is sure to arouse a suspicion of tubercular meningitis, and we must therefore investigate all the more carefully the accompanying symptoms, especially the character of the pulse and the state of the temperature.

Out of 200 cases, defæcation was perfectly normal in 24, the motions being at most a little softer than usual, or there were perhaps none at all on some days. In 22 cases, there was constipation during the whole course of the disease which necessitated the repeated use of enemata, castor oil, or calomel. In one of these children the constipation was so obstinate that 6½ grains of calomel (in two doses) and two evacuant enemata had to be given to move the bowels. This, however, had nothing to do with the gravity of the case; for we have had several cases of typhoid with a very protracted course, constant high temperature and comparatively severe nervous symptoms in which there was constipation from beginning to end. There is often constipation at the beginning, which is afterwards replaced by diarrhœa; this was the case, either at the beginning, or (more frequently) after the middle of the first week, or from the second week, in

[1] Montmollin (*loc. cit.*) observed vomiting in the earlier days in 88 cases, during the whole disease in 13 cases.

157 out of 200 cases. The motions varied much in number (generally only 1—5 daily, rarely 10—20), and they usually presented the well-known character; but they were also often greenish, or of the colour of *café-au-lait*. They generally retained this character into the intermittent stage, and only became quite normal when the fever had disappeared. Diarrhœa rarely sets in during convalescence without some recognisable cause, but in some children it does so with such violence that collapse seems imminent. During this period, we more frequently find a tendency to constipation, and with regard to this we have to be all the more attentive, because transient febrile attacks may arise, which pass off at once as soon as the hard scybala have been removed by a dose of castor oil or by copious injections of water into the large intestine (p. 327).

Intestinal hæmorrhage is very rare in typhoid among children compared with what we find in adults. All writers are agreed on this, and connect the fact with the rarity of extensive intestinal ulceration at this age (p. 320). I have myself only observed intestinal hæmorrhage 9 times, and in 5 of the cases it was slight, and only lasted for a short time. In one boy of 12 years, however, the hæmorrhage was violent and returned during a relapse, causing a very alarming degree of weakness. In one girl of 10 years, a slight intestinal hæmorrhage took place during a relapse, and was followed a few days afterwards by two very copious hæmorrhages ending in fatal collapse. In this case we had previously noticed the frequent passage of extremely offensive flatus which almost poisoned everyone round about when the bed-clothes were lifted, and which was to be attributed to the decomposition of quantities of blood in the intestinal canal. In a boy of 5 years, copious evacuations of fluid and clotted blood occurred on the 15th day of the disease and were followed by a rapid fall of the temperature from 103·5° to 98·1° within a few hours; but this only lasted till the following day, and then gave place to a renewal of the high temperature. The case ended fatally on the 20th day, and at the post-mortem we found extensive ulceration (p. 319, case 17). Only in one case, which was not examined post-mortem, was there bloody diarrhœa accompanied by hæmorrhages from the mucous membrane of the nose, mouth, and pharynx.

I have been struck by the extremely small number of cases in

which the motions and urine were passed into the bed involuntarily. Almost all the children more than a year old let the nurses know that they wanted to be attended to, even in cases where there was a considerable amount of apathy and dementia. Rilliet and Barthez have also noticed the same thing. Involuntary evacuations take place almost exclusively in severe cases where there is deep coma. It is all the more curious, therefore, that many children do not begin to pass their faeces and urine into the bed until the intermittent stage or until convalescence, when their intellect is perfectly unaffected; and I can only account for this by the weakness and the consequent aversion to the slightest movement. Retention of urine is also very uncommon, but I have met with it—*e.g.*, in a boy of 4 years who was completely anaesthesious and required a catheter to be passed frequently; this case ended in recovery.

It is of course more difficult to estimate the amount of tenderness of the ileo-cæcal region in children than it is in adults. I cannot, therefore, regard it as any more significant than the so-called "gargouillement" which is not at all uncommon in children who are merely suffering from ordinary diarrhœa. The form of the abdomen is generally normal or only moderately distended. I have rarely met with a great amount of meteorism, and it hardly ever occurred to such a degree as to displace the diaphragm and interfere with breathing—a circumstance to which the more favourable prognosis of typhoid fever in children is partly due. I have ascertained the presence of colic (especially before defecation) with certainty only in 16 cases. It was therefore all the more striking that in 3 of the children these pains only began during convalescence, and in 2 of them they were so very severe that one might have suspected that perforative peritonitis was setting in.

Otto M., 11 years old, convalescent from typhoid fever since October 19, 1874. On the afternoon of November 9th, sudden onset of very severe abdominal pain which continued to increase. This started from the right iliac region, spread to the hypogastrium and umbilicus and was accompanied by repeated vomiting. Abdomen tense and tender but not much distended. The patient complained much, but there was no fever (temp. 99·7°), and defecation was normal. Improvement followed the administration of 8 drops of tincture of opium and the use of an ice-bag. During the night, much eructation; on the following

day, nausea and several attacks of vomiting, which recurred on the 11th and 12th (without any error in diet). On the 14th, another violent attack of pain (with vomiting), which again originated in the ileo-cæcal region. After this, uninterrupted convalescence.

Max B., 7 years old, had been free from fever for 2 days after a severe attack of typhoid. At midday on December 28, 1875, exceedingly violent pain suddenly set in in the right side of the abdomen, with constant screaming and perspiration on the face. An injection of morphia (gr. $\frac{1}{11}$), at once produced relief and quiet sleep. Nothing abnormal found on examination of the abdomen. As there had been constipation for 10 days, 4 tablespoonfuls of castor oil were given at intervals, but these did not operate sufficiently. Free evacuation did not take place till the 30th after a few enemata. During this time, there was another short attack of pain (on the 29th) in the right side of the abdomen; this, however, passed off without treatment, only leaving behind it great tenderness of this region, and this disappeared on the 31st after the continuous application of an ice-bag.

There certainly could not have been any peritonitis in these cases; the pain was merely colic, due to the irritation of matter accumulated in the intestine. Besides the second case there was a third which likewise went to support this view, in which violent pains were set up in the course of the transverse colon by constipation during convalescence. Rilliet and Barthez also mention these pains and give the case of a boy of 11 years who was seized during typhoid fever by very severe pains in the abdomen, which lasted with intermissions for 36 hours.

In June, 1884, I met with the case of a girl of 6 years, in which typhoid fever began with constant complaints of violent pains in the abdomen, which lasted for days although the abdomen was normal except for some tenderness on pressure. The temp. rose to 105·8° and, as the typhoid symptoms became more well-marked, the abdominal pain disappeared. These had at first induced the doctor in attendance to diagnose the case as one of peritonitis.

In all my cases I have only once met with perforation of an intestinal ulcer. This occurred in the case of a boy of 11 years, and it took place in the 5th week, after convalescence had already begun. The rarity of perforation is confirmed by all writers, and Barrier's statistics (2 perforations out of 24 cases) are to be regarded as merely the result of a chance coincidence. In one boy of 10 years, we found recent peritonitis at the post-mortem, marked congestion of the entire peritoneum, which,

as well as the spleen, was scattered over with little purulent flocculi, and contained in its cavity some turbid brownish-yellow fluid. Still, we were not able to make out for certain any perforation of the numerous ulcers.

The occurrence of inflammation of the parotid is also comparatively rare; I have only seen it four times—in two girls, in one boy of 4 and another of 7 years. In the latter, an abscess of the left parotid developed in the 3rd week of an extremely severe attack of typhoid. In this case, the pus ruptured spontaneously into the external auditory meatus, and afterwards a counter-opening had to be made below the ear, which brought about recovery after a few weeks. In two cases the affection of the parotid took place a few days before death; in one case with a rise of temperature, and in the second the child died in a state of collapse (temp. 100°) before suppuration took place, while in the fourth, the abscess burst into the external auditory meatus and there was paralysis of the pes anserinus.

I now come to the symptoms connected with the respiratory organs, among which bronchial catarrh is the one most frequently met with, as in adults. I would however point out that in typhoid catarrh pretty deep inspirations are necessary (owing to the muscular weakness) to elicit rhonchi; the shallow respirations usual in these children occasion no abnormal sounds, but only weak vesicular breathing. Hence it comes that in a number of cases, in spite of the presence of cough, we cannot discover any catarrhal sounds unless the child happens to take a very deep breath while we are auscultating. Comparatively rarely (18 times) have I observed the catarrh passing on to a state of broncho-pneumonic consolidation distinctly recognisable on physical examination. But I do not deny that in many other cases lobular patches of broncho-pneumonia may have been present which it was yet impossible to make out on physical examination.

The broncho-pneumonia is almost always found on both sides at the posterior bases of the lungs. It usually sets in during the acme of the disease, less commonly when the temperature is falling, or even during the intermittent stage; and we may naturally suppose that the signs of consolidation are increased by hypostatic processes. The course of the attack may be much protracted by relapses of the broncho-pneumonia, and the

child's strength be reduced to an alarming degree. The extreme emaciation, the pale complexion, the loss of appetite and the remittent fever—which generally last for weeks in these cases—are certainly calculated to arouse apprehensions lest the infiltration has become caseous, or an attack of acute tuberculosis is about to develope. Fortunately, however, there is often no cause for such an apprehension, and the case ends in complete recovery (vol. i., p. 390).

Croupous pneumonia is much rarer than broncho-pneumonia, but the following case is an example of it.

Hedwig H., 13 years old, admitted on November 11, 1878, for epilepsy, which came on about every 3 weeks, especially during the night, in repeated severe paroxysms. On February 4, 1879, she became feverish (temp. 103·8°) and an attack of typhoid fever developed very acutely accompanied by roseola (which appeared as early as the 3rd day and was very copious, extending almost all over the body), delirium, coma, severe diarrhœa, &c. During the attack (which lasted for 16 days) the temperature was scarcely ever below 104° even in the morning; in the evening 104·9°—105·3°. As to the respiratory organs, we discovered first diffuse catarrh, later also extensive dulness of the right back with bronchial breathing. But the severity of the case, the deep coma and the steadily increasing collapse, prevented the progress of the pneumonic processes from being observed during the last few days. Death on the 20th from collapse.

Post-mortem: brain normal, epicardium and pleura studded over with little ecchymoses. Left lung normal. Only the apex of the right lung was air-containing. The rest of the upper lobe was tough, greyish-red and hepatised, containing disseminated hæmorrhagic areas. At the lower border, there were two cherry-red patches somewhat cubical in shape, ½ inches in diameter, surrounded by a sharp greyish-yellow line of demarcation. The lower lobe dark brownish-red, tough and hepatised throughout. Only at the lowest part of the middle lobe was there any portion of air-containing tissue left. Enlargement of the spleen; parenchymatous nephritis. (With regard to the changes in the intestine *cf.* p. 318, case 8). At the base of the arytænoid cartilage there was an ulcer extending down to the perichondrium. Superficial ulceration of the margin of the epiglottis. Thrush in the pharynx and œsophagus. Almost all the lymphatic glands enlarged and soft.

We found here almost complete hepatisation of the right lung, and at the border of its upper lobe two patches in the centre of the consolidation, which were separated from the surrounding

tissue, and which, had the patient lived longer, would either have passed on into complete gangrene (*i.e.*, circumscribed pulmonary gangrene) or abscess of the lung. The line of demarcation which surrounded it proved that its separation from the surrounding hepatised lung-tissue had already begun. In a few cases we found a moderate amount of purulent effusion in the pleural cavity. Another occurrence which is rare in the typhoid of children is ulceration of the larynx, which I have observed in four cases. In a fifth case we found no ulceration, but only marked swelling of the larynx, with inflammatory thickening of the perichondrium. All these children had suffered up till death from marked hoarseness and a harsh cough. One of them (a boy of 4) had also had, during the last nine days of the disease, increasing croupy symptoms, stridulous breathing, a croupy cough, and indrawing of the lower part of the thorax. At the post-mortem we found no trace of diphtheria anywhere, but the epiglottis was much reddened and swollen, and there were symmetrical ulcers on both arytænoid cartilages and inside the larynx. Hoarseness or even aphonia at the height of the disease may, however, be due also to atony of the voice-muscles. This may be recognised by the fact that the condition is partially overcome by a stronger exertion of vocal organs.

Actual gangrene of the lung I have observed in three cases :—

Franziska Sch., 4 years old, admitted July 7, 1877, with typhoid fever. At first, only diffuse bronchial catarrh could be made out. On the 13th, the patient coughed up for the first time some frothy blood-tinged sputum, most of which was swallowed again. The frequency of the respiration was not materially increased (about 30 in the minute). There was no dyspnœa. On the 17th, we discovered dulness, bronchial breathing and sharp medium râles all over the right back, especially from the spine of the scapula downwards. The breathing became irregular, the expiration noisy, the cough more distressing. Even now it was difficult to obtain a specimen of the sputum. Along with these respiratory symptoms, the regular typhoid symptoms ran their course but in a severe form. Death on 2nd August from collapse.

Post-mortem: the right pleural cavity contained a large quantity of discoloured brownish-grey exudation. Both layers of the pleura were covered with fibrin. The greater part of the upper portion of the right lung was of a leathery consistence, its

pleura was discoloured over an area the size of a hen's egg, and at this place fluctuation was felt. When the place was cut into, we opened into a cavity filled with offensive fluid and decomposed lung-tissue. The whole neighbouring tissue was hepatised, but just round the patch it was somewhat softened and discoloured. The layer of lung-tissue immediately bounding the cavity was distinctly gangrenous and putrid. The upper and middle lobes of the right lung bright-red and hepatised. (For the changes in the intestine see p. 318, case 7). Spleen somewhat enlarged; parenchymatous nephritis.

Fritz J., 9 years old, admitted with typhoid on March 11, 1881. On 4th April, œdema of the prepuce and formation of bullæ on the penis, the skin of which became gangrenous in a few days. Death from collapse on 6th April. Post-mortem: gangrene of the penis; Peyer's patches swollen and soft, not ulcerated. Both lower lobes hepatised, in the left a part of the lung the size of a pigeon's egg filled with offensive gangrenous matter and covered with discoloured pleura.

Marie T., 4 years old, admitted on April 23, 1880, with typhoid fever. Measles 14 days previously. On 5th May, fever gone, but child drowsy. Diarrhœa continuing. Resp. 60. On the 8th, return of the fever, dulness on the left side behind, numerous râles, cough. On the 10th, pulse 200, thready. For a long time had had a gangrenous bedsore over the sacrum. Post-mortem: left upper lobe slightly adherent, tough and hepatised; near the base a cavity larger than a pigeon's egg with greyish-yellow greasy offensive contents. Peyer's patches reticular, enlarged, with red spots.

All these were cases of croupous pneumonia in which gangrenous cavities had formed owing to septic decomposition, a process, the first stage of which I have already (p. 343) described as the formation of a separated patch ("sequestrum").

From a clinical point of view, I would emphasise the absence of any gangrenous fœtor of the breath (vol. i., p. 449), which as it is impossible to obtain any of the sputum prevents us being in a position to diagnose pulmonary gangrene. In the second case, the gangrene of the penis, and, in the third, the preceding attack of measles and the sloughing bedsore, may be looked on as factors predisposing to gangrene of the lung.

With regard to abnormalities of the urine, especially albuminuria, I find but few notes in the reports of my cases; but I admit that the examination of the urine, which in children can only be obtained with difficulty, if at all, may not have been carried out with the necessary exactness and regularity. In

one girl of 9 years, a moderate amount of albuminuria with slight œdema of the face and of the dorsal surfaces of the feet appeared on the 16th day after the disappearance of the fever; and these symptoms passed away within nine days under treatment with milk diet, warm baths, and doses of acetate of potash. In another case I am not sure whether the large amount of albumen found in the urine was not to be attributed to some doses of antifebrin. And in none of these cases was any bad result observed. In a girl of 7 years (private practice), I was struck by the very large quantity and the pale colour of the urine when the typhoid was at its height. As, however, we found neither sugar nor any other abnormal constituent, the enormously increased thirst, which was quite out of proportion to the degree of the temperature (which was not high), must be regarded as the cause of the polyuria. Ehrlich's diabenzol reaction was obtained in all cases in which it was examined for.

As to other complications and sequelæ, I have once seen erysipelas of the face with the formation of bullæ, in a boy of 8 years. The eruption was accompanied by high temperature and spread to the scalp, and the fever ended by crisis on the 5th day. I have found otorrhœa, generally unilateral, in 26 cases. It lasted 12—20 days, with or without impairment of the hearing and then disappeared gradually, leaving no bad results behind it. In one boy of 7, a hæmatoma of the right rectus abdominis muscle developed in the eighth week of a severe attack of typhoid. It was accompanied by severe pain, both spontaneous and on pressure and movement, and it formed a hard sharply circumscribed tumour ending just below the umbilicus. It disappeared by absorption within a few weeks. Very rarely do we find inflammation of the joints after typhoid; and therefore it seems worth while mentioning the case of a boy of 11 years who got synovitis of the left wrist three weeks after the subsidence of the fever. The joint was much swollen and extremely painful on every movement. The temperature was moderately high (100·8°), but it fell after two days, and the swelling rapidly diminished under the use of a splint and an ice-bag; but it reappeared within a very few days without rise of temperature, and was now successfully treated by tincture of iodine and a plaster of Paris bandage. One must just consider

whether this synovitis setting in three weeks after recovery from typhoid was properly to be regarded as a real sequela or merely as a chance affection. An analogous case affecting the right knee joint ended in recovery under the same treatment.

Among the sequelæ of typhoid, dropsy without any abnormality of the urine is also mentioned by different authors (*e.g.*, by Griesinger). This condition was frequently observed in children by Stoeber and by Rilliet and Barthez, and all their cases ended in recovery. I have only once met with dropsical symptoms following typhoid, apart from the case of nephritis mentioned on p. 346 and a few others in which the eyelids and the most dependent part of the scrotum seemed puffy after the temperature had fallen. This case was that of a very emaciated boy of 9, who, in the fifth week of the disease, had œdema of the hands and feet and a moderate amount of ascites with a persistent evening rise of temperature (101·1°—101·8), but without there being a trace of albumen in the urine. The fever ceased at once after the incision of three large abscesses under the scalp and the use of decoction of cinchona with wine and nourishing diet; and, with the improvement of the general health, the dropsical symptoms gradually disappeared so that after a month the child had completely recovered. It is doubtful whether in such cases we should (as I am inclined to do) regard the dropsy as atonic—*i.e.*, due to cardiac debility and venous engorgement—or as caused by parenchymatous changes in the kidneys. The absence of albuminuria affords no argument against the latter view; for, as you will remember, nephritis may exist even without any albuminuria (p. 153). I make a point of mentioning that this patient had never had any salicylate of soda, because some people are inclined to ascribe the occurrence of œdema and transudation to the influence of this drug.

The surprising rapidity with which these children recover their strength as compared with adults, has always seemed to me characteristic. Although cases are now and then met with, of convalescent children who are extremely emaciated and scarcely able to sit up in bed, still we have much oftener been surprised to find children who had lain in bed for weeks in a state of apathy and drowsiness, begin at once to sit up in bed and play, whenever the temperature fell or even during the intermittent stage; Rilliet and Barthez have also observed this.

On the other hand, as in most febrile diseases, we found a marked emaciation and loss of weight, and the child's complexion and nourishment did not return to their normal condition for months afterwards. The destruction of albuminous material caused by the prolonged high temperature and the deficient nourishment explain this fact, which causes so much anxiety to the relatives. In contrast to this, however, stands the fact of the unusually rapid growth in length which takes place during the disease and convalescence, especially affecting the long bones of the lower limbs. This is also observed in other infectious diseases, but never to the same extent as in typhoid fever. In connection with this we have another appearance, to which Prof. Köbner directed my attention some years ago in the course of conversation, namely, the formation of transverse fissures of the skin on the extensor surfaces of the lower limbs, especially above the patella. These fissures, which are red to begin with, gradually lose colour and finally come to resemble the lineæ albæ, which are seen on the skin of the abdomen in pregnant women. They are due to excessive stretching of the skin (especially when the knee-joints are bent), owing to its having become too small for the rapidly growing bones. But this phenomenon must be rare, for, although I have never omitted to look for it of late years, I have never yet seen a case of it. Auboyer[1] has described several cases of this condition occurring in severe typhoid fever.

Finally, a few more words on the relapses of typhoid fever. While Rilliet and Barthez have observed only 3 relapses out of 111 cases, I have met with 38 out of 302; and in these there were 20 cases in which neither cold baths nor any other cold applications had been used. An error of diet was only discovered in one case, that of a boy of 5 years, who after eating a large quantity of plum-cake during the intermittent stage at once had a return of the high temperature, followed by a relapse, of which he died. That the error of diet was really the cause of the relapse, I should not like to assert; for in all the other cases an excess of the kind could be positively excluded.

The severity of the primary attack gives no guarantee against the occurrence of a relapse; for although in most cases there has been a mild form of the disease to begin with, relapses

[1] *De la croissance et des rapports avec les maladies aiguës fébriles.* Paris, 1881.

do also frequently occur after severe cases of typhoid. As a rule, the relapse only sets in during the convalescence, about the 3rd—5th week of the disease, after the temperature has been quite normal for some time, 3—10 days on an average, and in one case even 18 days. Only in 2 cases have I observed a very short afebrile interval of 24—48 hours. In a smaller number of cases, there was certainly no period of complete apyrexia at all, and the relapse took the form of a gradual or sudden exacerbation of the intermittent stage of the fever. After the morning temperature had already been normal or subnormal, and only in the evening reached 101·3°—102·2°, a rapidly increasing exacerbation suddenly set in again, sometimes ushered in by a rigor.

Otto M., 11 years old, admitted on May 13, 1878, in the stage of defervescence of typhoid fever. Intermittent stage from 18th—25th. Temp. m. 97·7°—99·1°; ev. 101·3°. On the 25th, sudden rigor after which the temperature again rose; m. 101·8°; ev. 104°. Not till the 7th June was the temperature again entirely normal.

Carl Sch., 12 years old, admitted on November 8, 1878, in the intermittent stage of a severe attack of typhoid fever which had already lasted 3 weeks. Great emaciation and debility. On the 9th and 11th November, a rigor occurred between 2 and 3 p.m. Temp. ev. 104·5° and 103·1°. Complete disappearance of the fever during the next two days. On the 14th, 15th, and 16th, however, the temperature was again high in the evening although in the morning it was normal. On the 17th, the morning temperature also rose again, the spleen became palpable and painful, and a relapse now commenced which ended favourably after 9 days.

In these cases the relapse began in quite an unusual way. On two consecutive days we found attacks resembling those of intermittent fever, followed by two days quite free from fever. The next three days showed only evening rise of temperature, and after that for the first time the relapse began to run its usual course. The symptoms are usually quite the same as those of the primary attack. The roseola and the enlargement of the spleen (which has generally disappeared by this time) are wont to develope anew; and in a few cases the disease assumed even a more alarming character than before, so that 4 out of my 38 cases ended fatally. The duration of the relapses in my cases was as follows:—

| | | |
|---|---|---|
| In 2 cases | 4 | days. |
| 18 „ | 6—9 | „ |
| 14 „ | 10—14 | „ |
| 4 „ | 16—18 | „ |
| — | | |
| 38 | | |

Only in one case did a second relapse take place three weeks after recovery from the first; the temperature rose in the evening to 103·7°, the attack lasted for 9 days, and could not be explained by any local affection.

Let me add that I have several times seen chronic skin-eruptions (eczema and prurigo) disappear during an attack of typhoid, but reappear again soon after recovery. In one case, an eruption of varicella appeared immediately after the temperature had fallen; in a few others, scarlet fever or measles developed during convalescence. Diphtheria was the most formidable complication; in several cases it caused death, and in two it required tracheotomy, which unfortunately was unsuccessful.

It only remains now that I should give you my experience of the treatment of typhoid fever in children. As we have at present no means of combating directly the morbid poison (whether we regard it as the "typhoid bacillus" or not), the main importance attaches to the treatment of the fever. In using this "antipyresis" we must never forget that we have got to treat not the disease itself but the individual patient, and that the merciless adherence to a definite method leads to a merely mechanical routine, and certainly is not always for the good of the patient. This is especially true of the cold water treatment which, according to my experience, is generally not nearly so well borne by children as by adults, who are more strongly organised. In the first place, we possess no means by which to ascertain the tolerance of the particular child in regard to this treatment before it is tried; and least of all can we depend in this matter upon the appearance of the patient, for conclusions drawn from this are apt to be very misleading. I have seen children who seemed weakly and delicate yet bear the repeated use of cold baths very well, while on the other hand a boy of 12, who was extremely strong and robust and had hitherto enjoyed perfect health, became so collapsed after two baths of 77° F. that we had to give him a whole bottle of tokay within

the next 36 hours in order to restore warmth to his hands and feet and to bring back the small pulse to its normal condition. In other cases collapse took place after the very first bath, or even after the child had lain for an hour or two on a water-bed. Therefore if you wish to guard against the onset of those symptoms of collapse, which are certainly to be dreaded in typhoid, you must always regard the first bath merely as an experiment, on the result of which the nature of the further treatment is to depend. The ordinary custom, of giving a few spoonfuls of wine before and after the bath, is by no means sufficient to prevent its action from being injurious in unsuitable cases. Further, add to this the fact which one finds confirmed by daily experience, that so long as the fever is at its acme, or while there are only very small morning remissions, cold baths cause but a slight fall of temperature, and this lasts only for a few hours at most. Then again, for the reasons just given, I never care about going on giving baths whenever the temperature rises above 103·1°—as the fanatical supporters of the cold-water treatment would have one do. I have, therefore, gradually come to limit the use of baths in infantile typhoid to a much greater extent than I once did. Generally, I order a bath only when the evening temperature is 104° or more; and I do not give more than two baths in the 24 hours, of an average temperature of 88° F. and never below 81°. These baths have a distinctly favourable effect on many children, in so far as they give rise to a feeling of comfort such as can be caused by no other remedy in such cases, and relieve for the time any grave nervous symptoms which may happen to be present. The child must not be left in the bath for more than 5—8 minutes at a time. Should symptoms of collapse appear after the bath (trembling, coldness of the hands and feet, small pulse, collapsed appearance), and should they not disappear rapidly on the patient's being put back into bed, I regard this as a distinct contra-indication of the further use of the bath. In slighter cases where the temperature is not so high and the morning remissions are greater, I stop the baths altogether, and only apply an ice-bag to the head or perhaps to the abdomen; these are generally well borne, but they must be removed at once if the children begin to complain of cold. When the patients are very restless we may also try lukewarm baths of 90°—93° F., which I have often known to

have a calming effect. In the less severe cases there is no need whatever of any energetic antipyretic treatment by drugs. A bland fluid diet (milk, beef-tea), with 4 or 5 teaspoonfuls or dessertspoonfuls (according to the patient's age) of wine, is perfectly sufficient; and only where we are obliged (as in private practice) to write a prescription of some sort, we may order a hydrochloric acid mixture.

In serious cases, with a very high temperature, I used often to try the effect of giving large doses of quinine (grs. viiss—xvss), administered an hour or two before the evening rise of temperature, instead of the cold baths; and I have also frequently used this treatment along with the baths. According to its antipyretic effect, the dose of quinine was repeated every day or every second day. We were not deterred from pushing this drug either by its being vomited (which occurred even when it was given in half a wine-glassful of lemonade) or by the ringing in the ears, which frequently set in after it. Unfortunately, the same objection can be urged almost as strongly against quinine as against the cold baths. During the acme of the disease, especially in bad cases, its antipyretic action is slight, or at any rate passes off very rapidly. This mode of treatment only begins to be distinctly successful when the morning temperature begins to fall a little, and then indeed we are often able by a large dose of quinine to bring down the temperature of the following morning to a normal or even subnormal level, and even to cause a considerable fall for the next 24—36 hours. Salicylate of soda has been much recommended, but I cannot advise you to use it in typhoid in children. Although from my own experience I know that it has an antipyretic action equal to that of quinine, I have gradually given up using it owing to the vomiting often caused by large doses and especially to the alarming collapse which I have several times observed. Soon after taking grs. xvss—xxxi of this drug, the temperature fell about 4° or 5° F. There was copious perspiration, and such an alarming fall of the pulse, coldness of the extremities and collapsed appearance of the features, that we were obliged to resort to the use of strong stimulants (wine and musk), which I have never had to do after large doses of quinine. On the other hand, during the last few years we have used antipyrin with such good effect, and with an almost entire freedom from bad results that I now prefer this remedy

to all other antipyretics. After one dose of grs. iiiss.—ivss. (in older children, grs. viiss. at most), the temperature fell rapidly 4° or 5° F., and remained down for 5—6 hours. We have often found one or at most two or three doses in the 42 hours to be sufficient.[1] Only in three cases, did serious symptoms of collapse set in, requiring the use of stimulants. Recently we have also been trying antifebrin (grs. iss.—iii. according to the age) in the hospital with satisfactory results, although I should not prefer this remedy to antipyrin. We must always remember, however, that all these remedies merely lower the temperature for a time, thus improving the general condition, but that they do not influence the course of the disease as a whole.

In cases where the diarrhœa was so profuse as to require special treatment, subnitrate of bismuth (grs. iss.—iii. every hour) (Form. 30) or that along with tannic acid (gr. i.—iss., with extract of nux vomica, gr. $\frac{1}{10}$, or tincture of nux vomica, ♏ i.) (Form. 33) were generally successful. Intestinal hæmorrhage we have been in the habit of treating with liquor ferri perchloridi (gtt. viii., every 2 hours) and iced fomentations to the abdomen; constipation, with castor oil (dessertspoonful doses) or by injections of water into the intestine. The bronchial catarrh we have usually been able to leave without special treatment. Only when it was very diffuse or was developing into broncho-pneumonia did we give infusion of senega with aromatic spirit of ammonia (Form. 20) or benzoic acid with small doses of camphor (Form. 21) as stimulant expectorants. I have used dry cupping or fly-blisters only in a few cases of extensive pneumonic consolidation.

Whenever symptoms of collapse appeared, we endeavoured to meet them by giving large doses of tokay or port wine (a dessertspoonful every hour), musk, camphor (Form. 14), or subcutaneous injections of sulphuric ether. When there was great restlessness and sleeplessness I have often given chloral (grs. xvss.—xxxi. either internally, or in the form of an enema) with good effect; while morphia, given either internally or subcutaneously (gr. $\frac{1}{3}$—$\frac{1}{7}$), seemed less sure in its action. In all cases in which the fever lasted into the second week we gave regular doses of

[1] Bungeroth, *Charité-Annalen*, xi., 1886, S. 599.—Moncorvo, (*De l'antipyrine*: Paris, 1886) gives as much as grs. 46½ in the day.—Fereira, *Contributions à l'étude clinique des applications de l'antipyrine*: Rio, 1885.

always to be explained by renewed action of the malaria. The hypodermic use of quinine has this advantage, that smaller doses suffice, and we avoid the struggling resistance which the children offer to a very bitter medicine. But, with a few exceptions, the injections which I used caused so much pain and irritation that I should only have recourse to them in those cases of urgency, in which the internal use of the drug is out of the question.

I may take this opportunity of mentioning a few cases which I met with in Berlin in quite different districts of the town, and which could scarcely be regarded as due to anything but malaria, but which nevertheless offered the most obstinate resistance to treatment with quinine. Of the patients, two were 5 and 8 years old respectively, and only one had not passed the second year. In both there occurred daily attacks of fever lasting for hours, generally in the afternoon or towards evening, which either began with a hot stage at once, or were ushered in merely by a slightly marked and very short cold stage. The temperature in those attacks rose to 103° and over, and was not always absolutely normal even in the afebrile period. Apart from these attacks the children seemed well, but they became pale, flabby, and weak after the disease had lasted for weeks. In spite of repeated and most careful examination of all the organs, we could not find the slightest reason for the rise of temperature, and in no case was there any enlargement of the spleen. We were all the more inclined to suspect an insidious attack of endocarditis or miliary tuberculosis in process of development, as the steady use of quinine in large and small doses had absolutely no effect. In one of these cases the blood was examined, but it was found to differ in no way from the normal condition, and there was, especially, no leucæmia or melanæmia. Therefore as the condition remained quite unchanged for many weeks, and as nothing abnormal could be found on examining the heart, and as the idea of miliary tuberculosis had to be abandoned, I sent the children out of Berlin on the theory of a persistent malarial influence, and rapid recovery followed this change of residence. These children can hardly have been suffering from an attack of "intermittent malaria," because an attack of this nature would certainly not have resisted the action of quinine in this way. The source of the malaria, however, which may have been situated in the dwelling-house, was all the more difficult to verify

as no other member of the family had any similar sympto
had at any time suffered from such. Nevertheless, I
advise you not to be too hasty in assuming the pres
malarial infection in similar cases, for I have seen childr
died finally of tuberculosis or multiple lymphosa
after they had been subject for a long time to attacks o
of this kind, alternating with periods of absolutely
temperature.

## SECTION IX.

### CONSTITUTIONAL DISEASES.

### I.—*Rheumatism.*

Acute articular rheumatism (polyarthritis acuta rheumatica) which is now regarded by many as belonging to the class of infectious diseases, is by no means a rare affection in childhood. It differs from the same condition in adults in that its symptoms are generally milder. Both the local affection and the accompanying fever are usually less severe. With few exceptions the number of joints affected is smaller, and the swelling and painfulness less; the temperature does not, as a rule, rise above 102° or 103°. Also, the profuse sweating and the eruption of sudamina which are almost always present in adults, I have rarely seen occurring spontaneously in children, for they usually occur only after the use of salicylic acid. The joints most frequently affected were those of the ankle and knee, next, those of the upper extremity, and those of the fingers and metacarpal bones. In the latter case the fingers and back of the hands are sometimes slightly swollen and œdematous. I have only rarely seen the hip-joints become painful and immovable. In a girl of 5 years, both ankles, both wrists and the right knee-joint became swollen almost simultaneously, and the skin covering them was flushed in a way which is not generally seen or at most is only occasionally found over swollen finger-joints. I have frequently observed metastasis of the affection from one joint to another, and also its return to a joint which had previously recovered; and by this the course of the disease, which is generally 8—10 days, is protracted to 2—4 weeks as in adults. But the later recurrent attacks of the joint-affection always become less severe and shorter, and the accompanying fever also becomes less and shows only moderate elevations (100·8°) of temperature during the remainder of the course, alternating

with complete intermissions. Sometimes the children complain of pain in the abdomen and tenderness to pressure there; in others the rheumatic attack is accompanied by tonsillar sore-throat, and a certain degree of difficulty in swallowing.

Most of the cases of acute rheumatism that I have met with in childhood affected children between 9 and 13 years of age. Far less commonly I have seen younger children affected (5—8 years, or even smaller still),—and I have already[1] published a few cases of the latter kind. One of these was that of a child only 10 months old, in whom the symptoms of acute rheumatic polyarthritis (fever, painful swelling and immobility of the joints of the right hand and elbow, and of the left ankle and knee) were complicated by broncho-pneumonia and probably also by left pleurisy. After the attack had lasted 4—5 weeks we were able to make out a kind of crepitation on passive movement of the right elbow-joint (roughness of the articular surfaces) and also during the period when the joint-affection was diminishing there appeared a hardness and contracture of the adductors of the thigh, which only disappeared slowly after three weeks, and was probably to be regarded as due to rheumatic myositis.[2]

It is much rarer to find pneumonia and pleurisy as a complication (which occurred in a case already given in vol. i., p. 482) than it is to find endocarditis, whether alone or combined with pericarditis. Indeed, judging from my own experience, I should suppose that this complication is commoner in children than in adults. I have seen endocarditis even in cases where only one joint—for example, the knee—was affected. I may refer you to what I have already said about this complication (vol. i., p. 476) and shall only repeat here that the local symptoms, especially the sharp pain in the cardiac region (which is increased by pressure and percussion, and may even be so severe as to deprive the patient of sleep), likewise the dyspnœa, the irregularity of the pulse and the increase of the fever—are pre-

[1] *Beiträge zur Kinderheilk., N.F.*, S. 241.

[2] Several of the cases of acute rheumatism in children during the first weeks and months of life which have been published will not, I think, bear searching criticism. For they seem to be due to a confusion of this condition with syphilis of the epiphyses or with multiple periostitis of the articular ends of the bones. One indubitable case, however, has been published by v. Basch (*Prager med. Wochenschr.*, No. 46, 1884) affecting a boy of 13 weeks old. Schäfer (*Berl. klin. Wochenschr.*, 1885, No. 5) has seen new-born children infected by their mothers, who were suffering from rheumatism.

sent only in a small number of the cases. More frequently endocarditis (and even pericarditis when not severe) remains latent, being only discovered on local examination. I have had frequent opportunity of observing recent inflammation of affected valves (endocarditis recurrens) in children with old valvular disease—owing to a fresh attack of articular rheumatism. In addition to the example of this already given (vol. i., p. 479) I may adduce the following.

> Martha Schm., 11 years old. Had had an attack of acute articular rheumatism a year before and after recovery from this, incompetence of the mitral valve had been discovered at the polyclinic. On June 4, 1877, she was again affected with rheumatic swelling of both ankles; temp. 103·3°; pulse 140, small; dyspnœa, resp. 40. Salicylate of soda, grs. xxxi. The fever continued (100·8°—102·9°), and violent pains in the region of the heart appeared during the next few days, while the affection of the ankle-joint diminished and did not pass to any other joint. The pain kept the child from sleep, the præcordium was tender on palpation and percussion; resp. 52—68; pulse 144. The systolic murmur at the apex was considerably louder. On the 8th, there was also distinct pericardial friction following both sounds. This was still audible on the 12th, although the child felt better, and it only disappeared on the 23rd. This attack of recent peri-endocarditis recovered under antiphlogistic treatment — wet-cupping (6 cups), ice-bags, calomel (gr. ss.), mercurial ointment, blister. The pain recurred between the 24th and 29th, so that the ice-bag had to be re-applied. On 14th July, the patient was discharged with the old heart-trouble, the cardiac dulness reaching ⅔ inches beyond the right border of the sternum..

I need not return here to the connection between acute rheumatism and chorea, of which I have already spoken (vol. i., p. 207). I shall only add that I have as yet seen just one case of the so-called cerebral rheumatism, such as has been observed in adults and also in children by a few writers (Picot, Roger), and in this case chorea set in simultaneously, and death ensued from pericarditis. I cannot, therefore, decide whether Roger is right in supposing that all cases of cerebral rheumatism in children are combined with pericarditis.

The muscles which are most frequently affected by rheumatism are those of the throat and neck. You must not, of course, put down every case of stiff-neck or of torticollis in a child as a rheumatic affection, for you must always remember

that a number of other more serious maladies, especially disease of the cervical vertebræ and meningitis, may give rise to this symptom.[1] Still, cases of torticollis are not by any means rare, in which persistent contracture of the lateral muscles of the neck is undoubtedly to be attributed to the effect of a chill, or for which at least no other cause can be discovered. In these cases the use of iodide of potash, warm poultices and rubbing, massage or electricity, soon brings about recovery. In two infants of 12 and 15 months respectively, there was rheumatism, contracture of the muscles of the throat and neck, along with broncho-pneumonia; and some cases have been described (Picot) in which an attack of wry-neck was followed by chorea, just like acute articular rheumatism. Less commonly other groups of muscles are affected by painful rheumatic contractures, *e.g.*, the adductors of the thighs, as in two cases already given (vol. i., p. 479 and vol. ii., p. 359). Even in very young children who cannot speak I have several times observed symptoms which I could not explain by reference to anything save muscular rheumatism. These children, who had hitherto been quite well, suddenly became unwilling to use one of their lower or upper extremities. Pressure and passive movement of the limb were painful and at once excited violent screaming; there was also sometimes slight œdema of the backs of the hands and feet. The joints themselves remained quite unaffected, but yet the affection sometimes passed rapidly from one muscular area to another, then perhaps disappeared entirely for a time and returned afresh. The affection soon recovered when the child was made to rest in bed and the affected part was wrapped in cotton-wool. When it was one of the lower extremities that was affected, one was apt at first to suspect hip-joint disease. Sometimes the periosteum was gravely implicated. Two girls of 10 and 12 years respectively, who had been walking with bare feet on the cold pavement, were seized with such violent pain in the periosteum of the femur that every attempt at movement and any pressure on the swollen bones was unbearable, and, as the patients were also feverish, we were afraid that acute osteomyelitis was about to set in. In both cases, however,

[1] *Cf.* also the case of intermittent torticollis in vol. i., p. 190, and a case of pure spastic contracture of the muscles of the neck in my "Beiträge zur Kinderheilkunde," S. 24.

iodide of potash relieved the patient in a few days, and recovery soon took place.

After an attack of acute rheumatism, especially when it has affected the joints, the children are very apt to suffer from relapses. These may recur several years in succession and aggravate the already-existing valvular disease; they are also often followed by a return of the chorea. I have often observed vague articular pains returning from time to time for weeks or months after recovery from an acute attack. These may be accompanied by slight œdema in the neighbourhood of the joints or we may also have slightly febrile relapses. Only once, in a girl of 10 years, have I seen hydrarthosis occurring in the knee-joint, and requiring a long course of treatment.

Marie N., admitted on October 12, 1873, presenting the symptoms of accumulation of a large quantity of fluid in the left knee-joint—great swelling, distended outlines, floating patella. Violent pain had occurred in the left leg 14 days before, accompanied by swelling of the ankle-joint and fever. A few days afterwards the hip-joint was also painful and immobile. The pain then suddenly disappeared from the parts hitherto affected, and was succeeded by pain and swelling of the left knee, which had in the meanwhile increased in size. Patient seemed perfectly well in other respects; no fever. Treatment: confined to bed, ice-bag applied to the knee. On the 19th, after the pain had entirely ceased, joint painted with tincture of iodine, which set up an unusually severe inflammation and vesication of the skin. On 27th November, discharged perfectly cured.

Chronic rheumatism is much less common in children than the acute disease, indeed this affection is but rarely seen in its typical form. I have only seen very few instances in childhood of those permanent changes in the joints and tendonous structures which we so often meet with in adults in the form of "arthritis deformans."

Boy of 14 years, brought to the polyclinic on December 30, 1864. For the last 6 years had violent tearing pains in the hands and feet. He was said to have had no previous attack of acute rheumatism. On the left hand, almost complete anchylosis and nodular swelling of the joints between the 1st and 2nd phalanges of the thumb, index and middle fingers; on the right hand, similar changes (but less marked) in the index, middle and ring fingers. Swelling and tenderness of a few of the metacarpal bones. On the left foot, similar changes in the joints of the great and 4th toes.

Attacks of palpitation and dyspnœa without anything abnormal being discovered on examination. Further cause unknown.—The second case was quite similar. It was that of a Russian girl of 13 years, whom I only saw on one occasion, in my consulting-room.[1]

We more frequently see painful swelling and stiffness of various joints remaining in children, after one or more attacks of acute articular rheumatism, and resisting the most approved remedies for months and years.

In a boy of 7 years, who in the spring of 1876 had an attack of acute articular rheumatism mainly affecting the ankles, these joints were in October still so immovable that the child was supposed to have paraplegia. We found, however, on examination that there was nothing of the sort; for there was only a moderate amount of permanent swelling with great tenderness of the malleoli, but especially of the periosteum of both calcanea, and the plantar aponeurosis, which prevented the boy from planting the soles of his feet firmly on the ground. Recovery after treatment with iodide of potash continued for months.

Helene G., 12 years old, had suffered a year previously from an attack of acute articular rheumatism, and had complained ever since of pains in the joints of both hands and both feet. On admission into the ward (March 17, 1881), we found that the joints of both feet and knees, both elbows and shoulders and also those of the hand and fingers of the left side were swollen, painful and difficult to move. There was absolutely no fever. Treatment: iodide of potash and warm baths. Rapid improvement. On 3rd April, the patient left her bed and now only complained of stiffness of the ankles on walking. In the beginning of May, painful swelling of the wrists and ankle-joints again set in and continued for many months, alternately getting better and worse and sometimes accompanied by slight rises of temperature; and they had only been partially cured by March, 1883, in spite of the continuous use of iodide of potash, baths and painting. For a long time the affected joints were practically anchylosed, and it was only the persistent application of massage that after some months brought about a considerable improvement in the mobility, especially of the wrists.

In this last patient we observed a symptom which has been pointed out by Meynet,[2] and later by Rehn[3] and Hirschsprung.[4] The first case of this kind which I observed was in 1876.

[1] *Vide* P. Wagner, "Ueber Rheumat. chron. bei Kindern," *Münchener med. Wochenschr.*, 1888, Nos. 12 and 13.

[2] *Lyon médical*, 1875, No. 49.

[3] Gerhardt's *Handb. d. Kinderkrankh.*, 1878.

[4] *Jahrb. f. Kinderheilk.*, Bd. xvi., H. 3 and 4.

Anna M., 14 years old, appeared in my consulting-room on January 21, 1876. Within the last couple of years, two attacks of acute articular rheumatism. Incompetence of the mitral valve, with considerable hypertrophy and dilatation of the heart. About 6—8 weeks after these attacks, there appeared on both occasions several "exostoses" from the size of a pea to that of a pigeon's egg—first on the styloid process of both ulnæ and then on the margin of the patellæ. These were at first roundish, soft and tender, but gradually became hard, insensitive and pointed. There was a very considerable number of them and, on the second occasion, a similar nodule also formed in the left palmar aponeurosis.

These nodular new formations were also found in the case of Helene G. (p. 362) on July 10, 1881, above the olecranon on both elbows, likewise on the dorsal aspect of both wrists, internal to the styloid process of the ulna, and, finally, over the right sterno-clavicular joint, where the aponeurosis of the sterno-mastoid passes on to the manubrium sterni. The nodules were of the size of a small pea, were readily movable, and scarcely tender. Under treatment with salicylate of soda, the fever had disappeared by 9th August, and we now found that the nodules on the right elbow had also entirely disappeared. The remaining nodules gradually became smaller and flatter, and in the course of the autumn they disappeared entirely, and had not re-appeared by the time the patient was discharged. In a boy of 9 years with incompetence of the mitral valve, which had developed (June 1885) five months after an attack of articular rheumatism, we likewise found several hard, somewhat movable nodules, about the size of a bean, smooth on the surface and situated over the olecranon on both elbow-joints. The following case is, however, particularly important owing to its having been examined post-mortem.[1]

Auguste W., 12 years old, admitted on April 4, 1881. On examination, we found incompetence of the mitral valve with hypertrophy of the right ventricle. No history of the case was procurable. On a few of the joints, especially over the insertion of the tendons, we felt small diffuse elastic thickenings which were not tender. They thus occurred on both knee-joints at the insertion of the quadriceps into the upper border of the patella, and on both wrists above the styloid process of the ulna. On the 19th of April, there was slight fever and severe pain in

[1] G. Mayer, *Berl. klin. Wochenschr.*, 1882, No. 31.

both wrists which became markedly swollen and immovable. There was also headache, giddiness, increased action of the heart, pain in the cardiac region, and slight œdema of the face and legs. On 25th May, when the patient was again able to go about, the diffuse thickenings over the knee-joints had formed into resistant nodules the size of split peas. Quite similar nodules could now be felt over both external malleoli, over the right elbow above the olecranon, over both posterior superior iliac spines, and finally over the right shoulder-joint below the end of the clavicle. These were only a little tender and were somewhat movable. After death, which occurred with symptoms of general dropsy on 7th July, a post-mortem was performed by Prof. Grawitz, and we found (in addition to the heart-lesion and its results) oval tumours about the size of a cherry-stone and of tolerably hard consistence in the situation where we had felt the nodules. They were situated on the aponeuroses of the tendons and consisted mainly, as we found on microscopic examination, of fibrous tissue with fibro-cartilaginous fragments interspersed. Moreover, all the nodules had not the same structure; in one the connective-tissue predominated and in another the cartilaginous, and the nodule on the clavicle had become as hard as bone, owing to its containing calcareous masses. The nodules which we had felt over the patella, had disappeared entirely.

A nodule, the size of a pea, which we found over the olecranon in a girl of 11 years who had died of endocarditis verrucosa and cardiac hypertrophy following acute articular rheumatism (January, 1888), presented exactly the same characters. Likewise in one of Hirschsprung's cases, the nodules were found on examination after death to consist of a new formation of fibrous tissue. We must, therefore, regard them as the products of an inflammatory process which has been lighted up in the aponeuroses of the tendons by the acute rheumatism of the joints. These products may be absorbed and disappear owing to regressive metamorphosis (fatty degeneration) as is shown by my case and by another of Hirschsprung's; but, on the other hand, bone-like structures may be formed owing to calcification of the nodules. The tendons of the muscles and the aponeuroses may not, however, be the only points of origin of these inflammatory products, for the periosteum and perichondrium also seem capable of producing them. The nodule which was found on the clavicle in my last case was closely united to it, and was to be regarded as a true exostosis, as was also another hard growth of the size of a pea which we

found adhering to the head of the right ulna. To this class also belongs the case of a boy of 10 years, published by Ebert[1] and Virchow.[2] In this patient numerous hyperostoses and exostoses had formed, as the result of repeated attacks of acute articular rheumatism, on the articular ends of many of the long bones, and also on other parts of the osseous system. It is worthy of note that all these things have as yet only been observed in children between 3 and 14 years of age. Further observation is required to show whether they also occur in adults as the result of rheumatic polyarthritis.[3]

We must distinguish these "rheumatic fibromata or osteomata" from another kind of multiple exostoses, which are very rare in children, and for which either no cause at all can be discovered or only a hereditary predisposition, as in the case of the neuro-fibromata of the skin which have recently been much spoken about.

Boy of 10 years, brought on November 21, 1880; healthy. Since the 3rd year of life a large number of round or conical exostoses, which were not tender, had formed on the epiphyses of the right radius, of the left ulna, of the 9th rib on the left side, on the spines of both scapulæ and on the inner condyle of the right tibia. No hereditary predisposition.

Boy of 7 years, brought January 10th, 1882. Since his second year there had been exostoses on several of the costal epiphyses, and during the last few years also on both ulnæ and on the lower part of the left femur. The latter, on account of its size and hooked shape, could be easily felt through the child's trousers. The patient's grandmother was said also to have had small exostoses.—Similarly, a girl of 7 (brought February 7, 1874), like her father, presented a number of tolerably symmetrical exostoses on both sides and on various bones, especially about the epiphyses.

The most remarkable case was that of a boy who, although no hereditary tendency could be ascertained, had innumerable exostoses on almost all the bones, but was otherwise perfectly healthy and has now grown into a strong young man. In this case the formation and growth of several exostoses repeatedly took place under my actual observation; but by the period of puberty this process came to an end.

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[1] *Deutsche Klinik.*, 1862, No. 9.

[2] *Die krankhaften Geschwülste*, Bd. ii., S. 83.

[3] A case published by Scheele (*Deutsche med. Wochenschr.*, No. 41, 1885) seems to indicate that these nodules may also occur with a febrile rheumatism of the tendons, and even in connection with chorea.—Money, *Lancet*, 1883, No. 13.

Along with this formation of exostoses we occasionally find ossification of the tendons and muscles, which may attain to such a degree that a large number of the muscles and tendons in the body become changed into a mass of bone, and almost all movement becomes impossible (myositis ossificans). Just at the time that I became Director of the Children's Department in the Charité, there was a patient there (a girl of 12 years), the notes of whose case have unfortunately been lost, but who certainly presented one of the most remarkable examples of such almost universal ossification of muscles and tendons. In this child, as far as I can remember, we were unable to discover any connection with rheumatism. In a few cases (Skinner[1]) every bruise which the child gets seems to produce a bony formation of this kind in the muscles, accompanied by fever and pain.

I have but little to add regarding the treatment of rheumatism. In acute cases we have been accustomed (as in adults, and with equal success) to give either salicylic acid (grs. iii.—ivss., every two hours in wafer-papers), oftener salicylate of soda (grs. v. every two hours) or antipyrin (grs. ivss.—viiss.), and, in the chronic cases especially, iodide of potash.[2]

## II.—*Anæmia.*

The diminution of red blood-corpuscles and hæmoglobin which we call "anæmia" is extremely common in childhood, especially as a result of all diseases which are accompanied by loss of the fluids of the body. Children who are suffering from chronic diarrhœa, from extensive tuberculosis or from scarlatinal nephritis are invariably anæmic. But also those who become atrophic in consequence of defective nutrition and who are living in crowded rooms or in damp cellars, show in their faces the signs of poverty of blood. Of all these cases in which anæmia has only a secondary importance I need not speak further here. I shall refer only to that form which develops in otherwise healthy children and generally presents the same symptoms as the chlorosis of puberty. We observe this variety of anæmia

[1] Bouchut, *Maladies des enfants*, p. 903.

[2] I shall return, in considering purpura, to certain changes of the outer skin which sometimes appear as the result of rheumatic affections.

by no means uncommonly even in children of 8—10 years, and almost as frequently in boys as in girls. Every physician knows these cases, which are brought to him by the anxious parents, who say that nothing is wrong with the children but the want of a healthy complexion. The "green" appearance (a favourite Berlin expression) arouses the most lively apprehensions, although we find nothing further to justify them. Also, the pale yellowish colour of the surface of the skin is not always accompanied by an equal pallor of the visible mucous membranes, which may remain tolerably red in colour. All these children are unusually languid for their age, easily tired, ill-tempered, or extremely nervous and irritable. There is often also an entire loss of appetite, and especially a distaste for animal food, while the well-known unnatural appetite of chlorotic patients but rarely occurs in children. There are frequent complaints of painful sensations in the region of the stomach or in the intercostal spaces, without our being able to discover any sufficient cause for them. The anæmic venous murmur in the neck is common but not invariably present, and resembles in every respect that heard in chlorotic patients—*i.e.*, it is found mainly or exclusively on the right side of the neck, is increased considerably when the neck is turned round to the left and on pressure with the stethoscope, and is sometimes also heard at the upper part of the right border of the sternum, along the course of the common jugular vein, as a muffled murmur sounding as if from a depth. I do not regard this murmur as of any importance unless it is also audible when the head is held perfectly straight; for turning to the left may cause an analogous murmur even in healthy individuals, owing to the pressure of the muscles. I have never been able to make out abnormal murmurs over the heart so long as I was careful only to apply the stethoscope very gently; for any strong pressure on the costal cartilages is certainly capable of making the first sound at once impure and murmurish. And, as a fact, it has seemed to me as if this was more readily produced in anæmic than in healthy individuals.

Implication of the nervous system is very often indicated by attacks of headache or a flickering haze before the eyes, to which I have already alluded (vol. i., p. 349) in discussing migraine and its relation to excessive mental work. I have

already, however, repeatedly pointed out that more serious neuroses (chorea, hysteria, cataleptic conditions) may also develope from such beginnings.

Before these children came under my treatment, almost all of them had taken a large quantity of iron, without any lasting benefit. The reason of this is, that the causes of the trouble which (in my opinion) are commonest—constant confinement to the foul atmosphere of the town especially in overcrowded schoolrooms, and mental overwork—can rarely be got rid of. The schemes now in full operation in many cities, for sending even the children of the very poor to live in the country during the school holidays, are to be recognised most thankfully as, at least, an attempt to diminish this evil, and as one means of coping with this cause of anæmia in children. It is best, when it can be done, to send the children out of the town altogether, and to have them educated in boarding-houses and schools in a healthy, open part of the country; for the ordinary holiday change of a few weeks at the seaside or among the hills generally does little good. Should the extreme degree of anæmia necessitate a course of treatment with mineral waters or baths, the most suitable places to visit are the iron-springs of Elster, Franzensbad (especially when there is any dyspeptic complication), Pyrmont, Driburg, Schwalbach, &c. When, however, expense is no consideration I should especially recommend Tarasp or St. Moritz in the Engadine, which are to be recommended for relaxed states of the system, owing to their high situation among the mountains. From repeated experience I can recommend these health-resorts even for children of 7 or 8 years of age. The situation of St. Moritz, which lies open to the sun in every direction, renders it more beneficial to anæmic children (who so much stand in need of sunshine) than many of the mountain-districts "with the odour of pine-woods" which are resorted to as cool and shady summer retreats. For this reason, too, I can also recommend residence in high sunny localities apart from the use of mineral waters—*e.g.*, Krumhübel, Schreiberhau in the Riesengebirge, and Heiden, Gais, and other health-resorts in Appenzel. On the other hand, I always regard residence at the seaside, which is so much recommended by many, as a doubtful experiment. In a number of cases it certainly exerts a distinctly favourable influence; but

in many others it has no effect; and if the children are timid and are forced against their will to take the baths, it may even do positive harm. I therefore always prefer an elevated mountainous locality which is open to the sun. Rubbing down with cold water, which is so much used, is no better borne by many children than are the cold sea and river baths, and I believe that the popularity of this method of treatment is due more to tradition and the desire to be doing something, than to the good effect which it has been known to produce.

The natural or artificial mineral waters (Spa, Schwalbach, Pyrmont, &c.) are also better suited for internal use at home than the ordinary preparations of iron, because they contain only a very small quantity of iron, and are more digestible. The blackish colour which the motions often assume while iron is being taken always indicates that a portion of the medicine is not being absorbed, but is being discharged from the alimentary canal in the form of sulphide of iron and, therefore, that the dose requires to be diminished. I do not think it matters much which of the artificial preparations you use—whether reduced iron, lactate of iron, dialysed iron, or one of the tinctures. The principal thing always seems to me to be, that the dose should be small (from $\frac{1}{2}$ to, at most, $\frac{3}{4}$ of a grain of the solid preparations, 8—12 drops of the tinctures 2—3 times a-day) and given for months at a time. In order to prevent the teeth from becoming black it is best to give the iron in the form of pills; but these can only be given to older children. In a number of cases which offered obstinate resistance to the effects of iron or in which it was not borne, I have got very good results from the use of arsenic (Fowler's solution, Form. 11), and I therefore advise you to give this remedy a trial whenever the condition of the stomach permits of it.

## III.—*Purpura.*

Under this name are grouped together several morbid conditions of which the nature is unknown but which have in common the property of causing hæmorrhages into the skin, serous and mucous membranes, and even into the parenchyma of various organs. These hæmorrhages generally occur spontaneously or without external cause, and not, as in congenital hæmorrhagic

diathesis (the so-called "bleeder's disease"), mainly after injuries of the skin and mucous membranes.

I have already, on a former occasion, mentioned that one must examine very carefully, especially among the poor and in hospital patients, to guard against mistaking for purpura what are in reality just ordinary flea-bites, the signs of which are often found in the form of small petechiæ all over the body. In the case of infectious diseases especially (typhoid, scarlet fever), I have often found it difficult to decide whether the hæmorrhagic spots visible on the child's admission were caused by flea-bites or were connected with the disease itself; for, as you will remember, real petechiæ and larger hæmorrhages into the skin may occur as the result of infectious processes, of endocarditis, or septicæmia. You must therefore never forget to examine the heart in cases of purpura, especially if there is any fever. In one case of endocarditis after scarlet fever, in which there had been distinct murmurs, but only an impurity of the first sound, I founded my diagnosis (which was confirmed by post-mortem examination) especially on the presence of extensive purpura.

Here, however, we have only to consider those hæmorrhages which occur as an *independent* disease and have no connection with general febrile affections or with endocarditis. When these only affect the skin, the disease is called "purpura simplex;" when they are accompanied by bleeding from the mucous membrane it is called "*purpura hæmorrhagica*," or "*morbus maculosus*." Unfortunately we know very little about the nature of these morbid conditions, and even of the anatomical causes of the numerous hæmorrhages. The old view, that these cases were due to a separation of the constituents of the blood, cannot be proved either by chemical or microscopical examination. I have examined several cases of purpura hæmorrhagica in this particular (in one of them Dr. Litten was good enough to repeat my examination) and I found the red blood-corpuscles large, well filled and in no way changed in respect of their colour and number. Small corpuscles (microcytes) were only seen occasionally, and the number of white corpuscles was no greater than in the normal condition. These cases, however, were certainly slight, and made a rapid recovery. The old idea, that there was a smaller amount or a lessened coagulability of the fibrin, has received just as little

confirmation. And, therefore, as the blood is found to be normal, we are naturally disposed to attribute the condition to some affection of the solid parts, *i.e.*, of the small blood-vessels. As the hæmorrhages may be due either to rupture of the blood-vessels, or to an easier migration of the red corpuscles through the vascular walls, it has been supposed that there exists an abnormal friability of the latter; and, as a matter of fact, various observers (Hayem, Straganow, and others) have described microscopic changes of the small arteries and capillaries which certainly might have given rise to such a condition. Recent researches (von Kogerer[1]) have demonstrated the presence of endarteritic changes—especially of the larger arteries of the reticular layer of the skin—thickening, hyaline and fatty degeneration of their walls with narrowing of their lumen, proliferation of the endothelium and the formation of thrombi. I believe, however, that these degenerations are only to be taken into consideration in severe and fatal cases of purpura. For if we consider how suddenly the disease often arises and how quickly it may again disappear, we can hardly assume the presence of any considerable change of structure in the walls of the blood-vessels which could be capable of disappearing so rapidly. From this we see that there must be several different conditions grouped together under the common name of purpura or morbus maculosus, which have nothing in common but the fact that they set in under the form of a "hæmorrhagic diathesis." The severe forms which end fatally may be accompanied by those changes in the small blood-vessels; in other slighter cases which soon recover, we might even assume the presence of a vasomotor neurosis leading to paralytic dilatation of the smallest blood-vessels, stasis of the blood, and rupture of the vascular walls or diapedesis of the old blood-corpuscles. The addition of slight œdema which occurs in a number of cases is in favour of this hypothesis.

Simple purpura, in which there are no hæmorrhages from the mucous membranes, sometimes occurs in ill-nourished anæmic and rickety children living in damp cellars. More frequently along with leucocythæmia and enlargement of the spleen (p. 127). The hæmorrhagic spots in these cases are always few in number, and no larger than a lentil. In another form of

[1] *Zeitschr. f. klin. Med.*, Bd. x., Heft 3.

the disease the spots are more numerous and larger. This occurs in children, accompanied by pains in the limbs, especially in some of the joints, and there may also be some articular swelling; or the patient may have suffered from such symptoms a few days before (the so-called purpura or peliosis rheumatica). In these cases we find a number of dark red or bluish rounded spots of varying size, especially on the legs and feet, but often also on the abdomen and arms. In a boy of 4 years, I found them also on the scrotum. They remain unchanged on pressure with the finger, and in the centre of some of them we find a papular or diffuse hardness and projection, due to coagulation of fibrin. But in addition to the above-mentioned spontaneous pain, there is pain on pressure over the tibia, ankles, and soles, and on movement of the joints, so that walking may be rendered more or less difficult. Occasionally eruptions of wheals appear in addition to the purpuric spots (erythema nodosum), in the centre of which a bluish blood-extravasation can be seen or felt, and I have often observed slight œdema of the dorsum of the feet and of the ankles, although the urine never contained any albumen. In a boy of 7 years, who had also hæmorrhagic spots on his arms and face, the eyelids, cheeks, and alæ nasi became œdematous; in another there was œdema round the elbow, on the dorsal surfaces of the right hand, and on both feet, and on the eyelids. The spots generally fade after a few days, but they soon re-form again whenever the pain or articular swellings reappear, or even without these, as soon as the little patient gets out of bed and begins to walk about again. Thus several weeks may pass before these relapses (which may also be accompanied by œdema on each occasion) cease to occur, and complete recovery sets in. In most of the cases which I have observed, the affection was not accompanied by fever, and there only rarely occurred slight irregular rises of temperature, with little disturbance of the general health, or even none at all; and they all ended in complete recovery. In a girl of 11 years, who seemed perfectly well but for loss of appetite, we were struck by the pulse, which was extremely slow (68) for that time of life, and also somewhat irregular in rhythm, although nothing abnormal was found on examination of the heart. In one case, that of a child of 18 months, an eruption of pemphigoid bullæ the size of a pea, with

sero-sanguinolent contents, appeared on both feet with slight œdema simultaneously with the purpuric spots, after the patient had suffered for days from pains in the legs. After about four weeks it had quite disappeared, but five months later a fresh eruption of purpura appeared on the lower limbs.

A more complicated clinical picture may result from a number of abdominal symptoms (vomiting, intestinal hæmorrhage and colic) being added to those already mentioned, purpura and articular swellings. This form I first observed in 1868 and described on a later occasion.[1] The first case of this kind was as follows :—

> A strong boy of 15 years took an attack of gastro-duodenal catarrh with slight jaundice resulting from indigestion. A few days after, pain in the finger-joints of both hands without swelling. A day or two later, extensive eruption of purpura on the thighs, followed soon after by violent colic, vomiting and black stools. The pains in the belly were sometimes so very severe as to keep the child from sleeping; the region of the transverse colon was distended and tender. Slight fever (never above 101·7°). After 5 days, these symptoms disappeared, but within the next 3 days, a relapse occurred with exactly the same symptoms. Convalescence after one week. Within the next few weeks, 3 other relapses occurred, always accompanied by bloody motions which were either black or orange-coloured and contained more or less considerable masses of blood. There were 5 such attacks altogether within 7 weeks. Finally complete recovery. Opium seemed to give the best results.
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> In March, 1869, I met with my second case. A boy of 4 years was suffering from "dysenteric" symptoms—colic, tenesmus, a few bloody motions. At the same time there were some large patches of purpura on both elbows and thighs. Improvement after 3 days' use of castor oil and calomel, but some fresh purpuric spots appeared on the scrotum and prepuce. A few days after, a fresh attack of diarrhœa with streaks of blood in the motions and violent colic; then constipation; fresh relapses of purpura. Total duration 3 weeks.
>
> The third case (March, 1873), was that of a healthy girl of 12. "Rheumatic" pains in the limbs for the last week, presently accompanied by pain and swelling of the wrists and ankles with slight fever. Heart unaffected. A few days later, extensive purpura on the abdomen and lower extremities. Very violent colic, keeping the child from sleeping; repeated vomiting and

[1] "Ueber eine eigenthümliche Form von Purpura," *Berl. klin. Wochenschr.*, 1874, No. 51.

diarrhœa with much blood in the motions. Disappearance of all the symptoms after 5 days. Relapse followed. Within 4 weeks, 4 such attacks took place. Finally, complete recovery. No special treatment.

The fourth case was that of a healthy girl of 11, who, in the summer of 1872, had suffered from rheumatic pains in both ankles and in the right hip-joint. In July, 1873 (*i.e.*, about a year later), there was again pain in the wrist and ankles, but no swelling. This was immediately followed by purpura on the lower limbs, moderate fever, loss of appetite, vomiting and colic, with solid motions containing a large quantity of blood. Urine normal. In the course of 5 weeks, 3 such attacks took place at intervals of 8—9 days. The last attack unaccompanied by fever. An ice-bag was applied to the abdomen and seemed to have a good effect; the purpuric spots also faded. Suddenly the pains returned in the left arm and in the right elbow-joint and on the following night (that of 23rd July), there occurred very violent colic, vomiting of green matter, and 4 motions of a deep orange colour mixed with a large quantity of blood-clot. No fever, tongue clean. Ice-bag to the abdomen, iced milk as nourishment, emulsion of almond oil. On the 25th, another black motion. The child seemed perfectly well until the 30th, when another relapse of the purpura took place. There was then an interval until September. During this month a fresh violent attack occurred, which was quite similar to the preceding ones, and with this the disease terminated. There was nothing abnormal found in the heart, with the exception of arhythmia and slow rate of the pulse (as low as 60), which was sometimes observable.

I met with a fifth case on January 17, 1880. A boy of 7 years who had suffered for almost 9 weeks from a number of attacks of this disease which were always becoming less marked. These consisted of violent colic with tenderness of the right side of the transverse colon, bloody motions, purpuric spots on the fore-arms, and rheumatoid pains in the limbs, but without swelling of the joints or fever. Hands and feet sometimes became œdematous. Urine normal. Ergotin and iron given without success. Gradual recovery under general treatment.

The sixth case was that of a boy of 8 years (end of May, 1883). A highly febrile illness (temp. 105·8°) had occurred a year before and had been supposed to be typhoid. On the 5th day of this attack, there had been purpura and spots of erythema, hæmorrhage from the gum and swelling of many of the joints. Recovery after 8 weeks. There had been repeated vomiting during this illness, but the motions had not been examined. On May 19th, 1883, he had a second attack of a similar nature with violent colic; and when I saw him, numerous purpuric spots were still visible on the back, nates and thighs. Further course unknown.

It is evident that all these cases are of the same character, and I have seen other two (girls of 12 and 8 years respectively) during the last few years. The purpura in all these cases was always accompanied by colic, tenderness of the colon, vomiting, intestinal hæmorrhage, and (except in the second case) by rheumatoid pains; swelling of the joints was less constant. In a girl of 5 years who was brought to my polyclinic on January 10, 1882, there had during the last two months been attacks of purpura with swelling, pain and immobility of several joints, frequently accompanied by violent attacks of colic and great tenderness of the abdomen on pressure, but no bloody stools were observed. In the summer of 1887, I had another girl under treatment whose case was similar. Thus, then, we see that one of the links in the chain of symptoms may be absent. It is, however, characteristic of all that the symptoms come on in separate outbreaks, with intervals of several days or weeks, or even of a year, so that the duration of the disease is considerably prolonged. Fever is not invariably present, and is generally only moderate in degree. That the symptoms described are very closely connected, no one, I presume, will deny; but I am no more able to explain this connection than I am to explain ordinary rheumatic purpura. As my cases, however, recovered (with the exception of two, which were not examined post-mortem), I am not in a position to decide whether the symptoms are due to extravasations of blood into the mucous membrane of the stomach and bowel, or to the endarteritic processes already mentioned (p. 371). These were formerly described by Zimmermann[1] as occurring in the case of an adult—narrowing of the smaller intestinal arteries from proliferation of the cells and nuclei of the tunica adventitia and media, and consequent multiple patches of gangrene of the mucous membrane of the bowel.[2] In spite of the fact that almost all my cases ended favourably, I should not care to give an absolutely favourable prognosis. For in two of the cases there was a complication with acute nephritis. In one of these it appeared during the presence of the symptoms I have described, and ended fatally from anasarca and hydrothorax, in the other

[1] *Arch. der Heilk.*, 1874, Heft 2.

[2] Compare also Scheby-Buch, *Deutsches Arch. f. klin. Med.*, 1874, H. and 5.

it set in after the disappearance of the primary symptoms and ended in complete recovery.

As to the treatment: the best results seem to be got from the application of an ice-bag to the abdomen, iced milk as nourishment, and an emulsion of almond or other oil; to which, when the pains are severe, I usually add extract of opium (gr. $\frac{1}{20}$). Strict confinement to bed is to be ordered here, as in cases of ordinary purpura rheumatica. In many cases of the latter I have got good results from the use of iodide of potash (grs. i—ii).

From those forms of purpura which we have been discussing, we must distinguish another, for which I reserve the name purpura hæmorrhagica or morbus maculosus. This form is distinguished by an entire absence of pain, of articular swelling, and of the intestinal symptoms which I have just described. We find nothing but the purpuric hæmorrhages, and the latter occur, in most cases, only from the gums and nose. It is sometimes said that the urine very often contains blood or albumen, but I have very rarely found this to be so in my cases. We often find little blood-extravasations on the mucous membrane of the lips and cheeks, which do not merely lie loosely on the surface but are infiltrated into the superficial layer, so that after they separate a very shallow ulcer may be left. In almost all my cases the disease began suddenly, without any warning, and in the midst of apparently perfect health. There is a sudden eruption of blood-spots of a dark-red colour, occasionally with a brownish-red or bluish tinge, from the size of a millet or lentil-seed to that of a sixpence, or larger. It extends over the whole surface without any regular succession, so that within 24—36 hours the skin may become spotted all over, like a leopard's hide. Here and there we also find hæmorrhages in the form of streaks or blotches. This occurred, for example, in the case of a boy of 7 years who had an extravasation filling up the whole of the left inguinal region, at one part of which a hard fibrinous nodule, the size of a pigeon's egg, could be felt. These spots never disappear on pressure; but we sometimes find a red area round about the central fibrinous nodule, the outermost hyperæmic border of which fades momentarily on pressure. When there is hæmorrhage from the mouth, chewing may be rendered difficult owing to the presence of clots sticking between the teeth.

Rough friction of the gum excites hæmorrhage very readily, as does bruising of the skin; and even scraping the latter with the finger nail is generally quickly followed by a blood-spot or a red streak, which does not disappear on pressure. The small pricks, which we make in order to examine the blood, bleed very much; and the insertion of the needle of a hypodermic syringe almost always causes a pretty large blood-extravasation into the skin and subjacent connective-tissue, which only disappears slowly with general discolouration. In one of my cases hæmorrhage also occurred repeatedly from eczema of the cheek. At the same time the general health was usually so little affected that the children would have preferred not to have been kept in bed. I have never been able to discover with certainty any enlargement of the spleen, or abnormality of the heart, or hæmorrhage into the fundus of the eye. As a rule all the hæmorrhagic spots come out at once; less commonly the course is protracted by the appearance of fresh spots, and it is generally 10—14 days before all the spots entirely disappear. I have never observed any fever in my cases; indeed I often found the temperature below the normal (98·4°—99°).

It is very rare to find any occurrences calculated to arouse anxiety taking place at any period of the disease. In a boy of 5 years, however, such profuse epistaxis occurred on two occasions that the nasal cavity had to be plugged; and in a girl of 11 years, there was hæmorrhage, lasting 36 hours, from the socket of a tooth which had been extracted. The danger of exhaustion from the constant recurrence of profuse hæmorrhage is not, therefore, very great; and it is a characteristic feature of the severe form of purpura hæmorrhagica, which is far less common than the form which we are now describing, and—as I have already said—is perhaps connected with permanent molecular changes in the small blood-vessels. The mode of commencement, which is not sudden but almost always gradual, the frequent relapses, the chronic course, and the steadily increasing anæmia, constitute an essential distinction between this form and the ordinary one, which usually has an acute course. We also find, in addition to this, a constant recurrence of profuse hæmorrhage from the most diverse parts of the body—nose, mouth, stomach, intestine, kidneys, external ear, and lungs. These cases, which are fortunately rare, may end fatally after lasting for

a month or a year, either from exhaustion with anasarca and dropsy of the cavities of the body, or quite suddenly owing to effusion of blood into a vital organ, especially the brain (vol. i., p. 270). During this protracted course there usually occur pretty long intervals of apparently perfect health. Thus I have seen an anæmic girl of 12 years suffer three summers running from purpura, epistaxis, and also occasional hæmoptysis, although she was perfectly free from the complaint during winter. In a boy of 13, who had suffered for two years from morbus maculosus and had spots of blood in the mucous membrane of the palate and uvula, there sometimes occurred intervals of several months, during which there appeared neither purpuric spots nor other hæmorrhages. Such intervals are apt to arouse delusive hopes, which are belied by a sudden recurrence of the purpura and hæmorrhages. While in the acute form the whole eruption generally comes out at once (as I have already said), in the chronic cases we find spots of quite different colours on the skin, owing to the continual recurrence of relapses. Recent bright or dark-red spots are found existing side by side with older bluish-green and yellow ones; and between them we see in many places pale pigmented spots, these being the last traces of the re-absorbed hæmatin.

In none of my cases of purpura hæmorrhagica have I been able to be quite sure of the causes of the disease. Most of the children were between 8 and 14 years of age (only one—who presented purpuric spots on the face, hæmatemesis and black motions—was not quite 2 years old), and seemed otherwise in perfect health. I was never able to prove that the disease had been caused either by an unhealthy dwelling, or deficient quantity or bad quality of the food. As regards the possible influence of preceding diseases, the only two that I can regard as responsible are scarlet fever and measles; and I have already referred (pp. 231 and 259) to their connection with morbus maculosus.[1]

The treatment of the acute and less severe form may,

[1] The result of Petrone's observations—that the purpura was due to bacillary infection—has not been confirmed by Hryntschak (*Arch. f. Kinderheilk.*, 1884, S. 461). I cannot say whether the two cases published by S. Simon (*Revue mens.*, Nov. 1885), in which morbus maculosus occurred as the fore-runner of diabetes mellitus, are more than chance occurrences. At any rate, this fact is worthy of notice, seeing that Gerhardt (as he has told me himself) has made a similar observation.

according to my recent experience, be purely expectant. I have no longer any faith in the efficacy of ergotin in this disease,[1] since I have seen a series of cases in the hospital recover in a short time by rest in bed and without any medicine, and also since, on the other hand, I have seen ergotin fail completely in several severe chronic cases, although it was used regularly for weeks. Moreover, if you wish to use this remedy you must only give it internally (Form. 44); for subcutaneous injections almost always cause a considerable amount of extravasation of blood in these patients, and may even end in causing suppuration. A case of purpura hæmorrhagica, published by Shand,[2] which was cured by the use of the interrupted current, stands alone as yet. This treatment is based, I suppose, on the same idea as led me to try ergotin. In the chronic form, one may recommend preparations of iron, especially tincture of the perchloride (Form. 45) and residence in pure country or hill air, but only at a moderate elevation. In any case, cold water treatment may be also tried; for it has done me good (temporary) service in two cases of this kind. The separate attacks of bleeding when they become severe, require various kinds of treatment according to their locality—in the case of epistaxis, by plugging, in the case of hæmorrhage from the stomach and bowel, by ice-bags and by liquor ferri perchloridi, &c.

During the last few years I have met with two cases of very extensive hæmorrhage into the skin rapidly causing death, which I have described under the name "purpura fulminans."[3] A third case of the kind was communicated to me by Dr. Michaëlis, and a fourth has been published by Charron.[4] All these cases have in common the fact that there were absolutely no hæmorrhages from the mucous membrane, and that extensive ecchymoses occurred, which made all the extremities of a blue- and blackish-red colour within a few hours, and presented a tolerably hard infiltration of the cutis with blood. Also, in two cases some sero-sanguinolent bullæ appeared on the skin, but there was never any gangrene nor any offensive smell. The course of these cases is extremely rapid—scarcely 24 hours

[1] *Beitr. zur Kinderheilk., N.F.*, S. 405.
[2] *Lancet*, July 19, 1879.
[3] "Ueber Purpura fulminans," *Berl. klin. Wochenschr.*, 1887, S. 8.
[4] *Observations relatives à la pédiatrie:* Bruxelles, 1886, p. 27.

intervening between the formation of the first hæmorrhagic spot and the occurrence of death; the longest case lasted 4 days. There are also no complications, and at the post-mortem we find nothing at all save general anæmia, in particular no trace of embolism or thrombosis. The etiology is equally obscure. One of my cases commenced during an attack of pneumonia, two days after a complete crisis had occurred; the other 1½ weeks after quite a slight attack of scarlet fever. In the other two cases, there was nothing on which to found an opinion as to the cause.[1]

## IV. *Scrofula.*

Although the clinical picture of the disease which we designate as "scrofula" is so characteristic, we know nothing at all as to its real nature. Many physicians still hold that the malady is founded on a "dyscrasia," although no abnormality has as yet been discovered on examination of the blood. But this opinion is based merely on the simultaneous or successive implication of a number of organs, which justifies us in concluding that in these cases we have not to do merely with a local affection, but with a cause of disease acting injuriously on the nutrition of the various organs. Whether this "cause of disease" is to be found in an abnormal condition of the blood or tissue-elements, or of both of these simultaneously, we do not know. I therefore think it advisable in the meantime to look upon the matter simply from the clinical point of view, as being the only one which is of real importance for the practitioner. Regarded in this way, scrofula seems to me to mean nothing more than the simultaneous or successive onset of various forms of chronic inflammation in a number of different tissues, with a very marked tendency to enlargement of the neighbouring lymphatic glands, or even of those at a distance, which is apt to end in caseous degeneration of the glands, and the formation of abscesses round about them.

[1] Two analogous cases have been described since then by Strōm and Arctander (*Jahrb. f. Kinderheilk.*, xxvii., S. 180). The first of these followed scarlet fever. No post-mortems are given. According to Hervé (*Revue mens.*, &c., Avril, 1888, p. 170) three quite similar cases were formerly mentioned by Jelliot in the *Union méd. du Nord-Est.*

The modes of termination of scrofulous inflammation are, generally speaking, just the same as those of any other inflammation. Here, as elsewhere, we find suppuration, ulceration, induration, &c., taking place; and it is only in a few particulars —such as consistence of the pus, the form of the ulcers and their scars—that certain differences are noticeable in scrofulous inflammation. These, however, are by no means so characteristic as to justify us in claiming for them a really pathognomonic significance. We are therefore all the more inclined to ascribe such significance to a morbid process, which is often found in hypertrophied lymphatic glands in scrofulous subjects, and which is called "caseous degeneration." Since, therefore, we know (vol. i., p. 433) that this substance, the product of the necro-biotic destruction of the tissue, occurs more frequently in the various organs of tubercular individuals, and, according to the most recent researches, very often though not invariably contains tubercular bacilli in varying numbers—it is conceivable that the old dispute with regard to the connection between scrofula and tuberculosis is by no means as yet at an end, but has rather received new life through the discovery of the bacilli. Many people are inclined to regard the two diseases as entirely identical, and as being both due to the invasion of the bacilli. But for my own part I do not consider that this view is at all borne out by the clinical facts. If any one free from prepossession will observe a large number of sick children, he will soon be convinced that the morbid appearances of scrofula are extraordinarily different from those of tuberculosis; although it is of course a fact that a certain proportion of scrofulous patients finally die of caseous pneumonia or of general miliary tuberculosis, and especially of tubercular meningitis. This undoubted tendency of scrofulous subjects to tubercular affections does not, however, by any means prove that the two processes are identical. In my opinion it is rather due to the fact that any tubercular bacilli that have somehow got into the body find a particularly favourable nidus in the products of the scrofulous inflammation, which have such a tendency to become decomposed—especially in the glands and bones. In this material they continue to develope, and a more or less general infection of the body with tuberculosis may take place subsequently, starting from these centres.

The clinical picture of scrofula is tolerably characteristic taken as a whole. But its individual features present manifold differences, which vary according to the individual patient, the number and nature of the tissues and organs affected, and the patient's circumstances.

Although cases do occur in which individuals have distinct signs of scrofula, and are yet to all appearance perfectly healthy and well-nourished, yet these are exceptions, and in such patients the disease is nearly always just beginning, or else extremely slight in degree. Sooner or later flabbiness of the skin and muscles appears and pallor of the general surface; at the same time the fat does not necessarily disappear from the connective-tissue, and it may even be present in larger quantity than usual. The pallor of the skin, especially on the face, which indicates deficiency of red blood-corpuscles, cannot be regarded as at all characteristic of these cases; for in a certain number of them the cheeks may even be quite rosy. This led the old physicians to distinguish two forms of the habitus scrofulosus, the "erethistic" and the "torpid." The first of these they described as found in persons with dark hair and eyes, with a fine skin and a fresh complexion, and altogether pleasant appearance, indicating intelligence and vivacity. Patients of the "torpid" habit were said to be distinguished by blond hair, pale blue eyes, thick nose and upper lip, pale complexion and bloated features, with a stupid expression. Now there was undeniably much that was true in this distinction, although there are numerous gradations between the one form and the other. At any rate the so-called "torpid" habit is very much the more common of the two, and is seen most typically in those cases in which the upper lip is reddened and excoriated by acrid nasal discharge, and often much thickened by hypertrophy of its glands and inflammatory infiltration of its connective-tissue, so that it projects like a snout over the lower lip, and in which the inflamed eyelids are spasmodically closed when the light falls on them.

In many cases enlargement of the lymphatic glands in the neck, groins, or axillæ are the first signs of scrofula. Especially one feels or sees under the jaw at the side of the neck, at its uppermost part, groups of roundish glands which move freely under the skin; these vary in size from that of a pea to that of a hazel-nut, and are sometimes collected into large bunches the

size of a hen's egg or larger. They are painless, or else more or less tender, especially on pressure from without. We must bear in mind, however, that the enlargement of the cervical, occipital, and auricular glands often occurs quite independently of scrofula, *e.g.*, as the result of the irritation of teething, from already existing eczematous or impetiginous eruptions on the face, ears, or hairy scalp, and even from apparently slight injuries. Thus, for example, I saw enlargement of the glands of the neck occur at once in a little girl after her ears had been bored. I do not think that we are justified in at once assuming a scrofulous constitution in such cases, unless these symptoms are accompanied by others of a more conclusive character. Leucæmic and pseudo-leucæmic[1] hypertrophy of the lymphatic glands also occurs in children, and one must exclude these as well as all sympathetic enlargements before one is justified in assuming that the glandular enlargements are really of a scrofulous character. The latter may persist for many months and even for years, generally combined with other scrofulous symptoms, and they may also gradually disappear. In most cases, however, they give rise to repeated inflammations of the surrounding connective-tissue, especially in the neck, with extensive hard and painful infiltrations, which finally become reddened and fluctuating, and either burst spontaneously or are opened. This tendency of the hypertrophied glandular elements to "breaking down," necro-biosis (caseation) and suppuration constitutes, as Virchow has justly pointed out, an essential feature in the clinical picture of scrofula, and distinguishes it from the leucæmic and pseudo-leucæmic lymphomata, which generally persist unchanged till the end of life. Rapid recovery seldom follows the emptying of the abscess; much more frequently the opening closes on the surface, and a fresh accumulation of pus takes place beneath it, so that repeated incisions are required. After the abscesses have burst or been opened, there often form out of them more or less extensive ulcers with red infiltrated bridges of skin stretching over them and with undermined edges; and on the floor of these ulcers the affected glands may be exposed. Such ulcers are very difficult to heal, and often do so only after the affected glands

[1] Both kinds of glandular enlargement are entirely similar to those found in adults. I have published (*Charité-Annalen*, Bd. vi., *Jahrg.*, 1880) a typical case of pseudo-leucæmia with an enormous number of lymphomata, which was observed until death in one of my wards.

have been excised, and they always leave behind them cord-like cicatrices, very similar to those left by burns.

Next to the lymphatic glands, we often find the outer skin and the subcutaneous tissue affected, the latter in the form of circumscribed infiltrations on different parts of the body; these may be as large as a walnut, they almost always end in suppuration sooner or later, and their further course is similar to that of the glandular abscesses which I have described. These abscesses generally take an unusually long time to develope and they are often unaccompanied by any important signs of inflammation (so-called cold abscesses). Among others I have observed a child of 10 months who was suffering at the same time from osteomyelitis of the fourth left metacarpal bone, and who had, along with many other abscesses, a very large one over the left patella which had lasted 3 months without the skin over it becoming discoloured. In order to guard against mistaking the case for one of hydrops bursæ mucosæ, I made an exploratory puncture which proved the presence of pus. The affection of the outer skin takes the form of a larger number of chronic rashes, the symptoms of which are just the same as those of the non-scrofulous forms of eruption. We most commonly observe eczema impetiginosum on the face, less commonly on other parts of the body; and ecthyma on the back, nates, and thighs. The latter often leaves behind it more or less deep ulcers which have sharp margins, and heal with difficulty. We also often find eczema of the external ear and of the hairy scalp, combined with patches of erythema and red papules on the cheeks. Least common are the various forms of lupus, which is generally situated on the nose and less commonly on the cheeks and lips. The affected parts are hard, owing to their being infiltrated with exudation, and their surface is scattered over with red or livid nodules of varying size, which are either continually desquamating (lupus exfoliativus), or they break down into deep offensive ulcers, which go on increasing in depth and may even affect the cartilages and bones; and in this way the disease generally lasts for years (alternately improving and getting worse) and even in the most favourable cases leaves behind traces of large ulcers with deep radiating scars. Especially protracted is lupus serpiginosus, in which form some parts of the ulcers are always cicatrising while the borders are becoming

infiltrated with new nodules and breaking down into fresh ulceration. I have met with this form several times on the backs of the hands and on the fingers in scrofulous children. In these we observed the steady progress of the ulceration in one direction while the parts which were first affected were already forming radiating scars.

The thickened upper lip, which often looks red and raw owing to the acrid nasal secretion, and also the yellow or greenish-brown eczema-crusts adhering to the skin of the face (along with which are found red papules, vesicles and pustules)—give to the face a very characteristic expression, and this is often exaggerated by the inflammatory swelling and redness of the eyelids which are tightly pressed together whenever the light falls upon them. In many cases the eczematous inflammation spreads inwards from the external ear and gives rise to a sero-purulent discharge from the external auditory meatus.

The mucous membranes which are most frequently affected by scrofula in the form of chronic inflammation are that of the nose and the conjunctiva of the eye. Chronic rhinitis with redness and excoriation of the nostrils, sero-purulent discharge and often also swelling and thickening of the nose itself (the apertures of which are blocked by yellowish-green crusts of dried pus, so that breathing is rendered difficult) are among the commonest symptoms. Quite as common is conjunctivitis with formation of phlyctænæ on the margin of the cornea, much watering of the eyes and extreme photophobia, which often prevents the eyes being opened except in the twilight, and compels the children during the day to keep their face buried in the pillow or covered with their hands. The Meibomian glands are often affected by the inflammation, and then we have the symptoms of the well-known condition of blephar-adenitis, in which the eyelids are reddened, swollen, and excoriated, and become glued together during the night owing to secretion from the glands. We frequently observe tarsal cysts and dropping out of the eyelashes with hard thickening of the margins of the lids. Another extremely common symptom is offensive sero-purulent otorrhœa which is generally bilateral, and, when there is no eczema of the external ear, may be due to chronic inflammation of the auditory meatus, especially of the covering of the tympanum. In other cases it may be caused by caries of

the petrous bone or by the rupture of glandular abscesses, in front of or behind the ear, into the auditory meatus. In all cases where these inflammations have become chronic and last for years, the process may finally spread to the tissues immediately underlying the affected mucous membrane. Thus, we not unfrequently find rhinitis spreading from the mucous membrane to the perichondrium and nasal cartilages, also to the periosteum, the turbinated and nasal bones. It may also give rise to redness, swelling and pain of the whole nose and the discharge of offensive sanious pus containing fragments of dead bone; likewise to ulceration and destruction of the cartilaginous septum and alæ nasi, so that even after complete recovery more or less serious deformity of the nose remains. Indeed in many cases we observe on the one hand extension of the disease to the cribriform plate of the ethmoid and through this to the cerebral membranes, causing fatal meningitis; and on the other hand gradual carious perforation of the hard palate from the nasal cavity, giving rise to a more or less wide communication between the latter and the mouth. Similarly, the inflammation of the auditory meatus may slowly spread to the tympanum; and, when this has become perforated (as we may see by means of an aural speculum), to the mucous membrane of the tympanic cavity and its bony walls; finally, the petrous bone and the spongy substance of the mastoid process may become affected. The results of the disease extending in this way are as follows: there may be offensive blood-stained discharge from the ears containing fragments of necrosed bone, or as I have repeatedly observed, accompanied by the separation of some of the auditory ossicles from their connection, especially the malleus; less commonly we may have profuse hæmorrhage from the ear, and in one case this was accompanied by vomiting of black material (due to blood passing through the Eustachian tube into the pharynx and being swallowed); there may also result deafness, swelling and tenderness of the mastoid process and whole temporal bone; finally, redness of the superjacent skin, dislocation of the auricle (the concha of which becomes directed forwards) and the formation of sinuses leading into the interior of the carious mastoid bone. Indeed, the process may spread still further, on the one hand destroying the labyrinth and Fallopian canal and causing paralysis of the facial

nerve on the affected side (vol. i., p. 243), and on the other setting up inflammation and thrombosis of the petrosal sinus where it lies on the carious petrous bone, and ending in death with symptoms of meningitis or pyæmia. The eye is also often endangered by the scrofulous inflammation spreading to the cornea (keratitis), sometimes with unexpected rapidity. When this takes place there is violent photophobia and lachrimation, and we find a varying extent of the cornea becoming more or less opaque and of a greyish-green tinge, and the opaque area is often connected with a corresponding leash of enlarged conjunctival vessels. Ulceration of the cornea is very apt to occur under unfavourable circumstances, and this may lead to perforation, staphyloma, and atrophy of the eye. Even in the favourable cases in which perforation does not occur, more or less extensive opacities are generally left for years, or permanently, which often interfere with vision.

Whether other mucous membranes in addition to those I have mentioned are apt to be affected by scrofula is, I think, doubtful. According to my experience, at least, scrofulous children are not affected oftener than others by attacks of bronchial catarrh, broncho-pneumonia and diarrhœa. On the other hand it cannot be denied that these affections are particularly obstinate in scrofulous children, having a tendency to become chronic, that their chief danger consists in the fact that they are more likely in these patients than in others to be followed by secondary enlargement of the bronchial or mesenteric glands, and that these very readily caseate, and by taking up and cultivating the tubercular bacilli become the starting-point of miliary tuberculosis. Thus it happens that we often meet with enlargement and caseous degeneration of these glands in the bodies of scrofulous patients, along with caseous deposits in the lungs and tubercle in many situations. I cannot admit that catarrh of the vaginal mucous membrane (fluor albus) is also a sign of scrofula, as is often asserted. At any rate, among the many children whom I have treated for vulvitis and vaginal catarrh there were comparatively few who were scrofulous, and even in these the disease of the genitals was generally to be attributed to other causes (assault, masturbation, chill). On the other hand, it seems to me that scrofulous children have an undoubted liability to attacks of sore-throat, probably

to the fact that they suffer very frequently from hypertrophy of the tonsils, of which I have already spoken (p. 18). The nasal tone of the voice, the difficulty of hearing, and the habit (which these patients have) of keeping their mouths open, give them a somewhat characteristic aspect which will at once lead any experienced practitioner to examine the tonsils.

Besides the lymphatic glands, skin and mucous membranes, we also find the osseous system very often affected by scrofulous inflammation, the bones earliest affected being the phalanges of the fingers and toes and the metacarpal and metatarsal bones (pædarthrocace). On one or other phalanx, often on several at once, we find an olive-shaped swelling which grows slowly, is hard and of normal colour, and at first is not painful. This may remain in the same condition for many months until at last the skin which covers it becomes adherent to the bones, reddened and perforated by one or more sinuses, from which there trickles a little thin purulent secretion. Similarly, the metacarpal or metatarsal bones may be affected either alone or along with the phalanges. The inflammation commences in the interior of the bone and in its medulla (osteomyelitis) which finally breaks down into pus along with the adjacent layers of bone, while new layers of bone are being deposited on the outside by the periosteum (which is implicated in the inflammatory process)—only to be again destroyed by the process of softening and re-absorption which spreads to it from within. The same appearances may be found in connection with the long bones of the upper and lower extremities, while in other cases, the vertebræ (spondylitis) or the joints (especially those of the elbow, hip, and knee) are affected. These forms of inflammation, into the symptoms of which I cannot enter here in detail, are to be regarded as very serious consequences of scrofula, for various reasons. On the one hand, owing to their passing on to suppuration and causing hectic fever and amyloid degeneration of many organs, they may end fatally after lasting for years. On the other hand, even in favourable cases, anchylosis and deformity of the joints and permanent interference with movement may be left behind. Further, the inflammation of the vertebræ either spreads to the spinal meninges or to the spinal cord itself, causing paralysis of the limbs with its results (bedsores, &c.); or the affected vertebra may become suddenly displaced and

endanger life by compressing the spinal cord; or, finally, the case may end fatally from constant suppuration and the formation of so-called congestive abscesses and from hectic. I may be excused from giving a full description of this disease of the vertebræ, as it often occurs in children and even in adults quite independently of scrofula, and as it is described in all works on surgery. I shall only remark that both the vertebral disease and the above-mentioned inflammation of the joints is generally attributed by the children's parents to some traumatic cause—a fall, a blow, or something of that sort. Although I have no desire to deny the injurious effect of these injuries, still I believe that the bone-disease is often present in a latent form, and it has its course accelerated only by the injury; in other cases, however, every traumatic influence can be excluded with certainty. Of all these affections of the osseous system, pædarthrocace and scrofulous caries of the long bones have certainly the most favourable prognosis, as in them (although only after years) we may obtain complete elimination of the necrosed fragments of bone, and thorough cicatrisation with funnel-like indrawing of the superjacent skin. The analogous affections of the sternum and ribs—which are less common—may endanger life owing to the disease spreading to the mediastinum and pleura (vol. i., p. 425). Finally, I may also mention that the cranial bones, especially the temporal, may sometimes be affected by chronic inflammation; indeed the temporal bone may be affected quite apart from the otitis externa already described (p. 386), but it is equally incurable.

There can be no doubt that these forms of inflammation of bone which I have mentioned also occur in children who are free from any scrofulous tendency, or who show at least no other signs of this disease. This is true not only of osteomyelitis—which, as I have already mentioned (vol. i., p. 100), may also be due to syphilis—but also of other forms of multiple osteitis. Among other instances I may mention that of a child scarcely a year old who was perfectly healthy, and whose parents and brothers and sisters were also perfectly free from all signs of scrofula or syphilis. A number of swellings appeared in rapid succession on the epiphyses and bones of the left forearm, on the metacarpal bone of the left thumb, on the upper epiphysis of the right radius and on the left half of the frontal

bone, which soon presented fluctuation and had to be incised; all this time the child's general health was perfectly good. I scarcely think that we are justified in describing off-hand as "scrofulous" those cases in which only the osseous system, and neither the glands nor any other tissues are affected. It would rather seem as if the growth of the bones, and especially that of the epiphyses, which is so active in childhood may, under certain circumstances with which we are unacquainted, give rise to excessive hyperæmia ending in inflammation and suppuration.

Different cases of scrofula vary extremely in regard to the severity of the process and the number of organs affected by it. Sometimes the whole malady consists of swelling of the cervical glands, blephar-adenitis and eruptions on the head; sometimes of otorrhea, rhinitis, pædarthrocace or other combinations. Many years may pass before these affections finally recover under suitable care and treatment, as the patient grows older. But in many other cases, even in spite of the utmost care, the children's lives are endangered by the larger long bones, spine and joints becoming affected; or death may result from the development of acute tuberculosis, pulmonary phthisis, tubercular peritonitis or meningitis. Our prognosis, therefore, must vary accordingly. As long as the disease only causes chronic inflammation of soft parts (glands, mucous membranes, skin), life is not in any way endangered, although it may be many years before the patient recovers completely. But the prognosis becomes much more serious when the bones and joints are affected, and it is worst of all when symptoms of tuberculosis or of amyloid degeneration of the viscera have set in.

Of the causation of scrofula we know almost as little as we do of its real nature. That the disease may be inherited is a fact which no one can doubt who takes the trouble to go carefully into the family history of these cases. We are, indeed, unable, in a large proportion of the cases, to find this factor, and we then have recourse to all sorts of hypotheses—as for instance, that the parents are too old, or too young, or unequal in age, or that they are the subjects of some dyscrasia, as hydrargyria, syphilis, &c. That such parents may beget delicate children, cannot indeed be denied; and in so far as delicate children are certainly more apt to become scrofulous than strong ones—that is to say, are more readily affected by chronic inflammation of

various tissues—this hypothesis has certain valid grounds. The same may be said of want of the necessaries of life while the child is growing. Bad nourishment, and the unwholesome and stuffy air of damp and dismal rooms to which little light can penetrate—certainly do arrest the normal development of the organism, and bring about a condition of poverty of blood, and accordingly deficient power of resistance to morbid influences, which may finally result in the development of scrofula. The greater prevalence of this disease in large towns and among the poorer classes is sufficiently explained by these facts.

When a child has any tendency towards scrofulous disease, even although that tendency has hitherto hardly been indicated at all or only by the most trifling signs, it may suddenly come to the front under the influence of an acute attack of illness. The chief among these illnesses, as experience shows, are whooping cough, measles, smallpox, and even vaccinia; after an attack of these diseases one very often finds swollen glands, eruptions and inflammation of the mucous membranes, from which the patients have never suffered before. This is an indisputable fact known to every practitioner, but as yet unexplained. Many cases of so-called vaccinal syphilis are undoubtedly nothing but cases of such scrofulous affections after vaccination which have been mistaken for real syphilis.

The main condition of successful treatment is, that we should obtain for the patient the most favourable surroundings we can. The effect of pure air in healthy, light and well-ventilated rooms, the avoidance of chills and damp, a nourishing diet, the fresh air of country, hill, or seaside, gymnastic exercises, and careful attention to the skin (bathing)—are of far more value in this disease than all the vaunted anti-scrofulous remedies, and are in themselves sufficient to cure mild cases. These conditions, however, are only available in the minority of cases. In practice among the poor and in hospital we have to deal with disadvantageous circumstances which cannot be obviated, and therefore we need not be surprised that our treatment is so unsuccessful. On this account all attempts to obtain the benefit of these "air-baths" for the children of the poor and the scrofulous, are to be gladly welcomed—especially the establishment of children's homes at the seaside, which has already been done in many other countries. In Germany a beginning has now been made, and a

number of institutions of this sort have now been started on the coasts of the North Sea and Baltic, and they are worthy of our most active support.

Among the remedies which we ordinarily call "anti-scrofulous" the most important, according to my experience, is iodine, which I prefer to give along with iron as syrupus ferri iodidi (gtt. x.—xv. thrice a day) or combined with iodide of potash (Form. 46) as Lugol has recommended. The remedy must be persevered with for months, unless it causes digestive disturbances, but it is contra-indicated if there is tuberculosis of the lungs or even any suspicion of such a condition. I have never yet met with any symptoms of "iodism" such as have been often described. The worst that occurred was only a severe coryza or erythema on the face and on other parts of the skin. Salt baths have been much recommended, and they deserve their reputation on account of their powerful influence on tissue-change; but one must not expect any rapid action from them, and we must warn the parents that the courses of bathing can only be successful when they are long continued and frequently repeated. Further, the baths must not be persisted in too continuously, but must be stopped every few days; for their effects are too strong for many children. The extremely small quantity of iodine or bromine contained in certain salt springs (such as those of Kreuznach, Hall, and others) hardly needs to be taken into account; while the amount of chloride of sodium and of chloride of calcium (which constitutes the largest proportion of the mother salt) is of great importance with regard to the effect. In addition to the two salt baths just mentioned, we may recommend Œynhausen and Nauheim (which contain a large quantity of carbonic acid), also Kösen, Sulza, Wittekind, Frankenhausen, Harzburg, Salzungen, Arnstadt, Reichenhall, Ischl, Kissingen, Pyrmont (combined with chalybeate springs) Rheinfelden, Bex, Königsdorf-Jastrzemb, Soden (near Aschaffenburg), Dürkheim, Rothenfelde, and others. Berlin also has now entered the list of salt-bathing resorts, for a spring has been discovered which contains about 26½ per mille of salt. Unique of its kind is Colberg, for it affords not only salt baths, but also sea-baths and sea air; and the latter, as I have already said is to be regarded as an important factor in the treatment of scrofulous patients. Quite recently Greifswald has sought to claim these advantages with Colberg. Whichever of these resorts one

chooses, the most important factor in the treatment will in any case be the fresh air; and on this account the action of artificial salt baths used at home will always remain inferior to that of the natural baths. It is only where, owing to the patient's circumstances, the latter cannot be had that one must be contented with artificial salt baths. For such baths we use 1—5 lbs. of salt according to the patient's age (sea-salt, Strassfurt rock-salt, or some other "bath-salt"), and we also may add 1—2 lbs. of Kreuznach salt, or that from one of the other bathing-places.

We cannot suppose that the traces of iodine which exist in codliver oil really exert any specific action. This much extolled remedy certainly acts more as a nutrient in the same way as ordinary fatty foods; and, as Buchheim has demonstrated, it has a very material advantage over other forms of fat in that it contains a larger proportion of free fatty acids. It is mainly the darker sorts of codliver oil which have this advantage (about 5 per cent.), while the clearer varieties (which have been greatly recommended of late) contain much less. These free fatty acids become at once saponified when they reach the intestine, emulsify the remaining fat and favour its absorption.[1] In regard to direct action, codliver oil is in my experience considerably behind iodine, and I must further warn you not to give too large doses of it, as they are apt to interfere with the digestion. Two or three dessertspoonfuls in the day are quite sufficient. The inunction of codliver oil into the skin which is a favourite method with many physicians, I regard as objectionable owing to the unpleasant smell and the uncleanness of the proceeding. Nor have I been able to observe any favourable action from other vaunted remedies such as Plummer's powder, æthiopis, walnut-leaves, acorn-coffee, &c. One only wastes time in using such things. For scrofula in general, therefore, the only methods of treatment we can promise ourselves real success from, are improvement of the circumstances, the use of iodine or iodide of iron, and the baths already spoken of.

In addition to this course of treatment, which is to be adhered to as a general thing, we may find a number of indications for treatment from the various local affections—the inflammations of

[1] *Cf.* von Mering (*Therapeut. Monatshefte*, Feb. 1888) who recommends as a substitute for dark codliver oil a mixture of olive oil with fatty acids (5—6 per cent.) which he calls "Lipanin."

the eye and the affections of the bones, joints, skin, and mucous membranes. I need not enter into these more fully, as they mostly fall within the domain of ophthalmic or of general surgery. I ought to mention, however, that in pædarthrocace when the diseased bone is scraped out with a sharp spoon the course of the disease is shortened, and this is therefore to be preferred to purely expectant treatment under which the case may be protracted during many years. Still, I do not expect a great deal from this proceeding; at any rate some of my hospital cases resisted for years the repeated application of the sharp spoon. I have not sufficient experience of the treatment by inunction of potash soap which has been recommended by many practitioners, and which is said to have a remarkably rapid effect in causing diminution of the scrofulous swellings, especially those of the glands.[1] At any rate, if painting with tincture of iodine or iodoform collodion produces no effect, it is always worth while trying the treatment with soft soap (1—2 spoonfuls to be rubbed into different parts of the body daily) before one proceeds to any radical measures, that is, to excision of the indurated glands.

## V.—*Rickets.*

Rickets has been called "the English disease," owing to Glisson, the first writer of importance on the subject, having been an Englishman. The term "double-jointed" which is still popularly applied to patients suffering from this disease, is derived from the characteristic enlargement of the epiphyses. Rickets most frequently comes under observation in children during the second and third years of life; I say expressly "comes under observation," because it is about this period that the disease generally attains its greatest development and many parents, especially in the lower classes, only apply for medical advice when the symptoms have become very noticeable. Still it is a fact which I have myself very often verified, that the first symptoms of rickets may set in much earlier—within the first six months of life—especially in connection with the cranial bones and ribs. Many parents only have their attention drawn to the presence of the disease by the fact that the children, after having begun to walk, cease to be able to do so or that they

[1] Kappesser, Klingelhoeffer, Kormann, and others.

have never begun to walk or to stand alone on their feet. Much less commonly, the children are able to move about without support or with the mother's hand, but present in so doing a waddling gait like that of a duck. When we examine the child we are at once struck by the very large size of the head in proportion to the rest of the body, the broad forehead, which projects markedly when seen in profile (frons quadrata) and the very prominent parietal bones. Not infrequently the sagittal suture (and sometimes also the coronal one) runs, as it were in a grove which is bounded on each side by the thickened parietal bones. The anterior fontanelle, which in the normal condition is usually completely ossified by the 15th—18th months of life, continues, with few exceptions, more or less wide open in rickety children until well into the second or third year of life. The margins of the cranial bones yield readily to pressure, the sutures (especially the sagittal, often also the lambdoidal and coronal, and least commonly a portion of the frontal) are still widely open and have soft yielding borders. In many cases we also find the two posterior fontanelles still membranous. These symptoms, especially the size of the cranium, are often regarded by the laity as signs of "water in the head," but such a supposition is contradicted at once by the patients' power to hold the head quite erect and by the fact that the mental functions are perfectly unaffected and are often, indeed, unusually active (vol. i., p. 298). I have observed in a small number of cases that these children were unusually late in beginning to learn to talk. The development of the teeth is almost always delayed; such children often do not get their first teeth until the second year and the individual groups appear irregularly and at unusually long intervals. In many children the teeth become yellow, striated and blackish soon after their appearance, owing to the enamel covering them being defective, and they finally crumble away down to the margin of the jaw. I have sometimes found only the teeth of the upper jaw destroyed in this manner (and that even when they had just come through), while the lower ones remained unaffected; in other cases, all the teeth were just as good and as well preserved as in the healthiest children. The changes in the form of the jaw, to which Fleischmann has drawn attention in his excellent work,[1] are of great interest.

[1] *Klinik der Pädiatrik*, ii. Bd.: Wien, 1877, S. 168.

Even before and during the first dentition the lower jaw assumes a polygonal form instead of its usual arched curve, "owing to an approximation or contraction having taken place on both sides of the jaw behind the region of the canine teeth, while—owing to deficient deposit of calcareous salts on the anterior lamella of the middle portion of the jaw—the growth of that portion has been arrested and consequently the arch has not developed." The incisors are therefore situated alongside of one another in a straight line and the lateral portions of the jaw behind the canines do not retreat in a curve but run straight backwards diverging somewhat. At the same time the lower margin of the jaw is displaced a little outwards, and the alveolar margin somewhat inwards—so that the molars and sometimes also the incisors are not situated vertically but converge inwards. The changes in the form of the upper jaw are less striking; among these the lengthening of its long axis deserves particular mention, as well as certain asymmetries of the two halves of the jaw and abnormal growths of bone in certain situations on its inner or outer surface.[1] Further, on examining the thorax we find a very characteristic condition. The clavicles are often much curved or in some places bent at a sharp angle. The ribs present, at the point where their bony portion is united to the cartilage, a more or less prominent nodular swelling; and this on closer examination is generally found to consist of two swellings (one bony and the other cartilaginous) separated by a groove. In this manner there comes to be formed on each side of the thorax a row of nodular elevations running from above and within downwards and outwards, which is generally described as the "rickety rosary," and which is especially well seen in emaciated children. Accompanying this, we often find the sides of the thorax flattened, and in extreme cases of the disease they may even become concave. The portions of the 2nd—8th ribs lying between the angles and the epiphyseal swellings just described are markedly bent inwards towards the thoracic cavity and the lowest ribs bordering on these are bent outwards—an appearance which, taken along with the unusually projecting sternum makes the flattening and the concavity of the lateral walls all the more striking (pectus carinatum, pigeon-

[1] **Baginsky**, *Prakt. Beitr. zur Kinderheilkunde*, ii., Th., Rachitis.—**Herz**, *Arch. f. Kinderheilk.*, vii., S. 36.

breast). We also sometimes find asymmetry of the two sides of the thorax, indentations and incomplete rotation of the ribs on their axes, so that their outer surfaces are turned more inwards. The parents tell us that the child is short of breath, and this we always find to be correct in extreme cases; the respiration is more rapid and superficial than in healthy children, and we notice that the flattened, or even concave, sides of the thorax are still further drawn inwards with each inspiration, while at the same time the inspiratory action of the diaphragm becomes unusually prominent owing to the indrawing of the epigastrium. Attacks of bronchial catarrh are of frequent occurrence, during which the child's breathing is rather noisy and we hear more or less wide-spread crepitations, especially over the back. The strikingly globular abdomen, distended by the coils of intestine which are full of gas, is in strong contrast to the narrow and deformed thorax; and its upper part is more than usually filled up by the liver and spleen which are pressed downwards owing to the contraction of the thorax. The functions of the digestive apparatus are often quite undisturbed; only in a certain proportion of the cases is there any digestive disturbance, the most common being a tendency to attacks of diarrhœa. Among the most important appearances, however, are those which we find in connection with the bones of the upper and lower extremities. The epiphyses of the radius and ulna—especially of the former—are more or less noticeably thickened and broadened, so that in extreme cases (especially when the patients are thin) the hand seems as if it was marked off from the arm by a furrow; while the diaphyses of the bones of the fore-arm often present a convex curving towards the extensor aspect of the arm or even a very prominent bend. The curving of the tibia generally appears even more distinct, and its epiphysis at the ankle is also thickened like that of the fibula. The curvature is concave on the inner side and usually it is most distinctly marked at the lower third of the bone; occasionally it is so severe that the case looks like one of talipes varus. We often also observe curving and acute bending of the bones of the upper arm and thigh, and marked enlargement of the epiphyses in the neighbourhood of the knee- and elbow-joints. In a number of cases, even the shoulder-blades and ilia are found on careful examination to present more or less considerable th

their borders. Finally, some curvature of the vertebral column very often appears—either scoliosis or kyphosis; the latter most frequently affects the dorsal region and is combined with compensating lordosis of the lumbar spine. This kyphotic curvature is mainly distinguished from that due to spinal caries by the curve which it describes being greater, and by the fact that it is diminished or disappears entirely when the child lies on the abdomen, especially if moderate extension is applied to the spinal column at the same time.

All these changes are subject to wide variation in their degree and extent. We also very often find *some* of them entirely *absent*, so that the clinical picture of rickets is incomplete. The epiphyseal swellings on the ribs I have found to be the most constant symptom—indeed, they are scarcely ever absent and they are always among the first to appear and may indeed be noticeable by the third or fifth month of life. I have frequently failed to find signs of cranial rickets and in those cases the head was not increased in size, the fontanelles and sutures were closed at the proper time, and even the dentition sometimes proceeded in the normal way. Among others I have observed a girl of 9 months who, in spite of having rickets, had cut her first tooth during the sixth month and had already six teeth when I examined her. I found the same state of things in another child only 10 months old; which shows that dentition may in a few cases proceed with abnormal rapidity in spite of the presence of rickets. Generally, however, in such cases dentition has taken place *before* the rachitis set in, and *after* the disease appears, there is generally an abnormally long pause. Sometimes the most marked feature is the epiphyseal enlargement and the bending of the bones of the extremities, while the cranium and thorax are but slightly affected. Still, I have seen a few rickety children standing and walking at the age of 15—16 months just as well as perfectly healthy children. You will find the characteristic deformity of the thorax oftenest, and most marked in very young children who have been badly nourished and have suffered from many attacks of bronchial catarrh, whereas in those who are well-nourished and robust you generally find very little deformity or none at all. I shall presently return to the cause of all these differences.

Apart from these local variations, the general clinical picture

presented by rickety children also varies very much. In one set of cases (not a very numerous one) in which the characteristic bone-symptoms were but slightly marked, I have found the general condition undisturbed and the appearance healthy; but in the great majority of cases a profound affection of the organism is indicated by the anæmic complexion, the emaciation, the shrivelled condition of the skin and muscles, and the palpable enlargement of the lymphatic glands in the throat, neck, groins, and axillæ. In a few cases I have observed purpuric spots on the pale, waxy skin, or a scorbutic condition of the gums. The liver sometimes extends further below the margin of the ribs than it does in healthy children of the same age; but I have not been able to make out palpable enlargement of the spleen as often as other writers have done. In the numerous post-mortem examinations in the hospital we have found the spleen hypertrophied only in comparatively few cases, and I should describe the occurrence of enlargement of the spleen along with rickets (especially when very great) as merely fortuitous. The secretion of urine, when superficially examined into, presents no differences from the normal condition; but chemical analysis yields very different results, to which I shall again return. The secretion of sweat is increased in most cases, and such profuse perspiration occurs, on the head especially, that the pillow is quite wet in the morning. In many children sudamina and eczema, with small vesicles, appear as a consequence of this perspiration. A few only of the patients are irritable and restless, and the loud way in which they scream when one presses on the affected bones or lifts them up by catching them under the axillæ, seems to indicate the presence of pain.

I have never yet seen any cases of rickets with an acute feverish course, such as have occasionally been described; and as my cases have numbered many thousands, I therefore infer that this form of the disease must be very rare, if indeed it ever occurs at all. Whenever there was really a rise of temperature, I have always been able to discover some complication, especially bronchial catarrh. The rachitis itself has a chronic course throughout, and I therefore agree with Friedleben and Fürst,[1] who deny that there is such a thing as acute rickets. I do not regard Fürst's hypothesis—that an acute initial stage

[1] *Jahrb. f. Kinderheilk.*, xviii., 1882, S. 192.

may occur—as proved. We must certainly guard against hastily mistaking for acute rickets the multiple osteitis which I have referred to above (p. 388), and which may set in, especially at the epiphyses, owing to an extremely rapid growth of the bone, and, as we have seen, may even end in suppuration. Many cases of this kind may, as Friedleben and Fürst have also pointed out, pass into rickets after the acute symptoms have subsided, but in other cases this does not occur.[1]

It is nearly always difficult, or indeed impossible, to determine the duration of rickets; for we are seldom so fortunate as to be able to follow the whole course of the disease from beginning to end. The statements of the parents, who generally do not bring their children to us until they are in an advanced stage of the disease, are usually so unreliable that we can never form a conclusion as to the exact time when the disease actually began. This, however, is certain—that even in the most favourable cases, many months, often even several years, pass before we are in a position to regard the morbid process as at an end. The first favourable sign is when the children try to stand on their feet and to move about of their own accord: that is to say, when the strength and energy of the osseous and muscular tissues, of the spinal column and lower limbs, have been recovered, or, it may be, acquired for the first time. We now observe a rapidly progressing ossification of the fontanelles and sutures which are still membranous, also a more rapid eruption of the teeth. At the same time the growth in length of the body, which has hitherto been retarded, is accelerated noticeably, the child's general appearance steadily improves, and the deformity of the vertebral column and thorax (if not very extreme) gradually disappears. At last the only traces of the disease left are the curvature and bending of the long bones, especially in the form of "bow-legs" or "knock-knee" (genu valgum) and thickenings of the epiphyses, which often remain noticeable for years, but gradually disappear as the bones grow in length and breadth. Often also the normal position of the permanent teeth is unfavourably influenced by the arrested growth of the jaw (p. 395), and when the second dentition occurs various abnormalities are found—the teeth may

[1] The discussion on "Acute Rickets," which took place at the International Congress at Copenhagen, also ended in no definite result (*Jahrb. f. Kinderheilk.* xxv. S. 118).

come through at an angle to one another, or overlap like tiles on a roof, or may appear in a double row, and so on. Only in extreme cases, can traces of the disease (even the thickening of the cranial bones) be recognised as late as the time of puberty or later; and it is at these later periods of life that one of the results of rickets—namely, the deformed pelvis, which in childhood is of no importance whatever—may constitute the greatest danger. As a rule the pelvic inlet and the whole pelvic cavity become narrowed antero-posteriorly, owing to the rickety affection of the bones, while the promontory with the upper half of the sacrum is pressed forwards (there being at the same time lordosis of the lumbar vertebræ) and is brought near to the symphysis pubis, which is situated higher than usual, owing to the mechanical pressure. A more exact description of this deformity and of the manner of its occurrence would be out of place here, since little importance attaches to it from a pædiatric point of view.[1] The interest of the pelvic deformity only consists in the serious interference with labour which it causes owing to the shortening of the conjugate diameter, and which in extreme cases—as we learn from obstetrics—may necessitate the most serious operations. Under these circumstances we often also find considerable deformities in other situations, especially of the vertebral column (kypho-scoliosis) and of the thorax, which make the patient dwarfed for life.

While, then, in the very great majority of cases, rickets runs its course to the complete recovery of the patient (passing through all the stages of that process which might be expected from the nature of the disease), still we find this favourable course delayed in a number of cases by complications, or even turned into the opposite direction. The tendency to attacks of catarrh of the air-passages, which I have already spoken of, is especially fatal to these children. On the one hand, this is due to the fact that these attacks keep constantly recurring, and so increase the deficiency of the hæmatopoiesis already existing, and the consequent debility. On the other hand because, owing to the narrowness of the thoracic cavity resulting from malformation, a diffuse catarrh or catarrhal pneumonia, which in healthy children would be readily recovered from, is here very apt to

[1] *Cf.* Ritter von Rittershain, *Die Pathologie und Therapie der Rachitis*: Berlin, 1863, S. 181.

prove fatal. The narrowing of the space for the lungs, the weakness of the inspiratory muscles, and the filling of the bronchi with mucus, often give rise to extensive atelectasis in these cases, and this hastens the fatal termination (vol. i., p. 360). A large number of rickety patients die in this way. Very often we also observe attacks of spasmus glottidis and eclampsia, to which, as you will recollect, these patients are much more liable than healthy children are (vol. i., p. 182). The worst complication however is tuberculosis, which carries off a great many rachitic children whose surroundings are unhealthy.

I shall take this opportunity of recurring to a few points which are of importance in estimating the results of physical examination, and the disregard of which may lead to errors in diagnosis. I have already (vol. i., p. 6) referred to the influence which muscular contraction may exert on the percussion-note of the underlying parts of the thorax. And when the children struggle much while being examined and keep wriggling about, we are especially apt to find some impairment over one side of the back, which disappears again as soon as the child lies quiet, and when the muscular tension is equal on both sides of the body. In rickety children, however, in whom there is frequently some curvature of the spine, such impairment of the percussion (especially in cases of very marked scoliosis) may exist permanently though only slight in degree; and in such cases it has no significance unless we discover also signs of consolidation of the lung on auscultation over the area in question. Similarly, when the rickety deformity of the thorax is extreme, we may find the area of cardiac dulness on the front of the left side more extensive than in the normal condition, and the impulse of the heart is then generally to be felt beyond its usual limits. But these phenomena must not be at once set down as indicating hypertrophy of the organ, but may arise simply from displacement of the heart and deficient expansion of the lungs. Rilliet and Barthez say that they have also found the breath-sounds approaching the bronchial in character, and they refer this to the moderate amount of compression which the lung suffers owing to the sides of the thorax being pressed inwards. I have myself been often struck by the "indeterminate" character of the breathing; but I am inclined to think that in their explanation of the fact these authors have not attached sufficient importance

to the bronchial catarrh and the patches of atelectasis which so often occur in rickets.

The anatomical changes characteristic of rickets affect the bones exclusively. The bones are generally of a dark-red colour, and this is especially noticeable on the flat bones of the cranium. Their sharp corners and angles are blunted and rounded off, the long bones become shorter and somewhat cylindrical, enlarged at their epiphyseal ends, and present various longitudinal curves and bends. Their consistence is much softer than usual, so that we can easily make incisions into and sections through the bone, and we can sometimes even bend the diaphyses as if they were made of wax. The periosteum is thickened and hyperæmic, it is difficult to strip from the bone, and, when this is done, fragments of bone often come away with it. The softening is seen most markedly in the cranial bones, which on section are found to be extremely hyperæmic and very considerably thickened, especially towards the front of the head (frontal bone and part of the parietal bones); this accounts for the remarkable prominence of the frontal and parietal eminences during life. Under the thickened periosteum we find thin layers, porous and spongy like pumice stone, which in the diaphyses of the long bones alternate with compacter layers towards the centre, so that in the interior, near the medullary cavity, the latter becomes steadily harder, and the spongy layers become more like normal bone.[1]

In order to understand these appearances one must recollect, to begin with, the conditions of growth of the normal bone,[2] which, as is well known, takes place in two directions—in length and in thickness; the former from the cartilaginous epiphyses, the latter from the periosteum.

*a.* Growth in length. The hyaline cartilage of the epiphyses of the long bones is divided into two zones—one 1—2 mm. in breadth and bluish in colour, the other $\frac{1}{3}$—$\frac{1}{2}$ mm. broad, and of a pale yellow colour—where it passes into the spongy substance of the new-formed bone, which is full of medullary spaces filled with very vascular marrow. In the first,

[1] Virchow's *Archiv*, Bd. v.

[2] *Cf.* Ritter, *loc. cit.*, S. 27, *et seq.*—Rehn, Gerhardt's *Handbuch der Kinderkrankheiten*, iii., S. 54, 1878.—Kassowitz, *Die normale Ossification und die Erkrankungen des Knochensystems bei Rachitis und hereditärer Syphilis*: Wien, 1879—1882.

the so-called proliferation layer, we find a great number of large cartilage cells arranged in longitudinal lines, while in the second layer, the calcification takes place in such a way that the calcareous fragments are deposited in the inter-cellular substance surrounding the cartilage cells, and render it opaque. We then find formation of medullary spaces and of true bone occurring in the same layer; but as to the manner in which this process takes place, observers hold diverse opinions. Now, in rickets we find on the one hand an abnormal proliferation of the above-mentioned layers of cartilage with marked broadening; on the other hand, the calcification of the second layer is quite irregular and deficient, and the formation of medullary spaces extends from this layer into the proliferating cartilage, so that instead of the normal straight line of demarcation we find an indented line, marking the junction of the cartilage and the spongy bone. The occurrence of these processes explains the thickening of the epiphyses and the interference with the longitudinal growth of the bone, which generally causes the children to be stunted in their growth, and is noticeable in many of them till late in life.

*b.* Growth in thickness. In the normal condition the periosteum is very thick and vascular, and is firmly connected with the bone by a reticular fibrous tissue containing nucleated cells, and the growth of the bone takes place by these cells turning into stellate bone-corpuscles, and their matrix becoming calcified. Between the lamellæ of this new bone there are left roundish or elongated spaces communicating with one another, and filled out with soft reddish marrow, in which finally new blood-vessels form out of the marrow cells, and anastomose with those of the periosteum and the interior of the bone. This process lasts as long as the long bones continue to grow in thickness, but at the same time a large cavity filled with marrow is being formed in the interior by the bone-tissue becoming liquified and re-absorbed. But in rickets the periosteum and its cellular elements are considerably increased, the new-formed spongy layers of bone and trabecular reticulum are imperfectly ossified, and there is either comparatively little deposition of calcareous matter or none at all. Immediately under the thickened periosteum we find a hyperæmic trabecular network with medullary spaces, then a layer of compact substance, then again a spongy

layer. At the same time the normal re-absorption from the medullary cavity continues, and consequently the cortical layer must get steadily thinner. Exactly similar processes take place on the flat bones of the cranium and the shoulder-blades and iliac bones. The first step in the process of recovery is arrest of the cartilage-proliferation, followed by rapid calcification and ossification of the newly-formed layers which become even denser than in the normal condition (the so-called eburnation or sclerosis). As a result of this process, I found in a boy of 7 years a lenticular periostosis the size of a florin in the position of the large fontanelle, resembling the eminences of the frontal bone, which were very prominent.

But the osseous system has been examined chemically[1] as well as physically. All the examinations which have been made agree in showing that there is a more or less considerable diminution of the lime-salts in rickety bones, with lessening of the specific gravity (principally in those parts of the skeleton which are situated above the diaphragm) with relative increase in the amount of water contained in the cartilaginous parts, and in the organic matrix of the bones. The general body-weight diminishes *pari passu* with this change, and it does not begin to increase again to any great extent until some time after the child has begun to recover.

These anatomical and chemical changes, then, help to account for some of the main symptoms of rickets.

1. The arrested growth in length of the body is partly explained by the interference with the normal bone-formation at the epiphyses, partly by the flexibility of the long bones of the lower extremities, which have to bear the weight of the body.

2. The curving, bending, and fractures of the bones are mostly due to pressure and other traumatic influences acting on bones deficient in lime-salts. The angular bending (infraction) always affects the dense inner layers of the long bones; while the outer layers, which have remained soft, only yield along with the thickened periosteum, so that the bend resembles the fracture of a willow twig or quill. Although in this way, even where there is complete fracture of the inner layers, it is very difficult to make out crepitation or displace-

[1] Friedleben, *Jahrb. f. Kinderheilk.*, iii., Wien, 1860.—Baginsky, *Prakt. Beitr. zur Kinderheilk.*, ii.: Tübingen, 1882, S. 80.

ment of the broken ends; still, I have occasionally been able to feel crepitation distinctly. The weight of the body causes curvature and bending of the lower limbs, the convexity being generally outwards (bow legs), and the compression of the ribs and clavicles, which is caused when one takes hold of the child by the thorax, may result in fracture of these bones. A fall, or even turning round in bed, is apt to cause such fractures, and therefore we often find more or less angular bendings of the clavicles, ribs, bones of the fore-arm, thighs, &c., which are generally surrounded by callus, and in process of recovery. These fractures may occur singly, or there may be many of them, in which case a lamentable state of crippling results. In one such case (boy of 1¾ years) we found many of the ribs fractured just at their junction with the cartilages, and these fractures had recovered by the deposit of a large amount of fibrous callus in such a way that the epiphyses were situated almost on the outer surface of the diaphyses. It is also asserted that even violent muscular contractions may cause bending and fracture of the bones. Amongst other cases, I remember that of a very rickety child of 7 months (April, 1878), who after very violent convulsions repeated during a whole day, presented fractures of both radii requiring the application of a plaster of Paris bandage. Still, one can never, in such cases, exclude other injuries (especially rough handling) with absolute certainty. According to Kassowitz[1], the callus which forms after fractures remains cartilaginous and movable for a long time, and only becomes ossified when the rickets is recovered from.

Fleischmann is probably right in also attributing the alteration in the form of the jaw to the contraction of the mylo-hyoid and masseter muscles acting on bones which are deficient in lime-salts. On the other hand the characteristic deformity of the thorax is not the result of any single cause—*e.g.*, the paralysis or atrophy of inspiratory muscles (serrati, intercostals, &c.) which are so often blamed—but is rather due, as Ritter[2] has rightly observed, to the "combined action of a whole group of factors," and among these I may specially mention external atmospheric pressure, the contractions of the diaphragm, and the softness of the bones of the thorax. Even

[1] *Jahrb. f. Kinderheilk.*, xxii., 1884, S. 79.
[2] *Loc. cit.*, S. 167.

in healthy children, we observe that on very powerful and rapidly succeeding contractions of the diaphragm (such as take place during sobbing) the most yielding parts of the thoracic wall—*i.e.*, the anterior ends of the ribs—are distinctly retracted inwards at the beginning of every inspiration. This symptom is caused by the great flexibility of those portions of the ribs in childhood along with the comparatively weak and incomplete inspiration; for (on account of the latter) the quantity of air which obtains entrance into the lungs is not sufficient to counterbalance the action of the atmospheric pressure, which, therefore, forces inwards the more flexible portions of the thorax. This symptom is also found along with hypertrophy of the tonsils in very young children (p. 19), and may in these cases produce a quite similar deformity of the thorax. In rickety patients, however, in whom the chest-walls are exceedingly soft, and the action of the inspiratory muscles is often even feebler than is usual in children, it is all the more prominent, and in course of time it must occasion a permanent falling-in of the sides of the thorax along with corresponding protrusion of the sternum, and of the cartilages attached to it. Also the zonular depression which traverses the anterior surface of the rachitic thorax about three finger-breadths below the nipples, and below which the ribs lying over the liver, stomach, and spleen are turned outwards and project so as to form a kind of rim—must be ascribed partly to the concentric traction which the diaphragm exerts in a downward direction owing to its insertion, and partly to the atmospheric pressure.[1]

I have already mentioned that the increase in the size of the head and especially the prominence of the forehead and the parietal tuberosities is caused by periostitic deposits, and consequently the condition is sometimes mistaken for hydrocephalus. Cases of "hypertrophy of the brain" have, indeed, been described by various writers as occurring in rickets, but I have never myself met with a really indubitable case of this kind, nor yet have I been able to convince myself that hydrocephalic complications are as frequent, as is stated (*e.g.*, by Ritter).[2]

[1] Ritter, *loc. cit.*, S. 170, and Rehn, *loc. cit.*, S. 66.

[2] The deformities of the joints in rickets have recently been thoroughly investigated, especially by Kassowitz (*Jahrb. f. Kinderheilk.*, xxii., xxiii., and xxiv.). As to the correctness of his views I shall not presume to offer an opinion. I shall only mention that he attributes the detarded power of walking, standing

occipital protuberances, and in the intervals between these the cranial covering was membranous and traversed by thin bands of bone. The ribs and the bones of the extremities presented traces of many cured fractures, and there were changes in the cranial bones closely resembling those characteristic of rickets. Now, though the question as to fœtal rickets cannot be considered quite settled, still we must admit that the disease often develops very early and then it certainly has a right to be called "congenital," because the osseous affection appears at a time when external influences can hardly be taken into account.[1] In these cases the characteristic epiphyseal enlargement on the ribs and the very deficient ossification of the cranial bones appear within the first few months of life, whereas when the disease developes at a later age (*e.g.*, not until the beginning of the second year) the head may be quite unaffected, and only the thorax, extremities, and spinal column present the appearance of rachitis. I have never yet seen the disease commence later than the beginning of the second year; for in nearly all the cases in which it comes under treatment at a later age, it has already been long in existence, although the parents have only begun to recognise it when they found that the children did not learn to stand and walk at the ordinary time. I, at least, have never met with a case of rachitis tarda (corresponding to the so-called syphilis tarda) such as Kassowitz describes.[2]

I shall take this opportunity of considering somewhat more closely certain changes of the cranial bones which Elsässer described[3] under the name of "craniotabes or soft occiput." In these cases, the cranial bones, especially the occipital can easily be sawn, or even cut through. The substance of the bones is soft, succulent, vascular and yielding, and in many places rough and porous. Their earthy constituents are diminished. The loosening of the tissue is most marked near the fontanelles and sutures, while at the very border of these the bone seems more compact. The periosteum is thick, vascular, and difficult to strip off from the bone. In the occipital, but also in the parietal bones, espe-

[1] Schwarz (*Jahrb. f. Kinderheilk.*, xxvii., S. 454) found among 500 new-born children in Vienna, 75·8 per cent. already presenting signs of rickets on the cranial bones or ribs; Quisling, in Christiania (*Arch. f. Kinderheilk.*, ix.) found 23 cases out of 200 new-born infants.

[2] *Allgem. Wiener med. Zeit.*, 1885, No. 18.

[3] "Der weiche Hinterkopf," &c. Stuttgart and Tübingen, 1843.

cially along the lambdoidal suture, we find impressions and grooves corresponding to the convolutions of the brain; these are very thin and readily indented like cardboard; sometimes also the bony substance affected entirely disappears, leaving irregular, oval, or angular holes, or apertures which may even be as large as a hazel-nut, in which the pericranium and dura mater are in contact with one another. These appearances are occasionally found in children during the first months of life, but generally from the 4th to the 8th, or at most the 13th month. Only in one case is the occipital bone said to have presented the condition as late as the 3rd year. Elsässer, who regarded craniotabes as the form of rickets observed in young infants, supposed that the parietal and occipital bones, softened by rickets, became gradually absorbed, thinned, and finally perforated. As a matter of fact, on careful palpation of the head of children in the first year of life, one often finds spots on the occipital bone near the lambdoidal suture which are soft and yield to pressure with a crackling sensation like that of cardboard. But in some of these cases I have found that, as the delayed ossification progressed over the whole cranium, the thinned portions of bone also became filled up and consolidated without any other rachitic symptom being added. Therefore I do not think we can always regard this symptom offhand as a sign of rickets when it occurs alone; I should rather agree with Friedleben and Ritter in thinking that it may also occur within the limits of physiological development and without being accompanied by any other morbid change. According to the investigations of Friedleben, in all children in the 4th—6th months, especially those who have been hand-fed, the posterior portions of the cranium contain about 3 per cent. less of earthy salts than do the anterior portions, and are therefore softer, thinner, and more yielding. Under these circumstances constant lying on the back may favour the absorption of bone owing to the pressure acting on the occiput.[1] Nevertheless it cannot be denied that in the very great majority of such cases other rickety symptoms either exist simultaneously in some other part of the skeleton, or are added later. I have already (vol. i., p. 182) given my opinion as to the connection which Elsässer held to exist between craniotabes and laryngeal spasm.

[1] *Cf.* Parrot, *Revue mens.*, Oct. 1879.

Let me add but a few words on the pathogenesis of rickets. The most careful anatomical, experimental, and chemical investigations have not unfortunately by any means succeeded in clearing up the obscurity which envelops this subject; and the views of the authors who have worked much at this disease (and their number increases every year) differ so greatly from one another that it is as yet impossible to form definite conclusions. The examination of the blood has shown no important changes whatever; for a slight diminution of the red blood-corpuscles, or an increase of the white ones cannot be regarded as at all characteristic. The results of the analyses of the urine also present such important differences that we do not know which we are to accept. While according to earlier investigators a more or less considerable increase of earthy phosphates is to be found in rachitic urine, nearly all recent writers deny that there is any increase in the quantity of calcareous matter. Seemann[1] even found a considerable diminution in lime compared with the urine in healthy children, while Baginsky[2] was unable to discover any difference in the discharge of lime-salts between healthy and rickety children. Should this be correct it proves that the deficient amount of calcareous matter in the rickety bones cannot be caused, as was once supposed, by an acid (lactic acid) which dissolved the lime-salts (in which case the discharge of lime in the urine would necessarily be increased)—but simply by diminished supply of lime. Since, however, both human and, in a greater degree, cow's milk, generally contains a sufficient amount of lime, the want of lime in the bones cannot be due to insufficient supply of it in the food. Indeed Seemann himself discovered abundance of calcareous matter in the milk of two mothers whose children showed distinct traces of rickets. It is much more likely to be due to a diminished absorption of the lime-salts by the digestive organs; and this view is favoured by the fact that the fæces of these patients, according to the investigations of Petersen[3] and Baginsky, contain more lime than is found in those of healthy children at the same age.

[1] "Zur Pathogenese und Aetiologie der Rachitis," *Virchow's Arch.*, Bd. 67, 1879.

[2] "Ueber den Stoffwechsel in der Rachitis." *Veröffentlichungen der Gesellschaft für Heilkunde.* Pädiatrische Section: Berlin, 1879.

[3] Rehn, *loc. cit.*, S. 91.

Thus far we may almost be said to stand on firm ground; but all beyond this is matter of hypothesis. Why the lime-salts are not absorbed and assimilated in sufficient quantity by the digestive organs, is a question which has still to be answered; for if it is a fact that only 25 per cent. of the lime-salts of cow's milk is digested, but as much as 78 per cent. of those in human milk (Uffelmann[1]), and that the undigested remainder forms insoluble compounds with the fatty acids in the intestine, still one must bear in mind that even children nourished at the mother's or nurse's breast often enough become rickety. Although the theory of Seemann and Zander[2] (that there is a deficient formation of hydrochloric acid in the stomach, owing to which the lime-salts in the food are not dissolved and absorbed in sufficient quantities) would certainly afford a convenient explanation, still it is as yet merely a hypothesis, and serious objections have been urged against it.[3] It is an open question whether the deficient amount of salts supplied to the bone is sufficient in itself to give rise to the changes in the cartilage and bone characteristic of rickets, as Roloff and Seemann maintain; or whether in addition to this diminished supply of lime there must also be a relatively excessive formation of lactic acid or other organic acids; or whether, finally, the osseous affection is only due to a constitutional irritation acting on the osteogenous tissue, as Wegner[4] infers from his experiments. The anatomical appearances presented by the disease seem to justify us in thinking that we have to do with an irritative or inflammatory process in the regions of bone-growth—the epiphyses and periosteum. Kassowitz[5] has adopted this view with especial zeal, and considers that the very strong current of liquor sanguinis due to the extreme vascularity interferes with calcification and exerts a dissolving effect. The recent investigations of Pommer[6], however, seem to show that this "inflammatory" theory is quite untenable, and that the whole question is as yet not ripe for settlement. But for my part I can hardly attach any more importance to his hypothesis—that the original seat of the affection is in the central nervous system—than to that

[1] *Arch. f. klin. Med.*, 1881, S. 472.
[2] Virchow's *Archiv*, Bd. 83, S. 377.
[3] Baginsky, *ibid.*, Bd. 87.
[4] *Ibid.*, Bd. 55.
[5] *Die Pathogenese der Rachitis:* Wien, 1885.
[6] *Untersuchungen über Osteomalacie und Rachitis:* Leipzig, 1885.

of Oppenheimer[1], according to whom the disease is due to malarial affection—which my experience does not confirm in the slightest degree. As to the vexed question of the relation between rickets and osteomalacia, and also that concerning the occurrence of a special infantile form of osteomalacia, no decisive answer is yet forthcoming.[2]

Under these circumstances we are not in a position to base our treatment upon a thoroughly scientific foundation and we must generally be content with empirical remedies. It goes without saying that both for the prophylaxis and for the regular treatment a suitable diet and other hygienic measures are of the greatest importance, and it is only a pity that these cannot be thoroughly carried out except in the comparatively rare cases in which the patients are the children of well-to-do people. In the great majority of cases, hygienic measures are rendered impracticable by the poor circumstances of the patient. A nourishing and easily digestible diet (milk, beef-tea, yolk of egg, wine, and, later on, meat), fresh country or sea air, dry sunny rooms, and scrupulous care of the skin by means of lukewarm baths—all these are important factors both for prevention and for the treatment of the disease when it has once begun; but they are often quite unavailable. To forbid giving the child milk seems to me perfectly absurd; the prohibition is founded on a few unreliable experiments on the injurious effect of lactic acid, and it deprives the infant of the form of nourishment which is most suitable to it. In spite of the unfavourable external circumstances, however, rickets recovers in the very great majority of cases—unless an attack of tuberculosis or of some other complication sets in or the general cachexia has reached too advanced a degree. I generally commence the treatment with mild preparations of iron, especially tincture of the perchloride (gtt. viii.—x. thrice daily). Should this set up diarrhœa, as I have sometimes seen it do, I substitute lactate of iron or reduced iron (grs. $\frac{1}{2}$—$\frac{3}{4}$ twice daily). Of course the digestive organs must be in good order, to digest the iron. Therefore, if there is loss of appetite, foul tongue, constipation, or diarrhœa, we must first give hydrochloric acid, then mild bitters, especially tincture or wine of

[1] Oppenheimer, *Archiv f. klin. Med.*, xxx., H. 1 and 2.

[2] Rehn, *Jahrb. f. Kinderheilk.*, xix., 1882, Heft 3.—Kassowitz, *ibid.*, S. 439—Pommer, *loc. cit.*

rhubarb (gtt. x.—xii. thrice a-day); and the latter can be given along with iron. We should let the child have a warm bath every second day, with addition of salt (p. 393) or decoction of malt, or of an infusion of aromatic herbs (camomile or calamus —about a handful); and, in order to stimulate the flabby muscles, the lower extremities should be rubbed with flannel and kneaded (both during the bath and on other occasions) several times daily. For the profuse perspiration of the back of the head we should order regular bathing with cold water; and when there are softened spots in the occipital bone we should try (as Elsässer advised) to protect the affected parts as much as possible from pressure, by letting the head lie on a horse-hair cushion with a hole in it. In order to prevent, as far as possible, curvatures of the spine and bending of the lower limbs, we must not allow small children who are rickety to sit up without support, but must generally keep them lying on a hard pillow, and have them taken out on this in a perambulator, and should be very cautious about letting them attempt to stand or walk.

In a very large number of cases I have seen this treatment prove successful within a few months; not unfrequently I have seen the children begin to make attempts to stand on their feet and to move about even within a few weeks. In another set of cases, again, where recovery was not progressing under this form of treatment, I was able to convince myself of the real value of codliver oil, which has been so much lauded by others. I never give it, however, except in the cooler and cold seasons of the year, and then chiefly to emaciated children—never more than 2 dessertspoonfuls daily, either alone or in combination with iron. I have never seen any good result from the administration of lime-salts, which, after what has been said about the pathogenesis of the disease (p. 412), one is hardly inclined to put much faith in; and I have long since abandoned it. In spite of Cantani's recent recommendation[1], I adhere to the opinion I have expressed. Nor do I regard with any greater favour the use of phosphorus which Kassowitz[2] introduced, and praised so highly, on the strength of Wegner's experiments. I am willing to admit that such very small doses as

[1] *Spec. Pathol. u. Ther. der Stoffwechselkrankh.*, Bd. iv., translated by Fränkel: Leipzig, 1884.

[2] *Zeitschr. f. klin. Med.*, Bd. vii., Heft 2.

Kassowitz administers (gr. $\frac{1}{130}$ — $\frac{1}{65}$ daily, in codliver oil or sweet almond oil) scarcely ever do any harm, but the numerous experiments which were carried out in my polyclinic[1] and also several others in my wards, failed to show that this method had any advantage over that which I have recommended above. As regards treatment with phosphorus—which is the subject of controversy—I must, therefore, rank myself along with those who do not consider this remedy a specific for rickets.

The deformities of the limbs due to curving and bending of the bones require no special treatment when they are only slight in degree; for when recovery takes place and the limbs grow in length and bulk, these deformities steadily decrease and finally disappear. But after all, and in spite of what many say to the contrary, neither orthopædic treatment nor splints of any kind are capable of curing the curvature of bones after they have already become consolidated, or the consequent deformities. Only in recent conditions, where the bone is still soft and flexible, can any success be expected from this proceeding (redressement). Bandages and boots combined with suitable apparatus may, however, be useful in cases where, owing to a very marked curve of the lower part of the tibia the children are walking (as in pes varus) on the outer or inner edge of their feet. Similarly, orthopædic and gymnastic treatment need not be tried in old cases of rickety curvature of the spine. In order to lessen or entirely remove the deformity of the thorax, it has lately been recommended that we should use Hauke's "pneumatic bath" or cause the patient to breathe compressed air. In the first of these forms of treatment, the children are left for some time every day in chambers with a rarified atmosphere. I have never yet tried it, but a few good results have been obtained, and encourage us to give it a trial.[2]

The treatment of deformities of the extremities which have become consolidated, of genu valgum, &c., belongs to the domain of surgery, and very successful results have been obtained by bold operations—such as re-fracture of the bones which have been broken and have united in a bad position (followed by the application of plaster of Paris), osteotomy, &c.

[1] Schwechten, *Berl. klin. Wochenschr.*, 1884, No. 52.

[2] Kaulich, *Prager med. Wochenschr.* No. 2, 1880.—v. Laszewski, "Zur pneumat. Therapie im Kindesalter," [illegible], 1886.

## SECTION X.

### DISEASES OF THE SKIN.

The skin is extremely often the seat of morbid conditions in childhood, but these are almost all just the same as those in adults, both anatomically and clinically. I may, therefore, confine myself here to the consideration of those affections which are either much commoner in children or present in them certain peculiarities. First, however, I must touch upon a question which, on account of its great practical importance, has always engaged the attention of physicians and has received the most diverse and contradictory answers. I refer to the metastasis of skin-diseases. Under this name the older physicians understood the rapid disappearance of a skin-affection followed by the development of an internal or external disease immediately after. This they supposed to be due to the "morbid material" receding inwards from the surface, and they proposed to cure it by endeavouring to re-establish the discharge from the skin which had dried up. It is the custom, now-a-days, to discredit the existence of this metastasis. Hebra, especially, expressed himself with great decision against the occurrence of any such thing and consequently had no fear whatever that any effect on the general system would result from the rapid suppression of the skin-disease. And, indeed, a great many incorrect statements have been made on the subject; for example, some have overlooked the fact that the causal relation is often quite the other way, namely, that the skin-disease may disappear because the internal disease is in process of development. Thus chronic eruptions on the head frequently dry up when an attack of meningitis is coming on, just as in similar circumstances the nasal mucous membrane becomes dry, otorrhœa ceases and glandular enlargements may rapidly subside. These are all indisputable facts. On the other hand, experience has shown that the inflammatory irritation may spread from a suppurating

eruption on the scalp into the interior of the cranium owing to phlebitis or thrombosis of the small veins of the skin and bone, and there give rise to dangerous symptoms. In spite of all this, I do not regard the question of metastasis as by any means closed. To begin with, I do not consider that in this matter hospital cases can be regarded as having nearly the same value as they usually have; because the little patients are generally discharged from the wards immediately on recovery from their eruptions and the physician knows nothing about the subsequent history of most of them. I therefore regard private practice as far better fitted for the study of this question. And, personally, I am now much more chary of absolutely denying the occurrence of metastasis, and have been, ever since I observed without prepossession of any sort two or three cases in which a severe attack of pleurisy with effusion, bronchitis or diarrhœa set in almost immediately after the artificial suppression of a chronic eruption on the scalp, and in which marked improvement at once occurred on the spontaneous re-appearance of the eruption.[1] Besides these cases I afterwards observed two others in very young children in whom fatal convulsions set in 8—10 days after rapid recovery from an attack of eczema of the head and face. I am quite aware that these individual cases are not sufficient to settle the question and that they may be merely the result of a chance coincidence. Nevertheless, they made a deep impression on me and awoke in me doubts which had long been silenced as to whether sudden hyperæmia with its results might not arise in other parts owing to the rapid drying up of an extensive purulent or sero-purulent discharge which had existed for a long time. This possibility should not, I think, be ignored altogether in the treatment of such eruptions, and I shall return to the subject in speaking of eczema.

## I. *Erythema and Intertrigo.*

Erythema is one of the commonest of the skin-diseases which we meet with in childhood, especially during the first year or two of life. It is characterised by a varying number of red patches of different size and form which appear on the face and

[1] *Berl. klin. Wochenschr.*, 1864, No. 5.

also all over the body. The smaller of these are from the size of a lentil to that of a pea and rounded in form, and they are often described as roseola; the larger, which are irregular in form and extend over a greater surface, are more specially styled erythema. Occasionally the hyperæmia is accompanied by slight exudation, so that the reddened area of skin appears infiltrated and somewhat raised, either all over, or in some places, or just round the margins. Thus many varieties have arisen to which we apply the names erythema nodosum, papulosum, marginatum, or annulare, and which are identical with the forms occurring in adults. Sometimes erythema is combined with small blood-extravasations or with wheals like those of urticaria (erythema urticatum). The appearance of the rash is sometimes, but by no means always, accompanied by feverish symptoms (general malaise, loss of appetite, quickening of the pulse and rise of temperature), but these generally pass away when the eruption is completed. The eruption still continues for several days, the child seeming perfectly well; it then gradually fades and finally disappears without leaving a trace or with only a slight desquamation. The eruptions are sometimes very itchy so that the child keeps scratching them; and they are especially prevalent among children during the spring months, from March to May. I have hardly ever been able to ascertain the causes of the affection. Only in two or three cases—*e.g.*, in that of a child of 10 months weaned a fortnight before—an attack of erythema papulatum and nodosum accompanied by great itching set in as the result of a fit of vomiting and diarrhœa caused by an error in diet. I have especially often noticed erythema nodosum in children, generally on the lower extremities only, in the form of large nodules reddened at their apex, which disappeared after 2—3 days and which left behind a bluish or brownish pigmentation (remains of blood-colouring matter). This eruption was sometimes accompanied by a moderate amount of fever, œdema of the eyelids and of the backs of the hands and feet, often also by rheumatoid pains in the ankles, knees and wrists. Slight desquamation was frequently observed.

When erythema sets in with fever it may be mistaken for measles and, in a more diffuse form, even for scarlet fever. The comparatively low temperature, the absence of the characteristic sore-throat and the rapid course not followed by scaly

desquamation, are sufficient to differentiate it from scarlet fever. Still, one must not forget that cases of very slight scarlet fever (p. 199), sometimes occur in which the diagnosis is by no means an easy matter and can only be given with certainty after the characteristic peeling has begun. Many cases of recurrence of measles or scarlet fever in the same child may be due to erythema being mistaken for these affections. For I have frequently observed such attacks of erythema set in, especially in the convalescent stage of these diseases; it was sometimes diffuse, sometimes combined with urticaria extending more or less over the whole body, and sometimes accompanied by high fever (up to 104). But after the lapse of 24—36 hours the eruption has generally disappeared and the fever with it. Contagiousness is not a characteristic of these forms of roseola and erythema, although they may appear to a certain extent as an epidemic. Treatment is scarcely ever necessary, but when there are feverish prodromata we should keep the patient in bed and administer a mild purgative. No further treatment has been necessary in any of the cases of this sort of eruption observed by me, as there have never been serious symptoms of any kind.

Apart from the erythematous eruptions, which occur in children as well as in adults, accompanying general febrile diseases (rheumatism, typhoid, pyæmia, or diphtheria), or due to the action of drugs (quinine, antipyrin, chloral, &c.)—we not unfrequently find erythema occurring in the neighbourhood of excoriated or ulcerated areas of skin. This occurs, for example, round about vaccine pustules, and the whole arm may become red and swollen. Again, I have observed it in the neighbourhood of patches of eczema and intertrigo, and it is to be distinguished in such cases from erysipelas by its more "patchy" character, by the absence of fever and of wandering tendencies. Simple fomentations with lead-lotion are nearly always sufficient to cure this form of erythema, which is to be regarded as a simple inflammation of the skin and not as an infectious disease like erysipelas.

Along with these conditions are to be ranked the inflammatory affections of the skin which arise from direct irritation (pressure, chemical irritants), and are described under the name intertrigo. In very many young infants, especially such as are not properly cared for, we find more or less extensive

bright- or dark-red patches of erythema round the anus, on the genital organs or on the inner sides of the thighs, due to contact with the urine and fæces. We often also see similar patches on the heels and on the posterior surface of the thighs and legs, which are constantly in contact with the wet napkins when the child is lying on its back; or in places where folds of the skin are in contact with one another, as in the inguinal regions, on the front or back of the neck, the upper part of the chest, under the axillæ, behind the ears and in the flexures of the elbows. In many children, even when well taken care of, there is a marked tendency to intertrigo, and in such patients it spreads over large areas of the skin—*e.g.*, over the whole lower half of the body, sometimes also over the back or abdomen or even over the whole body, especially if proper attention is not given to cleanliness. At the same time we sometimes see little eczema-vesicles or dark-red papules appearing here and there on the reddened skin, and it then often assumes a moist shining and sticky appearance, owing to the erythema having passed on to dermatitis. The epidermis becomes softened and macerated by serous exudation, so that a great part of the body has a dark-red, shining excoriated appearance. The same thing also often occurs in the case of intertrigo when it is limited to the above-mentioned folds of skin; and when the epidermis separates, more or less deep, yellowish-grey, irregular ulcers may form in the centre of the reddened skin, and if they are situated about the anus and genitals are apt to mislead one into diagnosing syphilis. An inexperienced practitioner is liable to the same error when intertrigo about the anus and on the nates is accompanied by pretty large papules, the rounded apices of which are deprived of their epidermis and have the appearance of red or yellowish-red excoriations; and, in fact, these excoriations may have a considerable resemblance to ulcerated mucous papules. In a few cases I have seen the inflammation of the skin extending deeper and leading — especially in the neck — to the formation of phlegmon and abscess. All these forms of intertrigo may occur in the children of well-to-do families, but they are far more frequently met with in the neglected and atrophic children of the poor. Want of cleanliness, living in dark and crowded rooms, insufficient or unsuitable nourishment, chills, &c.—all combine to produce the condition which is appropriately called

cachexia pauperum. It is generally in such children that we see the intertrigo spreading over the greater part of the body. The epidermis may be raised up by a serous exudation underneath, and then there may be only fragments and shreds of it left on the reddened, moist and shining cuticle, as happens in extensive burns; while in other cases the skin is red but dry, and is covered all over with an enormous quantity of greyish-yellow desquamated scales consisting of epidermis and sebum. These cases, like extensive burns, end fatally owing to a complication with pneumonia or diarrhœa.

The treatment of intertrigo demands, above all, the most scrupulous cleanliness, frequent change of the linen and washing of the genital organs and the neighbourhood of the anus immediately after each discharge of urine or fæces. The reddened areas should be very frequently dusted with a powder composed of equal parts of oxide of zinc and starch, and affected folds of skin which are in contact must be separated by placing between them pieces of charpie or linen smeared with zinc- or lead-ointment. In the preparation of these ointments I always prefer pure vaseline (which may also be used alone) instead of ordinary lard, which is apt to become rancid and then gives rise to irritation. Warm baths, also, are apt to be too irritating; we must not use them hotter than 90° F., and we may add to them bran, gelatine, or bolus alba (1½—3½ oz.). In very extensive cases of intertrigo I have sometimes obtained good results from baths containing corrosive sublimate (grs. xvss.) even where there was no syphilis, while in obstinate intertrigo limited to the anus and genitals I have seen success follow daily painting with a solution of nitrate of silver (1:50), or corrosive sublimate (1:2000).[1]

## II. *Lichen-Strophulus and Prurigo.*

A. Lichen-Strophulus.—This skin affection is extremely common in childhood. It is characterised by appearance of numerous bright-red nodules on different parts of the skin, on the face, back, or extremities, which are either discrete, or, less commonly, clustered together in groups on a slightly reddened and somewhat infiltrated base; some of them are found to be perforated

[1] Wertheimber, *Ber[illegible]enschr.*, 1873, No. 15.

by a hair. They vary in size, some being so small that they are more easily felt than seen, some much larger, and reaching the diameter of a split-pea. Generally, but not always, there is violent itching, so that the children scratch the papules until they bleed. When the eruption is scanty the general health is not disturbed to any great extent; but when it is profuse, or when it extends over the greater part of the body, sleeplessness and great restlessness may arise from the itching. Some of the papules gradually fade and disappear by absorption; others present at their apex an inconspicuous vesicle or a small pustule, which dries up and finally leaves a thin scale on the diminishing papule. Relapses are common, so that the eruption may continue getting alternately better and worse for several weeks and even months before recovery finally takes place.

These papular eruptions are most frequently observed during the period of the first dentition, and are therefore regarded by many as consequences of the irritation of teething—that is, of a vascular neurosis reflexly excited from the dental nerves. The name "strophulus" is mainly applied to this form. Among the local irritations which may give rise to this affection, I should draw your attention specially to the influence of the sun's rays and of the heat, which besides producing a vesicular eruption (eczema solare) often also gives rise to a large number of extremely small red papules on the neck, back, chest, and face. In very many cases, however, we remain quite ignorant of the cause of the eruption. No connection with morbid conditions of the internal organs can be made out, and the assumption of a scrofulous constitution is almost always unfounded. The fact that we find these eruptions more frequently, upon the whole, among the children of the poor than among those of the higher classes, seems to indicate that here also unfavourable hygienic conditions, especially deficient cleanliness, are not without their influence.

The treatment of lichen-strophulus consists only in the use of warm baths, with the addition of bran or soap when the itching is violent. We may also recommend bathing twice daily with carbolic lotion (1—2 per cent.) on account of its soothing effect. Internal remedies, as far as my experience goes, have no influence on the skin-disease, and there is the less need for them as the trouble generally disappears of itself after a certain time.

B. Prurigo.—The prognosis in this disease is far less favourable. I have very often observed it even in children in the first few years of life, presenting the symptoms so strikingly described by Hebra. My experience goes to confirm his view that the great majority of cases of prurigo commence in childhood. The symptoms differ in no way from those observed in adults; in children also we find the prurigo-papules, partly pale, partly covered with a dark spot of blood (caused by scratching), situated mainly on the extensor aspects of the limbs, while the flexor aspects are little if at all affected, but also at the same time on the abdomen, back, and chest. The constant violent itching makes the children scratch themselves, and to this mechanical injury we must ascribe the further changes in the skin which gradually develope in the course of the disease—the eczematous eruptions and the roughness and thickening of the skin —which may materially modify the originally simple papular rash. I have hardly ever failed to find in children the unusually marked swelling of the lymphatic glands in the inguinal region and over the adductors of the thighs, which Hebra pointed out. The general health is unaffected, but the disturbance of the rest at night may in the end begin to tell on the child's appearance, especially as prurigo is always a very chronic complaint, continuing for years with slight intervals and almost always obstinately resisting treatment.

In some children with prurigo I have observed an eruption of small pemphigus-bullæ accompanying or preceding the disease, and in one case they preceded the prurigo in pretty considerable numbers; in the others only a very few appeared from time to time between the papules. Also in an old man whom I had treated in summer for very extensive acute pemphigus of eight weeks' duration, I observed during the autumn and following winter an eruption of very itchy prurigo-papules on different parts of the body which he had never suffered from before.

The etiology was obscure in all the cases, neither a hereditary disposition nor a tubercular tendency (to which Hebra attaches importance) could be ascertained with certainty. As regards treatment, I have been no more successful than others, for only in a single case (that of a boy of 9 years) have I succeeded in obtaining a cure which held good for several years; but this same patient's sister, who [illegible]ring at the same time

from prurigo, kept coming to the hospital with relapses. Daily rubbing of the body with sapo viridis in a warm bath, followed by baths of Vleminx's solution of sulphide of calcium (Form. 47), seemed to have a curative effect in the boy's case. But in other cases the same measures had no more effect than had the internal use of Fowler's solution of arsenic.

Finally, I should point out to you that on superficial examination old cases of scabies are apt to be mistaken for prurigo, if one has difficulty in discovering distinct burrows. You must always bear this possibility in mind, and that not only in practice among the poor. I have frequently observed scabies in children in well-to-do families, even in the first years of life and under circumstances which seemed to exclude the possibility of such an infection—where, therefore, the demonstration of an acarus caused no small horror.

## III. *Eczema and Impetigo.*

Of all the skin-diseases occurring in childhood, the vesicular and pustular forms are the commonest. By a purulent change in the contents of the vesicles, eczema passes into impetigo, the pustules of which at once become larger and when they burst generally dry up so as to form thick crusts of a clear yellow colour. We very often find vesicles, pustules, and their remains occurring together in the same situation on the skin (eczema impetiginosum).

This eruption often appears in early infancy, often indeed a few weeks after birth, chiefly on the face, and in this situation it is well known to the laity here by the name of "milk-scab" (crusta lactea). In the typical form of the disease we find the forehead, cheeks, nose, upper lip, and chin covered by a scab which is more or less continuous, or is broken at places through which we see the red excoriated skin. This scab is of a greenish or blackish-brown colour, and covers the face like a mask, through which the eyes look out bright and natural. In some places the scab is scratched off, and the blood which trickles from the excoriated skin becomes clotted and forms dark crusts. On closer examination we sometimes also find, round about the scab or on the parts of the skin still uncovered, small vesicles, either

standing singly or clustered together on a red surface, and little pustules, the dried secretion of which has formed the scab. Apart from the tormenting itchiness, these children usually feel perfectly well, and really look quite robust and well-nourished. Still, the neighbouring lymphatic glands under the angle of the jaw and the chin are generally slightly swollen, owing to the lymphatics being affected. The duration of the eruption varies greatly; as a rule it goes on getting alternately better and worse for 4—6 weeks at least, and it often lasts for months and even years, causing very great anxiety to the parents. We often notice that the eruption dries up during an attack of acute disease—*e.g.*, pneumonia, or any condition characterised by great loss of fluid, especially profuse diarrhœa—but returns again when the patient has recovered from the intercurrent disease. Recovery finally takes place by a cessation of the eczematous eruption and of the serous discharge secreted by the raw surface. Then the crusts dry up and fall off, leaving the skin reddened, but free from every trace of cicatrisation.

In a number of cases of this kind, the eczema spreads from the face over the scalp and the external ear into the interior of the concha and nostrils, and even as far as the lower eyelid, where it is apt to give rise to inflammation of the palpebral conjunctiva, even extending to the cornea.

The causes of crusta lactea are obscure. The assumption of a "scrofulous" constitution is generally quite arbitrary; still, it is an ascertained fact that the affection is hereditary in many families, so that almost all the children in them, even for several generations, suffer during infancy from eczema of the face. The disease is also ascribed to the nourishment, especially to the milk of some nurse being too rich and not suited to the tender age of the child; but confirmation of this theory—*i.e.*, recovery of child on change of nurse—has only been adduced for a very small number of cases. I agree with Bohn[1] that a very large number of children suffering from eczema of the face are unusually fat, and in such cases it is certainly advisable to make some change in the feeding. I have already referred (vol. i., p. 155) to its connection with dentition. I have certainly observed that each appearance of a group of teeth was accompanied by a recurrence of eczema on the cheek which had been

[1] *Jahrb. f. Kinderheilk.*, xx., 1883, S. 45.

recovered from, and that this again disappeared when the eruption of the teeth was complete.

Eczema is also common in later childhood, but it is then apt to affect the external ear, the concha, the region behind the ears and especially the hairy scalp, more severely than the face. Eczema capitis often forms extensive continuous crusts, covering the whole scalp. These are of a greenish-brown or greyish-green colour, are tolerably moist, cause the hair to become matted, and out of the fissures between them there trickles sero-purulent matter, which is very often offensive. These scabs are sometimes swarming with lice. In other cases the affection is more limited, and the scalp is only covered by these scabs in certain places.[1] They are either round in contour or quite irregular and become hard, like mortar, when dry. We then find loosened fragments elevated by the growth of the hair, and hanging on the hairs like beads on a string (tinea granulata). If we carefully remove the scabs by fomentations, we find the skin of the head red, raw, and covered with secretion. Vesicles and pustules often form round about the scabs, especially if—as often happens—the disease is breaking out afresh in places which had already healed. The violent itching makes the children scratch themselves, and this keeps up the inflammatory irritation. The neighbouring glands behind the ears, at the back of the head, under the jaws, and on the neck, become enlarged, and in many cases the secretion which accumulates under the scab becomes decomposed, causing an offensive odour. Not unfrequently the inflammation of the skin also spreads inwards from the surface. I have several times seen dense infiltrations of the skin arise in the centre of the eczematous areas, and finally pass into abscesses. Indeed, in several cases a large collection of pus formed under the pericranium of the parietal

[1] We must not confuse with this condition the well-known scaliness of the skin which we often find on the scalp of small infants, especially over the large fontanelle, forming a more or less thick yellowish or brownish crust. This consists of the inspissated product of an increased activity of the sebaceous glands (seborrhœa) mixed with desquamated epithelial scales. It often remains for months and sometimes forms afresh after having been removed. As a simple remedy I may recommend the rubbing-in of fresh butter or yolk of egg, followed by thorough washing with soap and water. Moreover, owing to the local irritation of the skin caused by the constantly accumulating layers of sebum and epidermis, eczema capitis is finally set up, and its sero-purulent discharge and crusts are mixed with the products of the seborrhœa.

bone, and when it was opened the probe reached right down to the bone, while at its border the well-known bony ring could be felt, as in cephalhæmatoma (vol. i., p. 37). In other cases erysipelas developed from the eczema capitis, which spread over the neck and face, and was accompanied by high fever (vol i., p. 48).

The duration of eczema capitis is generally very protracted. It frequently lasts (with intervals of varying length) for years, even up to the period of puberty—especially in poor children who are not kept properly clean. The hairs then usually undergo a morbid change, becoming lustreless and thin, and falling out; but they grow again after the disease is recovered from. Whenever this takes place, the secretion from the raw surface ceases, the dried scabs fall off, and for a long time afterwards small yellowish dry scabs keep forming on the reddened skin.

We often find eczema occurring on the body and limbs of children who are already suffering from it on the face and head. Still, the face and head may remain quite unaffected, especially in older children (at the age of the second dentition), and in them the disease presents the same features as in adults. In some cases the eruption has existed since the earliest childhood, as for instance in a girl of 6 years who had suffered continuously since she was 7 months old from an eruption of eczema involving the greater part of the body. In these chronic cases I have most frequently found the flexor surfaces of the elbows and knees affected, and also the inner surfaces of the thighs and calves. When the eczema is situated at the tips of the fingers, especially near the bed of the nails, not only may the affected nails separate, but the newly-formed ones may be deformed and claw-shaped, and may bleed when carelessly pared, owing to the hypertrophied papillæ of the skin being prolonged into them.

The course of these eruptions, wherever they may be situated, is generally chronic, extending over many months, and even years. Still, acute eruptions of this kind, lasting only a few weeks, often occur in otherwise healthy children. I have observed them repeatedly on the upper arm, and on the corresponding axilla, also on the lower extremities and face, especially on the chin, apart from any other morbid symptoms, especially without fever. In a boy of 14 (i[illegible]o me June 9, 1879) an

outbreak of eczema on both cheeks and ears had taken place regularly every spring for ten years, the eruption lasting 4—6 weeks and then completely disappearing. In a healthy girl of 11 (Feb. 7, 1881), an attack of acute eczema had occurred 6—7 times within one year; it was preceded by burning pains, and always appeared on the same place—namely over the right temporal region. It sometimes extended to the lower jaw, and generally lasted 3—4 days; examination did not reveal any local cause. Sometimes in the course of a chronic attack of eczema of the face, we suddenly find an acute outbreak taking place on the area of the skin which has long been in a raw condition, and round about it. The eyelids and the whole face become much swollen, and the fresh vesicles which appear on the face grow into large pustules, some of which have a central depression, like those of variola, and run into one another. I should specially draw your attention to this form of the disease, because I have frequently known errors to occur in the diagnosis of this rash. One case of the kind which occurred in private practice was diagnosed with perfect confidence as "variola," although the complete immunity of all the other parts of the body (there were only a few similar pustules on the wrists), the complete absence of fever and the general appearance of good health were against this from the beginning. And the further course of the disease actually showed that variola was quite out of the question. These acute exacerbations of chronic eczema were generally due to the children scratching the eruption until it bleeds.

I must here say a few words more about the hæmorrhages which may take place from an eczematous eruption on the face. These are not always due to traumatic influences, and I have myself met with three cases (all of which affected children between 3 and 4 months) in which the hæmorrhages were so severe as to lead to exhaustion, and end fatally. These seemed to be really cases of hæmorrhagic diathesis; but only in one of the cases did this show itself by the simultaneous occurrence of slight hæmorrhages from the stomach and bowels, while in the other two there was no hæmorrhage besides that from the surface. One of these two seemed perfectly healthy, while the other was suffering from rickets and spasmus glottidis, and appeared sickly and anæmic. In all the three cases the

blood oozed from eczematous surfaces and fissures almost constantly without their having been scratched, and formed soft black scabs which were soon washed away by the further flow of blood. All styptic remedies, including ergotin, were tried without success, and the children died from the steadily increasing exhaustion. Owing to these experiences I should advise you never to regard spontaneous hæmorrhage from eczema of the face recurring without a definite cause (scratching), as a symptom of trifling import.

We know very little of the etiology of eczema in general. Only in comparatively few cases can we arrive at the cause with certainty. Among these we may include the form known as acute eczema æstivum (solare, sudorale), which may result from excessive perspiration in rickety children (p. 399), but which chiefly appears during the summer-heat in very many children, especially young infants, in the form of a close eruption of extremely small vesicles and papules on a reddened surface and situated on the back, chest, and neck, but especially on the forehead and temporal bones. Occasionally larger papules and even pustules occur among these. In one girl of 3 years, I observed at the same time extreme erythema and œdema of the eyelids and forehead of the left side; in another child, there were even bullæ, like those in erysipelas. In other cases traumatic influences play an important part in the causation.

Child of 2 months, brought November 13th, 1879. Owing to the child having been delivered with forceps, the forehead had been bruised and excoriated and, for this, warm fomentations had been applied. A fortnight later eczema appeared over and near the bruised parts, reaching up to the parietal region and accompanied by severe œdema of the eyelids.—I have often known boring the ears give rise to an attack of eczema in very young children, which either remained confined to the outer ear or spread over the neck and back.—Other eruptions, especially psoriasis, may arise in the same way, as is well known. Thus, in a girl of 5 years who had formerly been quite healthy, I observed psoriasis develope on the nates immediately after the cicatrisation of a large burn in that situation, and it afterwards extended all over the body.

You will very often find vaccination declared to be the cause of eczema. The mothers say that immediately, or soon, after the child was vaccinated the eruption made its appearance on the face and on other parts of the body. Although I believe

that many cases of this kind mainly depend on a chance coincidence, still I should not care to deny that such a connection is possible; for other acute eruptions are frequently followed by eczema and impetigo—especially measles, but also scarlet fever, chicken-pox, and small-pox. In a boy of 5 years who had formerly been healthy, eczema commenced on both thighs immediately after scarlet fever, and lasted for months; I have frequently met with similar cases. On the other hand, the common assumption of a scrofulous constitution is often quite unfounded, and can only be justified by the presence of other symptoms of scrofula. The accompanying secondary enlargements of the neighbouring glands are not in themselves sufficient for this purpose.

It is asserted that eczema impetiginosum may be contagious, and Hebra and others have described a filamentous fungus by means of which the transmission is supposed to take place. I have met with several cases occurring in members of the same family, particularly noteworthy being that of a little child suffering from eczema of the face and head; he, after a few weeks, infected an older sister who carried him about and always leaned her cheek against his head.[1]

Most mothers urge you to cure the disfiguring eruption as soon as possible; but you must always consider whether such radical treatment is really advisable. For my part, having regard to past experience (p. 418), I consider it my duty not to be in too great a hurry about curing chronic cases of eczema—especially on the face and head—when the eruptions have already lasted for months and years. I prefer to cure them gradually by treating locally one part of the skin after another—as indeed one is often obliged to do owing to the extensive distribution of the affection. We must first remove the scabs by inunction with vaseline, or sweet oil, or by warm fomentations. For the head, it is best to apply the latter covered by a cap of oiled silk or guttapercha-tissue. After the scabs have been removed, the red and moist surface is to be washed once a day with soap and water (sapo viridis) and then dressed afresh every time with Hebra's ointment (diachylon) which is to be left on for 12

[1] The fear that eczema might afford an easy entrance to the tubercle-bacilli, does not, according to Demme's researches, appear to have very good grounds (21. *Jahresber. d. Jenner'schen Kinderspital*). Out of 17 cases which he examined, he only once obtained any positive results.

hours. Only when the inflammation is very severe, do I order fomentations of lead-lotion to start with. The most difficult thing is to keep the dressing firmly applied to the face in very young children and to prevent scratching. Neither the application of a linen mask with the ointment smeared on its inner side, nor tying up the hands and fingers with wadding and linen, are sufficient for this purpose, and we are generally obliged to protect the affected parts by firmly applied bandages leaving—as regards the face—only the eyes, nose, and mouth uncovered. Instead of Hebra's diachylon ointment, we may also use with advantage ointments of salicylic acid (Form. 48), tannin (Form. 49), boracic acid (Form. 50) or zinc; less commonly, and in limited patches of eczema, an ointment of white or red precipitate (1 in 30 of vaseline). It is not advisable to use tar ointments to begin with, for their effect is apt to be too irritating, and to aggravate the inflammation; on the other hand they are to be recommended after previous treatment with one of the above-mentioned ointments, in order to complete the cure. We generally use oleum cadinum, which is applied daily as a liniment (1 part to 2 or 3 parts of olive oil) after washing with soap and water. But in applying tarry ointments to large surfaces, you must always bear in mind the possibility of their exerting an irritating action on the kidneys (p. 167), and must therefore carefully examine the urine and discontinue the treatment should it become of a blackish tinge or contain albumen. During the last few years we have generally used (and with success) the mode of treatment recommended by Burchardt,[1] especially in cases of eczema of the face and head. The affected areas are painted with 2—3 per cent. solution of nitrate of silver once or twice daily, and later, only every second day—a somewhat painful proceeding which is apt to occasion small hæmorrhages. Very soon the secretion of matter and formation of scabs ceases, and the blackened parts are then dressed with an ointment of oleum cadinum (15 parts), oxide of zinc (20 parts), and vaseline (100 parts).

The duration of the treatment varies, of course, very much. While many cases of eczema—even those that have lasted for a very long time—recover after a few weeks' treatment, others have to be treated for months before recovery takes place, and even then

[1] *Monatshefte f. prakt. Dermatol.*, iv., No. 2, 1885.

we find relapses occurring often without any apparent cause. Eczema of the face is especially apt to keep recurring until dentition is complete, and then disappear without any treatment. In many very obstinate cases I have obtained excellent results from the use of arsenic in the form of Fowler's solution (Form. 11), principally, it would seem, from the alleviation of the itching and the consequent diminution of the scratching. Even little children of 2 or 3 years bear this remedy very well in small doses (gtt. 3—5 of the mixture given thrice daily on a full stomach). In cases with a scrofulous diathesis, I have seen good results from the use of syrup of the iodide of iron, or a mixture of iodine with iodide of potash (Form. 46). On the other hand, I cannot agree altogether with those who praise salt-baths so highly, for I have frequently found that they aggravate the eruption by causing a strong irritation of the skin. I should much rather recommend warm (90° F.) soap or sulphur baths; the latter may be prepared by adding 1½—3½ oz. of sulphurated potash to each bath.

## IV.—*Ecthyma and Rupia.*

We often observe in children large purulent pustules surrounded by a red margin, occurring singly or in groups; these may either be combined with eczema, or they may occur independently. They are most frequently situated on the nates, thighs, and legs; they may be as large as a pea, and they dry up so as to form blackish-brown crusts, and when these fall off, reddish spots or scars are left behind. Ecthyma is often found in scrofulous children, but it also occurs in those who are quite healthy, if they are not kept properly clean. It seems also to be often caused by the irritation of vermin (especially pediculi vestimentorum), and therefore one should look for this cause in such cases. In other respects the treatment is the same as that of eczema.

Ecthyma often occurs also, however, as the expression of a cachexia in sickly ill-nourished children, or those weakened by poverty and disease—especially by general tuberculosis, typhoid, measles, and scarlet fever—and then it has an unfavourable significance. Generally the condition in these cases arises from the

so-called cachexia pauperum which is due to the patient's wretched circumstances, and is manifested by extreme anæmia, emaciation and debility, and by a tendency to chronic inflammation of various tissues. The skin is also frequently implicated in the form of ecthyma or rupia cachectica, and these conditions may be combined. The difference between the two eruptions consists more in the size of the elevations of the epidermis than in their contents; for even the flaccid rupia-bullæ, which may attain the size of a sixpence or larger, are usually filled with opaque purulent matter like that in the ecthyma-pustules. In these cases deep ulcerations are apt to form out of the pustules and bullæ. I have seen them especially on the scrotum and in its neighbourhood, but also on the back; they were more or less numerous, round in shape with sharp borders as if punched out, and their size varied from that of a pea to that of a sixpence. As the general health improves, these ulcers may gradually heal, leaving cicatrices behind, but in unfavourable cases they go on steadily increasing in size, depth, and number. Worst of all, however, is the passage of ecthyma and rupia into gangrene, which often takes place in cases of this kind.[1] I believe I shall best illustrate both processes by giving the following cases.

Johann B., 1¼ years old, admitted on March 11, 1879. Very atrophic and wasted, presenting a number of cutaneous ulcers which, according to the parents' statement, arose from "pustules." They were almost entirely confined to the neighbourhood of the genitals—the scrotum, mons pubis, inguinal region and thighs; there were also a few on the nates. The ulcers varied from the size of a lentil to that of a sixpence, they were sharply defined, extended down to the papillary bodies of the cutis, they had a yellowish-grey floor and somewhat undermined edges. Several of them had coalesced so as to form larger ones. During the next few days, similar ulcers formed behind the right ear and coalesced, forming a deep ulcerated fissure which almost separated the auricle from the skull. Death in a state of collapse on March 21st. —*P.-M.* Double broncho-pneumonia, caseous degeneration of the bronchial glands, chronic intestinal catarrh.

Clara P., 2½ years old, admitted April 2, 1879; tolerably well-nourished. Several round ulcers on the right leg, 3 of which were

[1] According to Eichhoff (*Deutsche med. Wochenschr.*, No. 47, 1884) this gangrenous ulceration is due (?) to the action of a fungus (trichophyton tonsurans).

of the size of a shilling, with yellow sloughy floor and red punched-out margins. These were said to have arisen from pustules with dark red margins which had existed a fortnight before (ecthyma); recent pustules of a similar character were still visible here and there on the body. At the same time severe coryza, bilateral otorrhœa, eczema of the auricle and glandular swellings. After the 6th, a fresh eruption of pustules on the left leg, back and nates; these burst and quickly passed into ulcers, many of which, after the 16th, coalesced (especially on the back) so as to form large sores covered with a blackish-brown crust and emitting a distinctly gangrenous odour. The skin of the whole back, abdomen and part of the extremities was gradually eaten away by these deep gangrenous ulcerations. Almost all over the body we found small and large black sores with a red border, some of them round and some of them sinuous in contour owing to coalescence. Progressive emaciation and exhaustion, irregular rise of temperature, which on the evenings of the 26th and 27th reached 105·4°. Cough and diarrhœa. Owing to the extensive skin-disease, examination of the thorax was not possible. Death on 5th May.—*P.-M.*: In addition to the multiple gangrene of the skin we found chronic fibrinous pleurisy, double-bronchopneumonia, circumscribed gangrene of the lower lobe of the left lung, tuberculosis of the right lung, diaphragm and internal genital organs (salpingitis, perisalpingitis and perioopheritis tuberculosa).

In this case the wide-spread tuberculosis was the cause of the cachexia, from which arose the ecthyma cachecticum and the multiple gangrene resulting from it. The gangrene of the lung found at the post-mortem was probably of embolic origin (vol. i., p. 450). In spite of the most careful treatment with stimulants, local application of chloride of zinc, tannate of lead, camphor wine and carbolic oil, and immersion for many hours in warm aromatic baths—we did not obtain the slightest result. The following case, which was observed in private practice, ended in recovery.

A girl of 8 months, at the breast, perfectly healthy but pale. In the beginning of May, 1883, the disease began with the formation of a furuncular abcess of the size of a hazel-nut in the perineum. After this healed, there arose round about it a large number of bullæ, from the size of a pea to that of a hazel-nut, filled with serum. These were found on the perineum, labia, nates, groins, and on the inner surfaces of the thighs. The bullæ soon changed into dry black gangrenous scabs, some of which ran together and assumed sinuous outlines. The neighbouring skin was slightly infiltrated and reddened. Temperature to 102·2°.

Whimpering, as if from pain; restlessness, but good appetite. Treatment with fomentations of infusion of camomile, lead lotion, and carbolic lotion; internally, quinine and wine. After the scabs separated, ulcers were left, which looked just as if they had been punched out of the skin. Recovery after 3 weeks.

The etiology of the gangrenous rupia occurring in this healthy child is quite obscure.

### V.—*Abscesses of the Subcutaneous Tissue.*

The tendency to abscess-formation in the connective tissue is extremely marked in childhood, especially during the first years of life. I am not here referring to the patches of phlegmonous inflammation limited to a single spot which arise either from injuries or from irritation of neighbouring dermatitis (eczema impetiginodes) or from hypertrophy of the lymphatic glands, especially under the angle of the jaw, but to multiple abscesses which develop without any discoverable cause in various parts of the body either simultaneously or in succession, and are therefore regarded as "the expression of a diathesis." What this diathesis depends on, we do not know. This much, however, is certain—that these infiltrations and abscesses, although they are now and then found in healthy children, are much more liable to affect those who are atrophied or tubercular. The younger the children, the commoner are such abscesses. Even in the first few months of life we find multiple infiltrations developing on the most diverse situations, and varying from the size of a pea to that of a walnut or a hen's egg. Within a few days they become red and fluctuating, and then burst, and on healing leave behind bluish pigmented scars. The atrophy and debility already existing are considerably increased by the constantly recurring suppuration, and the abscesses sometimes form into deep ulcers, which, as I have often observed, may lay bare the muscles and cause extensive gangrene of the skin. I have frequently met with such deeply-spreading gangrene of the skin, especially on the neck and thorax, the areas affected were sometimes as large as one's hand, and the cases nearly always ended fatally. The same may be said of the abscesses which arise in the perineal con-

nective tissue, spread round the anus, rupture into the rectum and cause extensive necrosis.

While tubercle-bacilli have scarcely ever been found in these abscesses[1], Escherich[2] and Longard[3] have invariably found pyogenic staphylococci, and are probably quite right in maintaining that folliculitis is first set up by these organisms penetrating into the sebaceous and sweat glands, and that abscess-formation results from this. In any case, the advice given by these writers—that we should observe the utmost cleanliness with regard to the napkins (in which cocci have also been found)—deserves every attention. Next to this, the most important thing for the physician to prescribe is sublimate baths.

There is another kind of abscess often met with in scrofulous children and those who are suffering from affections of the bones. Round about the ankles, on the back of the hands and feet, over the ribs, on the head, &c., we often find abscesses covered by normally coloured skin; these may last many weeks before the skin becomes red, and when they are opened we feel carious bone with the probe. I have several times observed immense abscesses on the head which had developed between the bone and the pericranium, and which finally ruptured through the latter and through the outer skin. In these cases, just as in cephalhæmatoma (vol. i., p. 36) we could feel a bony ring encircling the abscess, which here as in the other cases is due to periosteal deposit at the margin of the abscess where the bone and the pericranium come into contact. We must not, however, mistake for such bony rings the cranial sutures which often project like a band in little children—a mistake which I once made myself in regard to the lambdoidal suture in a case of an unusual abscess over the occiput. The region behind the ear is also often the seat of large abscesses, which force the auricle away from the head so as to turn the concha forwards. If we defer opening the abscess, it is apt to rupture into the external auditory meatus, and when a deep incision is made we often find on passing the probe down to the bone that there is caries of the petrous bone or of the mastoid process.

[1] Ziesler, *Jahrb. f. Kinderheilk.*, xxiii., S. 79.
[2] *Münchener med. Wochenschr.*, Nos. 51, 52, 1886.
[3] *Archiv f. Kinderheilk.*, viii., S. 369.

But you must always examine with particular c
abscesses which are found at any part of the back,
nates, in the groin, or on the inner surface of the th
these are very often gravitation-abscesses arising from v
caries.

# FORMULÆ.[1]

*Referred to in the text as* (*Form.* 1), (*Form.* 2), *&c.*

1.
℞ Hydrarg. oxid. nig., gr. ½.
(*vel* Hydrarg. c. cretâ, gr. ½).
Pulv. sacchar. alb., grs. viii.
One powder twice daily.

2.
℞ Calomel, grs. ½ to ¾.
Pulv. sacchar. alb., grs. viii.
One powder twice daily.

3.
℞ Acid. hydrochlor. dil., mxx. to xl.
(Tinct. opii. gtt. 3 to 6)
Mucilaginis, ʒss.
Syr. simplicis, ʒss.
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

4.
℞ Creasoti, gtt. 2 to 4.
Syr. simpl., ʒss.
Aquam, ad ʒiss.
A teaspoonful every 2 hours.

5.
℞ Pepsini, grs. xv.
Pulv. sacch. alb., ʒiii.
Acid. hydrochlor. dil., ʒss.
Aquam, ad ʒiv.
A dessertspoonful 4 times daily.

6.
℞ Pulv. ipecac., grs. xv. to ʒss.
Antimon. tart., gr. ½ to ¾.
Oxymel. scillæ, ʒss.
Aquam ad, ʒiss.
A dessertspoonful every 10 minutes till vomiting occurs.

℞ Pulv. ipecac., grs. viii. to xv.
Antim. tart., gr. ½.
One powder every 10 minutes till vomiting occurs.

7.
℞ Calomel, gr. ½ to ¾.
Pulv. sacch. alb., grs. viii.
One powder every 2 hours.

℞ Sod. tartar., ʒi.
Mannæ, ʒiss.
Syr. rhamni, ʒvi.
Infus. sennæ ad, ʒiss.
A dessertspoonful every 2 hours.

8.
℞ Potass. bromidi, grs. xlvi.
Syr. simplicis, ʒss.
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

9.
℞ Chloral. hydrat., grs. xv. to xxv.
Syrup. aurantii, ʒss.
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

℞ Chloral. hydrat., grs. v. to viiss.
Aquæ, ʒiss.
To be given as an enema.

10.
℞ Morph. acet. vel hydrochlor., gr. ¼ to ½.
Syr. simpl., ʒss.
Aquam, ad ʒiss.
A teaspoonful 2—4 times daily.

11.
℞ Liq. arsenicalis, ʒss.
Aquæ, ʒii.
10—15 drops thrice daily.

11A.
℞ Acid. arsenios., gr. ½.
Pulv. glycyrrhizæ, grs. xxx.
Mucil. acaciæ q. s. ut ft. pil. xx.
One or two pills daily—caution.

[1] As several of Professor Henoch's prescriptions in their original form might have been found by many somewhat difficult to understand and to prescribe in this country, I have thought it best to reproduce them as nearly as possible by combinations of B. P. or other easily obtainable preparations. In doing so I have had most valuable help from Mr. J. Rutherford Hill, Assist. Sec. of the Pharmaceutical Society, whose name is a sufficient guarantee for their accuracy.—TRANSLATOR.

12.
℞ Ferri lactatis vel redacti, grs. ½ to ¾.
Pulv. sacch. alb., grs. viii.
One powder 2 or 3 times a day.

℞ Syr. ferri protochlor., N.F., ʒii. (1 gr. in 1 fl. dr.)
A half to one teaspoonful thrice daily.

℞ Vin. rhei, mxl.
Syr. ferri protochlor., N.F., ad ʒii. (1 gr. in 1 fl. dr.)
One to one and a half teaspoonfuls thrice daily.

13.
℞ Potass. iodidi, grs. xv.—xxx.
Aq. menth. pip., ʒss.
Aq. destill., ad ʒiv.
A teaspoonful 3 or 4 times a day.

14.
℞ Pulv. camphoræ, gr. i. to iii.
Pulv. sacch. alb., grs. viii.
One powder every 2 hours.

℞ Camphoræ, grs. xii.
Spirit. vini rect., ʒiiss.
Aquam, ad ʒss.
15 minims to be injected.

15.
℞ Ammon. chlorid., grs. xv. to xxx.
Vini antimon., ʒiii.
Ext. glycyrrhizæ. liq., ʒi.
Syrupi simpl., ʒiii.
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

16.
℞ Sodii nitrat., ʒss.
Aq. lauro-cerasi, ʒss.
Vin. ipecac., ʒiss to ʒiiss.
Syr. simpl., ʒss.
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

17.
℞ Calomel., gr. ⅛ to ¼.
Pulv. ipecac., gr. ⅛.
Pulv. sacch. alb., grs. viii.
One powder every 2 hours.

18.
℞ Vin. antimon., ʒiii. to ʒvi.
Syr. simpl., ʒiv.
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

19.
℞ Vin. antimon.
Oxymel. scillæ, aa ʒiiss.
A teaspoonful every 10 minutes till vomiting occurs.

20.
℞ Ol. anisi, mi.
Spirit. ammon. aromat., ʒss.
Syr. simpl., ʒss.
Infus. senegæ, ad ʒiv.
A dessertspoonful every 2 hours.

21.
℞ Pulv. camphoræ, gr. ½ to ¾.
Acid. benzoic., grs. ¾.
Pulv. sacchar. alb., grs. viii.
mitte in chart. cerat.
One powder every 2 hours.

22.
℞ Sodii vel potassii nitrat. vel potassii acetat., grs. xxx. to xlv.
Syrupi simpl., ʒss.
Infus. digitalis, ad ʒiv.
A dessertspoonful every 2 hours.

23.
℞ Syr. aurantii, ʒss.
Ext. cinchonæ liq., ʒiss. to ʒiiss.
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

24.
℞ Ext. cinchonæ liq., ʒiii. to ʒiv.
Syr. flor. aurantii, ʒss.
Aq. flor. aurantii, ʒiv.
A dessertspoonful 4 times a day.

25.
℞ Calomel., gr. ¼.
Pulv. digitalis, gr. ½.
Pulv. sacch. alb., grs. viii.
A dessertspoonful every 2 hours.

26.
℞ Potassii chlorat., grs. xlvi.
Syrupi simpl., ʒss
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

27.
℞ Potass. chlorat., grs. xlvi.
*vel.* Liq. chlori, ʒiv.
Ext. cinchonæ liq., ʒiss. to ʒiiss.
Syr. simpl., ʒss.
Aquam, ad ʒiv.
A dessertspoonful every 2 hours.

28.
℞ Confect. sennæ, ʒvi.
Pulv. acid. tart., grs. xviii.
Pulv. sacch. alb., ʒiiss.
Aquam, ad ʒiv.
About a dessertspoonful every 2 hours.

29.
℞ Vin. ipecac., ʒiss.
Tinct. opii, gtt. iii. to gtt. vi.
Mucilag. acaciæ
Syr. simpl., aa ʒii.
Aquam, ad ʒiv.
A de[illegible]ful every 2 hours.

30.
℞ Bismuth. subnit., gr. iss. to iii.
Pulv. acaciæ, grs. viii.
One powder every 2 hours.

31.
℞ Tinct. opii, gtt. vi.
Syr. simpl., ℥ss.
Infus. calumbæ, ad ℥iv.
A dessertspoonful every 2 hours.

32.
℞ Tinct. opii, gtt. vi.
Syr. simpl., ℥ss.
Infus. cascarillæ, ad ℥iv.
A dessertspoonful every 2 hours.

33.
℞ Acidi tannici, grs. xv.
Tinct. nucis vom., mxx.
Syr. simpl., ℥ss.
Aquam, ad ℥iv.
A dessertspoonful every 2 hours.

34.
℞ Argenti nitrat., grs. ½ to iss.
Mucilag. acaciæ, ℥ss.
Aq. destillat., ad ℥iv.
(in vitr. actinic.)
A dessertspoonful every 2—8 hours.

35.
℞ Plumbi acetat., gr. ½.
Pulv. acaciæ, grs. viii.
One powder thrice daily.

36.
℞ Ol. ricini, ℥i.
Ol. amygdal. essent., gtt. i.—ii.
Pulv. amygd. co., ʒiii.
Aquam, ad ℥iv.
A dessertspoonful every 2 hours.

37.
℞ Tinct. nuc. vomic., mlxx.
Syr. simpl., ℥ss.
Aquam, ad ℥iss.
A teaspoonful thrice daily.

38.
℞ Ext. ergotæ liq., ʒii.
Glycerini, ℥i.
Aquam, ad ℥ii.
15 minims to be injected hypodermically.

39.
℞ Potassii tartrat., grs. lxxxvii.
Syr. simpl., ℥ss.
Tinct. rhei., ℥iss to ℥iiss.
Aquam, ad ℥iv.
A dessertspoonful every 2 hours.

40.
℞ Quininæ sulph.
(vel hydrochlor.)
Ferri redacti, aa grs. ½.
Pulv. sacch. alb., grs. viii.
One powder twice or thrice daily.

41.
℞ Potassii acetat., grs. xxx. to xlvi.
Syr. simpl., ℥ss.
Aquam, ad ℥iv.
A dessertspoonful every 2 hours.

42.
℞ Potassii acetat., grs. xlvi.
Syr. aurantii, ℥ss.
Ext. cinchon. liq., ʒiss. to ʒii.
Aquam, ad ℥iv.
A dessertspoonful every 2 hours.

43.
℞ Acidi tannici, grs. ½.
Pulv. sacch. alb., grs. viii.
A powder every 2 hours.

44.
℞ Ext. ergot. liq., ʒii.
Syr. simpl., ℥ss.
Aquam, ad ℥iv.
A dessertspoonful every 2 hours.

45.
℞ Tinct. ferri perchlor., mxl.
Syr. simpl., ℥ss.
Aquam, ad ℥iv.
A dessertspoonful 4 times daily.

46.
℞ Iodi, grs. v. to viii.
Potass. iodid., grs. xv.
Syr. simpl.
(*vel.* Aq. menth. pip.) ℥iss.
Aquam, ad ℥iv.
A dessertspoonful 4 times daily.

47.
℞ Sulph. sublim., ʒiii.
Calcis, ℥iss.
Coq. c. aq. fervid, ℥xxviii.
ad remand., ℥xviii.
Cola et filtra.
To be used for inunction.

48.
℞ Acid. salicylic., grs. xl. to lxxx.
Spirit. vini rect.
Glycerini aa q. s. ad sol.
Vaselini, ℥i.

49.
℞ Acidi tannici, ℥ss.
Vaselini, ℥i.

50.
℞ Acidi borici, grs. xxiv. to xlviii.
Vaselini, ℥i.

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